Published in final edited form as:

Ann Epidemiol. 2015 March ; 25(3): 201–207. doi:10.1016/j.annepidem.2014.11.010.

Estimating population attributable fractions to quantify the health burden of obesity

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Abstract

Purpose—Obesity is a highly prevalent condition in the US and elsewhere and is associated with increased mortality and morbidity. Here we discuss some issues involved in quantifying the health burden of obesity using population attributable fraction (PAF) estimates and provide examples.

Methods—We searched PubMed for articles reporting attributable fraction estimates for obesity. We reviewed eligible articles to identify methodological concerns and tabulated illustrative examples of PAF estimates for obesity relative to cancer, diabetes, cardiovascular disease and all-cause mortality.

Results—There is considerable variability among studies regarding the methods used for PAF calculation and the selection of appropriate counterfactuals. The reported estimates ranged from 5% to 15% for all-cause mortality, −0.2% to 8% for all-cancer incidence, 7% to 44% for cardiovascular disease incidence, and 3% to 83% for diabetes incidence.

Conclusions—To evaluate a given estimate, it is important to consider whether the exposure and outcome were defined similarly for the PAF and for the relative risks, whether the relative risks were suitable for the population at hand, and whether PAF was calculated using correct methods. Strong causal assumptions are not necessarily warranted. In general PAFs for obesity may be best considered as indicators of association.

Keywords

Body mass index; obesity; body weight; epidemiologic methods; risk

Obesity and overweight are highly prevalent conditions in the US and elsewhere (1). The most recent estimates show a prevalence of 35% for overweight and 35% for obesity among

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adults. Obesity is associated with increased incidence of various diseases and risk factors, including cardiovascular diseases, diabetes, certain cancers, hypertension and dyslipidemia. (2). Thus it is of interest to quantify the health burden of obesity using population attributable fraction (PAF) estimates for obesity as a cause of morbidity or mortality. The word ‘attributable’ suggests a strong statement about causation. Levin’s original article describing the attributable fraction (3) put the word ‘attributable’ in quotes, but the quotes have long since been lost. Calculating attributable fractions adds no new causal information to the relative risks. Here the word ‘attributable’ will be used without necessarily implying causality.

Methodological issues

Several practical issues in calculating attributable fractions arise in obesity-specific contexts. These include the definition of obesity, the choice of an appropriate counterfactual exposure, the choice of appropriate relative risks, the interpretation of the results, and problems in how to compute attributable fractions.

Currently the US and international standard definition of obesity is a body mass index (BMI, weight (kg)/height squared (m) of 30 or greater (4, 5). This definition depends on body weight rather than on body fatness. Although BMI and percent body fat are highly correlated, they are not identical (6). A more general definition of obesity is as an excess of body fat. However there is no consensus on exactly what this level is (7), and few epidemiologic studies include direct measures of body fat. We limit discussion here to body mass index.

Identification of an appropriate counterfactual

Identifying the appropriate counterfactual in order to estimate and interpret PAF for obesity-related outcomes is challenging. An example of the relatively simple calculations to estimate attributable fractions is shown in table 1, with estimates calculated from the data on numbers of deaths and hazard ratios among men presented in a study by Adams et al. (8). Adams et al had selected a BMI of 23.5–<25 as the reference category. If we use that group as the counterfactual (non-exposed) category then the estimated PAF for obesity (a BMI of 30 or above) as a cause of death would be 3.7%. James et al (9) argue that the optimum BMI would be 21–22. If we adjust the relative risks to use BMI 21–23.4 as the counterfactual in this example, holding all else constant, then the PAF for obesity as a cause of death would be 0.7%. In contrast, if BMI 25–26.5 is used as the counterfactual, then the PAF for obesity as a cause of death would be 4.8%. Thus as observed elsewhere (10), the PAF varies with the choice of counterfactual.

Identification of appropriate relative risks

When attributable fractions are calculated within a single study (e.g. Wilson et al (11)), the relative risks and the attributable fractions use internally consistent definitions of exposure and outcomes. However if the relative risks are derived from one study (sometimes called the ‘derivation cohort’) and combined with prevalence estimates in a different population

(sometimes called the ‘target population’) to calculate the attributable fraction for the target population, inconsistencies can arise.

One potential problem is that the definition of exposure used to derive the relative risks may be different from the definition used to calculate the prevalence of exposure in the target population. For example, Katzmarzyk and Ardern (12) calculated attributable fractions by combining relative risks developed on measured height and weight data with prevalence estimates based on self-reported weight and height data from a different data set (13). The opposite procedure was followed by Calle et al (14) who combined relative risks from self-reported weight and height data with prevalence estimates based on measured weight and height data in a different population. The direction of any bias caused by this type of mismatch is unclear. Prevalence estimates based on self-reported weight and height are likely lower than prevalence estimates based on measured weight and height, but relative risks based on self-reported weight and height may well be higher than relative risk estimates derived using measured weight and height (9, 15–17).

Another potential problem is that the relative risks may be derived from a population that is different from the population under study. For example, Danaei et al (18) used relative risks (9) derived from the Asia-Pacific Cohort Studies Consortium to make estimates for overweight and obesity-associated ischemic heart disease mortality and hemorrhagic stroke mortality in the United States. These relative risks from primarily Asian populations may not be applicable for estimating attributable fractions for the US. A WHO consultation suggested that the relation of BMI to diabetes and to cardiovascular disease might be different in Asians than in other populations (19).

Another potential problem is that the relative risks from the derivation cohort may be for a health outcome that is not exactly the same as the outcome for which the PAF is being calculated in the target population. For example Calle et al (14) calculated obesity-related relative risks for cancer mortality among never smokers only and applied those relative risks in estimating attributable fractions for mortality from all cancers. All-cancer mortality includes a relatively large proportion of deaths from lung cancer. However, among never-smokers, mortality from lung cancer is generally quite low. In the Calle et al sample, 30% of cancer deaths were from lung cancer among men in the whole cohort, but among never-smoking men, only 6% of cancer deaths were due to lung cancer. Overweight and obesity have not been found to be associated with higher risks for lung cancer among ever-smokers (20). In the full sample of men, the hazard ratio for overweight relative to normal weight was 0.97 (95% confidence interval [CI] 0.94–0.99) (adjusted for smoking) but in the sample of never smoking men the corresponding hazard ratio was 1.11 (95% CI 1.05–1.18). In effect, the relative risks are for the composite outcome of the mix of cancers that occurs among never-smokers, but the attributable fractions are being estimated for the composite outcome of the mix of cancers that occurs among the entire population, which includes a relatively large proportion of cancers that have not been shown to be associated with overweight or obesity. This approach may exaggerate obesity-related deaths from cancer in the entire population. This general problem of a difference in the outcomes can take other forms. For example, Danaei et al (18) estimated the PAF of mortality from ischemic heart

disease (IHD) associated with overweight and obesity but used relative risks (9) for the combined outcome of IHD incidence or mortality.

Interpretation

PAFs use a number of assumptions and in general should be interpreted cautiously. Levin (3), who developed the attributable fraction, specifically noted that “This index is based on the assumption that smokers, if they had not become smokers, would have had the same incidence of lung cancer as that found among non-smokers.” This leaves the interpretation of PAF ambiguous. Are the counterfactual estimates based on the situation if the exposure had never existed (e.g. no one had ever smoked) or on the situation if there had been some intervention to eliminate exposure (e.g. all smokers had successfully quit smoking)? This assumes that removing the exposures also removes the effect, i.e. the risk in exposed individuals after an intervention will be the same as in those never exposed. This assumption may be problematic with observational data (and more robust in a randomized clinical trial setting), but it is necessary for making translational inferences for public health. Levin felt that the estimate was a maximum.

Interventions to prevent or reduce obesity might have other characteristics that could affect mortality independently of any effects on body weight. Hernan and Taubman [(21), p. 513] argue that “different methods to change BMI may lead to different counterfactual outcomes, even if they each achieve the same BMI value. Thus the counterfactual outcome at a given BMI value cannot be defined.” and conclude that “if the goal is to inform policy, it may be better to focus on modifiable lifestyle behaviors than on obesity itself.”

Changes over time in other factors might affect the association of BMI with mortality. As pointed out by Danaei et al [(18), (p. e1000058)] “The use of RRs from cohort studies that started a few decades ago may overestimate the effects of BMI on diseases such as IHD if ‘mediators’ such as SBP and cholesterol have been lowered over time in those with high BMI.”

Problems in calculation

Correct calculation of PAF is a critical component for each article that attempts to estimate this metric. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement (22) recommends that “Because of the semantic ambiguity and complexities involved, authors should report in detail what methods were used to calculate attributable risks, ideally giving the formulae used.”

According to Rockhill et al (10), the “most common error” in calculating attributable fractions is the use of adjusted relative risks in a computing formula that is valid only with unadjusted relative risks. Rockhill et al describe two commonly-used formulas for the calculation of PAF. One formula (designated as #2 by Rockhill et al) is

$$P(E) \cdot (RR - 1) / ((P(E) \cdot (RR - 1)) + 1)$$

where $P(E)$ is the proportion of the population exposed to the factor (in this case, to obesity) and RR is the relative risk of mortality associated with obesity. This formula is only valid when relative risks are not adjusted for confounding.

A different formula (designated as #4 by Rockhill et al.) is

$$pd * ((RR-1) / RR)$$

where pd is the proportion of cases exposed to the risk factor and RR is the adjusted relative risk of mortality associated with obesity. This is a formula appropriate for use with adjusted relative risks when confounding exists (1, 2). When relative risks are adjusted for confounding, the two formulas will give different results from each other. The second formula requires knowledge of the prevalence of the exposure among those with the outcome (e.g. the proportion of decedents who were obese). Other methods are available as well for calculating adjusted attributable fractions (23–26) but all similarly require additional information beyond the simple prevalence of exposure in the population. The use of the invalid formula sometimes occurs even when all the information is available (for example (27, 28), but is probably more likely to occur when a study extrapolates adjusted relative risks from one study to a different data set that lacks sufficient information on the confounding factors (for example (14, 29–31)).

This effect may be partially mitigated when the unadjusted formula is used within strata of some but not all of the confounding factors. For example, Preston and Stokes (31) calculated PAFs for obesity within age and sex categories using the formula for unadjusted relative risks. The relative risks they use are adjusted for age, sex and smoking. Since Preston and Stokes use age and sex specific estimates, this is likely to provide a degree of adjustment of the PAF estimates for age and sex. However their PAF estimates do not take into account the effect of smoking itself on mortality and are not properly adjusted for confounding by smoking.

The “comparative risk assessment” approach (e.g. Danaei et al. (18)) uses a method of calculating PAF with a minimum risk exposure distribution as the counterfactual rather than a specific category. Vander Hoorne et al. (32) state that to account for confounding correctly using this approach would require the availability of exposure and disease data stratified by the confounding variable. However, as Vander Hoorne et al point out [(32), p 239] : “In general, such stratified data are not available and therefore reliance on the formula with direct use of the adjusted relative risk estimates was necessary. ”

PAF estimates for obesity relative to selected health outcomes

We tabulated examples of PAF estimates for obesity or high BMI relative to selected health outcomes of cancer, diabetes, cardiovascular disease and all-cause mortality. Articles that might provide attributable fraction estimates for obesity were identified by a PubMed search for English-language articles with the words “attributable” and “obesity”, “overweight” or “body mass index” in the title or abstract, by PubMed searches for articles that cited key methodological articles about attributable fractions by Spiegelman et al. (25), Benichou (23), Bruzzi et al. (26), Rockhill et al. (10) or Steenland (33), and by examining the

reference lists of articles. Articles were excluded if PAFs were not reported clearly or where PAFs were expressed only as numbers and could not readily be converted into percent of a health outcome. No studies were excluded because of their findings. A more extensive search would probably yield a more complete accounting but a systematic review and appraisal of this literature was beyond the scope of this article. As discussed by Uter and Pfahlberg (34), devising appropriate search strategies for PAF estimates is complex. Instead, these tables are provided as illustrative examples and do not show extensive details about methods, samples or confounding factors.

Total mortality

A variety of estimates for all-cause mortality are shown in table 2. There are two main approaches to making estimates for the PAF for high BMI as a cause of total mortality. One approach uses relative risks for all-cause mortality to estimate PAF. This allows relative risks lower than 1 in some categories to decrease the overall PAF. Examples of this can be seen in table 1 and in articles by Calle et al. (14), Allison et al. (29) and Muennig et al. (35)

In another approach, exemplified by Kelly et al (36) and Danaei et al (18), researchers make estimates of attributable deaths only for obesity-related causes of death and aggregate the numbers to get an estimate of total deaths attributable to high BMI. Because this approach uses only causes of death that are monotonically associated with increasing BMI, then the lower the counterfactual BMI is set, the higher the number of attributable deaths becomes. This approach does not take into account excess mortality linked to lower rather than higher BMI levels and thus leads to a higher estimate. This effect can be seen in the article by Danaei et al (18) in which using a slightly lower counterfactual increased the proportion of attributable deaths from 7.5% to 9.2%.

For the United States, PAF estimates shown in table 2 were roughly similar for Flegal et al (37), Mehta et al (38) and Muennig et al, (35), i.e. approximately 5% of deaths. The studies by Allison et al (29) and Mokdad et al (30) arrived at considerably higher estimates for the US population. The reasons for this have been discussed elsewhere (24, 39), but one explanation is the use of incorrect attributable fraction methods in both the Mokdad et al and Allison et al papers, both of which used a formula that did not adjust for confounding factors.

An interesting case is the difference between the Van Dam et al study (40) and the Iversen et al study (41), both studies of middle-aged women that used very similar methods, including the same SAS macro to calculate attributable fractions. The PAF was 14% in the van Dam study and 2% in the Iversen study. Iversen et al comment (p. 569): “Although the prevalence of being overweight or obese was identical (48%) in both studies, the absence of significant increased mortality risk associated with overweight or obesity at midlife in the present study accounts for the different population attributable risk estimates.”

Cancer

Some PAF estimates for high BMI as a cause of all-cancer mortality and incidence are shown in table 3. Similar to the situation for all-cause mortality, some of these estimates

were constructed by using results for all-cancer mortality to derive an overall estimate. For others, the researchers aggregated results for individual obesity-related cancer sites. Of the studies in the table, four studies (40, 42–44) adjusted the PAF estimates correctly for the confounding factors; in 6 studies it was not clear (45–50) and in the remaining studies, PAF was not adjusted properly for confounding factors.

According to the National Cancer Institute fact sheet on cancer and obesity (51), cancers of the esophagus, breast (postmenopausal), endometrium (the lining of the uterus), colon and rectum, kidney, pancreas, thyroid, gallbladder, and possibly other cancer types are associated with obesity and high BMI. Many studies have estimated obesity-attributable PAF for individual cancers. For example, Wilson et al estimated from a study in Australia that 12.1% of breast cancer cases were attributable to obesity (52) and Arnold et al estimated that 7.2% of pancreatic cancer in whites (53) was due to obesity. Trichopoulos (54) found that obesity (BMI 30+) but not overweight was associated with increased risk of hepatocellular carcinoma with a PAF of 16.1%. Danaei et al (55) estimated that in high income countries 14% of colon cancer 13% of breast cancer and 43% endometrial cancer was due to obesity. Lukanova et al (48) in a study in Sweden estimated that for women 30% of endometrial cancer, 22% of ovarian cancer, 20% of colon cancer and 16% of colorectal cancers were attributed to obesity. In men, about 30% of all kidney cancer cases could be attributed to overweight and obesity. Bergstrom et al (56) found that in Europe the highest attributable fractions of cancer due to obesity were for cancers of the endometrium (39%), kidney (25% in both sexes) and gallbladder (25% in men and 24% in women).

Not all cancers are associated with BMI, and some cancers are inversely associated with BMI. In a large US study Smith et al (20) found that lung cancer risk was reduced at higher BMI among current and former smokers. Chang (57) addressed issues of lower breast cancer rates at higher BMIs in younger women vs higher rates at higher BMIs in older women. In a large prospective study in China and a meta-analysis, Smith et al (58) found that low BMI, rather than high BMI, was associated with increased risk of squamous cell carcinoma of the esophagus. Thus, for example in the study by Lukanova et al (48), although obesity was associated with increased risk for some cancers in men, nonetheless in men, there was no association of BMI with overall cancer risk.

Results were generally similar for cancer incidence and for mortality, although few studies addressed both simultaneously. The low estimates for studies in China and Japan may reflect the very low prevalence of high BMI in those studies.

Cardiovascular disease

The association of cardiovascular disease with obesity is well known (59). Our search turned up relatively few examples of PAF calculations. Some estimates of PAF for different aspects of cardiovascular disease outcomes are shown in table 4. Most studies were appropriately adjusted for confounding factors. In four studies (60–63) the degree and type of adjustment for confounding factors was not clear and only one study (27) used an incorrect approach to confounding. Estimates were variable across studies, reflecting differences in populations

studied, the outcome studied, the prevalence of obesity and differences in the methods used. Only one study (11) addressed both incidence and mortality simultaneously.

Diabetes

Type 2 diabetes mellitus has been observed to have a strong association with obesity. Because several articles presented PAF estimates for diabetes prevalence, those PAF estimates are shown in table 5 as well as PAF estimates for diabetes incidence or mortality. Almost all studies show relatively high attributable fractions for diabetes. Two studies were incompletely adjusted (36, 64), and in two cases the degree of adjustment was not clear (65, 66). In one case (67), we were able to calculate the PAF from the information provided in the article. Martiniuk et al (66) estimated obesity-attributable diabetes mortality for 15 countries in the Western Pacific and South East Asia regions. Age-standardized male PAFs ranged from 11% for India to 98% for American Samoa. Age-standardized female PAFs ranged from 9% in India to 95% in American Samoa. According to the authors, their paper demonstrates that theoretically between 9% and 98% of deaths from diabetes could be prevented by tackling obesity in the Asia Pacific region.

Summary and conclusions

Attributable fractions provide a useful way to express the burden of morbidity and mortality associated with obesity, taking into account both the prevalence of obesity and the relative risks of health outcomes associated with obesity. Correct application of attributable fraction methods is most easily done within a single study because definitions of exposure, outcome and the population will be internally consistent.

To evaluate a given estimate, it is important to consider whether the exposure and outcome are being defined similarly for the PAF and for the relative risks, whether the relative risks are suitable for the population at hand, and whether the PAF was calculated using appropriate methods, particularly in regard to the appropriate adjustment for confounding factors. Despite numerous cautions (e.g. (10, 23, 26, 33, 34, 68–73) about bias in attributable fractions arising from using the incorrect formula for PAF when relative risks are adjusted, this practice continues to appear in the literature.

Strong causal assumptions are not necessarily warranted, and in general PAF for obesity may be best considered as indicators of association.

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Table 1

Example of within-study PAF calculations with data from Adams et al.(8)

BMI category i	N	Number of deaths	Percent of all deaths (pd)	Adjusted hazard ratio (RR)	Counterfactual of BMI 23.5–24.9			Counterfactual of BMI 21–23.4			Counterfactual of BMI 25–26.4		
					PAF (%) *	Number of “attributable” deaths †	PAF (%) *	Number of “attributable” deaths †	PAF (%) *	Number of “attributable” deaths †	PAF (%) *	Number of “attributable” deaths †	
<18.5	1,119	333	0.8	1.97	0.39	164	0.34	143	0.41	173			
18.5–20.9	6,992	1,513	3.6	1.54	1.26	531	0.93	392	1.38	582			
21–23.4	36,788	5,229	12.4	1.14	1.52	641	0	0	2.07	873			
23.5–24.9	46,486	5,768	13.7	1.00	0.00	0	–1.87	–789	0.65	274			
25–26.4	56,236	6,657	15.8	0.95	–0.83	–350	–3.24	–1366	0	0			
26.5–27.9	49,882	6,021	14.3	0.95	–0.75	–316	–2.93	–1236	0	0			
28–29.9	49,029	6,355	15.1	1.00	0.00	0	–2.06	–869	0.72	304			
30–34.9	51,857	7,488	17.8	1.10	1.62	683	–0.74	–312	2.46	1037			
35–39.9	11,306	2,028	4.8	1.35	1.24	523	0.73	308	1.42	599			
40+	3,352	781	1.9	1.83	0.86	363	0.72	304	0.92	388			
Total	3,13,047	42,173											
For BMI	30				3.72	1569	0.71	300	4.8	2024			

^{*} Calculated as $pd[(RR_i - 1)/RR_i]$; Hazard ratios are recalibrated to use the counterfactual BMI level as the reference category

[†] Calculated as $PAF \times \text{total number of deaths}$

Table 2

Examples of PAF (%) for obesity or high BMI as a contributor to all-cause mortality

First author (reference)	Mortality	Population	PAF formula valid for adjusted relative risks
Allender (74)	12%	United Kingdom	Uncertain
Allison (29)	11%	United States	No
Banegas (75)	8%	Europe	No
Danaei (18)	9%	United States	No
Flegal (37)	5%	United States	Yes
Iversen(41)	2%	Women, United Kingdom	Yes
Katzmarzyk (13)	9%	Canada	No
Kelly (36)	9%	United Kingdom	No
Mehta (38)	3%	Men, United States,	Yes
Mehta (38)	4%	Women, United States,	Yes
Mokdad (30)	15%	United States	No
Muennig (35)	5%	United States	Uncertain
Ringback Weitoft (61)	3%	Men Sweden	Uncertain
Ringback Weitoft (61)	4%	Women, Sweden	Uncertain
Van Dam (40)	14%	Women, United States	Yes

Table 3

Examples of PAF (%) for obesity or high BMI as a contributor to all-cancer incidence and mortality

First author (reference)	Mortality	Incidence	Population	PAF formula valid for adjusted relative risks
Bergstrom (56)		5%	Europe	No
Boffetta (76)		2%	France	No
Boswell-Purdy (45)	4%	6%	Canada	Uncertain
Calle (14)	4%		Men, United States	No
Calle (14)	14%		Women, United States	No
Danaei (55)	3%		High income countries	No
Dryer (46)	<1%		Norway	Uncertain
Flegal (42)	4%		United States	Yes
Inoue(47)	<1%	1%	Japan	Uncertain
Kuriyama (43)		-0.2%	Men, Japan	Yes
Kuriyama (43)		4.5%	Women, Japan	Yes
Lukanova (48)	7%		Women Sweden	Uncertain
Ott (77)		5%	High income countries	No
Pan(49)		8%	Canada	Uncertain
Parkin (78)		6%	United Kingdom	No
Polednak (79)		6%	United States	No
Reeves(44)		5%	United Kingdom	Yes
Renehan (80)		3%	Men, European	No
Renehan (80)		4%	countries	No
Van Dam(40)	8%		Women, European countries	Yes
Wang (50)	<1%	<1%	Women, United States	Uncertain

Table 4
Examples of PAF (%) for obesity as a contributor to cardiovascular disease (CVD) incidence and mortality

First author (reference)	Mortality	Incidence	Outcome	Population	PAF formula valid for adjusted relative risks
Dunlay (60)		12%	Heart failure	United States	Uncertain
Faeh (81)	12%		CVD	Switzerland	Yes
Flegal(42)	9%		CVD	United States	Yes
Flint (82)		39%	CHD *	Men, United States	Yes
Flint (82)		44%	CHD *	Women, United States	Yes
Huxley (83)		18%	Atrial fibrillation	United States	Yes
Loehr (84)		28%	Heart failure	United States	Yes
Mann (85)	8%		CHD *	United States	Yes
Ringback Weitoft (61)	9%		Circulatory disease	Men, Sweden	Uncertain
Ringback Weitoft (61)	17%		Circulatory disease	Women, Sweden	Uncertain
Schnohr (62)		10%	CHD *	Men, Denmark	Uncertain
Schnohr (62)		7%	CHD *	Women, Denmark	Uncertain
Seidell (63) Men	21%		CHD *	Men, Netherlands	Uncertain
Seidell (63) Women	28%		CHD *	Women, Netherland	Uncertain
Van Dam(40)	31%		CHD *	Women, United States	Yes
Van Dis (27)	35%		CVD	Netherlands	No
Wilson (11)	2%	15%	CVD	Men, United States	Yes
Wilson (11)	1%	10%	CVD	Women, United States	Yes

* CHD-coronary heart disease

Table 5
Examples of PAF (%) for obesity or high BMI as a contributor to diabetes prevalence, incidence or mortality

First author (reference)	Mortality	Incidence	Prevalence	Population	PAF formula valid for adjusted relative risks
Atlantis (86)			37%	Australia	Yes
Chan (67)		66%		Men, United States	Yes
Dal Grande (87)			41%	Australia	Yes
Flegal (42)	41%			United States	Yes
Hart (65)		56%–60%		United Kingdom	Uncertain
Hart (65)		74%–83%		United Kingdom	Uncertain
Hosseiniapanah (64)		60%		Iran	No
Hu (88)		61%		United States	Uncertain
Jiang (89)			44%	Men, Canada	Yes
Jiang (89)			53%	Women, Canada	Yes
Kelly (36)	77%			United Kingdom	No
Laaksonen (90)		77%		Finland	Yes
Martiniuk (66)	9%–98%			15 countries of Western Pacific, SE Asia	Uncertain
Wilson (11)		21%		Men, United States	Yes
Wilson (11)		3%		Women, United States	Yes