Could targeted food taxes improve health?

Oliver Mytton, Alastair Gray, Mike Rayner and Harry Rutter

Updated information and services can be found at:
http://jech.bmj.com/cgi/content/full/61/8/689

These include:

References	This article cites 17 articles, 10 of which can be accessed free at: **http://jech.bmj.com/cgi/content/full/61/8/689#BIBL**
Rapid responses	One rapid response has been posted to this article, which you can access for free at: **http://jech.bmj.com/cgi/content/full/61/8/689#responses**
	You can respond to this article at: **http://jech.bmj.com/cgi/eletter-submit/61/8/689**
Email alerting service	Receive free email alerts when new articles cite this article - sign up in the box at the top right corner of the article

Notes

To order reprints of this article go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to *Journal of Epidemiology and Community Health* go to:
http://journals.bmj.com/subscriptions/

EVIDENCE BASED PUBLIC HEALTH POLICY AND PRACTICE

Could targeted food taxes improve health?

Oliver Mytton, Alastair Gray, Mike Rayner, Harry Rutter

....

See end of article for
authors' affiliations
....

Correspondence to:
Dr O Mytton, Queen's
Medical Centre, Nottingham
NG7 2UH, UK;
olivermytton@doctors.org.uk

Accepted 11 November
2006
....

Objective: To examine the effects on nutrition, health and expenditure of extending value added tax (VAT) to a wider range of foods in the UK.

Method: A model based on consumption data and elasticity values was constructed to predict the effects of extending VAT to certain categories of food. The resulting changes in demand, expenditure, nutrition and health were estimated. Three different tax regimens were examined: (1) taxing the principal sources of dietary saturated fat; (2) taxing foods defined as unhealthy by the SSCg3d nutrient scoring system; and (3) taxing foods in order to obtain the best health outcome.

Data: Consumption patterns and elasticity data were taken from the National Food Survey of Great Britain. The health effects of changing salt and fat intake were from previous meta-analyses.

Results: (1) Taxing only the principal sources of dietary saturated fat is unlikely to reduce the incidence of cardiovascular disease because the reduction in saturated fat is offset by a rise in salt consumption. (2) Taxing unhealthy foods, defined by SSCg3d score, might avert around 2300 deaths per annum, primarily by reducing salt intake. (3) Taxing a wider range of foods could avert up to 3200 cardiovascular deaths in the UK per annum (a 1.7% reduction).

Conclusions: Taxing foodstuffs can have unpredictable health effects if cross-elasticities of demand are ignored. A carefully targeted fat tax could produce modest but meaningful changes in food consumption and a reduction in cardiovascular disease.

E vidence of rapidly rising levels of obesity has led to increasing concerns about the effect of diet on health, both globally and in Europe.[1–4] Diet is implicated in many major western diseases, such as ischaemic heart disease (IHD), stroke, some cancers and type 2 diabetes.[1 3] In the UK, it is estimated that 10% of all disability-adjusted life years lost can be attributed to poor diet.[5] Changes in diet, such as reductions in the intake of fat, particularly saturated fat, and sodium, have been advocated to reduce the incidence of some of these diseases.[6–8]

A variety of approaches have been advocated, including altering the price of food.[3 9] In the UK, there have been calls for taxation on food to improve health, often termed a fat tax,[9] and a recent Parliamentary Select Committee recommended that "the Government should keep an open mind on this issue".[2] At a European level, the European Heart Network has called for a comprehensive and integrated food policy, which includes a pricing strategy.[10] The World Health Organization recognises that the pricing of healthy food is important for improving diet in order to prevent diseases.[3]

Taxation to increase price and reduce consumption has been an important part of reducing tobacco consumption in order to improve health in the UK.[11 12] Increasing tax on alcohol is also an effective means to reduce consumption.[13] However, there are important differences between tobacco, or alcohol, and food. The success of any fat tax depends on changes in food-purchasing patterns with the outcome of a healthier diet. A healthier diet could improve health by a variety of means—for example, reducing serum cholesterol to reduce the incidence of cardiovascular disease. A recent literature review found a wide variety of taxes levied on food, but that the intention normally was to raise revenue rather than to change diet and improve health.[9] Moreover, there was little information on the effects of food taxes on behaviour and health to inform the ongoing debate on a fat tax. Only one paper, by Marshall,[14] was identified which attempted to estimate the effects of extending value added tax (VAT) to products high in saturated fat on

health. Like others, we also feel that extending VAT to additional food categories might be the preferred means for legislators to introduce a targeted food tax to improve health.[14 15] Currently, in the UK, VAT at the standard rate of 17.5% is charged on a range of different foods including confectionery, ice cream, savoury snacks and most drinks, foods typically sold by catering outlets. Using empirical economic and dietary data, together with estimates of the effect of diet on health, we sought to estimate the potential health and dietary effects of extending VAT in the UK to additional categories of food.

METHODS

We used empirical economic data to predict the effect of a rise in price (by 17.5%, the current UK rate of VAT) on changes in consumption. The own price elasticity of demand predicts the percentage change in consumption (quantity brought) of that item for a 1% rise in price: for example, if the price elasticity of bread is –0.40, a 10% rise in the price of bread, all other things being equal, will result in a fall in consumption of bread by 4%. In addition, if the price of bread rises, the consumption of margarine may fall. The cross-price elasticity of demand predicts this effect. For example, a cross-price elasticity of –0.1 for margarine with respect to the price of bread means that for a 10% rise in the price of bread the consumption of margarine would fall by 1%. Typically, items that complement each other have a negative cross-elasticity, while items that can be substituted have a positive cross-elasticity. For each item of food taxed, the effect on consumption is calculated using each item's own price elasticity of demand. In addition, the effect on consumption of other items is calculated using the appropriate cross-price elasticity value. We assumed that changes in consumption (quantity purchased) led to changes in what was actually eaten. We then estimated the effect of changes in nutritional intake, changes in salt intake and the intake of

....

Abbreviations: IHD, ischaemic heart disease; VAT, value added tax

different types of fat (by their effect on serum cholesterol), on mortality from cardiovascular disease (both strokes and IHD), using empirical data derived from meta-analyses.

As there were many calculations to make for any item on which VAT was introduced, in order to predict the effect on diet and health, the calculations were performed using a spreadsheet model (Microsoft Excel 2000). Within this model, it is possible to alter taxed food items and observe the effect on a range of health and diet outcomes. The different configurations of food items taxed were termed ''approaches''. The three approaches we investigated were the following.

Saturated fat

Marshall proposed extending VAT to the main sources of dietary saturated fats (whole milk, butter, cakes and pastries, biscuits, puddings and ice creams, cheese and buns).[14] We replicated Marshall's proposal as best we could with the categories provided by our data.

SSCg3d

SSCg3d scores are a quantitative estimate of how unhealthy a food is.[16] Eight nutritional parameters determine the food's score. Points (up to a maximum of 10) are scored for energy density, saturated fat, sodium and non-milk extrinsic sugar and subtracted for fruit and vegetable content, iron, calcium and n-3 polyunsaturated fat. Scores range from −12 (spinach) to +29 (chocolate digestive biscuits). We applied VAT to all foods classified as less healthy by this model (ie, foods with a SSCg3d score of ≥9).

Best outcome

We sought to establish the maximum potential gain with a taxation level of 17.5%, while trying to minimise the additional cost to the consumer. We used the model to test the effect of taxing different food items on the following outcomes: reduction in serum cholesterol by changes in dietary fat; reduction in salt, non-milk extrinsic sugar and calorie intake; increase in fruit and vegetable consumption. By judicious use of trial and error, we sought to identify the configuration of taxed items that maximised these outcomes, which we call best outcome.

Table 1 shows which food categories, taken from the National Food Survey,[17] are taxed in each model, together with their SSCg3d scores.

DATA

We took data for food consumption, expenditure and elasticity values from the National Food Survey 2000.[17] The National Food Survey was an annual survey using interviews and diaries to record food bought for consumption at home in Great Britain. The National Food Survey for 2000 suggested that food and drink consumed outside the home was responsible for 26% of expenditure (excluding alcohol). Therefore, we assumed that food bought for consumption at home represents about 75% of total food consumption by energy, and that food consumption outside the home is not affected by the tax changes examined. The National Food Survey categorises foods into groups (table 1). Because the subcategory items are similar, we assumed firstly the own price elasticity to be the same as the main category of which it is a member, and secondly the cross-price elasticities between subcategories to be 0.6 (similar items are relatively substitutable: eg, butter or margarine). This was subjected to sensitivity analysis.

We took estimates of the effect of changes in intake of dietary fat and cholesterol on serum cholesterol from a quantitative meta-analysis of metabolic ward studies, shown in table 2.[6] We estimated the effect of serum cholesterol on mortality from IHD by two means: first, using the summary data from a meta-analysis of 58 randomised trials of cholesterol-lowering by any

means and IHD events, which predicts that a 1.6 mmol/l reduction in low-density lipoprotein would cause a fall in IHD by around 50%[18]; second, using the method employed by Sacks and Katan.[19] Their estimates are based on four large prospective American studies[20–24] and assume that a 1 mg/dl reduction in low-density lipoprotein reduces IHD by 1%[25] and that a 1 mg/dl increase in serum high-density lipoprotein cholesterol reduces IHD by 2% in men and by 3% in women.[26] Marshall used a similar method, but relied on older data[7] together with Clarke's estimates.[6] We did not consider the relationship between serum cholesterol and fatal cerebrovascular disease, because this is less well established.[18 27 28]

We used data derived from a quantitative meta-analysis, shown in table 3, to estimate the effect of salt intake on mortality from stroke and IHD.[8] We took data from the British Heart Foundation's statistical database (www.heartstats.org.uk) for estimates of the number of deaths from cardiovascular disease in the UK, for 2003.

RESULTS

Table 4 shows results from the three taxation regimens examined, comparing expenditure, nutrient intake and effects on cardiovascular disease. In addition, the table shows the results previously obtained by Marshall in his analysis of a fat tax. Both models taxed a similar proportion of dietary saturated fat. The change in dietary saturated fat intake predicted by our model is about half that predicted by Marshall, and with changes in other fats considered, noticeably a rise in serum cholesterol from a fall in polyunsaturated fat, shown in table 5, the net effect on serum cholesterol is negligible. Our saturated fat estimate suggested that such a tax could cause a small rise in salt intake, as a result of cross-price elasticities of demand, and might overall result in more deaths than it averts. Sensitivity analysis showed that the magnitude of this effect is dependent on the cross-price elasticity between subcategory items, and varies from 1800 to 4000 extra deaths, depending on the cross-price elasticity chosen between 0 and 1. If the effect of salt intake on cardiovascular disease was half of the lower estimate of He and MacGregor,[8] then the number of extra deaths would fall to between 1300 and 1900.

Our second estimate, taxing items with a high SSCg3d score, predicted a small increase in serum cholesterol. However, overall the SSCg3d estimate predicted a reduction in deaths, due to cardiovascular disease, of between 2100 and 2500 every year, primarily owing to a substantial reduction in salt intake. Our best outcome estimate predicted a reduction of between 2600 to 3200 cardiovascular disease deaths every year, again largely by reducing salt intake. These estimates of number of lives saved were not strongly affected by altering the within-category cross-price elasticity value between 0 and 1 (ranging from 1900 to 2700 for SSCg3d, and from 2600 to 3200 for best outcome. If the effect of salt intake on cardiovascular disease was half of the lower estimate of He and MacGregor,[8] then the number of lives saved would be 700 for SSCg3d and 1100 for best outcome.

All three taxation estimates predicted a fall in fruit and vegetable consumption of approximately 2–4%, again as a result of cross-elasticity effects. The saturated fat tax would increase weekly household food expenditure by approximately 3.2%, the SSCg3d tax would increase expenditure by 4%, and the best outcome approach would increase expenditure by 4.6%, equivalent to an extra 67 pence per person per week or approximately £2 billion annually across the UK (2000 prices).

DISCUSSION

Our estimate of the effect on cardiovascular disease of introducing taxes on the major sources of dietary fat is

Table 1 Categories of food used within the model, together with consumption and nutritional data for the UK

Foodstuff	Items taxed in different models			Contribution to total dietary consumption	
	1: Saturated fat	2: SSCg3d	3: Best outcome	Saturated fat (g)	Salt (mg)
Milk and cream					
Whole milk	Yes	–	Yes	8.6	–
Skimmed milks	–	–	Yes	5.5	–
Yogurt and fromage frais	–	–	–	1.0	–
Others	–	Yes	Yes	3.1	5.4
Cheese	Yes	Yes	Yes	10.3	4.3
Carcase meat					
Beef and veal	–	–	–	3.4	0.4
Mutton and lamb	–	–	–	1.7	–
Pork	–	–	–	1.7	0.4
Other meat and meat products					
Bacon and ham	–	Yes	Yes	2.4	7.8
Poultry, uncooked	–	–	–	2.1	0.8
Others		Yes	Yes	10.7	11.6
Fresh fish	–	–	–	0.2	0.6
Processed and shell fish	–	–	–	0.1	0.3
Prepared fish	–	–	–	0.4	1.0
Frozen fish	–	–	–	0.3	0.8
Eggs	–	–	–	1.0	0.8
Fats					
Butter	Yes	Yes	Yes	10.3	1.2
Margarine	–	Yes	Yes	2.1	0.8
Low fat and dairy spreads	–	Yes	Yes	4.5	2.3
Vegetable and salad spreads	–	Yes	Yes	2.1	–
Other	–	Yes	Yes	2.1	0.4
Sugar and preserves	–	Yes	Yes	–	–
Fresh potatoes	–	–	–	–	–
Other fresh vegetables	–	–	–	–	–
Processed vegetables					
Processed potatoes, inc frozen	–	–	–	4.1	3.1
Others	–	–	–	0.7	5.4
Fresh fruit	–	–	–	0.3	–
Other fruit and fruit products	–	–	–	0.7	–
Bread	–	–	–	0.7	13.6
Cereals					
Cakes, pastries and biscuits	Yes	Yes	Yes	11.4	5.0
Breakfast cereals	–	Yes	–	0.3	5.0
Others	–	Yes	Yes	4.5	27.9

Main categories are shown in bold type; subcategories are shown in plain type.
Data taken from the National Food Survey, 2000, for the UK[17].

significantly different from that reported previously by Marshall.[14] The change in dietary saturated fat intake that we predicted would result from such a fat tax is smaller than Marshall predicted. Marshall's work had a number of limitations. Firstly, it used the author's own estimates of the effect of taxation on the consumption of food, rather than empirically obtained values. The empirical elasticity values are less than his estimates and the empirical data suggest there will be greater substitution with other high-fat products than he predicted. Secondly, the analysis was restricted to saturated fat and did not take account of the impact of a tax on the intake of other nutrients, such as polyunsaturated fat and salt. Our model suggests that beneficial reductions in saturated fat are partly offset by reductions in polyunsaturated and monounsaturated fat and

that a small rise in salt intake from such a tax would have a detrimental effect on mortality from cardiovascular disease.

The cross-elasticity effects show that food consumption is highly interdependent and difficult to predict. Our model suggests that there could be a variety of unintended potentially detrimental effects, caused by the estimated cross-price elasticities of demand. For example, we observed that reducing saturated fat consumption tended to increase salt consumption and that fruit consumption tended to fall as a result of taxation on milk and cream.

When designing the best outcome taxation strategy, we found it hard to achieve a reduction in serum cholesterol: when taxing broad categories of foods, if the intake of saturated fat is reduced the intake of other fats such as polyunsaturates and monounsaturates is also reduced. The reduction in intake of

Table 2 Effect of changing intake of different dietary fats and dietary cholesterol on serum cholesterol

	Change in HDL (mmol/l)	Change in LDL (mmol/l)	Total cholesterol
1% increase in energy derived from saturated fat	0.013	0.036	0.052
1% increase in energy derived from monounsaturated fat	−0.008	0.006	0.005
1% increase in energy derived from polyunsaturated fat	−0.022	0.005	−0.026
1 mg increase in cholesterol per day	0.0005	0.0001	0.0007

From Clarke et al,[6] 1997.
HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 3 Effect of reduction in salt intake on the incidence of stroke and ischaemic heart disease

Reduction in salt intake (g/day)	Reduction in risk of stroke (%)*	Reduction in risk of IHD (%)*
3	12–14	9–10
6	23–25	16–19
9	32–36	23–27

IHD, ischaemic heart disease.
*Lower estimate based on changes in the systolic blood pressure and higher estimate based on changes in diastolic blood pressure. Based on He and MacGregor, 2003.[8]

Table 5 Estimate of the effect of changes in intake of different types of fat and dietary cholesterol on population mean serum cholesterol, achieved by extending value added tax to the principal sources of saturated fat

	Change in cholesterol (mmol/l)		
	HDL	LDL	Total cholesterol
Saturated fats	−0.0017	−0.0045	−0.0069
Monounsaturated fat	−0.0027	0.0036	−0.0022
Polyunsaturated fat	−0.0020	0.0090	0.0106
Cholesterol	0.0001	0.0003	0.0005
Total effect	−0.006	0.008	0.002

HDL, high-density lipoprotein, LDL, low-density lipoprotein.

polyunsaturates and monounsaturates causes a rise in serum cholesterol that counters the fall achieved from the reduction in saturated fat. In addition, we observed a trade off between the reduction in the proportion of energy derived from saturated fat and a reduction in salt intake; some food products high in salt, such as some cereal products, also tend to be high in sugar and therefore energy dense; so reducing salt intake reduces non-fat calorie intake, increasing the proportion of energy derived from saturated fat, which tends to increase serum cholesterol. The additional benefit gained from taxing items outside the least healthy category was small. Moreover, as the more healthy food items were taxed, in addition to the less healthy food items (ie, extending VAT to nearly all food items), the potential number of lives saved tended to fall slightly and a further reduction in fruit and vegetable consumption was observed. Further gains would best be achieved by a higher rate of taxation on least healthy food items, rather than extending the VAT at 17.5% to healthier food items. Food consumption is relatively insensitive to price changes, such that a taxation rate of 17.5% is likely to reduce the intake of nutrients such as salt and saturated fats by no more than 5–10%. So the scope for significantly altering the national diet by judicious use of VAT seems limited. Greater change could be achieved with a higher level of taxation, but this is unlikely for political and economic reasons. To achieve large changes, such as 3–6 g per day reduction in salt intake[8] or 0.60 mmol/l reduction in serum cholesterol,[7] which have been advocated, it may be necessary for the food industry to produce foods with less salt and less saturated fat. Rose argued that small changes in risk factors for diseases of high prevalence can produce meaningful change at a population level,[29] and our results suggest that, although the percentage changes in the incidence of cardiovascular disease are small, the actual number of lives saved could be substantial because of the high

incidence of cardiovascular disease in the UK. Fat taxes would not eliminate dietary-related diseases, but could be one of several tools used to achieve that goal.

Our data should be interpreted cautiously. Firstly, our estimates of the effect of targeted food taxes on the number of deaths are crude and limited to the effects of dietary fat and salt on cardiovascular disease; they give only a rough guide to the magnitude of the effects that we may see. We have not looked at the effect of other nutrients on health, which are not well characterised; about one third of the cancer mortality could be related to diet.[7] Secondly, the model assumes that all food purchased for consumption at home is eaten: we make no allowance for the proportion of food that is not eaten, but discarded. We have also assumed that food eaten outside the home is unaffected by the taxation. Thirdly, the food categories used in the model contain a wide range of products, and it may not be reasonable to assume that all products within each category will behave in the same way: our experience, from using the model, suggests that better targeting of taxation to smaller more precise food categories produces better results. Fourthly, the relationships between price changes and changes in health have been captured using aggregate population data. A variety of factors other than price influence individual food purchase behaviour, including the palatability of foods, attitudes to and knowledge about foods, and some of these factors are captured in price elasticity of demand. Similarly, a variety of factors other than diet, such as age, sex and lifestyle factors, like smoking, influence health. A more complex model could in theory incorporate these factors. Fifth, our elasticity data are based on the UK population, so making predictions for other countries, particularly with respect to possible cross-price elasticity effects, may be inappropriate.

Table 4 A comparison of the results for the three estimates from our model

	1: Tax foods high in saturated fats (Marshall's proposal)		2: Tax based on SSCg3d score	3: Tax to obtain best outcome
	Our estimate	Marshall's estimate [14]		
Change in household food expenditure (%)	3.2	Not estimated	4.0	4.6
Percentage of total food expenditure taxed	9.9	Not estimated	33.3	44.5
Percentage of dietary saturated fat taxed	41	44	64	80
Change in % calories derived from saturated fat	−0.13	−0.67	0.09	0.05
Change in salt intake (%)	5.2	Not estimated	−5.8	−6.6
Change in non-milk extrinsic sugar intake (%)	−1.5	Not estimated	−7.3	−7.6
Change in calories consumed (%)	2.2	Not estimated	−4.3	−6.1
Change in fruit and vegetable intake (%)	−1.2	Not estimated	−3.9	−3.9
Mean change in serum cholesterol	0.002	−0.044 to −0.052	0.009	0.005
Change in mortality from IHD (%)	1.3 to 2.0	−1.8 to −2.6	−0.8 to −1.1	−1.2 to−1.5
Change in mortality from stroke (%)	1.5 to 1.7	Not estimated	−1.6 to −1.9	−1.8 to−2.1
Overall change in annual number of CVD deaths (UK)	Increase 2500 to 3500*	Decrease 2100–3100	Decrease 2100 to 2500*	Decrease 2600 to 3200*

CVD, cardiovascular disease; IHD, ischaemic heart disease.
*Lower estimate based on He's lower estimate for the effect of salt on CVD and the lower estimate of Katan's or Law's method. Higher estimate based on He's upper estimate for the effect of salt on CVD and the higher estimate of Katan's or Law's method.

What is already known

- Fat taxes have been proposed to alter consumption patterns and reduce the incidence of diet-related disease.
- Fat taxes will be regressive and resisted by the food industry. Most taxes levied on food are aimed at raising revenue, and the effect on health is uncertain.

What this paper adds

Fat taxes may be used to produce modest changes in diet, which at a population level would have a meaningful effect on mortality. More research is needed to better understand the potential effects of any fat tax, particularly the effect on the poor.

Policy implications

Fat taxes could be used, by governments to improve national diet and reduce the incidence of diet-related disease. Careful consideration needs to be given to designing fat taxes, examining the effect of any taxation on a range of nutrients.

Other approaches to fat taxes, such as taxes on specific ingredients such as salt or added sugar, are beyond the scope of this paper. The type of fat tax we have modelled in this study could be portrayed as a restriction on personal freedom. However, it could also be considered as a counterweight to other market influences, such as advertising, that affect our choice of food and the associated health benefits and harms.

One common criticism of fat taxes is that they are regressive—that is, that low-income households would pay a greater percentage of their income on fat taxes than higher-income households.[30] An analysis of the effects on different income groups was not possible using our model, because the National Food Survey does not provide price elasticity data split by economic group. However, theoretically, those on low incomes should be more price sensitive in their pattern of demand and therefore may be more likely to change their consumption patterns and obtain larger proportional health benefit, as seems to happens with cigarette consumption.[11]

CONCLUSIONS

The factors that affect food consumption patterns are highly interdependent, and careful consideration of unintended cross-elasticity effects and changes across a range of nutrients need to be considered when assessing the effect of any fat tax. The potential changes in nutrition that would result from an extension of VAT to further categories of food are modest. However, given the high incidence of cardiovascular disease and the acknowledged contributory role of dietary salt and fat, inducing even small changes in diet has the potential to produce worthwhile population level changes in its incidence. A well-designed and carefully targeted fat tax could be a useful tool for reducing the burden of food-related disease.

. . . .
Authors' affiliations

Oliver Mytton, Queen's Medical Centre, Nottingham, UK
Alastair Gray, Department of Public Health, University of Oxford Old Road Campus, Health Economics Research Centre, Headington, Oxford, UK
Mike Rayner, Department of Public Health, University of Oxford Old Road Campus, British Heart Foundation Health Promotion Research Group, Headington, Oxford, UK
Harry Rutter, South East Public Health Observatory, 4150 Chancellor Court, Oxford Business Park, Oxford, UK

REFERENCES

1 **Donaldson L**. *Health check: Annual report of the Chief Medical Officer 2002*. London: DoH, 2002.
2 **House of Commons Health Committee**. *Obesity: third report of session 2003–4*. London: The Stationery Office, 2004.
3 **World Health Organisation—Food and Agricultural Organization**. *Diet, nutrition and the prevention of chronic diseases. Technical report series 916*. Geneva: WHO, 2003.
4 **Organisation for Economic Co-operation and Development**. *Health data 2005: obesity*. Paris: OECD, 2005.
5 **Rayner M**, Scarborough P. The burden of food-related ill health in the UK. *J Epidemiol Community Health* 2005;**59**:1054–7.
6 **Clarke R**, Frost C, Collins R, *et al*. Dietary lipids and blood cholesterol: quantitative meta-analysis of metabolic ward studies. *BMJ* 1997;**314**:112–17.
7 **Law MR**, Wald NJ, Thompson SG. By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? *BMJ* 1994;**308**:367–73.
8 **He FJ**, MacGregor GA. How far should salt intake be reduced? *Hypertension* 2003;**42**:1093–9.
9 **Caraher M**, Cowburn G. Taxing food: implications for public health nutrition. *Public Health Nutr* 2005;**8**:1244–51.
10 **European Heart Network**. *Food, nutrition and cardiovascular disease prevention in the European region: challenges for the new millennium*. Brussels: EHN, 2002.
11 **Townsend J**, Roderick P, Cooper J. Cigarette smoking by socio-economic group, sex, and age: effects of price income and publicity. *BMJ*, 1994;309, 923–7.
12 **Godfrey C**, Maynard A. Economic aspects of tobacco use and taxation policy. *BMJ*, 1988;297, 339–43.
13 **Institute of Alcohol Studies**. *Alcohol and tax*. St Ives: Institute of Alcohol Studies, 2003.
14 **Marshall T**. Exploring a fiscal food policy: the case of diet and ischaemic heart disease. *BMJ* 2000;**320**:301–5.
15 **Jacobsen MF**, Brownell KD. Small taxes on soft drinks and snack foods to promote health. *Am J Public Health* 2000;**90**:854–7.
16 **Rayner M**, Scarborough P, Stockley L. Nutrient profiles: options for definitions for use in relation to food promotion and children's diets. London: Food Standards Agency, 2004.
17 **Ministry of Agriculture Fisheries and Foods**. *National Food Survey: household food consumption and expenditure 2000*. London: Ministry of Agriculture Fisheries and Foods, 2000.
18 **Law MR**, Wald NJ, Rudnicka AR. Quantifying effect of statins on low density lipoprotein cholesterol, ischaemic heart disease, and stroke: systematic review and meta-analysis. *BMJ* 2003;**926**:1423–30.
19 **Sacks FM**, Katan M. Randomized clinical trials on the effects of dietary fat and carbohydrate on plasma lipoproteins and cardiovascular disease. *Am J Med* 2002;**113**:13s–24s.
20 **Dawber TR**, Meadors GF, Moore FE. Epidemiological approaches to heart disease: the Framingham Study. *Am J Publ Health* 1951;**41**:279–86.
21 **Gordon DJ**, Ekelund LG, Karon JM, *et al*. Predictive value of exercise tolerance test for mortality in North American men: the Lipid Research Clinics Mortality Follow-up Study. *Circulation* 1986;**74**:252–61.
22 **The Lipid Research Clinics Program**. The lipid research clinics coronary primary prevention trial results: design and implementation. *J Chronic Dis* 1979;**32**:609–31.
23 **The Lipid Research Clinics Program**. The lipid research clinics coronary primary prevention trial results: I Reduction in the incidence of coronary heart disease. *JAMA* 1984;**251**:351–64.
24 **Multiple Risk Factor Intervention Trial Research Group**. Multiple risk factor intervention trial: risk factor changes and mortality results. *JAMA* 1982;**248**:1465–77.

25 **Gordon DJ**, Rifkind BM. High-density lipoprotein—the clinical implications of recent studies *N Engl J Med* 1989;**321**:1311–16.
26 **Gordon DJ**, Probstfield JL, Garrison RJ, *et al.* High-density lipoprotein cholesterol and cardiovascular disease: four prospective American studies. *Circulation* 1989;**79**:8–15.
27 **Oliver M**. Cholesterol and strokes. *BMJ* 2000;**320**:459–60.
28 **Baigent C**, Keech A, Kearney PM, *et al.* Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of randomized data from 90,056 participants in 14 randomised trials of statins. *Lancet* 2005;**366**:1267–78.
29 **Rose G**. *The strategy of preventive medicine*, Oxford University Press, 1992.
30 **Crawford I**, Leicester A, Windmeijer F. *The 'fat tax'*. London: The Institute of Fiscal Studies, 2004.

BMJ Clinical Evidence—Call for contributors

BMJ Clinical Evidence is a continuously updated evidence-based journal available worldwide on the internet which publishes commissioned systematic reviews. *BMJ Clinical Evidence* needs to recruit new contributors. Contributors are healthcare professionals or epidemiologists with experience in evidence-based medicine, with the ability to write in a concise and structured way and relevant clinical expertise.

Areas for which we are currently seeking contributors:
- Secondary prevention of ischaemic cardiac events
- Acute myocardial infarction
- MRSA (treatment)
- Bacterial conjunctivitis

However, we are always looking for contributors, so do not let this list discourage you.

Being a contributor involves:
- Selecting from a validated, screened search (performed by in-house Information Specialists) valid studies for inclusion.
- Documenting your decisions about which studies to include on an inclusion and exclusion form, which we will publish.
- Writing the text to a highly structured template (about 1500–3000 words), using evidence from the final studies chosen, within 8–10 weeks of receiving the literature search.
- Working with *BMJ Clinical Evidence* editors to ensure that the final text meets quality and style standards.
- Updating the text every 12 months using any new, sound evidence that becomes available. The *BMJ Clinical Evidence* in-house team will conduct the searches for contributors; your task is to filter out high quality studies and incorporate them into the existing text.
- To expand the review to include a new question about once every 12 months.

In return, contributors will see their work published in a highly-rewarded peer-reviewed international medical journal. They also receive a small honorarium for their efforts.

If you would like to become a contributor for *BMJ Clinical Evidence* or require more information about what this involves please send your contact details and a copy of your CV, clearly stating the clinical area you are interested in, to CECommissioning@bmjgroup.com.

Call for peer reviewers

BMJ Clinical Evidence also needs to recruit new peer reviewers specifically with an interest in the clinical areas stated above, and also others related to general practice. Peer reviewers are healthcare professionals or epidemiologists with experience in evidence-based medicine. As a peer reviewer you would be asked for your views on the clinical relevance, validity and accessibility of specific reviews within the journal, and their usefulness to the intended audience (international generalists and healthcare professionals, possibly with limited statistical knowledge). Reviews are usually 1500–3000 words in length and we would ask you to review between 2–5 systematic reviews per year. The peer review process takes place throughout the year, and our turnaround time for each review is 10–14 days. In return peer reviewers receive free access to *BMJ Clinical Evidence* for 3 months for each review.

If you are interested in becoming a peer reviewer for *BMJ Clinical Evidence*, please complete the peer review questionnaire at www.clinicalevidence.com/ceweb/contribute/peerreviewer.jsp