LETTERS

Letters are selected from rapid responses posted on bmj.com. After editing, all letters are published online (www.bmj.com/archive/sevendays) and about half are published in print
To submit a rapid response go to any article on bmj.com and click "respond to this article"

SATURATED FAT IS NOT THE MAJOR ISSUE

What we substitute for saturated fat matters

Malhotra questions the wisdom of continuing public health recommendations to limit dietary saturated fat for the prevention of cardiovascular disease and went on to suggest that dietary saturated fat may decrease the risk of cardiovascular disease.[1] His assessment seems to be missing a crucial point—the displacement source of energy for saturated fat. Data suggest that replacing saturated fat with carbohydrate has no effect on the risk of cardiovascular disease.[2 3] However, replacement by polyunsaturated fat protects against the risk of cardiovascular disease.[2-4] These associations hold regardless of whether the conclusions are drawn from pooled analyses or meta-analyses.[2 3]

At this time, on the basis of all the data, the best dietary advice that we can give to reduce the risk of cardiovascular disease is to eat a moderate fat diet. We should also advise replacing saturated fat with polyunsaturated fat—that is, replacing animal fats (meat and dairy) with vegetable oils—within the context of an energy intake that is consistent with achieving and maintaining a healthy body weight.

Alice H Lichtenstein Gershoff professor of nutrition science and policy, Tufts University, Boston, MA, USA
alice.lichtenstein@tufts.edu

Competing interests: None declared.

1 Malhotra A. Saturated fat is not the major issue. *BMJ* 2013;347:f6340. (22 October.)
2 Jakobsen MU, O'Reilly EJ, Heitmann BL, Pereira MA, Balter K, Fraser GE, et al. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. *Am J Clin Nutr* 2009;89:1425-32.
3 Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. *PLoS Med* 2010;7:e1000252.
4 Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Saturated fatty acids and risk of coronary heart disease: modulation by replacement nutrients. *Curr Atheroscler Rep* 2010;12:384-390.

Cite this as: *BMJ* 2013;347:f6844

Public Health England endorses limiting saturated fat intake

Malhotra's article is based on limited evidence.[1] Government recommendations of limiting saturated fat intake to no more than 11% of total food energy to reduce the risk of cardiovascular disease are based on longstanding advice from the Committee on Medical Aspects of Food Policy.[2] These recommendations are also endorsed by the Scientific Advisory Committee on Nutrition, which provides the UK with independent scientific advice on nutrition. The advice is in line with recent thorough assessments made by the Institute of Medicine (2005), WHO (2008), and the European Food Safety Authority (2010).

Good evidence from randomised controlled trials shows that saturated fat consumption influences cholesterol concentrations and increases the risk of cardiovascular disease. For example, a meta-analysis of 16 trials found that heart attacks and deaths from heart disease were significantly reduced in studies that significantly lowered serum cholesterol by replacing saturated fat with polyunsaturated fat.[3] A Cochrane systematic review concluded that reducing or modifying fat intakes to reduce saturated intake lowered the risk of cardiovascular events by 14%.[4] The combined results of 14 trials investigating statins also found that a sustained 1 mmol/L reduction in low density lipoprotein-cholesterol over five years reduced major vascular events by 23%.[5]

On the basis of all the evidence, Public Health England will continue to advise people to eat a diet that is low in saturated fat and it supports the Department of Health's responsibility deal incentives to reduce saturated fat in foods. Public Health England also supports broader changes to the diet, including reductions in salt, sugar, and energy intake and increases in fruit and vegetable consumption.

Alison Tedstone director of diet and obesity
Vicki Pyne public health nutritionist, Public Health England, London SE1 8UG, UK

phe.enquiries@phe.gov.uk

Competing interests: None declared.

Full response at: www.bmj.com/content/347/bmj.f6340/rr/669645.
1 Malhotra A. Saturated fat is not the major issue. *BMJ* 2013;347:f6340. (22 October.)
2 Committee on Medical Aspects of Food Policy. Nutritional aspects of cardiovascular disease. Stationery Office, 1994.
3 Skeaff CM, Miller J. Dietary fat and coronary heart disease: summary of evidence from prospective cohort and randomised controlled trials. *Ann Nutr Metab* 2009;55:173-201.
4 Hooper L, Summerbell CD, Thompson R, Sills D, Roberts FG, Moore HJ, et al. Reduced or modified dietary fat for preventing cardiovascular disease. *Cochrane Database Syst Rev* 2012;5:CD002137.
5 Baigent C, Keech A, Kearney PM, Blackwell L, Buck G, Pollicino C, et al. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90056 participants in 14 randomised trials of statins. *Lancet* 2005;366:1267-78.

Cite this as: *BMJ* 2013;347:f6850

Evidence favours link between saturated fat intake and CHD

Malhotra provides useful reminders about the causes of coronary heart disease (CHD),[1] which are complex, with no single factor being "the major issue."[1] Many patients who now present with myocardial infarction are overweight and have features of the metabolic syndrome.

However, contrary to Malhotra's statement that "advice [to reduce saturated fat intake] has, paradoxically, increased our cardiovascular risks,"[1] the past several decades have seen substantial reductions in mortality from CHD.[2] Such reductions have occurred in parallel with reductions in saturated fat intake and serum cholesterol concentrations in much of the Western world.[3] These observations are supported by findings of cohort studies and clinical trials.[4]

A meta-analysis of observational studies in nearly 900 000 adults in Western countries showed a linear association between total cholesterol and CHD mortality.[4] A similar association has repeatedly been shown for cholesterol and non-fatal CHD.[5] Clinical trials of cholesterol lowering by diet or drugs confirm that CHD risk reduction is proportional to the extent of cholesterol lowering.[6]

Direct evidence for an association between saturated fat intake and CHD is less clear; this is not surprising because cohort studies can be confounded by problems such as not accounting for sources of replacement energy and misreporting of dietary intakes. Nevertheless, a data pooling study of 11 prospective cohort studies showed that replacing saturated fat with polyunsaturated fat significantly reduced CHD risk, whereas substitution with carbohydrate modestly increased the risk.[7] Arguably, the strongest confirmation of association comes from randomised controlled trials. A meta-analysis of eight relatively long term trials showed a significant reduction in CHD events in studies where saturated fats were reduced mainly by substitution with polyunsaturated fat.[8]

Jim Mann professor, Departments of Human Nutrition and Medicine, and Edgar National Centre for Diabetes and Obesity Research, University of Otago, Dunedin 9054, New Zealand
jim.mann@otago.ac.nz

Rachael McLean senior lecturer, Departments of Human Nutrition and Preventive and Social Medicine, University of Otago, Dunedin 9054, New Zealand
Lisa Te Morenga research fellow, Department of Human Nutrition, and Riddet Institute, University of Otago, Dunedin 9054, New Zealand
Competing interests: None declared.
Full response at: www.bmj.com/content/347/bmj.f6340/rr/669477.

1 Malhotra A. Saturated fat is not the major issue. *BMJ* 2013;347:f6340. (22 October.)
2 Tunstall-Pedoe H. The decline in coronary heart disease; did it fall or was it pushed? *BMJ* 2012;344:d7809.
3 Farzadfar F, Finucane MM, Danaei G, Pelizzari PM, Cowan MJ, Paciorek CJ, et al. National, regional, and global trends in serum total cholesterol since 1980: systematic analysis of health examination surveys and epidemiological studies with 321 country-years and 3.0 million participants. *Lancet* 2011;377:578-86
4 Prospective Studies Collaboration. Blood cholesterol and vascular mortality by age, sex, and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55 000 vascular deaths. *Lancet* 2007;370:1829-39.
5 Asia Pacific Cohort Studies Collaboration. Cholesterol, coronary heart disease, and stroke in the Asia Pacific region. *Int J Epidemiol* 2003;32:563-72.
6 Cholesterol Treatment Trialists' (CTT) Collaboration. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170 000 participants in 26 randomised trials. *Lancet* 2010;376:1670-81.
7 Jakobsen MU, O'Reilly EJ, Heitmann BL, Pereira MA, Bälter K, Fraser GE, et al. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. *Am J Clin Nutr* 2009;89:1425-32.
8 Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. *PLoS Med* 2010;7:e1000252.

Cite this as: *BMJ* 2013;347:f6851

Sugar, not fat, is the culprit

For decades, a diet high in fat (particularly, saturated fat) has been cited as the cause of cardiovascular disease.[1] But not everything stacks up. Epidemiological studies of populations such as the Inuit, who traditionally survive almost entirely on animal food sources (and consume large amounts of saturated fat) have a very low incidence of cardiovascular disease. By demonising fat for decades, we have been side tracked from targeting the real culprit, refined carbohydrates, particularly sugar.

Numerous randomised controlled trials have shown that a low carbohydrate diet (versus a low fat diet) results in higher concentrations of high density lipoprotein (protective against heart disease) and lower triglyceride concentrations.[2-4] A prospective cohort study of 75 521 women followed for 10 years found that a diet high in refined carbohydrate is an independent risk factor for cardiovascular disease.[5]

Despite this, the official NHS guidance on sugar is inadequate and contradictory. It recommends us to cut down on the amount of sugar we consume yet encourages us to consume five portions of fruit and vegetables a day. Under this guidance, juices, smoothies, canned, and dried fruit—all high in sugar—count as part of the "five a day" (www.nhs.uk/

Livewell/5ADAY/Pages/Whatcounts.aspx). Adherence to this advice can easily lead to excessive sugar consumption. To illustrate, a 150 mL glass of orange juice, a 250 mL smoothie, a 30 g portion of raisins, and 80 g of canned peaches amounts to 69 g of sugar or about 16 teaspoons of sugar. There is no maximum amount of sugar recommended but clearly 16 teaspoons of sugar a day is far from healthy. We have lost sight of the real culprit, and our public health message really needs to focus on sugar.

Danny C G Lim cardiology and medical registrar, Healthcare Locums, North East England, Durham, UK
dcglim@gmail.com
Competing interests: None declared.

1 Malhotra A. Saturated fat is not the major issue. *BMJ* 2013;347:f6340. (22 October.)
2 Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, et al. A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med* 2003;348:2082-90.
3 Shai I, Schwarzfuchs D, Henkin Y, Shahar DR, Witkow S, Greenberg I, et al. Weight loss with a low-carbohydrate, Mediterranean, or low-fat diet. *N Engl J Med* 2008;359:229-41.
4 Stern L, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, et al. The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. *Ann Intern Med* 2004;140:778-85.
5 Liu S, Willett WC, Stampfer MJ, Hu FB, Franz M, Sampson L, et al. A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *Am J Clin Nutr* 2000;71:1455-61.

Cite this as: *BMJ* 2013;347:f6846

Too soon to reject the saturated fat hypothesis

Judging by UK newspaper headlines this week I think that Malhotra and the *BMJ* have done a disservice to the public debate on nutrition.[1] Just because glycaemic load and soft drinks are associated with obesity and type 2 diabetes, it does not mean that saturated fat does not cause disease.

Hundreds of experiments have shown that saturated fat increases low density lipoprotein-cholesterol to a small degree.[2] In addition, clinical interventions that replace saturated fat with large amounts of polyunsaturated fat reduce cardiac events, even if it is unclear which polyunsaturated fat is best.[3] The Lyon Diet Heart Study found that an intervention which, along with many other dietary changes, lowered saturated fat and increased polyunsaturated fat protected against heart disease.[4] The benefits of a low glycaemic load diet on cardiovascular events are yet to be shown because no randomised trials have been done. The PREDIMED study found that a large amount of virgin olive oil or nuts reduces strokes in the context of a low saturated fat Mediterranean diet, but it could not show whether a low fat diet was harmful because none of the diets were low in fat.[5]

We need more data before rejecting the saturated fat hypothesis and confusing the public more.

Peter Clifton endocrinologist, University of South Australia, North Terrace, Adelaide, SA, Australia
peter.clifton@unisa.edu.au
Competing interests: None declared.

1 Malhotra A. Saturated fat is not the major issue. *BMJ* 2013;347:f6340. (22 October.)
2 Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am J Clin Nutr* 2003;77:1146-55.
3 Ramsden CE, Hibbeln JR, Majchrzak SF, Davis JM. n-6 fatty acid-specific and mixed polyunsaturate dietary interventions have different effects on CHD risk: a meta-analysis of randomised controlled trials. *Br J Nutr* 2010;104:1586-600.
4 De Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation* 1999;99:779-85.
5 Estruch R, Ros E, Salas-Salvadó J, Covas MI, Corella D, Arós F, et al; PREDIMED Study Investigators. Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med* 2013;368:1279-90.

Cite this as: *BMJ* 2013;347:f6847

Author's reply

Saturated fat is known to increase low density lipoprotein (LDL)-cholesterol. However, unlike carbohydrates, saturated fat also raises high density lipoprotein (HDL)-cholesterol and reduces triglycerides, with little effect on total cholesterol:HDL ratio—thought to be a better predictor of coronary heart disease (CHD) events than total cholesterol alone.[1 2]

The food source of saturated fat may be more important. A recent multi-ethnic study found that high intake of dairy saturated fat was associated with a lower CVD risk but high meat intake was associated with increased risk.[3] Although evidence supports replacing saturated fat with polyunsaturated fat to reduce CHD events, this benefit may be specific to omega 3, with omega 6 polyunsaturated fatty acids in vegetable oils implicated in raising proinflammatory atherogenic LDL. This may be why a reanalysis of unpublished data found that cardiac patients who replaced butter with safflower oil and margarine containing omega 6 had increased all cause and cardiovascular

mortality despite a 13% reduction in total cholesterol.

Furthermore, the food industry's exploitation of the "low fat" mantra has resulted in diets high in refined carbohydrate. It has fuelled worsening obesity and atherogenic dyslipidaemia, a metabolic state defined by increased triglycerides, reduced HDL-cholesterol, and increased proportions of small dense LDL particles. A reduction in carbohydrate intake but not saturated fat seems to improve this dyslipidaemic profile. The food industry has also exploited the obsession with total energy consumed rather than nutritional value by adding sugar to many processed foods. A nine teaspoon sugared cola has under 150 calories but the EPIC study found that drinking one can a day increased the risk of type 2 diabetes independently of body mass index. Conversely, in the PREDIMED study, consumption of four tablespoons of extra virgin olive oil a day (500 calories) significantly reduced risk of heart attack and stroke.

After a two year review of 16 000 studies, Sweden is the first Western nation to reject the "low fat" dietary dogma—advocating a diet that is high fat and low in refined carbohydrates as the best for cholesterol profile and weight loss. Promoting a Mediterranean diet would reduce the intake of processed food and added sugar, which unlike fat and protein has no nutritional value, causes dental caries, and is driving the metabolic syndrome. Such a policy might offer the best dietary solution to improving public health.

Aseem Malhotra interventional cardiology specialist registrar, Croydon University Hospital, London, UK
aseem_malhotra@hotmail.com

Competing interests: None declared.

Full response at www.bmj.com/content/347/bmj.f6340/rr/671980.

1 Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am J Clin Nutr* 2003;77:1146-55.
2 Assmann G, Schulte H, von Eckardstein A, Huang Y. High-density lipoprotein cholesterol as a predictor of coronary heart disease risk. The PROCAM experience and pathophysiological implications for reverse cholesterol transport. *Atherosclerosis* 1996;124(suppl):S11-20.
3 De Oliveira Otto MC, Mozaffarian D, Kromhout D, Bertoni AG, Sibley CT, Jacobs DR Jr, et al. Dietary intake of saturated fat by food source and incident cardiovascular disease: the Multi-Ethnic Study of Atherosclerosis. *Am J Clin Nutr* 2012;96:397-404.

Cite this as: *BMJ* 2013;347:f6855

AIRCRAFT NOISE AND HEALTH

$PM_{0.1}$ particles may increase risk of vascular disease

Hansell and colleagues reported an association between exposure to aircraft noise and increased risks of stroke, coronary heart disease, and cardiovascular disease in areas close to Heathrow airport.[1] They considered several confounding variables, including air pollution. However, air pollution was represented by PM_{10}—the mass concentration of suspended particles smaller than 10 µm—which does not reflect air pollution from aircraft.

Aircraft emit $PM_{0.1}$ particles, which are so low in mass that variations in $PM_{0.1}$ or even $PM_{2.5}$ do not correlate well with those of PM_{10}.[2 3] Yet $PM_{0.1}$ particles can penetrate deeper into the lungs and translocate more easily into the bloodstream than PM_{10},[4 5] so they may have led to the reported increased risks of vascular disease.

Unlike noise, which dissipates immediately, $PM_{0.1}$ emissions persist for hours. When emitted at low altitude, $PM_{0.1}$ would reach ground level within tens of minutes owing to turbulent mixing. Over the following hours, $PM_{0.1}$ would coagulate into $PM_{2.5}$ particles and persist in the air for several days.[3] These $PM_{2.5}$ particles have adverse health effects but are not equivalent to $PM_{0.1}$[4]—for example, their larger size means reduced lung penetration. Therefore the $PM_{0.1}$ coagulation lifetime of hours is comparable to Hansell and colleagues' 8-16 hour averaging periods, and the reported association between vascular health risks and aircraft activity may have reflected exposure to aircraft emitted $PM_{0.1}$ particles.

The authors probably used PM_{10} data because data on other pollutants were not available. Even when available, such data may reflect ground traffic rather than aircraft activity. Thus the aircraft-activity data they used to estimate noise exposure might have been the best available proxy for aircraft $PM_{0.1}$ pollution. The distinction between noise and $PM_{0.1}$ is important for policy makers: noise disperses immediately, but $PM_{0.1}$ particles may have diluted effects over a longer period and wider area.

Joel C Corbin atmospheric scientist, Institute for Atmosphere and Climate Science, ETH Zurich, 8092 Zurich, Switzerland joel.corbin@env.ethz.ch

Competing interests: None declared.

Full response at: www.bmj.com/content/347/bmj.f5432/rr/667230.

1 Hansell, AL, Blangiardo M, Fortunato L, Floud S, de Hoogh K, Fecht D, et al. Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study. *BMJ* 2013;347:f5432. (8 October.)
2 Liu Y-J, Harrison RM. Properties of coarse particles in the atmosphere of the United Kingdom. *Atmos Environ* 2011;45:3267-76.
3 Seinfeld JH, Pandis SN. Atmospheric chemistry and physics: from air pollution to climate change. John Wiley & Sons, 2012.
4 Heal MR, Kumar P, Harrison RM. Particles, air quality, policy and health. *Chem Soc Rev* 2012;41:6606-30.
5 Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease an update to the scientific statement from the American heart association. *Circulation* 2010;121:2331-78.

Cite this as: *BMJ* 2013;347:f6783

Study missed opportunity to confirm link

Might Hansell and colleagues explain the process for selecting confounders in their study of aircraft noise and cardiovascular disease?[1]

In acute clinical settings we estimate cardiovascular risk by accounting for age, sex, smoking, diabetes, blood pressure, hypercholesterolaemia, and family history. The article did not explain why smoking, ethnicity, deprivation, age, and sex were chosen as confounders over other well defined associations.

In particular, the authors went to the effort of working back from lung cancer to create a proxy marker for smoking prevalence. Yet there is no evidence of an attempt to include hypertension as a confounding factor. In the discussion, hypertension is postulated as the missing link in causality between noise and coronary heart disease. This is supported by many human studies that show a transient rise in blood pressure with noise and animal models that exhibit a chronic association between the two. It is a shame that the opportunity to confirm (or refute) this theory of causality was missed by not including hypertension in the calculation.

The effect of classic risk factors on cardiovascular disease is substantial, and a disparity in the prevalence of any of these factors could account for the results seen here. Until these confounders have been considered, the results of this well powered study show a correlation only.

But who knows? Perhaps one day "miles from Heathrow" will be considered alongside pack years of smoking as a risk factor for cardiovascular disease in acute clinical clerking.

Nicholas Moore doctor, NHS, Royal London Hospital, London, UK nickyamoore@googlemail.com

Competing interests: None declared.

1 Hansell, AL, Blangiardo M, Fortunato L, Floud S, de Hoogh K, Fecht D, et al. Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study. *BMJ* 2013;347:f5432. (8 October.)

Cite this as: *BMJ* 2013;347:f6788

Working at Heathrow may be a risk factor

Residual confounding is a possibility in Hansell and colleagues' study on cardiovascular disease and aircraft noise, and correlation is not causation.[1] I would like to suggest a

confounder. Living in the vicinity of Heathrow airport, I am often struck by the irregular hours worked by its employees, many of whom presumably live under the flight path. Shift work is known to be associated with vascular events and could be a residual confounder.[2] It is also possible that the aircraft noise disturbs their sleep even further.

If the effect of noise on cardiovascular events is immediate, a big if, then perhaps the effects of variation in flight paths or the six days of silence during the ash cloud in 2010 could be studied.

Jan Coebergh consultant neurologist, Ashford and St Peter's Hospitals NHS Trust, Chertsey KT16 0PZ, UK
jcoebergh@hotmail.com

Competing interests: I live under a flight path of Heathrow.

1 Hansell AL, Blangiardo M, Fortunato L, Floud S, de Hoogh K, Fecht D, et al. Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study. *BMJ* 2013;347:f5432. (8 October.)
2 Vyas MV, Garg AX, Iansavichus AV, Costella JC, Donner A, Laugsan LE, et al. Shift work and vascular events: systematic review and meta-analysis. *BMJ* 2012;345:e4800.

Cite this as: *BMJ* 2013;347:f6794

Authors' reply

In our recent paper on aircraft noise and cardiovascular disease near Heathrow airport we concluded, "As well as the possibility of causal associations, alternative explanations such as residual confounding . . . should be considered." Responses to our paper put forward possible candidates.

Corbin suggests that ultrafine particulate air pollution ($PM_{0.1}$) from aircraft could be a possible explanation for our findings. Although increased concentrations of such particles have been found near the runway during take-off,[1] we are unaware of data showing that this is the case in well mixed air up to tens of kilometres away from the airport. $PM_{0.1}$ particles are not a regulated pollutant in the UK, so there are no readily available data to investigate this.

Moore questions our choice of confounders. We adjusted for age, sex, area level ethnicity, and lung cancer (as proxy for smoking), but information on hypertension, cholesterol, and family history are not currently available within routine datasets at small area scale. Also, care would be needed in adjusting for hypertension if raised blood pressure lies on the causal pathway between aircraft noise and cardiovascular disease.

Coebergh raises the possibility that our findings reflect occupational hazards related to Heathrow. We think this is unlikely—stroke and coronary heart disease are most common in older people who are less likely to be still working.

Our paper concluded that "Further work to understand better the possible health effects of aircraft noise is needed," and these rapid responses support this notion.

Anna L Hansell assistant director
John Gulliver lecturer in environmental science, UK Small Area Health Statistics Unit, MRC-PHE Centre for Environment and Health, Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London W2 1PG, UK
Sean Beevers senior lecturer in air quality modelling, Environmental Research Group, MRC-PHE Centre for Environment and Health, King's College London, UK
Paul Elliott director, UK Small Area Health Statistics Unit, MRC-PHE Centre for Environment and Health, Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London W2 1PG, UK
p.elliott@imperial.ac.uk

Competing interests: See www.bmj.com/content/347/bmj.f5432.

1 Zhu Y, Fanning E, Yu RC, Zhang Q, Froines JR. Aircraft emissions and local air quality impacts from takeoff activities at a large international airport. *Atmos Environ* 2011;45:6526-33.

Cite this as: *BMJ* 2013;347:f6795

POLICY ON TOBACCO FUNDED RESEARCH

You should also shun research funded by the drug industry

I was the *BMJ* editor when we decided after much discussion that we would publish research funded by tobacco companies, but I respect the decision of the current editors of *BMJ*, *BMJ Open*, *Heart*, and *Thorax* not to do so.[1]

I do, however, have what I think is a difficult question for the editors, and I'd appreciate it if each of them could answer.

The two arguments for stopping publishing research funded by the tobacco industry are that the research is corrupted and that the companies are publishing research in journals mainly to advance their commercial aims, oblivious of the harm they do.

NICHOLAS RIGG/GETTY IMAGES

I suggest that exactly the same is true of the drug industry and that we probably have even more evidence on the misconduct of drug companies than of tobacco companies. Both Ben Goldacre and Peter Gøtzsche have gathered together this evidence in important books.[2 3]

So will the editors stop publishing research funded by the drug industry, and if not why not? Knowing the heavy financial dependence of journals on the drug industry, I shall be looking for sophistry in the explanations.

Richard Smith chair, Patients Know Best, London SW4 0LD, UK
richardswsmith@yahoo.co.uk

Competing interests: I was the editor of the *BMJ* and chief executive of the BMJ Publishing Group, in which role I was responsible for *Thorax* and *Heart*. I was a member of the board of the Public Library of Science from 2004 to 2011. I now work for a for-profit company, UnitedHealth Group, and am the chair of the board of a for-profit company, Patients Know Best, in which I have equity. I receive a pension from the BMA, the owners of the BMJ Group.

1 Godlee F, Malone R, Timmis A, Otto C, Bush A, Pavord I, et al. Journal policy on research funded by the tobacco industry. *BMJ* 2013;347:f5193. (15 October.)
2 Goldacre B. Bad pharma. Fourth Estate, 2012.
3 Gøtzsche P. Deadly medicines and organised crime: how big pharma has corrupted healthcare. Radcliffe, 2013.

Cite this as: *BMJ* 2013;347:f6732

Editor's reply

Smith makes a good point. Like the tobacco industry the drug industry has repeatedly been found to have manipulated and suppressed research data to make its products seem better than they are. Non-industry funded research also suffers from such misconduct, but the scale and influence of pharma funded research make its current state of particular concern.

I am firmly of the view that clinical practice should be based on independent research. Drug and device companies should not evaluate their own products. All phase III trials should be designed, analysed, and reported independently of the manufacturers. Clinical practice guidelines should be free of industry bias.

The *BMJ* is working on its own (www.bmj.com/open-data),[1] and in support of others (www.alltrials.net),[2] to increase transparency and accountability in clinical trials, to reduce the influence of industry in medical education and clinical guidelines,[3] and to unpick the legacy of research bias and misconduct.[4 5] If these efforts do not soon bring about a necessary sea change in the way industry funded trials are performed, the *BMJ* may decide to stop publishing such trials. Whether an editor would survive such a decision is a question I may have to test.

I would like to hear readers' views on what more we and others should be doing, especially on whether journals should continue to publish research funded by drug and device manufacturers.

Fiona Godlee editor in chief, *BMJ*, BMA House, London WC1H 9JR, UK fgodlee@bmj.com

Competing interests: See www.bmj.com/content/347/bmj.f5193/rr/671904.

1 Godlee F, Groves T. The new BMJ policy on sharing data from drug and device trials. *BMJ* 2012;345:e7888.
2 Kmietowicz Z. European regulator appeals against order stopping it from releasing clinical study documents. *BMJ* 2013;347:f4728. (24 July.)
3 Lenzer J. Why we can't trust clinical guidelines. *BMJ* 2013;346:f3830 (14 June.)
4 Abramson JD, Rosenberg HG, Jewell NJ, Wright JM. Should people at low risk of cardiovascular disease take a statin? *BMJ* 2013;347:f6123. (22 October.)
5 Doshi P, Dickersin K, Healy D, Vedula SW, Jefferson T. Restoring invisible and abandoned trials: a call for people to publish the findings. *BMJ* 2013;346:f2865. (13 June.)

Cite this as: *BMJ* 2013;347:f6859