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LETTERS

GLOBAL BAN ON TRANS FATS

Voluntary approaches work

Voluntary reformulation has virtually eliminated artificial trans fatty acids (TFAs) from processed foods in the UK. This demonstrates the effectiveness of voluntary policies, a fact that has been disputed.[1] Voluntary approaches enable the industry to move more quickly than legislative ones, allowing for a rapid response to consumer demand and scientific evidence.

Independent monitoring and evaluation of public health policies is needed whether voluntary or legislative action is taken and is fully supported by the food manufacturing industry. The results of the national diet and nutrition survey show the effectiveness of the voluntary removal of artificial TFAs.[2] Average TFA intakes (natural and artificial) were less than 2 g per day for all age groups (0.7-0.9% of food energy)—well below the maximum UK and World Health Organization recommendations.[3 4] The Department of Health has suggested that most TFAs consumed come from natural sources rather than artificial ones as a result of the efforts of the food industry.

Furthermore, in August the Department of Health published work led by the Institute of Food Research on TFAs in processed foods.[5] The analysis cannot distinguish between artificial and natural TFAs, but the total values were low; for example, TFAs in chocolate digestive biscuits have dropped from 1.60 g/100 g in 1992 to 0.04 g/100 g in 2008.

The Food and Drink Federation was a founding signatory of the government's public health responsibility deal, demonstrating our ongoing commitment to a multi-stakeholder and coordinated approach to improving public health.

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Competing interests: BG is a director of the Food and Drink Federation, the voice of the UK food and drink manufacturing industry.

1 Coombes R. Trans fats: chasing a global ban. *BMJ* 2011;343:d5311. (7 September.)
2 Department of Health. National diet and nutrition survey: headline results from years 1 and 2 (combined) of the rolling programme, 2008/9-2009/10. 2011. www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsStatistics/DH_128166.
3 Scientific Advisory Committee on Nutrition. Trans fatty acids and health. 2007.
4 WHO/FAO. Diet, nutrition, and the prevention of chronic diseases. 2002. www.fao.org/DOCREP/005/AC911E/AC911E00.HTM.
5 Department of Health. Nutrient analysis of a range of processed foods with particular reference to trans fatty acids. 2011.

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NUTRITIONAL CHANGE AND NCDs

Not as complex as it may seem

Preventing non-communicable diseases (NCDs) via nutritional change is not as complex as Yach asserts.[1] Firstly, he ignores the use of taxes on unhealthy foods, which are effective and can raise revenue. Such taxes have been successful in several high income countries, with some evidence of benefit (on weight and chronic disease risk).[2] Denmark, Finland, Norway, and Hungary all apply excise taxes on high sugar and high fat foods.[3] Modelling work suggests that generalised "unhealthy food" taxes are probably cost effective.[4] These moves would probably be feasible in many developing countries, especially those that successfully tax tobacco and alcohol. There is already evidence for the benefit of soft drink taxes in some developing countries,[5] and data from Egypt suggest that higher sugar prices help decrease body mass index.[2]

Yach also pays little attention to legislative measures, such as banning some ingredients outright (for example, trans fats in Denmark), limiting or banning food advertising directed at children, and appropriately labelling food packaging (front of pack traffic light nutrition labelling[4] and specific warnings such as "high salt"). These moves would be feasible in many developing countries, especially those that have successfully banned or restricted marketing of tobacco and other hazardous products, such as asbestos and leaded petrol.

Perhaps after these key steps are achieved, some of the complex issues that Yach raises could be tackled. By why not go first for the cost effective taxes and smart regulation that have been shown to work?

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Competing interests: None declared.

1 Yach D. Nutritional change is not a simple answer to non-communicable diseases. *BMJ* 2011;343:d5097. (11 August.)
2 Thow AM, Jan S, Leeder S, Swinburn B. The effect of fiscal policy on diet, obesity and chronic disease: a systematic review. *Bull World Health Organ* 2010;88:609-14.
3 Holt E. Hungary to introduce broad range of fat taxes. *Lancet* 2011;378:755.
4 Sacks G, Veerman JL, Moodie M, Swinburn B. "Traffic-light" nutrition labelling and "junk-food" tax: a modelled comparison of cost-effectiveness for obesity prevention. *Int J Obes (Lond)* 2011;35:1001-9.
5 Thow AM, Quested C, Juventin L, Kun R, Khan AN, Swinburn B. Taxing soft drinks in the Pacific: implementation lessons for improving health. *Health Promot Int* 2011;26:55-64.

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REDUCING POPULATION SALT INTAKE

The sodium phantom

Cappuccio and colleagues' analysis is unbalanced.[1] Firstly, the mean sodium intake in population studies that found a lowering effect of sodium reduction on blood pressure was above 250 mmol. Therefore these results cannot be applied to most of the world's populations, which have a mean intake of about 150 mmol.[2] In a population with a normal sodium intake, sodium reduction had no effect on blood pressure.[3]

Secondly, the fall in blood pressure and increased survival in Finland probably have nothing to do with reduced sodium intake, because in other parts of the world a 50% increase in sodium intake was associated with a fall in hypertension prevalence from 30% to 15%.[4]

Thirdly, "healthy physiological requirements of salt" may not be 1 g per day. Populations on minimal salt intake have extreme activation of the renin-aldosterone system and an increase in lipids and catecholamines.[5]

Fourthly, Strazzulo and colleagues' meta-analysis did not report an analysis of all cause mortality, which would have shown an increased trend with low sodium intake.[1]

Fifthly, the calculations of lives to be saved by sodium reduction are based on dubious assumptions from selected studies and contradicted by new population studies,[6] which show increased all cause mortality with low sodium intake.

Blood pressure is normal in 85% of the population,[4] so it is essential to determine whether sodium reduction significantly affects blood

pressure in normotensives before advising general sodium reduction. Given the possible side effects of sodium reduction,[5] this effect is not sufficient to recommend general sodium reduction. It is surprising that many countries have uncritically adopted sodium reduction, which probably is the largest delusion in the history of preventive medicine.

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Competing interests: None declared.

1 Cappuccio FP, Capewell S, Lincoln P, McPherson K. Policy options to reduce population salt intake. *BMJ* 2011;343:d4995. (11 August.)
2 McCarron DA, Geerling JC, Kazaks AG, Stern JS. Can dietary sodium intake be modified by public policy? *Clin J Am Soc Nephrol* 2009;4:1878-82.
3 Staessen J, Bulpitt CJ, Fagard R, Joossens JV, Lijnen P, Amery A. Salt intake and blood pressure in the general population: a controlled intervention trial in two towns. *J Hypertens* 1988;6:965-73.
4 Gregg EW, Cheng YJ, Cadwell BL, Imperatore G, Williams DE, Flegal KM, et al. Secular trends in cardiovascular disease risk factors according to body mass index in US adults. *JAMA* 2005;293:1868-74.
5 Stolarz-Skrzypek K, Kuznetsova T, Thijs L, Tikhonoff V, Seidlerová J, Richart T, et al; European Project on Genes in Hypertension (EPOGH) Investigators. Fatal and nonfatal outcomes, incidence of hypertension, and blood pressure changes in relation to urinary sodium excretion. *JAMA* 2011;305:1777-85.

Cite this as: *BMJ* 2011;343:d6119

Authors' reply

Graudal and Jürgens's first statement is incorrect. The TOHP Trial, for example, was carried out on a background of a contemporary American diet with an average baseline daily sodium intake of 150-180 mmol. An average moderate reduction of 33-44 mmol/day reduced cardiovascular disease outcomes by 52% and 21% and all cause mortality by 24% and 17% at 18 months and 3-4 years, respectively.

The Belgian study achieved no difference in salt intake, so unsurprisingly no effect was seen on measured outcomes. The example of Finland is unique. It highlights the substantial health benefits, without adverse effects on cardiovascular outcomes (as erroneously suggested by some), of a long term reduction in salt intake and shows how mandatory actions can be extremely effective in the implementation of public health policies.

Their third point has already been extensively dismissed in detailed scientific rebuttals in response to the 2008 publication in the Cochrane library,[1] and their lack of acknowledgment risks misleading readers not conversant with the literature. Strazzullo and colleagues' meta-analysis did not analyse all cause mortality because most of the studies included did not report such results. We invite Graudal and Jürgens to reconsider the doubtful value of the recent published study cited in support of their views, given the overwhelming international criticisms raised.[2-4] We are surprised at these omissions.

Finally, blood pressure is continuously distributed and the definitions of "normotension" and "hypertension" are not biologically justified. These definitions are constantly amended only to guide the cost effective use of drug treatment. Preventive medicine deals with all people, not just the ill. In conclusion, further denial and procrastination about dietary salt reduction will be costly in terms of avoidable illness and costs; it will also be ethically irresponsible.

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Competing interests: For full statement see www.bmj.com/content/343/bmj.d4995.full.

1 Jürgens G, Graudal NA. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglyceride. *Cochrane Database Syst Rev* 2004;1:CD004022.
2 Campbell N, Cappuccio FP, Tobe SW. Unnecessary controversy regarding dietary sodium: a lot about a little. *Can J Cardiol* 2011;27:404-6.
3 He FJ, Appel LJ, Cappuccio FP, de Wardener HE, MacGregor GA. Does reducing salt intake increase cardiovascular mortality? *Kidney Int* 2011;80:696-8.
4 Aleksandrova K, Pischon T, Weickert C, Rebholz CM, He J, Bochud M, et al. Urinary sodium excretion and cardiovascular disease mortality. *JAMA* 2011;306:1083-6.

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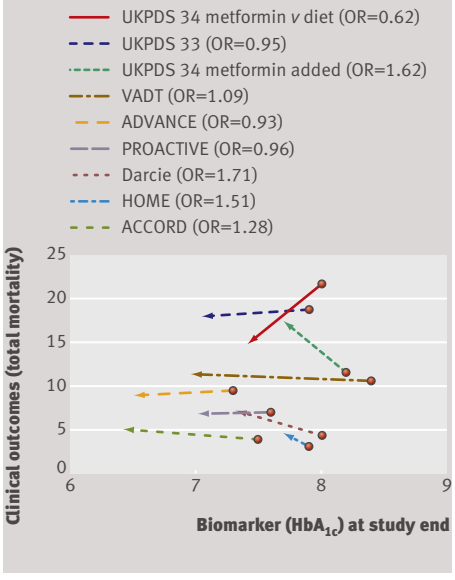
PLOTTING SURROGATE OUTCOMES

Arrow plots show way forward

Boussageon and colleagues conclude that relying on surrogate end points for treating people with type 2 diabetes is a fallacy.[1] Forest plots in meta-analyses do not, however, show well the relation between a surrogate outcome such as a biomarker and a clinical outcome such as mortality. We suggest that two dimensional arrow plots are better and display more of the information garnered from meta-analyses of biomarkers and clinical outcomes.[2]



Arrow plot of aggressive treatment of diabetes. Arrows start with results of control group and end with results of intervention group. Solid arrows indicate significant change in clinical outcome

In the figure the arrows start with the results of the control groups and end with the results of the intervention groups. The slopes of the arrows represent the relation between change in the biomarker (glycated haemoglobin, HbA_{1c}) and the change in clinical outcome (mortality).

The figure shows no relation between changes in HbA_{1c} and mortality. No definite clinical recommendation is possible. However, on the basis of results from the metformin compared with diet arm of the UKPDS 34 trial, metformin may significantly reduce mortality in patients with increased risk of mortality and when not combined with a sulphonylurea.[3]

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Competing interests: None declared.

1 Boussageon R, Bejan-Angoulvant T, Saadatian-Elahi M, Lafont S, Bergeonneau C, Kassai B, et al. Effect of intensive glucose lowering treatment on all cause mortality, cardiovascular death, and microvascular events in type 2 diabetes: meta-analysis of randomised controlled trials. *BMJ* 2011;343:d4169. (26 July.)
2 Badgett R, Dong F, Aung K. Meta-analysis for health care data. Joint Statistical Meetings, Miami, Florida, July-August 2011.
3 UK Prospective Diabetes Study (UKPDS) Group. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). *Lancet* 1998;352:854-65.

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ROLE OF COMPARATIVE EFFICACY

Requires legislation

Data on the comparative efficacy of drugs—or relative efficacy, as it is termed in the EU[1]—are certainly needed.[2] Apart from the pitfalls addressed by Sorenson and colleagues,[2] the political reality is that comparative efficacy is required for national reimbursement decisions, which, not surprisingly, are fiercely defended by member states. In 2009 the EU consensus was that we needed "principles that could be used as a basis for a toolbox" for assessing relative effectiveness, studying relevant data, overviewing existing networks, and developing collaboration.[1] Two years later we don't seem to have moved much.

Open dialogue between all interested parties is fundamental. Streamlining the authorisation of medicinal products has been proposed, [3] but a way to enforce it has not, to my knowledge,

been suggested. The highest possible degree of transparency and access to information and data is also fundamental, but achieving that has pitfalls too.[4]

Would an advisory board independent of the European Medicines Agency do the trick? In principle yes, but without a legally binding consensus it would have a modest effect at best. Making it legal will be a long and difficult road for the European Commission.

How should such a committee work? Scientific debate and arbitration should be transparent with a moderated open peer review system. Might this be an opportunity to involve the scientific and medical community of the EU, or even the rest of the world, and not just a few experts?

But first things first: if comparative efficacy is deemed necessary, an EU body is needed to guide the exercise.

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Competing interests: MM has no competing interests other than working with one of the many national medicines agencies in the EU providing scientific resources to the European Medicines Agency.

1 European Commission. The Pharmaceutical Forum. http://ec.europa.eu/pharmaforum.
2 Sorenson C, Naci H, Cylus J, Mossialos E. Evidence of comparative efficacy should have a formal role in European drug approvals. *BMJ* 2011;343:d4849. (6 September.)
3 Barker R. A flexible blueprint for the future of drug development. *Lancet* 2010;375:357.
4 Jefferson T, Doshi P, Thompson M, Heneghan C. Ensuring safe and effective drugs: who can do what it takes? *BMJ* 2011;342:c7258.

Cite this as: *BMJ* 2011;343:d6134

Should reduce prescribing pool

As a GP with some conscience about the cost to the taxpayer of my prescribing decisions, I found it heartening to read Sorenson and colleagues' article about the need to measure "comparative efficacy" for European drug approvals.[1]

In 1995 an article that questioned the need for nine angiotensin converting enzyme inhibitors concluded that three would have covered all indications.[2] Ten years later the *BMJ* published an article that found that 80% of the increase in drug expenditure in British Columbia between 1996 and 2003 was explained by the use of new patented drugs that offered no substantial improvements on less expensive alternatives available before 1990.[3]

If so many new drugs are not necessary and drug bills are spiralling, we have a moral duty to analyse how these drugs slip into the prescribing pool. A large part of the answer lies with our profession's relationship with the drug industry. In Ireland, we rely on the industry to sponsor virtually all education for prescribers and part fund many of our professional organisations. Sorenson and colleagues' proposal would go some way towards restricting the ever expanding prescribing pool.

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Competing interests: None declared.

1 Sorenson C, Naci H, Cylus J, Mossialos E. Evidence of comparative efficacy should have a formal role in European drug approvals. *BMJ* 2011;343:d4849. (6 September.)
2 Consumer Association. Who needs nine ACE inhibitors? *Drugs Ther Bull* 1995;33:1-3.
3 Morgan SG. "Breakthrough" drugs and growth in expenditure on prescription drugs in Canada. *BMJ* 2005;331:815-6.

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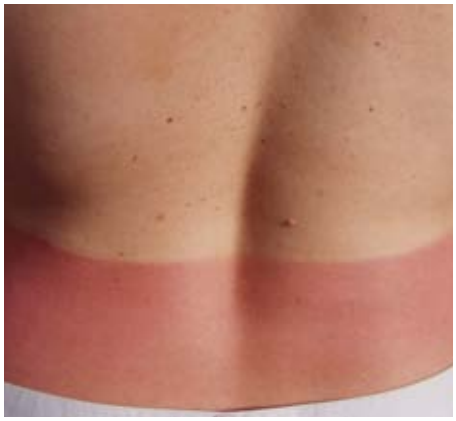
BAD MEDICINE: MELANOMA

Full information needed



Spence may be guilty of the same "whiff of propaganda" and contradictions that he alleges from others.[1]

UK melanoma figures are 54% for head, neck, and limbs (38% trunk) in men and 77% (17% trunk) in women. In the west of Scotland, 69% of melanomas occurred on the face, whereas only 55% did so in Queensland—the epicentre for melanoma.[2]

Melanoma is the most common and the second most common cancer in adults aged 25-29 years and 15-29 years, respectively.[3] The greatest increase is in young women, and this is possibly related to the use of sunbeds.

Incidence is directly related to rates of biopsy, and the allegation that dermatologists may well remove lesions that will never progress, but how can we tell? Many illnesses "may never progress," including syphilis, as shown in the infamous Tuskegee Study of Untreated Syphilis, where over 40 years "only" 100 of 400 participants died of syphilis or its complications.[4]

Evidence for the benefits of sun block is good. In 1992, 1621 Australian adults were enrolled in a clinical trial, and data on the occurrence of melanomas over 15 years clearly showed the protective effect of sunscreen.[5]

Vitamin D deficiency may not all be due to sunscreens. It was endemic in Glasgow before sunscreens were commercially available (in 1928).

Melanoma can be cured only by surgery and if diagnosed early enough (in situ), it is a 100% cure.

I think that early diagnosis of melanoma and increased awareness of the deleterious, as well as beneficial, effects of ultraviolet radiation will see mortality from melanoma gradually reduced.

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Competing interests: None declared.

1 Spence D. Bad medicine: melanoma. *BMJ* 2011;343:d5477. (31 August.)
2 Green AC, Kimlin M, Siskind V, Whiteman DC. Hypothesis: hair cover can protect against invasive melanoma on the head and neck (Australia). *Cancer Causes Control* 2006;17:1263-6.
3 Bleyer A, O'Leary M, Barr R, Ries LAG, eds. Cancer epidemiology in older adolescents and young adults 15 to 29 years of age, including SEER incidence and survival: 1975-2000. National Cancer Institute, 2006.
4 McLellan D. Ernest Hendon, 96; Tuskegee syphilis study's last survivor. *Los Angeles Times* 2004 Jan 25; http://articles.latimes.com/2004/jan/25/local/me-hendon25.
5 Green AC, Williams GM, Logan V, Strutton GM. Reduced melanoma after regular sunscreen use: randomized trial follow-up. *J Clin Oncol* 2011;29:257-63.

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The truth, such as it is

Spence's thought provoking article spurred me into a little investigation of my own.[1]

Data from the South West Cancer Information Service show that the age standardised rate for the incidence of malignant melanoma in England rose from six per 100 000 population in 1985 to 18 per 100 000 population in 2006; mortality similarly rose from two per 100 000 population to three per 100 000 population.[2] Both increases are statistically significant.

National Cancer Data Repository data show that over 95% of diagnoses in 2006-8 were confirmed by histology.[3] Because the death rate is rising and diagnoses are confirmed, it does not seem that melanomas are being overly diagnosed.

We would expect a fall in death rate (or numbers) if, as dermatologists suggest, diagnoses are being made earlier and are better; the proportion of people diagnosed with melanoma who die as a result of the disease is decreasing even though the incidence of diagnosis and mortality are increasing. This apparent anomaly can be explained by the fact that the rate of diagnosis increases more quickly than the mortality rate and the five year survival rate is increasing.

I cannot proffer a biological explanation for these data. We may be observing an occurrence of catastrophe theory—a branch of mathematics in which sudden shifts of behaviour (melanoma diagnosis) arise from small changes in circumstances (average age of population). I am

unaware of a biological mechanism in humans that would allow for such a phenomenon to exist.

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Competing interests: None declared.

1 Spence D. Bad medicine: melanoma. *BMJ* 2011;343:d5477. (31 August.)
2 South West Public Health Observatory. Malignant melanoma: incidence, mortality and survival rates in the South West region and England. 2009. www.swpho.nhs.uk/skincancerhub/resource/view.aspx?RID=46813
3 South West Public Health Observatory. Skin cancer data quality. 2010. www.ncin.org.uk/view.aspx?rid=627.

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Author's reply

I referenced my facts from national data sources, but like all "facts" they are debatable and often contradictory. The key issue, however, is whether the observed increased incidence of melanoma is real or an overdiagnosis artefact.

Overdiagnosis occurs when "cancers" that would never progress to overt disease are removed. This can reflect variation in histological practice. Doctors might be more likely to call "cancer" on samples in these more litigious times than run the risk of being wrong. Or we might simply be removing early cancers than are non-progressive. Overdiagnosis is likely when incidence rises rapidly but the outcome (like death) remains stable. Overdiagnosis is a major problem in breast and prostate cancer.

Since 1980 the incidence of melanoma has increased by nearly 300%,[1] most commonly early stage lesions, but the death rate from melanoma for people under 65 remains stable. There is no plausible biological explanation or model for such a rise except that it is not real but an overdiagnosis artefact. Make no mistake, being told that you have cancer when you don't causes major suffering and lasting harm.

Although the overall death rate from melanoma has increased, this could be because melanoma is a disease of elderly people and life expectancy during this period increased by eight years. Also vascular disease, the most common cause of death, has declined dramatically. People have to die of something. I do believe that a "catastrophic theory" is at work,[2] the catastrophic effect of overdiagnosis.

Finally, the prevention of melanoma cannot be used to justify the proposed draconian sun avoidance policy in the UK. We have no idea what this policy would do to vitamin D values and the impact on the long term health of our children.

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Competing interests: None declared.

1 Office for National Statistics. Cancer trends in England and Wales 1950-1999: An update. 31 March 2005.

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WILLETT

POLITICS OF NHS RECONFIGURATION

Closing hospitals with warm sympathy

Am I alone in being struck by a discrepancy in our current approach to organising healthcare?[1] I accept there may be a minimum size for hospitals to be effective. There is nothing new in this sentiment; Aneurin Bevan's said he "would rather be kept alive in the efficient if cold altruism of a large hospital than expire in a gush of warm sympathy in a small one."

Is there not an inconsistency in our wish to close local district hospitals? Bevan was contrasting the lower quality of medical facilities in community hospitals with those in district general hospitals. We are now planning to close some acute hospitals, with a concurrent drive to maintain ill people in the community when possible. Although local "intermediate care" set-ups can be excellent for rehabilitation after surgery or treatment for acute illness, pressure is increasing to use them to prevent patients ever reaching an acute hospital. In my experience, such schemes usually have inadequate facilities and staffing, have little regulatory consistency between one scheme and another, and scant attention is paid to clinical governance issues.

Shutting smaller hospitals will mean that fewer ill patients will gain access to acute hospitals, because beds lost from one axed hospital are never fully compensated elsewhere. The argument that smaller district hospitals lack the facilities to look after ill patients safely will apply even more to intermediate care facilities. We seem to be on a fast track to pre-NHS medicine—personally, if I succumb to illness, I wish to be admitted to even a small NHS hospital rather than run the gauntlet of obtaining a correct diagnosis and appropriate management in the community, with or without a gush of warm sympathy.

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1 Lister S. The politics of NHS reconfiguration. *BMJ* 2011;343:d5311. (7 September.)

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FREEDOM OF INFORMATION REQUESTS

Research on the cheap?

Christie recently reported that a tobacco company was using the Freedom of Information Act 2000 to compel Stirling University to give it access to detailed research results, despite the researchers' objections.[1] We have encountered a variation on this theme—namely, researchers using the act to compel hospitals to provide data to further their research. In recent years we have received three freedom of information requests in which the questions and the source implied that the purpose was scientific research and publication.

This development raises questions of ethics and research governance. Usually, providing information for someone else's research is voluntary. People can assess the value of the project and the time available. Contributors are usually thanked for giving information. Now the Freedom of Information Act allows researchers to force other clinicians to provide data, even if the project is scientifically worthless or the clinicians have other priorities.

Guidance on the act states that requests costing less than £450 must be complied with.[2] A request made to all 167 acute NHS Trusts in England might cost up to £75 150, equivalent to 75 working weeks. Thus, by invoking the Freedom of Information Act, a researcher can divert the equivalent of a consultant's yearly salary to support their project, bypassing the normal governance mechanisms for research funding.

National and international guidance states that participation in research must be voluntary.[3 4] This was written with research subjects in mind but should apply equally to work colleagues in the NHS. The General Medical Council, research ethics committees, and journal editors should consider the ethical and resource implications of compelling colleagues to participate in research, even if such compulsion is permitted in law.

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Competing interests: None declared.

1 Christie B. Tobacco company makes freedom of information request for university's research. *BMJ* 2011;343:d5655. (5 September.)
2 Information Commissioner's Office. What are the cost limits for a Freedom of Information Act request? www.ico.gov.uk/Global/faqs/freedom_of_information_act_for_the_public.aspx#fA48D7E50-37A4-44EB-8E70-FF3919B14A27.
3 General Medical Council. Good practice in research. www.gmc-uk.org/guidance/ethical_guidance/6001.asp.
4 World Medical Association. WMA Declaration of Helsinki. www.wma.net/en/30publications/10policies/b3/index.html.

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