Letters

Open letter to health ministers at Framework Convention on Tobacco Control

EDITOR—Tobacco exacts a devastating toll on health. To stem the epidemic, the World Health Organization proposes the first international treaty for public health—the WHO Framework Convention on Tobacco Control. The convention, to be adopted by the World Health Assembly on 21 May, offers a golden opportunity to save lives. But a handful of countries threaten to send the treaty up in smoke.

The convention commits governments to a set of policies to cut tobacco consumption. Developing countries look to the treaty to protect their citizens from the worst excesses of a powerful global industry.

Every day, doctors see the suffering and death caused by tobacco. More than 130 medical associations have joined forces to spell out their prescription for the convention in the doctors' manifesto for global tobacco control (www.doctorsmanifesto.org/). Doctors are satisfied that the proposed treaty offers a valuable blueprint for protecting health.

The consensus text to be presented to the World Health Assembly represents the outcome of three years of negotiation. But throughout the process a handful of rich countries—notably the United States, Japan, and Germany—have sought to water down the treaty. Even at this late stage, they still threaten to scupper it.

Tackling the global tobacco epidemic requires international solidarity. Doctors worldwide urge their governments to make one last effort to ensure an effective convention. At the World Health Assembly, health ministers must stand by the consensus text, adopt the treaty, and seize this historic opportunity. The lives of our patients—and their citizens—depend on it.

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Case fatality rates after hospital admission for stroke

Covariates were not measured

EDITOR—The article by Roberts et al on stroke mortality in hospital and at one year after stroke raises several issues.[1] The case-mix ratio, presence or absence of on-site neurosurgical services, and stroke unit care should be emphasised as important and easily measured explanatory covariates.

Unfortunately, the most important predictor of 30 day mortality is stroke severity at baseline, and this variable is not captured by using administrative data. Nevertheless, intracerebral haemorrhage is accurately coded in hospital discharge abstracts (ICD-9 code 431). Therefore, grouping the cases by diagnosis—intracerebral haemorrhage versus ischaemic stroke (ICD-9 (*International Classification of Diseases*, 9th revision) codes 433, 434, 436)—is valid for intracerebral haemorrhage.

Although intracerebral haemorrhage makes up only about 10% of all strokes, it has a higher acute mortality than ischaemic stroke. In Toronto the 30 day mortality at a neurosurgical centre for primary intracerebral haemorrhage was 27.4%.[2] Centres that receive patients with intracerebral haemorrhage may therefore have higher aggregate mortality rates and adjustment for case mix would be relevant.

Although it remains unclear whether acute neurosurgical intervention improves overall long term outcome in intracerebral haemorrhage, it may reduce early mortality.[3] At experienced centres, aggressive interventional care shows promise in improving both morbidity and mortality.[4] One surrogate for neurosurgical availability is teaching hospital status. Observational evidence that differences in mortality exist by hospital type, after adjustment for case mix, is worth exploring.

The only intervention that reduces mortality for ischaemic stroke (and probably also for intracerebral haemorrhage) is care in a stroke unit. The publication of randomised trial evidence for and development and implementation of stroke units have occurred over the time period of the current study. Evaluation of 30 day mortality by hospital, stratified by presence or absence of a stroke unit, could provide important observational evidence of the "real life" effectiveness of stroke units.

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Competing interests: None declared.

1 Roberts SE, Goldacre MJ. Case fatality rates after admission to hospital for stroke: linked database study. *BMJ* 2003;326: 193-4. (25 January.)
2 Hill MD, Silver FL, Tu JV, Austin P. Rate of recurrence of stroke after primary intracerebral hemorrhage. *Stroke* 2000;31:123-7.
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Definition of stroke defines stroke mortality

EDITOR—Stroke is a clinical syndrome characterised by rapidly developing clinical signs of focal (or global) disturbance lasting 24 hours or longer or leading to death with no apparent cause other than of vascular origin. The definition of stroke incorporates haemorrhagic and ischaemic lesions, with additional subtypes in both categories. The incidence and mortality vary widely across stroke subtypes, likely reflecting underlying differences in etiology and pathophysiology.

Estimates of stroke mortality that do not consider these inherent characteristics are misleading. Statistical analyses of stroke incidence and mortality are critically dependent on the definition of stroke.

Roberts et al report case fatality rates after hospital admission for "stroke" based on ICD-9 (*International Classification of Diseases*, 9th revision) codes.[1] This method of coding for stroke diagnosis is known to be inaccurate owing to lack of specificity, and analyses should be adjusted for published positive predictive values of each ICD-9 code. The authors exclude subarachnoid haemorrhage (ICD-9 430), yet they include other and unspecified intracranial haemorrhage (ICD-9 432). Ischaemic strokes (ICD-9 433, 434, 436) are then lumped with intracerebral haemorrhage (ICD-9 431).

This lumping leads to an exaggeration of ischaemic stroke mortality. Roberts et al report unadjusted case fatality for in-hospital stroke at 29.7% between 1988-97 in the former Oxford health region of Eng-

land.[1] In-hospital mortality for ischaemic stroke admissions in the United States during 1997 has been estimated at 5.3%, compared with 30.9% for intracerebral haemorrhage.[2] The mortality figures reported by Roberts et al are probably skewed by the increased in-hospital mortality associated with intracerebral haemorrhage. The in-hospital stroke case fatality rate in the database analysed by Roberts et al seems inflated owing to their selected definition of stroke. Determination of stroke mortality is meaningless, unless the definition of stroke is reasonably specific or representative of a particular clinical syndrome.

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1 Roberts SE, Goldacre MJ. Case fatality rates after admission to hospital with stroke: linked database study. *BMJ* 2003;326:193-4. (25 January.)
2 Liebeskind DS, Sayre JW. Decreasing incidence of stroke in the United States: 1996-2000 [abstract]. *Stroke* 2003;34:287.

Authors' reply

EDITOR—We agree with the points made about contrasting case fatality rates for intracerebral haemorrhagic and ischaemic stroke. In using administrative databases for comparisons between hospitals and over time, however, many strokes are not specified as haemorrhagic or ischaemic.

We analysed codes for stroke in accordance with the groupings used in NHS indicators of hospital performance in England and Scotland.[1 2] In our study 7.5% of strokes were recorded as haemorrhagic, 9.9% as ischaemic, and 82.6% as stroke that was not specified as either. The case fatality rates at 30 days after admission were 48% for haemorrhagic, 25% for ischaemic, and 35% for unspecified stroke. They varied considerably by age (web tables for our original paper and this letter).

Several factors relevant to outcome influence whether a stroke is investigated or recorded specifically as haemorrhagic or ischaemic. The percentage recorded as specific types in our database was higher in young than old people, changed over time, and varied between hospitals. As we reported, case fatality rates fell significantly over time and differed significantly between hospitals. Importantly, these patterns were found for each type of stroke—haemorrhagic, ischaemic, and unspecified stroke (web tables).

The extent of investigation is also probably related to case severity, and therefore to outcome, in ways that are difficult to measure. For example, investigation is more likely in patients whose prognosis may be improved by specific intervention; by contrast, death followed by postmortem examination may add to the likelihood that the stroke type is specified.

For all these reasons, analysis of stroke overall is more complete than restricting analysis to the minority of cases of specified type when comparing hospitals over time. We agree, however, that information about the individual types of stroke is very important when it is available and reliable.

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Competing interests: None declared.

1 Department of Health. NHS performance indicators: February 2002. Available at: www.doh.gov.uk/nhsperformanceindicators/2002 (accessed 1 May 2003).
2 Clinical Outcomes Working Group. *Clinical outcome indicators*. Edinburgh: Scottish Executive Health Department, 2002.

Three additional tables appear on bmj.com

Thyroid function tests and hypothyroidism

Reducing concentrations further would be harmful

EDITOR—In their editorial on thyroid function tests and hypothyroidism, Toft and Beckett discuss the target concentration of thyroid stimulating hormone concentration for patients receiving thyroxine replacement for primary hypothyroidism.[1]

They say that patients with continuing non-specific symptoms (despite a thyroid stimulating hormone concentration within the laboratory reference range) are "under-replaced."

They conclude that simply satisfying the recommendations of the American Thyroid Association and restoring thyroid stimulating hormone concentrations to normal is inadequate.

They say that some patients achieve a sense of wellbeing only if their thyroid stimulating hormone is low or undetectable and that most patients feel well only with a low normal thyroid stimulating hormone concentration.[1]

Primary care doctors in the United Kingdom provide sole continuing clinical care for most patients (82%) with primary hypothyroidism.[2] Before considering changing their clinical practice doctors should know that almost no empirical evidence exists to support the assertion that hypothyroid patients feel better with a thyroid stimulating hormone concentration that is "low normal," undetectable, or suppressed.[1] Two systematic reviews with meta-analysis have found that reducing concentrations of thyroid stimulating hormone with thyroxine treatment increases the risk of osteoporosis, particularly in postmenopausal women.[3 4] This is of considerable clinical importance as women over the age of 50 years constitute 84% of patients prescribed thyroxine replacement in primary care.[2] Suppression of thyroid stimulating hormone also increases the risk of atrial fibrillation.[5]

Many hypothyroid patients are currently over-replaced with thyroxine in the United Kingdom. The proportion of patients with suppressed concentrations of thyroid stimulating hormone is over 20% in primary care, while undetectable thyroid stimulating hormone concentrations (on ultra-sensitive assay) are 12%.[2] A change in routine clinical practice that further reduces concentrations of thyroid stimulating hormone in women receiving thyroxine replacement is likely to increase both osteoporosis and atrial fibrillation, without any good evidence that these women will feel any better.

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Competing interests: None declared.

1 Toft AD, Beckett GH. Thyroid function tests and hypothyroidism. *BMJ* 2003;326:295-6. (8 February.)
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5 Sawin CT, Geller A, Wolf PA, Belanger AJ, Baker E, Bacharach P, et al. Low serum thyrotropin concentrations as a risk factor for atrial fibrillation in older persons. *N Engl J Med* 1994;331:1249-52.

Restoring serum TSH to reference range should be goal of replacement

EDITOR—The article from Meier et al concludes that judicious initiation of thyroxine treatment in overt hypothyroidism should be guided by clinical and metabolic presentation and thyroid hormone concentrations and not by serum thyroid stimulating hormone concentrations.[1] This conclusion seems to contradict overwhelming evidence that serum thyroid stimulating hormone represents the most sensitive marker of primary hypothyroidism; furthermore, it is not supported by the findings of the study.

The cohort was a selected group of 49 patients with clinically significant hypothyroidism as evidenced by mean serum concentrations of thyroid stimulating hormone higher than 40 mU/l, and the study used a series of poorly specific and sensitive markers of thyroid status. Duration of hypothyroidism is an important factor determining the clinical symptoms and signs, which were not controlled for in this study.

Whether data from this study can be extrapolated to patients receiving thyroxine treatment is questionable. The accompanying editorial by Toft and Beckett says that some patients receiving thyroxine replacement continue to have symptoms despite restoration of normal concentrations of serum thyroid stimulating hormone and that exogenous subclinical hyperthyroidism is less harmful than endogenous subclinical hyperthyroidism. Few, if any, data support this view.

This statement conflicts with increasing evidence that excessive thyroxine replace-

ment results in adverse symptoms and quality of life scores, increased bone loss, and adverse cardiovascular outcomes.[2–5] It is our belief that restoration of serum concentrations of thyroid stimulating hormone to within the reference range should be the goal of replacement. If symptoms persist despite adequate replacement then an alternative explanation should be sought.

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1 Meier C, Trittibach P, Guglielmetti M, Staub JJ, Müller B. Serum thyroid stimulating hormone in assessment of severity of tissue hypothyroidism in patients with overt primary thyroid failure: cross sectional survey. *BMJ* 2003;326:311-2. (8 February.)
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Authors' reply

Editor—It is surely a sign of the increased pressures on doctors' time that they now tend to read only the titles or abstracts of manuscripts (designed to have maximum impact) rather than the body of the text. The two papers common to the criticisms of our position on thyroxine replacement treatment by both Crilly and Vanderpump and Franklyn illustrate the problem.

In the Framingham study a low serum thyroid stimulating hormone concentration was shown to be a risk factor for the development of atrial fibrillation,[1] but the patients in question were a heterogeneous group, the minority of whom were taking thyroxine. A similarly misleading conclusion has been drawn from the recent Birmingham study in which a low serum thyroid stimulating hormone (<0.5 mU/l) was associated with an increase in cardiovascular mortality.[2]

This finding has been used to argue that a suppressed serum concentration of thyroid stimulating hormone should be avoided in patients taking thyroxine replacement,[3] although thyroxine treatment was one of the exclusion criteria. The weakness of the meta-analysis, showing that thyroxine induced suppression of thyroid stimulating hormone led to reduced bone mineral density, was recognised by the authors themselves, who said that their design (cross sectional studies) was not appropriate because the many risk factors for bone loss do not allow correct matching of controls with cases.[4] This realistic assessment accords with the earlier views of Franklyn et al that thyroxine treatment alone does not represent a significant risk factor for loss of bone mineral density.[5]

We have long taken the view that most hypothyroid patients are content with a dose of thyroxine that restores serum concentrations of thyroid stimulating hormone to the low normal range. However, some achieve a sense of wellbeing only when serum thyroid stimulating hormone is suppressed, when we take care to ensure that serum triiodothyronine is unequivocally normal.

Until valid evidence shows that such a policy is detrimental we will continue to treat patients holistically rather than insist on adherence to a biochemical definition of adequacy of thyroxine replacement. The issue of whether a little too much thyroxine is dangerous is likely to evaporate when appropriate preparations become available to allow treatment of hypothyroidism with both tri-iodothyronine and thyroxine.

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1 Sawin CT, Geller A, Wolf PA, Belanger AJ, Baker E, Bacharach P, et al. Low serum thyrotropin concentrations as a risk factor for atrial fibrillation in older persons. *N Engl J Med* 1994;331:1249-52.
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Computerised evidence based guidelines in primary care

Interactive decision support is here and essential for 21st century primary care

Editor—I am worried that the paper by Rousseau et al on computerised evidence based guidelines in primary care may inhibit the development of primary care in the 21st century.[1]

My practice been using interactive decision support as an important facet of modern general practice for several years and received a "Doctor of the Year" award a couple of years ago, but we would not have been able to demonstrate the high quality general practice without the locally created doctor and nurse chronic and acute disease decision support software that we use.

Most chronic illness including hypertension, diabetes, and heart failure and acute care of illnesses such as back pain, asthma, and mental illness are managed successfully within the confines of routine general practice. Rousseau et al should make it quite clear what software has been used to allow primary care to distinguish this project from decision support software which is working well in every day practice. The analogy of ploughing a field may be useful. If a formula one racing car had been used to pull the plough, the readers would not be surprised if it were not up to the job. This could then have been reported as "internal combustion engine is less effective than the horse."

We must not tar all decision support with the same brush—we couldn't work successfully without locally written decision support in our practice.

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Competing interests: Bewdley Medical Centre has been a beacon practice for cardiovascular disease. Its practice manager is on the Torex user group.

1 Rousseau N, McColl E, Newton J, Grimshaw J, Eccles M. Practice based, longitudinal, qualitative interview study of computerised evidence based guidelines in primary care. *BMJ* 2003;326:314-8. (8 February.)

Computerised decision support and reflection in action

Editor—Eccles et al have shown not only the lack of effectiveness of a computerised decision support system based on evidence based guidelines but also some of the reasons why general practitioners found it unusable in everyday clinical practice.[1 2]

General practitioners value information on demand but need to be able to recognise the need for information and to possess the skills to obtain and interpret it at the point of use. These skills (usually taught as evidence based medicine)[3] are difficult and not widespread,[4] so it is understandable that efforts to implement the findings of research should have concentrated on implementing the recommendations of expertly produced evidence based guidelines in a linear, top-down process. The use of research evidence is thus simplified and codified as a checklist of tasks in response to predefined prompts.

The issue with this approach is not whether its computerised form is user friendly but whether it is a practical or desirable way of making clinical decisions about individuals. Experienced clinicians mostly make decisions in an apparently intuitive way—"reflection in action"—rather than by proceeding through a rational technical process such as that exemplified by computerised guidelines.[5]

In a setting such as general practice, where doctors are attempting to address patients' agendas in psychological and social as well as biological terms, the intrusion of a checklist (whether computerised or not) may disrupt the smooth flow of a consultation and lead neither to implementation of a guideline nor to tackling the patient's own concerns.

Information on demand is now available online through the National Electronic Library for Health to all general practitioners. All guidelines from the National Institute for Clinical Excellence, the

Cochrane Library, *Clinical Evidence*, and much else may be accessed while the patient is present. Clinicians need to acquire confidence in using information technology and evidence based medicine skills.

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Competing interests: TL is the co-organiser of an evidence based practice workshop.

1 Eccles M, McColl E, Steen N, Rousseau N, Grimshaw J, Parkin D, et al. Effect of computerised evidence based guidelines on management of asthma and angina in adults in primary care: cluster randomised controlled trial. *BMJ* 2002;325:941.
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5 Schon DA. From technical rationality to reflection-in-action. In: Dowie J, Elstein A, eds. *Professional judgement: a reader in clinical decision making.* Cambridge: Cambridge University Press, 1983:60-77.

Authors' reply

EDITOR—The enduring message from researching methods to promote the uptake of research findings is that no one strategy that will work for all individuals, settings and behaviours,[1] yet this is what both Prince and Lipman imply.

It is understandable that Prince promotes a locally developed computerised decision support system addressing local needs and priorities, particularly as he and his team seem highly motivated both to use it and to carry out the actions that it recommends. Unfortunately, it is unproved whether their solution will work for others' problems. There is a greater challenge in constructing and evaluating a computerised decision support system that will work across a range of computer systems, individuals, settings, and behaviours. We feel that our paper provides important messages for those developing future computerised decision support systems.

Although we support what Lipman says about on-demand information, we think that increasing information technology and evidence based medicine skills will not provide a "one size fits all" solution to promoting the uptake of research findings. Those in our study who liked on-demand information were more likely to mention finding information useful in digested formats such as evidence based guidelines. Even if trained in the skills of evidence based medicine few general practitioners will have time within their daily practice to conduct reviews of available evidence. Furthermore, such an approach does not address the challenge of alerting clinicians to new developments or gaps between their own or best practice.

As we are unlikely to find a magic bullet that works for all, it seems wise to continue to develop and evaluate a range of methods to promote the predictable uptake of research findings into routine care.[2]

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Competing interests: None declared.

1 Bero L, Grilli R, Grimshaw JM, Harvey E, Oxman AD, Thomson MA. Closing the gap between research and practice: an overview of systematic reviews of interventions to promote implementation of research findings by health care professionals. *BMJ* 1998;317:465-8.
2 Foy R, Eccles M, Grimshaw J. Why does primary care need more implementation research? *Fam Pract* 2001;18:353-5.

Transplantation has no rival

EDITOR—I read the editorial by Anyanwu and Treasure on heart transplantation with great interest.[1] I had just the same feeling about organ transplantation in 1959 when Roy Calne, Isobel Beswick, and I did the first organ (kidney) transplants, using effective chemotherapy to deal with rejection. Although organ transplantation was the great new hope at the time, I felt it was too cumbersome a weapon of treatment to last for very long.[2]

Curiously, nearly all the other operations I performed during my career at the Royal Free Hospital have been abandoned and become part of the history of medicine, while organ transplanation seems to have no immediate rival.

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Competing interests: None declared.

1 Anyanwu A, Treasure T. Prognosis after heart transplantation. *BMJ* 2003;326:509-10. (8 March.)
2 Calne R, Hopewell J, Beswick I. Three clinical cases of renal transplantation. *BMJ* 1964;1:411-3.

Findings for looked after children are not generalisable

EDITOR—Hill et al's paper on meningococcal C immunisation in looked after children showed that looked after children were more than twice as likely not to receive meningococcal vaccine as were children at home.[1] They say that their findings are likely to be generalisable. This is not the case.

As part of the children's services planning process in the Southern Health and Social Services Board in Northern Ireland, we tagged the records on the child health system of looked after children in one of our health and social services trusts (Craigavon and Banbridge Health and Social Services Trust) in October 2001. The table shows the findings.

These findings do not support Hill et al's findings. In Northern Ireland, health and social services have been integrated since the 1970s, which may partly explain the differences. Out of area placements are also less common in Northern Ireland compared with other parts of the United Kingdom.

The child health system can provide up to date "snapshot" pictures of the health of looked after children. In the Southern Health and Social Services Board we are introducing protocols to ensure information from the child health system is available to the looked after team shortly after the child becomes "looked after." Integrated health and social services can be an effective means of protecting the health of vulnerable children in society.

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Competing interests: None declared.

1 Hill CM, Mather M, Goddard J. Cross sectional survey of meningococcal C immunisation in children looked after by local authorities and those living at home. *BMJ* 2003;326: 364-365. (15 February.)

Child health system records of looked after and other children, Craigavon Banbridge Health and Social Services Trust, October 2001. Values are numbers (percentages) of children born 1 January 1987 to 31 December 1999

Record	Looked after children (n=75)	Other children (n=23 936)
Breast fed	0 (0)	4869 (20.3)
Mothers who smoked	21 (28)	4011 (16.8)
Completed immunisations:		
Primary	67 (89.3)	22979 (96)
Meningococcal C	68 (90.6)	22260 (93)
Measles, mumps, and rubella	69 (92)	22979 (96)
Statement of special educational needs	20 (26.6)	230 (0.96)

Obituary for David Horrobin

Medicine has lost something unique and irreplaceable

EDITOR—I submit a shortened version of an obituary I wrote for David Horrobin for the *BMJ*.[1]

David Frederick Horrobin, director of Laxdale (b Bolton, England, 1939, q Oxford 1968; MA DPhil BM BCh), died from mantle cell lymphoma on 1 April 2003.

David Horrobin was one of the most productive clinical scientists of his generation, a highly successful businessman, and an inspirational figure in the world of medicine.

As a brilliant Oxford student, David achieved a scholarship to Balliol, the top physiology degree, and a prize fellowship

before an unconventional career move to a chair in physiology at Nairobi Medical School, Kenya. He later became professor of medicine at the University of Montreal.

To establish independent funding for his research on lipid biochemistry and nutrition, David set up the pioneer biotechnology company Efamol in 1979 (later re-named Scotia), which grew to be worth more than £400m (and made David wealthy enough to feature in the "rich list" of the *Sunday Times*). He founded Laxdale in 1997 to concentrate on developing psychiatric treatments.

David pioneered the therapeutic use of glucocorticoids in pregnancy, was the first to document the stifling effects of peer review, and originated the thesis that clinical research has declined in its ability to generate major breakthroughs. He believed that medical science had become indifferent to the need for new and more effective treatments and was a practical democrat who worked with patient groups, supported innovative scientists, and founded the journal *Medical Hypotheses* as a forum for radical ideas.

If David's intellect was remarkable, so was an energising personality which motivated people to positive action. His life had an epic quality, which sometimes made him seem like the last representative of a freer and more creative era. With his premature death, medicine has lost something unique and irreplaceable.

He leaves a wife and business partner, Sherri; two children; and three grandchildren.

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Competing interests: None declared.

1 Richmond C. David Horrobin. *BMJ* 2003;326:885. (19 April.)

Work inspired and continues to nurture positive clinical outcomes

EDITOR—In April 2003 the scientific community lost one of its greatest treasures, David Horrobin.[1] Born in England on 6 October 1939 he was a scholar of Oxford, where he obtained a first class honours medical degree. To this he added a clinical medical degree and a doctorate in neuroscience.

David began his research on essential fatty acids in 1972 at the University of Newcastle Medical School and continued his work at the University of Montreal. He set up a pharmaceutical company, Efamol, in 1979, which later became a public company, Scotia Pharmaceuticals. His innovative approach to research led to the discovery of many lipid products for the treatment of disease. Laxdale was set up in 1997 for the development of novel lipid pharmaceuticals for psychiatric and neurodegenerative disorders.

David was the founder and editor of two journals, *Medical Hypothesis* and *Prostaglandins, Leukotrienes and Essential Fatty Acids*. He was a prolific writer who wrote and edited numerous books and over 800 scientific publications.

His research opened a doorway into the profound influence of lipids towards health, sparking a minor revolution of lipid research around the world. Today, research into essential fatty acids in the US has evolved to gold standard analysis at Johns Hopkins. David's work inspired and continues to nurture positive clinical outcomes in our patients by respecting the critical balance of targeted application of essential fatty acids and its impact upon the cell membrane, the brain, and the human body.

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Competing interests: None declared.

1 Richmond C. David Horrobin. *BMJ* 2003;326:885. (19 April.)

Original mind will be missed

EDITOR—On behalf of the staff at Laxdale I submit a more recognisable view of David Horrobin than was evident in the obituary by Richmond.[1]

David Horrobin was one of the most original scientific minds of his generation, applying his knowledge and understanding of human physiology and disease to investigate lipids as therapeutics in medicine.

Born in 1939 in Bolton, England, David obtained a first class honours medical degree from Balliol College, Oxford, before adding a clinical medical degree and a doctorate in neuroscience and becoming a fellow of Magdalen College. Work with the Flying Doctor Service led to an interest in Kenya and an appointment as professor of medical physiology at its new medical school. David's travels culminated in his publication *A Guide to Kenya and Northern Tanzania* and later to the book *The Madness of Adam and Eve*. This was shortlisted for the 2002 Aventis science book of the year. Subsequent positions were held at the Universities of Newcastle and Montreal.

In 1979 David set up a small pharmaceutical company, Efamol. This became Scotia Holdings, which by his departure in 1997 had 450 employees and a market capitalisation of over £400m. David then started a new company, Laxdale, developing novel pharmaceuticals for psychiatric and neurodegenerative disorders. Laxdale's work will continue.

David was the founder and editor of the journals *Medical Hypotheses* and *Prostaglandins, Leukotrienes, and Essential Fatty Acids*. He was a prolific writer himself and wrote and edited numerous books on a wide range of subjects, although his main interest was always schizophrenia. He inspired people with his unique combination of enthusiasm and tenacity, humility and friendliness, open-minded creativity, huge breadth, and depth of knowledge and outstanding analytical power.

Two years ago David fell ill with mantle cell lymphoma, and he recently wrote a moving account of his illness in the *Lancet*. David's final resting place is on the Isle of Harris. He will be greatly missed.

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Competing interests: SMcG is finance director of Laxdale and a friend of David Horrobin.

1 Richmond C. David Horrobin. *BMJ* 2003;326:885. (19 April.)

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Summary of rapid responses

The response to David Horrobin's obituary is unprecedented for an obituary and among the biggest for any article.[1] At the time of going to press (8 May) we had posted 108 responses, 59 of them within a week of publication in the paper journal. We posted responses on every day but two from the 18 April, when the obituary became available on bmj.com. By comparison, legalising euthanasia and physician assisted suicide received 110 responses, and defining a good doctor 102.[2 3]

"Star" obituaries have caused controversy before.[4] We published a correction to that of Dame Sheila Sherlock in the light of its seven responses.[5] In this issue we publish a prominent correction (p 1091) to David Horrobin's obituary, as well as the three shortened obituaries (above), produced at short notice for the paper journal from longer versions on bmj.com.[1]

The response on bmj.com was heated—largely outrage and dismay—catalysing editorial debate on the role of medical obituaries and the ultimate tribute of a negative obituary. Respondents debated opinion dressed up as fact, defamation, and the competing interests of obituary writers and demanded a correction and an apology. As these seemed not to be forthcoming by 30 April, one respondent called for a complaint to be filed with the Press Complaints Commission. A week later another respondent reported that this had happened.

Who responded to David Horrobin's obituary? Family and friends wrote eloquently, strongly, and evocatively about the man and his work, as did work colleagues and fellow scientists and acquaintances. Some respondents attracted to the debate commented on the deeper issues evoked and the editorial ramifications, speaking both for and against the *BMJ* and its editor, but mostly against.

The controversy seems far from over. David Horrobin was obviously a big man, which was exemplified for me (who did not know him) in the concluding words of a former student and now full professor: "I was no longer in [David's] shadow but richer for having been."

Sharon Davies *letters editor, BMJ*

Competing interests: None declared.

1 Electronic responses. David Horrobin. bmj.com 2003. bmj.com/cgi/eletters/326/7394/885 (accessed 8 May).
2 Correspondence. Legalising active euthanasia and physician assisted suicide. *BMJ* 2002;324:846-8.
3 Correspondence. What's a good doctor and how do you make one? *BMJ* 2002;325:711-5.
4 Richmond C. Dame Sheila Sherlock. *BMJ* 2002;324:174 (responses available at bmj.com/cgi/eletters/324/7330/174).
5 Correction: Dame Sheila Sherlock. *BMJ* 2002; 324: 366 (responses available at bmj.com/cgi/eletters/324/7333/366/i).