Letters

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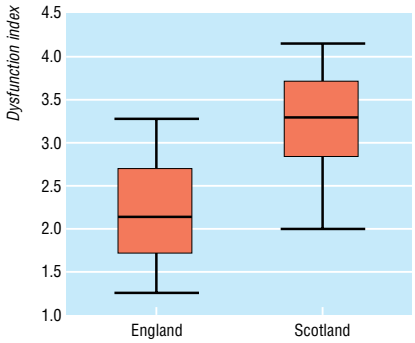
Schools' experience of league tables should make doctors think again

EDITOR—The BMA discussion paper *Clinical Indicators (League Tables)* touches on the issue of how performance indicator systems can have dysfunctional behavioural and managerial implications.[1] Winston shows how this applies to the Human Fertilisation and Embryology Authority's league tables of in vitro fertilisation clinics.[2] Our research looks at the dysfunctional effects of league tables in education and may be relevant to some of the issues surrounding league tables in the health sector.

We have recently reported a comparison between two systems, one with league tables (English primary schools) and one without (Scottish primary schools). In most other respects the two systems are similar. The research entailed a questionnaire survey of heads and teachers from 54 randomly selected primary schools in England and Scotland during 1999.

Some of the key findings were:

- English schools were more likely to report concentrating on meeting their targets, at the expense of other important objectives



Boxplots showing mean (SD) degree of dysfunction for each country (the lower the number the greater the dysfunction)

- The target setting and testing process apparently had a narrowing effect on the curriculum in England
- English schools were more like to say that they had concentrated resources on "borderline children"—those close to reaching the threshold—who would improve their league table position
- English schools particularly thought that the target setting and testing process had increased the "blame culture"

These findings were not entirely surprising and had been predicted (for example, by Smith[3]), but what was surprising was the substantial difference between the two countries. Seven questions were combined to form a dysfunction index. On a scale of 1 (maximum dysfunction) to 5 (no dysfunction), English primary schools scored 2.17 and Scottish 3.19 ($P < 0.001$; effect size 1.68). The figure shows the results.

As well as the differences, we found several important (though not significant) similarities:

- Both groups wanted access to performance data for their internal use—that is, they were not against performance indicators as such
- Schools in both countries seemed to be under similar pressure to meet targets. This suggests that having league tables does not necessarily apply greater pressure than other less public techniques
- Parents in England were no more likely to make reference to school test results than their Scottish counterparts. This questions one of the fundamental justifications of league tables: giving consumers information that they can use to put pressure on "their" schools

Although there were difficulties in allowing for other factors that may have an influence on the responses to the questionnaire, the data should give pause for thought. Careful consideration should be given to the unintended consequences of league tables.

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This letter is based on a paper presented to the European educational research conference, Edinburgh, September 2000, available on line: www.leeds.ac.uk/educol/documents/00001555.doc

1 British Medical Association. *Clinical indicators (league tables)—a discussion paper*. London: BMA, 2000.
2 Winston R. League tables of in vitro fertilisation clinics misinform patients. *BMJ* 1998;317:1593.
3 Smith P. Outcome-related performance indicators and organizational control in the public sector. *Br J Management* 1993;4:135-52.

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Male circumcision and HIV prevention

Some science would not have gone amiss

EDITOR—While a number of studies suggest an association between the foreskin and HIV infection, a simple tallying of studies, such as performed by Szabo and Short,[1] is unscientific and misleading. Meta-analysis suggests that men engaging in high risk behaviours may be placed at further risk by having a foreskin, but in the general population circumcision status is not a significant factor. It also showed an important degree of heterogeneity between studies, calling into question the validity of the summary results.[2] The multiple confounding factors influencing sexual behaviour and HIV susceptibility make it irresponsible to place blame on normal anatomy.

Langerhans' cells in the preputial mucosa are nothing new: all mucosal tissues have Langerhans' cells. Szabo and Short did not report Langerhans' cell concentrations in comparison with other mucosal tissues, their concentration in the glans, foreskin remnant, and circumcision scar in circumcised men, the presence of associated T cell infiltration (which may be necessary for viral transmission), or how findings in elderly cadavers correlate to sexually active young men. Szabo and Short state that the inner surface of the foreskin and the frenulum must be regarded as the most probable sites for viral entry of primary HIV infections in

men; but without quantitative comparative data their statements are pure speculation.

The only reports of preputial Langerhans' cells have been in specimens from neonates[3] and elderly cadavers. If normal genital mucosa is at risk, we need to know the concentration of Langerhans' cells in healthy men, men with multiple sexual partners, men with genital infections, men with HIV, and men of differing races and ages before any recommendations can be made.

Szabo and Short dismiss the complications of circumcision as having a low incidence; but the rate of immediate complications in the United States is between 3.1% and 9%,[4] and another 5% will later develop meatal stenosis.[5] A higher rate of complications is believed to follow circumcisions performed in the developing world, where circumcision has been linked to tuberculosis, tetanus, penile amputation, and death.

HIV transmission is heavily dependent on certain sexual behaviours, not anatomy. The authors have not provided any new information to alter this fact but have taken the discussion off on a needless tangent. Indiscriminate mass circumcision, which is currently popularised by some Western researchers, is unproved and does not address the core behavioural issues that have fuelled this pandemic. Therefore, it will not alter the course of AIDS in Africa.

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1 Szabo R. Short RV. How does male circumcision protect against HIV infection? *BMJ* 2000,320:1592-4. (10 June.)
2 O'Farrell N, Egger M. Circumcision in men and the prevention of HIV infection: a "meta-analysis" revisited. *Int J STD AIDS* 2000;11:137-42.
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Nature has not made a design error

Editor—Szabo and Short's article on male circumcision and HIV places them in splendid solidarity with Victorian notions of sex and hygiene, together with tribal initiation rituals.[1 2] They are completely isolated from their major peer medical organisations, not one of which endorses routine infant circumcision as a prophylactic measure despite over 100 years of pressure brought to bear by circumcisers.

To accept that circumcision is a really good idea, we first have to believe that nature made some huge design error in human anatomy that requires removal by force. This is a great leap of faith given the fact that not just humans but all mammals, both male and female, have evolved over millions of years to end up with a prepuce. But for some reason known only to religious types and medicalised capitalism the only mammal to be benefited by summarily removing this omnipresent organ through surgery is the human male.

The history of medicalised circumcision is a fascinating study in Victorian medicine and anti-sexuality.[4] Amputating the normal prepuce of human beings started in the English speaking countries as a measure to prevent masturbation. It did not work, but circumcisers have learnt that the pretexts for penile pruning are inexhaustible. Simply by playing on the fears of the culture they can keep the practice going—and the income flowing. At the turn of the 20th century better hygiene was the big issue, followed by penile cancer in the 1930s,[3] cervical cancer in the '50s, sexually transmitted diseases in the '60s, urinary tract infections in the '80s,[4] and, perhaps the most dreaded of all, AIDS in the '90s. If it looks as if routine infant circumcision is an operation in search of a disease, that's because it is. Every single claim for legitimate medical benefit justifying this routine has been discredited.[5]

But still the amputations go on. Every 26 seconds another penis is reduced in the United States. This is in sharp contrast with the rest of the world, where over 80% of the male population are left whole and intact—including all of Europe, most of non-Muslim Asia and Latin America—their genitals as nature designed them before the collective wisdom of Szabo and Short and other pro-circumcision proponents had a "better" idea.

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Competing interests: The International Coalition for Genital Integrity is an alliance of 18 organisations dedicated to protecting the normal anatomy of males and females. Its members include healthcare professionals, psychologists, researchers, lawyers, journalists, ethicists, academicians, and citizen activists dedicated to ending unnecessary genital cuttings.

1 Szabo R. Short RV. How does male circumcision protect against HIV infection? *BMJ* 2000,320:1592-4. (10 June.)
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No case was made for circumcising unconsenting children

Editor—Szabo and Short have concluded that circumcision of male children should be seriously considered as an additional means of preventing HIV.[1] Whether they have a valid argument for the circumcision of consenting adults, they have certainly not made a case for circumcising unconsenting children who are not sexually active. Furthermore, there are certain failings of the article that should not have escaped the attention of the peer reviewer.

Szabo and Short conducted a Medline search for relevant literature, but they present no full listing of the search results. An objective review of the literature would have shown that there was no consensus that male circumcision protects against HIV.[2] One meta-analysis showed circumcised men to be more at risk of HIV than those with the normal, intact penis.[3]

No evidence is presented by Szabo and Short to confirm their claim that HIV enters the body through CD4 and CCR5 receptors on Langerhans' cells located in the penis. As such their proposed mechanism for prevention of HIV by male circumcision is little more than supposition.

It is unacceptable for Szabo and Short to claim that circumcision has a low incidence of complications on the basis of a booklet favouring circumcision that has had no peer review.[4] Although a complication rate as low as 0.06% has been claimed for circumcision, rates as high as 55% have also been reported.[5] A detailed literature review of the complication rate for circumcision concluded that a realistic rate of significant complications is 2-10%.[6] It seems possible that any programme of child circumcision would cause more serious complications than it would prevent cases of HIV.

We believe that we live in an enlightened age. What is most surprising is that we still believe that we should ward off disease by cutting children's genitals. Publishing the opinion of Szabo and Short will do more to perpetuate non-therapeutic circumcisions of unconsenting children in North America and Australia than it will for the prevention of HIV in Africa.

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Competing interests: Dr Dalton has no competing financial interests, but he is a trustee of NORM-UK, a registered charity whose objects relate to the subject matter of this letter.

1 Szabo R. Short RV. How does male circumcision protect against HIV infection? *BMJ* 2000,320:1592-4. (10 June.)
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More studies need to be done before widespread circumcision is implemented

Editor—Szabo and Short suggest that the increased number of Langerhans' cells on the surface of the foreskin explains why circumcised men are less likely to become infected with HIV.[1] They did not mention an issue that has long dogged debate on the protective effect of circumcision on the incidence of cervical cancer and now increasingly prostate cancer[1 2]—that is, the extent to which improved hygiene and affluence are confounding variables to the benefits of circumcision. This is exemplified by the lower incidence of cervical cancer in

Frequency of HIV infection by circumcision and retractability of foreskin

	No of cases	% who were HIV positive
Circumcised		
All	21	29
Pre-puberty	9	22
Post-puberty	12	33
Non-circumcised		
All	62	55
All circumcised *v* all non-circumcised	χ^2=4.33, P=0.037	
Retractability of foreskin		
Exposed glans, easy retraction	31	61
Long foreskin, difficult retraction	31	48
Short *v* long foreskin	χ^2=1.04, P=0.308	

educated high caste women in India whose husbands were not circumcised than in less educated Muslim women with circumcised husbands.[3] Undoubtedly the increased numbers of Langerhans' cells with HIV receptors in the foreskin may well contribute to an increased susceptibility to HIV.

Evidence that nutritional state and other sexually transmitted diseases also play a part in acquiring HIV infection prompted us to examine the role of the foreskin in the occurrence of HIV infection in a series of 83 new patients (40 positive for HIV) attending a urethritis clinic at East and West Drakefontein Gold Mines Carltonville, Gauteng, South Africa, as part of a study of the impact of HIV and sexually transmitted diseases on serum concentrations of prostate specific antigen.[3] After giving their signed informed consent the miners received a questionnaire and were examined to ascertain whether they were circumcised (including whether the glans penis was visible) and the retractability of the foreskin. In addition, a limited history of sexual activity was recorded.

As expected, the frequency of HIV infection was significantly lower in those who were circumcised (table). The small subgroup who had been circumcised after puberty seemed to show some benefit in reducing the incidence of HIV infection. Even more interesting in the light of Szabo and Short's hypothesis about the increased numbers of Langerhans' cells in the foreskin, we found, contrary to what might be expected if their hypothesis was correct, that the frequency of HIV infection was less in men with long foreskins that were difficult to retract than in those with short easily retractable short foreskins.

Clearly this observation is based on too small a sample size for us to be totally confident in the results. However, these observations, added to those on the role of hygiene versus circumcision in reducing cervix cancer from India,[4] suggest that further studies would help to clarify Szabo and Short's hypothesis and need to be done before widespread use of circumcision is implemented to try to reduce the spread of HIV infection. Furthermore, work needs to be done on the influence of circumcision after puberty because performing such a procedure after the first infection of a sexually transmitted disease could be more effective than circumcision based on the total population.

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1 Szabo R. Short RV. How does male circumcision protect against HIV infection? *BMJ* 2000,320:1592-4. (10 June.)
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4 Gajalakshmi C, Shanta V. Association between cervical and penile cancers in Madras, India. *Acta Oncologica* 1993;32:617-20.

Authors' reply

Editor—Our review article was primarily concerned with exploring the mechanisms by which male circumcision protects against heterosexually acquired HIV infection in men. We concluded that it is the inner aspect of the foreskin, which is poorly keratinised but well supplied with Langerhans' cells, that is likely to be one of the principal sites of HIV entry into the penis.

We have now developed an active research programme on this topic, and, together with our collaborators, we hope to publish a number of papers in the near future on the distribution of Langerhans cells in the foreskins of young men with and without balanitis, the degree of keratinisation of the various penile epithelia, and the uptake of live HIV virus applied to the inner and outer aspects of adult human foreskins in vitro. Some of our histological findings were shown on the BBC *Horizon/Discovery* television programme "The Valley of Life or Death" on 16 November.[1]

The claim by Van Howe et al that a meta-analysis of the many papers that show a significant correlation between lack of male circumcision and HIV infection is unscientific and misleading makes little sense, since most of the 40 studies that show such an association have incorporated multivariate analysis to correct for confounding variables such as different sexual practices. Furthermore, Van Howe's own meta-analysis has been invalidated because of several major methodological errors.[2 3]

Male circumcision, like all minor surgical procedures, carries a small risk of postoperative complications. But this should not detract from the twofold to eightfold protective effect that circumcision provides against HIV infection, which, unlike the surgical complications, is almost invariably fatal. Other than recommending that male circumcision should be seriously considered as an additional means of preventing HIV in all countries with a high prevalence of infection, we have avoided all discussion about the relative advantages and disadvantages of neonatal male circumcision as a routine procedure in developed countries, where the prevalence of HIV infection is low. We do not intend to enter that debate, where objectivity is hard to find.

It is pleasing to note that organisations are now beginning to give serious consideration to the policy implications arising from the protective effect of male circumcision against HIV infection. In June 2000 the Horizons Project of the Population Council published a report of an international discussion meeting entitled "Male Circumcision and HIV Prevention: Directions for Future Research,"[4] and in July the World Health Organization held a similar consultation in Durban at the time of the international AIDS conference, although its findings have yet to be published.

It would be unfortunate if the zealous opponents of neonatal male circumcision in developed countries, however well meaning, distracted attention from the glaring fact that in central and southern Africa, where 24.5 million people are infected with HIV,[5] circumcision could offer some immediate protection against spread of the disease until such time as effective vaccines become available.

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1 Jackson T. No news is bad news. *BMJ* 2000;321:1419. (2 December.)
2 Van Howe RS. Circumcision and HIV infection: review of the literature and meta-analysis. *Int J STD AIDS* 1999;10: 8-16.
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Summary of rapid responses

In all, 41 correspondents or groups contributed 50 responses to this education and debate article. Of the 28 correspondents who gave their address, 10 were from Canada, eight from the United Kingdom, four from the United States, four from Australia, one from new Zealand, and one from India. Broadly speaking, most of the rapid responses posted were against male circumcision and the hypothesis that it protects against HIV infection, with the remainder calling for more research on the subject.[1]

1 Electronic responses. How does male circumcision protect against HIV infection? bmj.com 2000;320 www.bmj.com/cgi/content/full/320/7249/1592#responses; accessed 29 Nov 2000.

Midwife led debriefing to reduce maternal depression

Effectiveness of individual midwives is unclear

EDITOR—I should like to comment on the randomised controlled trial of midwife led debriefing to reduce maternal depression by Small et al.[1] The first randomised clinical trial was of a drug treatment, streptomycin for pulmonary tuberculosis,[2] which has provided the model for clinical trials ever since. In a drug trial we are not usually concerned with who is giving the drug because the effect of the drug itself is being measured. The treatment is impersonal, and we should be justified in assuming that the effect of a drug given by one person will be the same as it would be given by another.

When we carry out trials of more personal treatments, however, as in the trial by Small et al, we should be aware that the treatment given by one operator might not be the same as that given by another. Surgeons are not all equally skilful, for example. So if we were to carry out a trial comparing two surgical techniques the surgeons would be a non-random sample from the wider population of surgeons. This might not be too bad if each surgeon carried out both techniques because there would be some sort of balance. If different groups of surgeons carried out each technique this would not be so. We could randomise surgeons to treatments, but this would probably be difficult to achieve. We could, and I think should, take surgeon variation into account—for example, by multilevel modelling.[3] The inevitable result would be to make confidence intervals wider and P values bigger, as happens when cluster randomised trials are analysed correctly.[4]

In the trial by Small et al the situation is even more complicated. The intervention is debriefing by a midwife—a very personal intervention. It is easy to believe that the individual skills of midwives in this complex task vary greatly. Clearly, the mothers in this trial are a sample from which we want to draw some conclusions about mothers in general. But surely the midwives are a sample too. We are asking whether debriefing by midwives is helpful. The two midwives here have somehow to represent the effectiveness of midwives everywhere. It may be that these particular midwives are not very good at debriefing rather than that debriefing is ineffective. Half of us are below average, after all.

It is difficult to see how we could analyse the trial to take the midwife variation into account as such variation exists only in one arm. Although stratification by midwife is mentioned, the midwife's intervention is received by women in only one arm of the trial. Stratification therefore cannot allow for variation between midwives.

I cannot criticise researchers for not applying a statistical technique yet to be invented, or at least to be noticed by myself. Medical research is in its infancy. There are many unanswered questions and, I suspect, many that are yet to be asked. We do not really know how to do it yet. It will be an interesting challenge to find out.

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1 Small R, Lumley J, Donohue L, Potter A, Waldenström U. Randomised controlled trial of midwife led debriefing to reduce maternal depression after operative childbirth. *BMJ* 2000;321:1043-7. (28 October.)
2 Medical Research Council. Streptomycin treatment of pulmonary tuberculosis. *BMJ* 1948;ii:769-82.
3 Goldstein H. *Multilevel statistical models*. 2nd ed. London: Arnold, 1995.
4 Kerry SM, Bland JM. Analysis of a trial randomised in clusters. *BMJ* 1998;316:54.

Authors' reply

EDITOR—Bland's point about practitioner variables in implementing interventions that are not drugs is important. Our approach to implementing the trial of debriefing after operative birth was to identify the key elements, summarised by Wessely et al as identifying emotional responses, encouraging their expression, and legitimising them.[1] We also sought to define the necessary skills—active listening; reflection; encouraging the expression of women's experiences; accepting distress, anger, and pain; being able to name and normalise the experience; and being able to avoid offering solutions. We then selected two midwives who had these skills to a high degree so that our trial of debriefing would give the intervention the best possible chance of showing whether it was effective in reducing depression. During the run-in period all debriefing sessions were taped (with the women's written consent) to assess the quality of the intervention against the key elements. We saw the trial as a phase III trial, in the language of the recent Medical Research Council paper,[2] and had foreshadowed in the grant application subsequent work to develop a manual and training programme for midwives if the trial were effective, to be followed by well-designed cluster randomised dissemination trials (phase IV).

Bland is misleading when he writes, "We are asking whether debriefing by midwives is helpful." We were indeed asking that, and women responded overwhelmingly that it had been helpful or very helpful. This is one of the principal intentions of debriefing, to reduce the immediate psychological distress after a traumatic experience, so women's responses were reassuring. The trial was not designed to answer the helpfulness question but to see whether debriefing could prevent the subsequent development of depression. It did not—despite the more than average skills of the two midwives.

Bland's critique (the practitioners weren't up to the task) has often been used to explain away trial findings in perinatal work (antenatal cardiotocography, routine antenatal ultrasonography) in which interventions in widespread use have performed poorly within trials. One contribution would be for journals to require (and publish) enough detail about the implementation of complex interventions for readers to make informed judgments. As for practitioner and institutional variation, there seems to be no alternative but a wider use of cluster randomisation, despite the difficulties and challenges.

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1 Wessely S, Rose S, Bisson J. A systematic review of brief psychological interventions ("debriefing") for the treatment of immediate trauma related symptoms and the prevention of post traumatic stress disorder. Cochrane Review. In: *Cochrane Library*. Issue 3. Oxford: Update Software, 2000.
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Survival and reduction in mortality from breast cancer

Impact of mammographic screening is not clear

EDITOR—We should all rejoice that there has been an improvement in survival and reduction in mortality for carcinoma of the breast, but Richards et al in their paper perpetuate the myth that this is related to the breast screening programme.[1] The periods for comparison were 1981-5 and 1986-90.

The Forrest report on mammographic screening was published in 1986,[2] the first screening centres were established in 1988, and the country was not covered by the programme until 1990. Even the greatest zealots for mammographic screening would not expect an impact on mortality until 1997. The fall in mortality could therefore be attributed only to improvements in treatment, and it is relevant to note that the first overview of the trials of adjuvant systemic treatment were published in 1985.[3] The only support for the assertion that the reduction in mortality can be attributed to the breast screening programme was a personal communication from S M Moss. Many people are of the opinion that mammographic screening is saving thousands of lives, but opinion alone does not provide sufficient data to support a publication in a prestigious journal such as the *BMJ*.

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1 Richards MA, Stockton D, Babb P, Coleman MP. How many deaths have been avoided through improvements in cancer survival? *BMJ* 2000;320:895-8. (1 April.)
2 Forrest P. *Breast cancer screening: Report to the Health Ministers of England, Wales, Scotland and Northern Ireland*. London: HMSO, 1986.
3 Early Breast Cancer Trialists Collaborative Group. Effects of adjuvant tamoxifen and of cytotoxics on mortality in early breast cancer: an overview of 61 randomised trials amongst 28,896 women. *N Engl J Med* 1988:319:1681-92.

Diagnostic practice in the United States is different

EDITOR—Richards et al seem to have made inferences about "deaths avoided" using data on five year survival.[1] This measure is, however, powerfully affected by diagnostic practice and is not a reliable indicator of mortality.[2]

In the United States the problem is best exemplified by prostate cancer. Five year survival has increased from about 40% in the 1950s to about 95% currently.[3] Although it is tempting to conclude that we Americans have made major medical advances (and left the United Kingdom in the dust), the truth is that this largely reflects our diagnostic practice. As we aggressively seek and find early stage (and often innocuous) tumours, the incidence of prostate cancer in the United States has risen almost threefold over the same period.[3]

The method used by Richards et al might lead one to conclude that the deaths of tens of thousands American men have been avoided through improvements in early diagnosis and treatment. The fact of the matter is that prostate cancer mortality in the United States is now slightly higher than it was in 1950.[3]

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1 Richards MA, Stockton D, Babb P, Coleman MP. How many deaths have been avoided through improvements in cancer survival? *BMJ* 2000;320:895-8. (1 April.)
2 Welch HG, Schwartz LM, Woloshin S. Are increasing 5-year survival rates evidence of success in cancer? *JAMA* (in press).
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Improvements in survival may be an illusion

EDITOR—Richards et al discuss the number of cancer deaths avoided as a result of improvements in survival.[1] There is a logical flaw in their argument as survival is defined as death more than five years after diagnosis. The population of patients in the five year window in which death from cancer can occur represents a mixed distribution of early, mid-phase, and advanced tumours. Advanced tumours are usually symptomatic and quickly fatal, whereas early cancers are often small, unnoticed, and have a longer potential survival independent of treatment. The introduction of screening tests or public awareness campaigns will increase detection rates for early, "survivable" tumours rather than late tumours. A patient whose tumour will kill them in four years and 11 months' time and who receives a diagnosis after a wait of seven weeks before seeing a consultant would be classified as a cancer death. By introducing a fast track, maximum two week, waiting time to see the consultant, their cancer will be diagnosed five weeks earlier, allowing them to be categorised as a cancer "survivor" even though their actual survival time was identical. Most impact will therefore be made on the easiest tumour types to detect. This is confirmed by Richards et al's data as survival has improved for breast cancer (mammography), colon cancer (faecal occult blood), melanoma (visual surveillance), cervical cancer (cytology), cancer of the testis (self examination). No improvement has been seen for lung or laryngeal cancers because they are either difficult to detect or aggressive and hence survival is less than five years at detection.

In the data for breast cancer, assuming a uniform distribution for potential survival over a five year window, 4822 (sic) deaths could be avoided simply by accelerating diagnosis by 6.5 months. However, the survival distribution is not even and improved diagnosis has most effect on early cancers. Mammography is claimed to increase the diagnosis of early treatable breast tumours, but screening may only be obscuring a lack of any progress on overall long term tumour survival by an artefact of earlier diagnosis. Longer term—for example, 10 year—survival statistics are required to address this issue properly. We therefore contend that improvements in survival may be an illusion and that "deaths avoided" is a term that should be replaced by "deaths postponed."

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1 Richards MA, Stockton D, Babb P, Coleman. How many deaths have been avoided through improvements in cancer survival? *BMJ* 2000;320:895-8. (1 April.)

"Avoided deaths" may not be useful for predicting mortality reductions from cancer

EDITOR—Richards et al in their paper suggest a method for estimating the number of deaths from cancer avoided as a result of improvements in five year relative survival and use this to predict future reductions in mortality from cancer.[1]

We have a number of reservations about the usefulness of this method.

Firstly, survival is not always independent of incidence. For example, an increase in screening activity, such as occurred with the introduction of breast screening and more widespread testing for prostate specific antigen, will both increase incidence and improve survival by advancing the date of diagnosis without necessarily postponing date of death. In these circumstances, selection, lead time, and length biases will confound attempts to draw inferences on mortality reductions from survival estimates.

Secondly, under certain conditions, the application of the method used by Richards et al will yield counterintuitive results. For example, suppose the method was used to estimate the number of "avoided" deaths in the next quinquennia, 1991-5, again using 1981-5 as the baseline. Then, were incidence to fall and survival to increase between 1986-90 and 1991-5, both highly desirable outcomes, the number of deaths avoided in 1991-5 will be less than in 1986-90 if the fall in incidence is proportionately greater than the increase in survival between the two periods. In other words, progress towards the government target, as measured by avoided deaths, seems to be worse, although true progress is being made.

The government's target, referred to in the paper by Richards et al and the accompanying editorial,[2] is for a reduction in mortality from cancer in people aged under 75 by at least 20% by the year 2010, from a 1997 baseline. It is therefore sobering to reflect that, for many sites of cancer, the net effect of an increase in incidence together with a small gain in survival will be not only to increase the number of avoided deaths but also to increase the actual number of cancer deaths. For example, Richards et al estimate that more than 2000 deaths were avoided in patients with bladder cancer and non-Hodgkin's disease registered in 1986-90, as a result of improvements in survival. Reference to Coleman et al, however, also shows that for these sites of cancer, the number of deaths actually occurring within five years of diagnosis increased between 1981-5 and 1986-90 by over 3000.[3]

Inspection of age specific mortality statistics may yet offer a more timely and direct method of monitoring progress towards the government's mortality target.

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Authors' reply

EDITOR—We agree with Baum that any impact of screening on breast cancer mortality in women would not be expected for several years after screening begins, mainly because women who die in a given year will have had their cancer diagnosed up to 10 or more years earlier. Death rates do not respond rapidly to a sudden shift in diagnosis or treatment. By contrast, the survival rates for women diagnosed in a given year will obviously reflect recent improvements in diagnosis and treatment, including any effect of screening.[1] About a third of the decline in breast cancer mortality since 1988 is still likely to be due to screening,[2] but we agree that breast cancer was being diagnosed earlier, treatment improved,[3] and mortality was falling well before the screening programme was fully implemented.

Breast cancer survival has risen for women of all ages during 1971-98,[4] but among women aged 50-69 diagnosed since 1991 five year survival has risen more than one year survival—this is not typical of lead

time bias. We agree with Welch, Reynolds and Wierzbicki, and Threlfall et al that interpretation of this rapid increase is complex because it reflects earlier diagnosis (lead time) and the inclusion of some slow growing tumours that may never have been diagnosed during the patient's lifetime (length bias), as well as the survival benefit of more effective earlier treatment.

We agree with Welch that survival rates are affected by new diagnostic tests, such as prostate specific antigen for prostate cancer, which lead to patients being diagnosed with very early or even innocuous disease. This can also affect mortality because death may be incorrectly attributed to prostate cancer.[5] Testing for prostate specific antigen was not widespread in England and Wales until the early 1990s, and survival rose by less than 1% for men given a diagnosis during 1981-90, the period covered by our estimate, and it accounted for less than 2% of the overall estimate of avoided deaths. Prostate cancer survival has risen since 1991.[4]

Reynolds and Wierzbicki said that we defined survival as death more than five years after diagnosis. Cancer survival estimates for a given time since diagnosis simply reflect the probability of survival up to that point in time. We used relative survival rates up to five years after diagnosis.

We agree with Threlfall et al that if incidence is rising faster than survival (as with non-Hodgkin's lymphoma), mortality will increase. Our estimate of avoided deaths within five years of diagnosis remains logical in this context, however, because it reflects the number of additional deaths that would have occurred if five year survival had not increased at all. The estimate of avoided deaths does not depend on trends in incidence.

Threlfall et al comment that improvements in incidence and survival will be required to achieve the government's target of reducing cancer deaths under age 75 by 20% by 2010. We are inclined to agree. Close surveillance of incidence, survival, and mortality will be needed to assess progress toward this target.

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Aspirin for primary prevention

Treatment policy should be based on all trial evidence, not subgroup analysis

Editor—We have suggested that aspirin for primary prevention is safe and worthwhile when the estimated 10 year coronary risk is >15%, provided that any hypertension is controlled.[1] This conclusion comes from conservative interpretation of a meta-analysis examining the balance of benefit and risk in four large randomised controlled trials of aspirin for primary prevention, and fully supports recommendations in the Joint British Societies and British Hypertension Society guidelines.[2] One assumption central to this analysis, and to these guidelines, is that relative risk reduction by aspirin is constant, so that the magnitude of benefit from aspirin is determined by pretreatment coronary risk.

Unfortunately, Meade et al did not examine this assumption in their subgroup analysis of the thrombosis prevention trial.[3] Rather, they present subgroup analyses according to individual risk factors (systolic blood pressure, age, and cholesterol concentration). These analyses are not really apposite to the guidelines and may even be misleading. For example, their results suggest little benefit (6% reduction in coronary heart disease) or even harm (8% increase in all cardiovascular events) from aspirin when systolic blood pressure exceeds 145 mm Hg. In the physicians' study in the United States there was a substantial (35%) reduction in coronary events at systolic blood pressure >150 mm Hg.[4] In the hypertension optimal treatment study, men with hypertension that was controlled from 168/106 mm Hg to an average of 140/83 mm Hg, which is still "high normal," had a coronary reduction of 42% (P=0.001) and a 13% reduction in all cardiovascular events.[5] The important point is that subgroup analysis of the thrombosis prevention trial is certainly not representative of all the trial evidence available.

Similar discrepancies are present in the findings for age. At age 65 and over this subgroup analysis suggests a 29% increase in coronary heart disease, but a 41% reduction in stroke, with aspirin. As the authors note, this is totally inconsistent with the physicians' study, which showed coronary reductions of 44% at ages 60-69 and 41% at ages 70-84.[4] In the hypertension optimal treatment study, treated hypertensive patients aged 65 and over had reductions in coronary heart disease of 38% and all cardiovascular events by 21%.[5]

The treatment policy for aspirin for primary prevention should be based on all the trial evidence and estimation of absolute risk of coronary heart disease,[2] not on subgroup analysis of a single trial or on a single coronary risk factor.

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Doctors and patients should understand potential benefits and risks of aspirin treatment

Editor—I echo Meade et al's concern that the subgroup findings in their paper must be interpreted with caution.[1] In the hypertension optimal treatment trial the use of 75 mg aspirin was associated with the prevention of 1.5 myocardial infarctions (2.5 in patients with diabetes) per 1000 patients treated for one year.[2] It will be interesting to learn if a subgroup analysis of the diabetic patients in Meade et al's study is possible. In the nurses' health study the reduction in occlusive infarction of a large artery was greater for older or hypertensive women.[3]

Although the nurses' health study is a prospective cohort study, it is the best available evidence for the use of aspirin in primary prevention of stroke and coronary artery diseases in women. Confirmatory data on the role of aspirin in primary prevention in women await results of ongoing randomised controlled clinical trials.

In He et al's meta-analysis—a large meta-analysis of 16 trials, including the British doctors' and the United States physicians' trials—aspirin treatment was associated with an absolute increase in risk of haemorrhagic stroke of 12 events per 1000 people (95% confidence interval 5 to 20; P<0.001).[4] The mean dose of aspirin in this meta-analysis was 273 mg and the mean duration of treatment 37 months.

The meta-analysis showed that the increase in the absolute risk of haemorrhagic stroke is not related to patient characteristics, such as age, hypertension, and hyperlipidaemia. The pooled odds ratio for haematemesis was 1.5 in Roderick et al's

overview of 21 randomised controlled trials, with average follow up of 3.85 years.[5]

The Canadian and the United States Preventive Services Task Forces do not make recommendations for or against the use of aspirin in asymptomatic patients for the primary prevention of cardiovascular diseases. If aspirin treatment is considered, doctors and patients should understand the potential benefits and risks of the treatment before starting it.

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Analysis did not account for cluster randomisation

EDITOR—We congratulate Chapman et al for their paper on prevention of dog bites in children.[1] The study was a cluster randomised trial; schools were allocated to intervention or control groups, and data were collected at the level of the individual schoolchild. The analysis did, however, not account for cluster randomisation. The assumption of independence of observations required for standard statistical tests, such as the χ^2 test used by Chapman et al, is violated in cluster randomised trials. It is not unreasonable to assume that the actions of the children from one school towards the dog were more similar to each other than to the actions of children from another school.

Standard statistical methods that do not account for cluster effects in data from cluster randomised trials will result in the overestimation of the significance of an intervention. For dichotomous outcomes, Donner and Klar recommend a two sample *t* test based on cluster level event rates when the numbers of clusters in each arm of the trial number is less than 10.[2] Analysing the data ignoring clustering gives a difference between the groups of 70% ($P<0.0001$; 95% confidence interval 62% to 77%). Reanalysis of the school level event rates using a *t* test with equal variances gave a mean difference of 70% ($P=0.0018$; 36% to 100%). This showed that although the evidence is still in favour of the intervention to prevent a bite, it is not as strong as when clustering is ignored. A revision of the CONSORT statement for reporting of cluster randomised trials should help researchers avoid the potential pitfalls of such unit of analysis errors.[3]

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Peterborough hospital has tissue bank on not for profit basis

EDITOR—Josefson writes that academic hospitals in the United States are to ask patients for the right to sell their tissue.[1] This comes soon after publication in the United Kingdom of a review of the issues related to setting up a human research tissue bank in the NHS (based on our experience in Peterborough)[2] and new guidance on patient confidentiality by the General Medical Council.[3]

Originally established in 1996, our tissue bank has expanded rapidly and now supplies more than 30 commercial biomedical research organisations. It is located in a department of cellular pathology in a district general hospital. Human tissue is procured and processed subject to relevant law and ethical and safety procedures, and the bank is operated on a not for profit basis and is subject to audit.[4] The bank would not succeed without the enthusiasm and commitment of the small team or without a commercial client base.

Before surgery, research nurses from the tissue bank visit patients and obtain consent to the use of tissue for research. Patients are told that the research will have no influence on the planned surgery, the tissue used will be surplus to diagnostic requirements, the research will be carried out in the commercial biomedical sector, and their details will be anonymised.

The bank has a donor register for people who wish to leave their bodies for research after death, but most cadaveric donations are referred under an agreement with the United Kingdom National Blood Service's tissue services. Of 1068 patients interviewed in the past two years, only nine refused to let us bank their tissue; 31 cadaveric retrievals were undertaken in that time.

The demand for human tissue is increasing. Currently there is no national system or control for banking anonymised human tissue for biomedical research.

Acquiring, processing, storing, and distributing donated human tissue is labour intensive. These costs should not be borne by the NHS, particularly if tissue is supplied to the commercial sector. Nor can the NHS be seen to profit directly. Working with the commercial biomedical sector can lead to new and exciting collaborations that may ultimately benefit patients.

Finally, we prefer to use the term "not for profit" rather than "selling"—but then we don't write news headlines.

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Public health service needs to be independent

EDITOR—With the publication of the Phillips report on bovine spongiform encephalopathy (BSE),[1 2] the state of the public health system is revealed. For almost 30 years the independent voice of public health in England has been weakened at all levels, national, regional, and local.

The pressures to cooperate and not to rock the boat have clearly affected even chief medical officers and senior civil servants. It has been a particular problem for we, as regional directors of public health in regional health authorities, who found ourselves to be civil servants when regional health authorities were abolished seven years ago.

We then become constrained in our ability to represent the public interest on health matters in cities and boroughs. A proud tradition, dating back to the 1840s, has been attenuated, and district directors of public health no longer engage in the same robust way in public debate on health matters as did their predecessors.

Now is the time to seriously consider establishing an independent public health service and giving statutory protection to directors of public health when commenting in good faith on matters of public health.

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