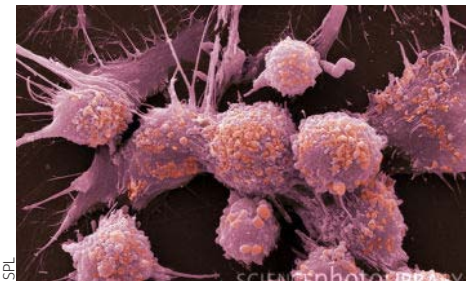

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PROSTATE CANCER SCREENING

Study raises five questions

Sandblom and colleagues' 20 year follow-up study of prostate cancer screening raises five questions.[1]

(1) Screening test methods evolved during the study. Why were men from the screening group aged 69 or older excluded only from the fourth screening session rather than consistently throughout?

(2) Digital rectal examination was the first screening test, prostate specific antigen (PSA) testing being included only in the third screening session. In addition to the combined utility of rectal examination and PSA testing, what are the individual contributions of these tests to the early detection of prostate cancer?

(3) Overdiagnosis of indolent tumours has been rightly emphasised as a significant drawback to screening, but unnecessary investigations, the need for early repeat testing, and patient anxiety from a false positive result of examination or testing have not. How many false positive results arise from PSA testing, rectal examination, and the two combined? We could not deduce whether the combined tests reduced or increased the number of false positive results.

(4) What are the predictive values of the individual tests and their combination? We could not calculate them from the information given.

(5) Figures 2-4 show overall and disease specific mortality only in patients diagnosed with prostate cancer in the two groups, which may be misleading. What are the rates in the two groups overall as per allocation?

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1 Sandblom G, Varenhorst E, Rosell J, Löfman O, Carlsson P. Randomised prostate cancer screening trial: 20 year follow-up. *BMJ* 2011;342:d1539. (31 March.)

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What is mortality denominator?

Sandblom and colleagues state: "In a Cox proportional hazard analysis, the hazard ratio for death from prostate cancer was 1.23 (0.94 to 1.62; P=0.13) and 1.58 (1.06 to 2.36; P=0.024) after adjustment for age at start of the study."[1]

Does the hazard ratio of 1.23 mean that being assigned to the uninvited group increases prostate cancer mortality by 23% compared with the invited group, or that only those in the uninvited group with prostate cancer have 23% more deaths than the invited group with prostate cancer? In other words, what is the denominator?

Is the hazard ratio of 1.23 unadjusted for age at assignment while 1.58 was adjusted for age? Was adjustment for age pre-specified before the investigators analysed their death rates or was this post hoc analysis? If post hoc analysis, how many mortality risk factors were tested for an effect on case rate mortality? If the assignment to group was based on date of birth, the age distributions of the two groups should be nearly the same. Why then was the Cox proportional hazard analysis adjusted for age at the start of the study?

Screening detects more slow growing or comparatively benign cancers such that case rate mortality with prostate cancers alone in the denominator will favour screening over no screening with fewer slow growing prostate cancers. Moreover, screening symptom-free men is intended to detect at an earlier stage before metastasis. Detecting any cancer at an earlier stage prolongs survival after diagnosis even if no treatment is given to any case in either group. Therefore both prostate cancer mortality and all cause mortality should be compared by assigned group beginning on the day of assignment, not day of diagnosis.

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1 Sandblom G, Varenhorst E, Rosell J, Löfman O, Carlsson P. Randomised prostate cancer screening trial: 20 year follow-up. *BMJ* 2011;342:d1539. (31 March.)

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Study has major shortcomings

Although Sandblom and colleagues' study is the longest follow-up to date of prostate cancer screening,[1] it has some major shortcomings.

(1) The group of men randomised to screening is small. Of the 1494 men, only 1161 participated in the initial screening (78%). Those who refused are apparently excluded from the intention to screen analysis, which is not correct in such analysis.

(2) Information on the sample size calculation is contradictory. In the methods section the sample size was calculated to allow assessment of the acceptance and feasibility of a prostate cancer screening programme. In the statistics section the study was designed to detect a plausible reduction of prostate cancer specific mortality within 20 years. Which is correct?

(3) Of the 85 prostate cancers diagnosed in the screening arm, 42 were detected as interval cancers. This implies ineffective screening by rectal examination during the first two screening rounds. These interval cancers, if you look at table 2, are likely to have a high adverse prognostic impact if their unfavourable stage and grade distribution and the imbalance in applied treatments are taken into account.

(4) Kaplan-Meier projections are used to present trial results on mortality as survival curves. The authors do not take account of the lead-time bias, which is inherent in this comparison of survival.

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1 Sandblom G, Varenhorst E, Rosell J, Löfman O, Carlsson P. Randomised prostate cancer screening trial: 20 year follow-up. *BMJ* 2011;342:d1539. (31 March.)

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Underscreened and undertreated

Three points question Sandblom and colleagues' conclusion that screening for prostate cancer conferred no significant survival benefit.[1]

(1) A quarter of the men randomised to the screening cohort did not attend. Two thirds of the attendees were not invited for a second prostate specific antigen (PSA) determination because of age. Thus, on average, the "screened" population had a single PSA determination. This was probably only slightly more often than in the control group (until 1994, less than one PSA test per man was performed in Sweden). Can a single PSA test seriously be construed as a screening programme?

(2) Only a quarter of the men diagnosed with prostate cancer in the screened cohort were treated with curative intent (radical prostatectomy, brachytherapy, or external radiotherapy).
(3) About half of the men diagnosed with prostate cancer, whether in the screened or control group, died of their disease.

An equally valid reason for the dismal prostate cancer survival among these men is that they were underscreened and undertreated.

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1 Sandblom G, Varenhorst E, Rosell J, Löfman O, Carlsson P. Randomised prostate cancer screening trial: 20 year follow-up. *BMJ* 2011;342:d1539. (31 March.)

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Report bias favours screening

While aiming to assess prostate cancer specific mortality, Sandblom and colleagues indicate a "prostate cancer specific mortality" in the screening group of 35% and in the control group of 45%, which actually is the case fatality rate.[1] The true prostate cancer specific mortality was 2% (30/1494) in the screening group and 1.7% (130/7532) in the control group—an absolute excess risk in the screened versus the unscreened group of 0.3% and a relative excess risk of 16.3%.

The authors also present two misleading figures that suggest screening results in higher (although non-significantly) survival. However, survival of patients diagnosed with prostate cancer is not an adequate outcome measure in the context of screening because it is prone to lead time bias and overdiagnosis bias,[2] which distort the results in favour of screening. In this study, an estimated 32% of the prostate cancer cases in the screening group were overdiagnosed. Because of the severe negative effects (such as incontinence, erectile dysfunction) of overdiagnosis, due consideration should be given to a balanced presentation and interpretation of results.

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Cite this as: *BMJ* 2011;342:d3703

Evidence of overdiagnosis

Sandblom and colleagues' paper compared the case fatality rate of prostate cancer between men randomly assigned to screening and those who were not invited for screening.[1] A more reliable outcome of such a randomised controlled trial is the difference of mortality as a result of prostate cancer between intervention and control groups. Of 1494 men invited for screening, 30 (2%) died from prostate cancer. Of 7532 men not invited, 130 (1.7%) died from prostate cancer. These proportions are not given in the paper. The outcome in the invited group is not significantly worse than in the control group, but it provides a negative answer to the question about screening efficacy for reducing prostate cancer mortality. Survival analysis limited to diagnosed cases is biased by overdiagnosis of clinically insignificant prostate cancer.[2] In table 1 of Sandblom and colleagues' paper we found a significant 1.8% (95% confidence interval 0.6% to 3.0%) (χ^2=10.23; P=0.0014) absolute increase in prostate cancer as a result of overdiagnosis in the invited group. Figures 2 and 3 show an illusionary improved prognosis among cases in the intervention versus control group because of an excess of 27 overdiagnoses among the 85 cases in the intervention group.[1] Both case fatality rate and survival are overdiagnosis dependent: patients who have been overdiagnosed cannot die from prostate cancer. Evidence based medicine must be independent of misleading influence and use well recognised principles, such as analysis by intention to treat.[3]

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Authors' reply

In answer to Burn and colleagues:
(1) The decision to exclude men older than 69 was taken towards the end of the study to avoid overdiagnosis in older men. Even if this could have been done consistently from the beginning, the increasing age of the study population during the first three screening rounds would not affect the outcome substantially.
(2) To investigate whether the detection rate and subsequent mortality changed after the introduction of prostate specific antigen (PSA) testing, we modelled diagnosis before 1993 versus diagnosis after 1993 as a dichotomous time-varying covariate. We found no significant difference in mortality.
(3) The problem of false positive findings at the primary investigation has been analysed.[1]
(4) Although we did not address predictive values, they have been considered briefly before.[2]
(5) The figures indeed showed survival only of the men who were diagnosed with cancer; figures based on the whole cohort would not show any deviation between the curves.

In answer to Tolan, mortality in the control group is the denominator—that is, prostate cancer mortality was slightly higher in the screening group. Age was adjusted a priori, and we have not tested for any covariates we did not present. The analyses began from 1 January 1987, not from the day of diagnosis. The hazard ratio of 1.23 is without adjustment for age, whereas that of 1.58 is adjusted for age. The mean age at the start of the study was 61.25 and 62.10 years in the screening and control group respectively (NS).

We based the Cox proportional hazard analysis and Kaplan-Meier analysis on men with a diagnosis of prostate cancer. Adjustment for age at diagnosis minimises the lead time bias. The main outcome measure, however, was the risk ratio for death from prostate cancer, which is based on the whole cohort.

To address Schroder's concerns:
(1) The number of men in the screening group is much smaller than in the ERSPC and PLCO studies, but the long follow-up gives statistical power to detect major hypothetical differences in prostate cancer mortality. All refusers were included in the analysis of prostate cancer mortality ratio.
(2) The study protocol was not designed with mortality as primary end point but the long follow-up renders analyses with mortality as outcome meaningful, albeit not with the same statistical power as the ERSPC trial.
(3) The many interval cancers (occurring also with PSA as the screening tool) undoubtedly have a large impact on the outcome. We are sure that screening as it is performed nowadays is more effective.
(4) Even if lead time bias cannot be taken into account in Kaplan-Meier projections, they still provide important information.

In answer to Aronowitz:
(1) Most men attended at least two screening rounds (1268 men attended at least once and 726 at least three times).
(2) Radical prostatectomy and radiotherapy was practised much less when the study was performed than it is today.

(3) The high mortality in the screening group is probably explained by the low percentage of men undergoing treatment with radical intent, as well as relapse after radical treatment.

Given the problem of lead time bias and overdiagnosis mentioned by Dreier and colleagues, the Cox proportional hazard analyses were based on time from the start of the study, not from diagnosis. Overdiagnosis probably accounts for the significant hazard ratio seen in the Cox proportional hazard analysis with adjustment for age.

We used prostate cancer mortality in both groups as the major end point—that is, including men without prostate cancer diagnosis in the denominators. The risk ratio for death from prostate cancer was estimated as 1.16 ((30/1494)/(130/7532)), which was not significant, as pointed out by Junod and colleagues. Our intent was not to show an illusionary improved prognosis in the intervention group but to depict the long term survival of men diagnosed with prostate cancer in a screening programme, without neglecting the problem of overdiagnosis and lead time bias.

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Competing interests: None declared.

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Cite this as: *BMJ* 2011;342:d3710

INCIDENTAL EOSINOPHILIA

Always take a travel history

We emphasise the need to take a travel history from all patients with eosinophilia and to clarify the appropriate investigations for parasitic infections, the most common cause of eosinophilia worldwide.[1] Delay in taking a travel history can have serious consequences, such as failure to diagnose and treat asymptomatic strongyloidiasis before immunosuppressive therapy is given.[2] However, travel histories are obtained from less than 20% of British adults with acute clinical syndromes that might be travel related.[3]

Eosinophilia is typically caused by helminths, not giardiasis or most other protozoal infections. Examination of the famous "hot stool" for ova, cysts, and parasites is only needed when amoebic dysentery is suspected, in which case faeces, mucus, or rectal scrapings should be examined immediately for motile trophozoites of *Entamoeba*

DR RICHARD KESSEL+DR GENE SHIH/VISUALS UNLIMITED/SPL

histolytica with ingested erythrocytes. Amoebiasis is not usually associated with eosinophilia, although this is a common misconception.[4]

For other parasites, the examination of fresh or preserved faecal specimens is not quite so urgent, and the choice of other investigations, including serological screening, depends on many factors. Strongyloidiasis is ubiquitous in the tropics, but eosinophilia in travellers or immigrants from Africa is more likely to be caused by schistosomiasis or loiasis. Comprehensive British guidelines on the diagnosis and management of travel related eosinophilia have recently been published.[5]

We recommend the automatic generation of reminders in laboratory reports on full blood counts with raised eosinophils, such as, "Note the presence of eosinophilia. Have you taken a travel and drug exposure history?" These could be added to the learning points tabled by Sims and Erber.[1]

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Competing interests: None declared.

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Cite this as: *BMJ* 2011;342:d3688

INFECTIVE ENDOCARDITIS

NICE *v* world on prophylaxis

Articles defending the National Institute for Health and Clinical Excellence's (NICE) advocacy of no antibiotic prophylaxis tend to suggest that the contrary position is universal antibiotic prophylaxis.[1] But the essential difference between American, European, and Australian guidelines and those from NICE is in advising antibiotics for high risk cardiac patients (those with prosthetic heart valves or previous endocarditis) having high risk dental procedures (mainly extractions).[2]

In this high risk group, endocarditis is more common and more dangerous than in native valve lesions,[3 4] with an acute mortality of 25% and a survival rate of only 55% at five years.[2] Furthermore there is evidence, albeit no proof, that antibiotics are effective.[2] In one study,[5] six cases of endocarditis occurred in 304 patients with prosthetic valves who were not protected by antibiotics, but none in 229 protected patients.

The number of high risk patients having high risk procedures in the recent study is unknown, but probably small.[1] The incidence of endocarditis is low, so the study was not designed to detect an effect in the group under debate. Furthermore, unpublished local audits suggest that patients at high risk tend to continue taking antibiotics.

The authors are wrong to conclude that their findings support the near total cessation of antibiotic prescribing recommended by NICE. We urgently need a national registry of new cases of endocarditis and a randomised controlled trial of antibiotic prophylaxis in high risk patients before we change from almost universally accepted international guidelines in favour of NICE.

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NEW MANAGEMENT OF GONORRHOEA

Impact of antibiotic resistance

The British Association of Sexual Health and HIV (BASHH) guidelines on the management of *Neisseria gonorrhoeae* have changed.[1 2]

Analysis of the UK Gonococcal Resistance to Antimicrobials Programme data showed decreasing sensitivity to cephalosporins.[3] The BASHH clinical effectiveness group therefore drafted guidelines advising ceftriaxone 500 mg intramuscularly as first line treatment, with cefixime 400 mg orally, as second line treatment if intramuscular injection is contraindicated or declined.

Ciprofloxacin is no longer recommended unless culture shows the isolate to be sensitive, or the regional prevalence of resistance is less than 5%, because 2009 data showed an increase in resistance to 35%, with 54% of isolates in men who have sex with men being resistant.[4]

When diagnosing gonorrhoea in a male patient outside of a genitourinary medicine clinic, a urethral swab sent in transport medium (Amies or Stuarts) for culture, or a dual chlamydia or gonorrhoea nucleic acid amplification test (NAAT), on first void urine, are advised. Because of worrying trends in antibiotic resistance, patients with positive gonorrhoea NAAT results should have repeat testing using culture, to identify antibiotic sensitivities.[5] A test of cure is recommended in all positive patients.[2]

When epididymo-orchitis is suspected, exclude a urinary tract infection by performing urine analysis and culture on a midstream urine sample only.

We strongly recommend that all patients with suspected or confirmed gonorrhoea be referred to a genitourinary medicine clinic for full sexually transmitted infection screening, treatment, partner notification, and test of cure. This will ensure correct management of the index patient and any sexual partners, thereby preventing onward transmission.

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Competing interests: None declared.

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NHS REFORMS

Responsibility for public health?

Concern about the health reforms has focused on the NHS.[1] We have concerns about responsibility for public health. If implemented, the bill will remove statutory responsibility to protect the public's health from any local organisation.

The NHS Act 2006 makes primary care trusts (PCTs) responsible for protecting and improving population health. This underpins the current public health system. Although partnership working is crucial, the "buck stops" with the PCT to ensure current NHS responsibilities for public health are managed. This has helped ensure that responsibility is taken for health protection incidents involving several organisations and in mobilising NHS resources to swab, vaccinate, and treat people during pandemics. It also maintains focus on public health programmes, such as tuberculosis, immunisation, and screening, which require a population approach across organisations.

The bill abolishes PCTs and confers extra responsibilities on local authorities; it adds a new section to the NHS Act—each local authority must take steps that it considers appropriate for improving local people's health (there is no obligation to do so); it allows the secretary of state to take such steps as he or she considers appropriate for improving the health of the people of England; and it creates GP commissioning groups with no geographically defined public health responsibilities.

The effect is that the secretary of state has no obligation to protect and improve the health of local populations and that health protection is excluded as a responsibility of any local health organisation.

Public health professional organisations have, in a letter to David Cameron, proposed that local authorities should be responsible and accountable for protecting and improving the health of their local populations and should be supported in this by Public Health England.[2] We support this view and hope it is included in the bill after the listening exercise.

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Competing interests: All authors are employed by the Health Protection Agency. The views expressed are those of the authors and not necessarily those of the HPA.

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PATIENT PARTICIPATION GROUPS

The *BMJ* and the Big Society

Was Nagraj and Gillam's editorial on patient participation groups just a tiny bit uncritical?[1] And was the strapline added by the *BMJ*'s editorial team (NHS reforms offer new and wide ranging opportunities) politically naive to the point of being irresponsible?

Input of service users to publicly funded healthcare can be a tool of the political right ("choice") or of the left ("voice").[2] It is more than 20 years since patient participation was first mooted by a Conservative government and seized on by civil servants as the lever that would drive up standards, inform service [re]design, and save money in the NHS[3]; 13 years since this same ideology was embraced by New Labour[4]; and 10 years since Shifting the Balance of Power called for the introduction of a new Patient Advocacy and Liaison Service (PALS), which was going to place the patient at the centre of far reaching reforms.[5] Interestingly, when the PALS services were set up, the "A" in the acronym had been mysteriously changed to "Advice," with the effect that the advocacy on behalf of the vulnerable (voice) element of these statutory bodies was downplayed in favour of an emphasis on informing consumption of health as a commodity (choice).

These latest proposals for "patient participation" promise much but are as vacuous as the previous ones. There is no evidence that politically driven structures oriented to supporting patient choice achieve anything other than redistributing resources towards the articulate and information rich and shifting responsibility for health inequalities from the state to the citizen.[6] A more fitting summary soundbite for this editorial might have been "what comes around goes around."

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Competing interests: None declared.

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PERNIOSIS OR CHILBLAINS?

What's in a name?

It is fascinating to compare the different approaches to the same medical problem between primary and secondary care.[1] Chilblains are a common self limiting disorder, managed by most GPs every winter in a 10 minute consultation. In secondary care the same condition warrants blood tests, a skin biopsy, presumably multiple consultations, and a fancy name. Are GPs under-investigating? Or are secondary care doctors over-investigating? Who will be the arbiter?

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Competing interests: None declared.

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