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LETTERS

JIM VARNEY/SPL

MILDLY ABNORMAL LIVER TESTS

Don't forget coeliac disease and drug history

Cobbold and colleagues omitted two important considerations when evaluating mildly raised serum aminotransferase concentrations.[1]

Firstly, abnormal liver biochemistry can be a presenting feature of coeliac disease.[2 3] This has long been recognised,[4] but the prevalence of coeliac disease is often underestimated, leading to delay in diagnosis and treatment. The prevalence in Western populations is about 1:100 (1:10 with an affected first degree relative), so it makes sense to ask patients with persistently raised aminotransferase concentrations about symptoms and family history and to consider serological and biopsy investigation. About 15-55% of patients with coeliac disease have raised aminotransferases, and in one study this measure alone indicated the development of coeliac disease in two of 158 patients.[5]

Secondly, the authors mention that prescription drugs and herbal remedies should be considered in the differential diagnosis. But the use of illegal drugs can also cause derangement of aminotransferase concentrations.[3] The use of anabolic steroids, cocaine, ecstasy, glues, solvents, and other substances should be sought in the history.

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Competing interests: None declared.

1 Cobbold JFL, Anstee QM, Thomas HC. Investigating mildly abnormal serum aminotransferase values. *BMJ* 2010;341:c4039. (30 July.)
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Cite this as: *BMJ* 2010;341:c4603

Myth of γ glutamyltransferase

Cobbold and colleagues state that γ glutamyltransferase should be measured in all patients with raised serum alanine aminotransferase because if raised it would indicate alcohol related liver disease.[1] Because of its ubiquitous distribution within the liver, γ glutamyltransferase is raised in all types of liver disease.[2] In the absence of serious liver disease, the myth that a raised γ glutamyltransferase is sensitive and specific for alcohol excess persists. In these circumstances, for example, γ glutamyltransferase is raised in only 52% of alcoholic patients,[3] but it is also raised in 50% of patients with non-alcoholic fatty liver disease.[4] Guidelines recommend that it is measured only to identify the likely origin of an isolated raised alkaline phosphatase, because if raised it indicates a hepatic rather than bony origin.[2 5] In this case study, γ glutamyltransferase was raised but the final diagnosis was non-alcoholic fatty liver disease.[1] We therefore suggest that measuring γ glutamyltransferase when investigating raised serum transaminases is unnecessary and potentially misleading.

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HYPERINTENSITIES ON MRI

White matter and depression

The editorial by Wallin and Fladby and systematic review by Debette and Markus suggested a link between white matter hyperintensities on magnetic resonance imaging and stroke, dementia, and death.[1 2] However, they did not mention the relation between white matter changes and late onset depression, also called "vascular depression."[3] Depression may present as a prodromal syndrome of dementia, and the affective and cognitive changes are thought to be part of a continuum seen in cerebrovascular disease.[4] It is important that future studies investigating the risk of white matter changes should also investigate the effects of these changes on mood and depressive symptoms.

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Competing interests: None declared.

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NICE ON BACTERIAL MENINGITIS

Possible additions to summary

Visintin and colleagues provide a useful summary of the guidance from the National Institute for Health and Clinical Excellence (NICE) on managing bacterial meningitis and meningococcal septicaemia in children and young people.[1] Further information relating to organisms causing bacterial meningitis other than *Neisseria meningitidis* is available in the more detailed guidelines.

One point that could have been added (briefly mentioned in the full guideline only) is that primary meningococcal conjunctivitis can occasionally progress to systemic meningococcal disease. This may occur especially in children, and systemic treatment is indicated.[2]

A footnote about salmonella meningitis could also have been useful. This infection was probably not specifically considered because *Salmonella* spp are not a common cause of

bacterial meningitis. However, neonates and infants are more at risk than older age groups from invasive salmonella infections, and the mortality, complication rate, and potential for relapse from salmonella meningitis are high.[3 4] The antibiotic treatment is generally longer than that recommended by the guidelines for meningitis caused by Gram negative bacilli such as *Escherichia coli*, and the most recent advice from the American Academy of Pediatrics should be consulted. As potential benefit outweighs risk for serious salmonella infection, addition of ciprofloxacin should be considered even for children. Reports also suggest that the risk of relapse is less with ceftriaxone (where not contraindicated) than if cefotaxime is the cephalosporin used.[3 4]

When the bacteria mentioned in the guidelines are likely to have caused meningitis but are without laboratory confirmation (possibly because of previous antibiotic treatment), the full length of treatment for the suspected organism is advisable to avoid relapse.

When the infections considered in this guidance occur, discussion with a microbiologist or infectious diseases physician/paediatrician is also indicated.

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Competing interests: None declared.

1 Visintin C, Mugglestone MA, Fields EJ, Jacklin P, Murphy MS, Pollard AJ, on behalf of the Guideline Development Group. Management of bacterial meningitis and meningococcal septicaemia in children and young people: summary of NICE guidance. *BMJ* 2010;340:c3209. (28 June.)
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Cite this as: *BMJ* 2010;341:c4466

COLORECTAL CANCER SCREENING

Randomised trials of flexible sigmoidoscopy

Bretthauer's editorial contains two incorrect statements about the outcomes of the UK flexible sigmoidoscopy trial (UKFSST).[1 2]

Firstly, the subtitle, "Flexible sigmoidoscopy shows promise, but randomised trial data are needed," is misleading because UKFSST is a randomised controlled trial. It involved 170 000 people and reduced the mortality of colorectal cancer by 43% and incidence by 33% in screening attenders.

Secondly, the absolute reduction among the 40 000 people screened in UKFSST was 211 cases and 85 deaths rather than 49 cases and 19 deaths.[1] The figures quoted in the editorial refer to differences in rates per 100 000 person years, not absolute numbers prevented.

Bretthauer's suggestion that further randomised trials are needed relates to the idea that flexible sigmoidoscopy should be introduced into the population in a randomised fashion, allowing comparison with standard screening tests. We agree that this would provide data on uptake rates, but it would require 10 years of follow-up to estimate effectiveness while depriving half the population of the benefit of a test with proved efficacy.

Effectiveness data are country specific (NORCCAP is specific to Norway), whereas efficacy data are generalisable to all populations when adjusted for local participation rates. Thus the 43% reduction in mortality from colorectal cancer in UKFSST attenders translates into a 21.5% reduction in a population with 50% attendance.[3]

The benefit of a single flexible sigmoidoscopy was sustained over 11 years. Interpretation of longer term effects may indeed be complicated by exposure to the NHS bowel cancer screening programme, but using the excellent NHS records, we can adjust for this.

Single flexible sigmoidoscopy in UKFSST did not reduce incidence of proximal cancers. Bretthauer expects a different outcome from similar trials (Italy, Norway, United States) with lower thresholds for offering colonoscopy, but their results may confirm that endoscopy is comparatively ineffective in preventing proximal cancers.[4]

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Competing interests: None declared.

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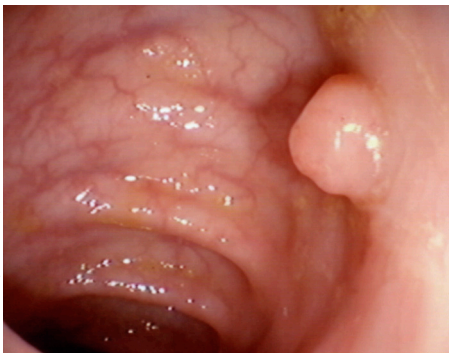
Cite this as: *BMJ* 2010;341:c4618

Author's reply

Atkin and colleagues point out that the subtitle in the print version of my editorial is misleading.[1] I agree that it is poor and does not accurately reflect the content of my editorial.

When I approved the manuscript the subtitle was "New evidence suggests flexible sigmoidoscopy should be included in national screening programmes," which remains in the canonical version online. The editorial change in print introduced a misunderstanding.



DR LARPENT/GREHGEP/SPL

In the last revision I sent to the *BMJ*, I added an introductory sentence to the second paragraph explaining that the numbers stated are based on 100 000 individuals (and do not represent actual numbers obtained in Atkin and colleagues' study[2]). This sentence (including the rates per 100 000 in both groups) was not included by the *BMJ* in the published version. Therefore, it became unclear that I was talking about cases per 100 000, not actual numbers.

Although I agree that the method of adjustment for non-compliance by Cuzick et al (which was applied in Atkin and colleagues' study) seems attractive,[3] I would prefer to postpone the final answer on effectiveness until the results from the ongoing randomised trials on flexible sigmoidoscopy are available.

I do not necessarily "expect" an effect on proximal colorectal cancer in the ongoing trials.[1] However, because these trials have a lower threshold for full colonoscopy after flexible sigmoidoscopy screening and thus more individuals have undergone full colonoscopy, the results may differ from those shown in the UK trial.

As stated in my editorial, Atkin and colleagues' trial is a landmark study, but we should not yet close the case for flexible sigmoidoscopy screening completely. Results from the other studies will be out fairly soon and should provide even stronger evidence to guide individuals on colorectal cancer screening.

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Competing interests: None declared.

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Cite this as: *BMJ* 2010;341:c4620

SMALL STUDIES IN META-ANALYSES

Making the best of a little

Nuesch and colleagues confirm that the effects of small studies can distort the results of meta-analyses.[1] More important than this potential distortion, however, is the potential for getting completely the wrong answer from a meta-analysis that contains only small trials.

The problem, of course, is that many, if not most, meta-analyses comprise small trials and even in aggregate they do not amount to sufficient numbers from which to draw conclusions, even if everything else were perfect. Too much is often made of too little whether complementary therapies such as acupuncture[2] or conventional treatments in difficult disciplines such as palliative care[3] are considered.

Meta-analyses of small studies should not be allowed to reach conclusions without pointing out that there is too little information to be sure of a result. One approach would be to agree a minimum number of events—beneficial and harmful—below which a result cannot be trusted. Two hundred events is a useful rule of thumb for believability.[4] Size is an important source of bias that needs to be considered alongside study quality and validity.[5] It is not routinely covered in the Cochrane risk of bias table: perhaps it should be.

The focus on events, particularly in pain studies, concentrates on clinically useful outcomes, which is important when the distribution of results is anything but Gaussian, the average result is obtained by only a few, and duration bias is substantial.[5]

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Competing interests: None declared.

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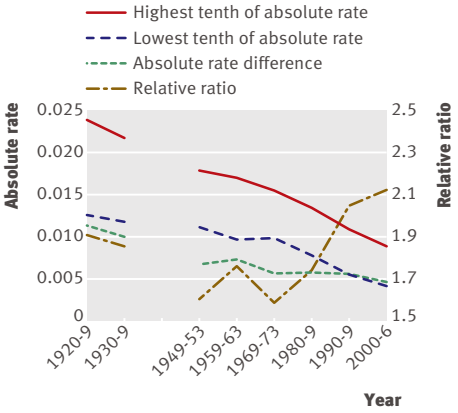
Cite this as: *BMJ* 2010;341:c4463



Absolute rates of mortality in lowest and highest tenths with absolute rate differences and relative ratios

INEQUALITIES IN MORTALITY

Study rates, not standardised mortality ratios

In their study from 1921 to 2007 Thomas and colleagues conclude on the basis of standardised mortality ratios that inequalities in mortality continue to rise and are now almost as high as in the 1930s.[1] Relative ratios are, however, misleading when absolute rates change strongly. I calculated the differences in absolute rate between the highest and lowest tenths using the authors' standardised mortality ratios and mortality for those aged 55-59 in the United Kingdom.[2]

The figure shows that differences in absolute rate were high before the second world war, declined in the third quarter of the 20th century, remained steady in the last quarter, and were lowest in 2000-6. Equality was highest in 1969-1973, when mortality was highest in the best off. Tobacco was the great social equaliser (hardly an ideal to strive for).

Mortality declined rapidly in all social classes, to a greater extent in the poor and more rapidly in the rich. This cause for celebration is the natural consequence of a great, but unavoidably heterogeneous progress in populations at diverse risks of mortality. In a dynamic system increasing differences between forerunners and laggards are to be expected when change increases. This reverses when change stops, the slow catching up. This is illustrated by the life expectancies of the extremes, as shown in this paper. It is comparable to the times of the first and last riders in a stage of the Tour de France: the faster the stage the bigger the difference.

Mortality differences by social class, income, and educational attainment are as ubiquitous as death itself. The relative differences are not informative, but how much of the social health divide is avoidable at reasonable cost is. We should keep in mind that the evidence of effectiveness of targeted health policies is tenuous compared with the many deaths that are avoided by general progress in rich and poor alike.

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Competing interests: None declared.

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Cite this as: *BMJ* 2010;341:c4614

ORPHAN DRUGS

Relating price determination to disease prevalence

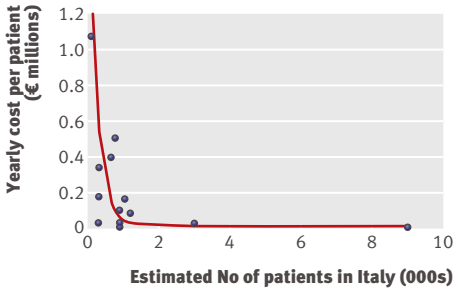
For orphan drugs, the yearly cost per patient is inversely related to the prevalence of the disease.[1-5] Although this relation is widely recognised qualitatively, to our knowledge, no attempt has been made to define it mathematically.

We examined the decisions made for non-cancer orphan drugs approved in Italy over the past years and developed the following equation to define how the yearly cost per patient is related to disease prevalence:

$$[\text{yearly cost per patient}] = 2\,000\,000 \times e^{-0.004 \times [\text{patients}]} + 10\,000$$

where "yearly cost per patient" is in € and "patients" is number expected to receive the treatment in Italy. The equation is intended to be used only when the disease prevalence is fewer than two cases per 10 000 people.

The equation was determined by fitting the data pairs of [yearly cost per patient] *v* [patients] for 17 non-cancer orphan drugs (figure). We calculated yearly costs on the basis of a "typical" patient receiving the dosage indicated by the registration leaflet. The weighted non-linear least squared fit that generated this equation was carried out using software from pharmacokinetic calculations (PCNONLIN-4.0; Scientific Consulting, NC, USA). Although the function does not perfectly fit the



Data pairs of [yearly cost per patient] *v* [patients] for 17 non-cancer orphan drugs (four values were identical) and best fit curve according to equation. Data sources: prevalence from www.orpha.net; drug prices from Italian national drug regulatory agency; Italian population 60.3 million; further details, including names of the drugs http://bmj.com/cgi/eletters/337/aug11_2/a1225#239966

original data, this curve provides a useful reference to help improve the homogeneity of future decisions in this area.

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Competing interests: Although AM, AC, and LP are members of the drug reimbursement committee of the Italian drug agency (AIFA), this letter reflects their personal views.

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FUNDING CARE FOR OLDER PEOPLE

Government guidance caused rise in long term care spending

The Audit Commission's 2008 report on fair access to care services (FACS) noted that with the tightening of FACS bands "there is no directly observable impact from a council's choice of FACS policy on emergency admissions to or delayed discharges from hospital."[1] Thus it is difficult to see how Featherstone and Whitham can claim that "as local authorities tighten eligibility criteria for long term care funding costs shift into the NHS."[2 3]

The true cause of the recent dramatic rise in NHS spending on long term care is the 2007 and 2009 guidance from the Department of Health on eligibility for NHS continuing healthcare funding. The Commission for Social Care Inspection recently noted that the guidance was expected to increase numbers of people supported by NHS funded continuing care by 7000 a year to 31 000 in total.[4] But in England the number receiving such care rose from 24 952 in 2007-8 to 37 920 in 2008-9 after the guidance was introduced, and it reached 44 924 in the first quarter of 2009-10. By the fourth quarter of 2009-10 it had risen to 50 426.[5]

We need a fair way to support long term care funding, but simply merging local authority and NHS spending on care for elderly people will not keep costs in check.

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Competing interests: None declared.

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SURGERY AND SPONDYLOSIS

Inappropriate partners

The pursuit of a surgical solution to a problem such as spondylosis whose pathology is poorly understood is frustratingly disappointing. "First do no harm" should drive decision making in clinical practice, yet clinicians still seem besotted with the idea that surgery is the answer to back pain.

Fouyas and colleagues admit the paucity of evidence to justify surgery as a therapeutic option for cervical spondylosis even with neurological dysfunction.[1] This lack is all the more notable when long term follow-up is considered to be five years, whereas patients with spinal pain commonly live with their predicament for 5-10 times longer than this.[2] The mistake is assuming a direct relation between symptoms and structural deviation as shown by investigations such as magnetic resonance imaging. In fact, many people, perhaps everyone if age for age comparisons were made, have such changes, which are often associated with either insignificant or accepted symptoms.[3]

Surgery produces irreversible change even without unwanted and unexpected effects. It hungrily consumes healthcare resources. It promulgates and reinforces the widely accepted idea that a medical model is the most efficient in managing chronic disease.[4] This philosophy is a large obstacle to the reform of health care, if not universally, certainly in the Western world.

Aggressive analgesia, closely followed by paced, functional restoration of activity and completion of tasks while addressing relevant psychosocial factors is a logical pattern of care. It is low risk and probably more efficient in its consumption of healthcare resource.[5]

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Competing interests: None declared.

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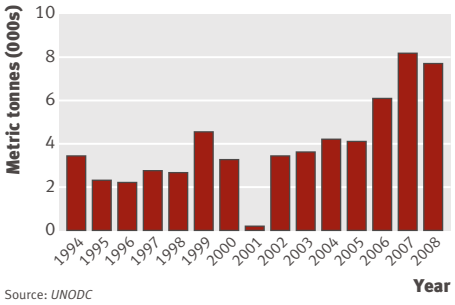

Source: *UNODC*

Opium poppy cultivation in Afghanistan[5]

WAR ON DRUGS

Is drugs policy the great game?

Why do governments maintain the pretence of possible victory in the war on drugs?

The retiring president of the Royal College of Physicians, Sir Ian Gilmore, stated in an email: "Everyone who has looked at this in a serious and sustained way concludes that the present policy of prohibition is not a success." This endorses the recent article in the *BMJ* from the Transform Drugs Policy Foundation.[1] Nicholas Green QC, chairman of the Bar Council, said that it was "rational" to consider "decriminalising personal drug use."[2]

Ken Clarke has recently noted that prison does not work. The medical evidence is that the whole war policy is a costly failure, in personal illness and national finances. Why no change in approach? Perhaps this is part of the great game. The poppy fields have proved useful in foreign policy ever since the opium wars.[3]

Occasionally the covert use of funds is exposed to the light of day. The Iran Contra affair involved drug deals. Truth is stranger than fiction. The casual dismissal of any complicated deal as being a conspiracy theory is effective, but here the charges were substantiated.[4] The Taliban ceased the opium trade in 1991, but the UK/US invasion has restored supplies and increased production (figure).[5] One explanation could be that the gains to the international and banking interests of states trump the clear warnings of their doctors and judges. Let us hope the forthcoming moves away from long term harm minimisation to short timescale cures in the United Kingdom will not serve to increase the addiction problem and its profitability.

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Competing interests: JMO has one session a week in an NHS clinic supporting people on a heroin replacement programme.

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