# Letters

## Calcium and vitamin D in preventing fractures

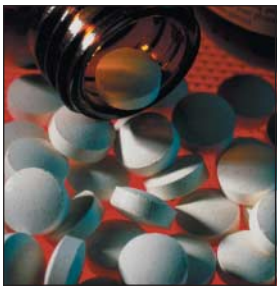
### Vitamin K supplementation has powerful effect

EDITOR—Porthouse et al conducted a good randomised controlled trial of calcium and supplementation with cholecalciferol (vitamin $D_3$) for prevention of fractures in primary care.[1] However, vitamin D and calcium are not enough.

No mention was made of the extensive research from Japan and the Netherlands, which shows that vitamin K supplementation has a powerful effect in decreasing osteoporosis and osteoporosis related fractures. Combining vitamin K, vitamin D, and calcium seems ideal.

Researchers from Osaka Medical College showed that vitamin K and vitamin D together increased bone density much better than vitamin K alone.[2]

When comparing calcium and vitamin D alone with placebo, researchers at the University of Maastricht found little benefit on bone loss. But those randomised to take vitamin K in addition to calcium and vitamin D had significantly less femoral neck bone loss after three years.[3]



STEVE HORRELL/SPL

The Yamaguchi osteoporosis prevention study showed that vitamin K alone reduced vertebral fractures by 56% compared to placebo, comparable to the benefit found from etidronate.[4]

Researchers at Hirosaki University in Japan showed that vitamin K lowered bone fractures in elderly female patients with Parkinson's by 90%.[5] The same research team showed an 86% decrease in fractures in elderly patients with Alzheimer's treated with a combination of vitamin K, vitamin D, and calcium compared with placebo.[w1]

If the medical standard became to first use vitamins D and K with calcium before using bisphosphonates or selective oestrogen receptor modulators, the public would save billions of dollars a year.

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Competing interests: None declared.

1 Porthouse J, Cockayne S, King C, Saxon L, Steele E, Aspray T, et al. Randomised controlled trial of calcium and supplementation with cholecalciferol (vitamin D3) for prevention of fractures in primary care. *BMJ* 2005;330:1003. (30 April.)
2 Ushiroyama T, Ikeda A, Ueki M. Osaka Medical College. Effect of continuous combined therapy with vitamin K(2) and vitamin D(3) on bone mineral density and coagulofibrinolysis function in postmenopausal women. *Maturitas* 2002;41:211-21.
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P+ *Additional reference on bmj.com*

### Dietary intake of calcium needs to be considered

EDITOR—In their prospective randomised trial of calcium and vitamin D supplementation, Porthouse et al found no evidence that this reduced the risk of clinical fractures in women at risk.[1] However, several aspects need closer examination before a therapeutic role can be defined.

Although these women all had risk factors for hip factors, they were highly self selected. Of 48 987 originally invited, only 3314 (7%) were eventually randomised. Median follow-up overall was only 25 months, and the design of the recruitment process means that this must have differed between the two groups. In the "unequally allocated group," followed up for longer, the evidence of a benefit of the intervention is greater.

Vitamin D concentrations were not measured to see the prevalence of insufficiency and whether this degree of supplementation was able to produce a significant improvement in values. It would also have been informative to see whether improved calcium absorption reduced parathyroid hormone concentrations.

Perhaps the most important factor is dietary calcium. The self reported estimated intake in both groups was over 1000 mg daily, which is greater than in many other studies, and both groups received literature on adequate calcium and vitamin D intake. High quality data from the US Study of Osteoporotic Fractures Research Group on dietary calcium, intestinal calcium absorption, and hip fractures, which the authors do not cite, put these findings into context.[2]

Ensrud et al studied 5452 women with a mean follow-up of 4.8 years. Some 33% had a dietary calcium intake less than 400 mg daily. In this group, when fractional calcium absorption was below the mean, the risk of hip fractures was two and a half times greater than when absorption was above average. Therefore this combination of factors (low dietary calcium intake and low fractional absorption) seems to identify women most likely to benefit from calcium and vitamin D supplements.

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1 Porthouse J, Cockayne S, King C, Saxon L, Steele F, Aspray T, et al. Randomised controlled trial of calcium and supplementation with cholecalciferol (vitamin D3) for prevention of fractures in primary care. *BMJ* 2005;330:1003. (30 April.)
2 Ensrud KE, Duong T, Cauley JA, Heaney RP, Wolf RL, Harris E, et al. Low fractional calcium absorption increases the risk for hip fracture in women with low calcium intake. *Ann Intern Med* 2000;132:345-53.

### Data are not sufficient to show inefficacy

EDITOR—The study by Porthouse et al had two major design flaws.[1] Firstly, the dose of vitamin D (800 IU per day) is subphysiological and therefore subtherapeutic. Secondly, their use of "self report" as a measure of compliance is unreliable.

The dose of vitamin D at 800 IU daily was not determined scientifically but determined arbitrarily before sufficient scientific methodology was available.[2–4] Heaney et al determined the physiological requirement of vitamin D by showing that healthy men use 4000 IU cholecalciferol daily,[2] an amount that is safely attainable with supplementation[3] and often exceeded with exposure of the total body to equatorial sun.[4]

We provided six guidelines for interventional studies with vitamin D.[5] Dosages of vitamin D must reflect physiological requirements and natural endogenous production and should therefore be in the range of 3000-10 000 IU daily. Vitamin D supplementation must be continued for at least five to nine months. The form of vitamin D should be $D_3$ rather than $D_2$. Supplements should be assayed for potency. Effectiveness of supplementation must include measurement of serum 25-hydroxyvitamin D. Serum 25(OH)D concentrations must enter the optimal range, which is 40-65 ng/ml (100-160 nmol/l).

Since the study by Porthouse et al met only the second and third of these six crite-

ria, their data cannot be viewed as reliable for documenting the inefficacy of vitamin D supplementation.

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Competing interests: AV is a researcher at Biotics Research Corporation, a drug manufacturing facility in the United States that has approval from the Food and Drug Administration.

1 Porthouse J, Cockayne S, King C, Saxon L, Steele E, Aspray T, et al. Randomised controlled trial of calcium and supplementation with cholecalciferol (vitamin D3) for prevention of fractures in primary care. *BMJ* 2005;330:1003. (30 April.)
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5 Vasquez A, Manso G, Cannell J. The clinical importance of vitamin D (cholecalciferol): a paradigm shift with implications for all healthcare providers. *Altern Ther Health Med* 2004;10:28-36.

**Author's reply**

EDITOR—Radecki notes that there is evidence for vitamin K in preventing fractures. This evidence is insufficient to recommend routine vitamin K supplementation in a primary care population. We need pragmatic trial evidence of vitamin K to test the hypothesis that it prevents fractures.

Walters observes that we recruited only a small proportion of those originally invited. Like nearly all randomised trials we can randomise only those who consent to take part. Therefore, we cannot avoid "selecting" our population. Nevertheless, our population did have a raised risk of hip fracture.

Walters refers to observational data indicating a benefit of calcium and vitamin D supplementation. We also used data from this study in our study design to identify clinical risk factors. This study was not a randomised trial. Although people with low calcium consumption and absorption may benefit from calcium and vitamin D supplementation, we would question its practical implementation at this stage in primary care, and our trial was not designed to answer that question.

We used the same calcium and vitamin D supplements as the Medical Research Council's RECORD trial, which did measure parathyroid hormone and vitamin D concentrations in a subsample. In that trial supplementation significantly changed both measures but still found no effect on the incidence of fracture.[1]

Vasquez and Cannell argue that we did not use high enough doses of vitamin D. Vitamin D supplementation may be effective at the doses they suggest, although there is no randomised evidence, with fracture end points, to support this. A recent study of high dose vitamin D injection in the south of England gave an indication of harm.[2] In terms of compliance rates our use of self report may be "unreliable," but this does not detract from our main study findings of an absence of evidence for benefit.

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Competing interests: DT has received funding from Shire and other pharmaceutical companies for research and sponsorship to attend conferences and meetings.

1 RECORD Trial Group. Oral vitamin D3 and calcium for secondary prevention of low-trauma fractures in elderly people (randomised evaluation of calcium or vitamin D, RECORD): a randomised placebo-controlled trial. *Lancet* 2005;365:1621-28.
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## Vitamin D deficiency may have role in chronic low back pain

EDITOR—The optimal management of patients with chronic low back pain remains a challenge for healthcare services, as discussed by Koes,[1] but the importance of vitamin D is not widely appreciated.

Many studies have shown the high prevalence of vitamin D deficiency in various populations. For example, 93% of 150 patients presenting to a university affiliated inner city primary care clinic in Minneapolis with persistent, non-specific musculoskeletal pain had deficient concentrations of vitamin D.[2]

Most patients (83%) attending spinal and internal medicine clinics in Saudi Arabia over six years who had experienced low back pain that had no obvious cause for more than six months had an abnormally low level of vitamin D.[3] After treatment with vitamin D supplements, clinical improvement in symptoms was seen in all of those who had a low initial concentration of vitamin D. The authors concluded that screening (of patients with chronic low back pain) for vitamin D deficiency should be mandatory.

A report in the *Medical Journal of Australia* described two patients with failed spinal fusion for chronic low back pain who were subsequently found to have severe vitamin D deficiency.[4] Both responded positively to vitamin D supplementation. The authors highlight the need for attending surgeons and physicians to be aware of the potential for vitamin D deficiency in their patients since failure to recognise this easily reversible problem may result in complications of treatment, including failure of spinal fusion surgery, additional morbidity, and the substantial costs of further surgery and hospitalisation.

All patients with persistent, musculoskeletal pain are at high risk of the consequences of unrecognised and untreated vitamin D deficiency. Current clinical guidelines for managing chronic low back pain should include assessment of vitamin D status (by measuring serum 25-hydroxyvitamin D concentrations), together with advice on appropriate vitamin D supplementation in those found to be deficient.

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Competing interests: None declared.

1 Koes BW. Surgery versus intensive rehabilitation programmes for chronic low back pain. *BMJ* 2005;330:1220-1. (28 May.)
2 Plotnikoff GA, Quigley JM. Prevalence of severe hypovitaminosis D in patients with persistent, nonspecific musculoskeletal pain. *Mayo Clin Proc* 2003;78:1463-70.
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4 Plehwe WE, Carey RPL. Spinal surgery and severe vitamin D deficiency. *Med J Austr* 2002;176:438-9.

## Participants in research

**Routine extrapolation of randomised controlled trials is absurd**

EDITOR—For more than a decade it has been an article of faith in evidence based medicine that randomised controlled trials are "best evidence" and their findings can routinely be extrapolated to clinical situations.[1] In his editorial Sackett, the founder of evidence based medicine, seeks retrospectively to reassure clinicians that this practice was justifiable, but the accompanying study by Vist et al fails to tackle the question.[2]

Most randomised controlled trials are not hypothesis testing experiments but epidemiological comparisons of rival treatments that measure the effect size of the treatment in a sample drawn from a population.[3] For the measurement to be precise, a sample needs to be sufficiently large; and to be extrapolated to the population, a sample needs to be representative (a census of all cases or a random sample).[4] Problems of small samples can be overcome by megatrials, but ever larger randomised controlled trials and meta-analyses neither overcome nor quantify the problem of biased sampling.[3 5]

To answer the question of whether a measurement from a randomised controlled trial is representative requires enumeration of the population base.[5] Since the selection biases and recruitment percentage would (if known) vary widely between specific trials, randomised controlled trials must be evaluated individually and contextually before considering extrapolation.[3]

It is absurd to imply that the routine extrapolation of treatment effect sizes is okay because measurements from randomised controlled trials are (somehow?) independent of the specific characteristics of a trial sample. Unless the principal confounders (the exact pathology and severity of disease, age, sex, precise drug and dosage, concurrent treatment, etc[2 4]) are known to be sufficiently similar, then it must be assumed that the magnitude of treatment effect size will differ between trials and clinical populations. To assert otherwise is to replace science with dogmatic theology.[1]

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Competing interests: None declared.

1 Charlton B, Miles A. The rise and fall of EBM. *Q J Med* 1998;91:371-4.
2 Sackett DL. Participants in research. *BMJ* 2005;330:1164. (21 May.)
3 Charlton BG. Fundamental deficiencies in the megatrial methodology. *Curr Contr Trials Cardiovasc Med* 2001;2:2-7.
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### Patients in clinical trials are protected by consumer regulation

EDITOR—Sackett discussed the role of participants in trials.[1] By volunteering as participants in clinical trials, patients enter a legal contract with the trial sponsor. The information sheets for patients, and a consent form signed by both parties, contain the contract terms. The contract creates legally binding obligations for both patient and sponsor.

What is supplied to the patient under the contract is additional treatment, which may be a new and beneficial drug. The patient permits valuable data to be collected and used.

The contract, counter-intuitively, seems to be a "consumer contract" (because it is a contract with standard terms, between an individual acting for purposes which are outside his business (the patient), and a supplier (the sponsor) acting in his business or profession).

All consumer contracts are governed by the Unfair Terms in Consumer Contracts Regulations 1999.[2] Those regulations, for the protection of the consumer, make fairness, plain language, and good faith legal requirements in such contracts.

The Central Office for Research Ethics Committees urges the following words on ethics committees for use in clinical trial contract documents, concerning injuries to patients in the trial: "Broadly speaking … 'the sponsor,' without legal commitment, should compensate you without you having to prove that it is at fault."[3]

Non-lawyer patients would understand that the sponsor had arranged for the certain and easy availability of compensation in the event of injury in the trial. In reality, the words used give nothing beyond the hope of an unenforceable *ex gratia* payment. So the words are not plain language; and consumer law would not permit them (because they do not meet the legal requirement of fairness and openness).

If the contract actually is not a consumer contract, then it must be said that the public has less legal protection when it volunteers for a clinical trial than when it buys double glazing.

Survivors of serious adverse events can only hope that clinical trial contracts are indeed governed by consumer protection legislation.

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Competing interests: DRL is a prospective patient volunteer in a clinical trial.

1 Sackett DL. Participants in research. *BMJ* 2005;330:1164. (21 May.)
2 Office of Fair Trading. Unfair terms in contracts—what is an unfair term? www.oft.gov.uk/Consumer/Unfair+terms+in+contracts/what+are+unfair+terms.htm (accessed 28 Jun 2005).
3 Central Office for Research Ethics Committees. Guidelines for researchers. para15. What if something goes wrong. February 2001. www.corec.org.uk/applicants/help/docs/Guidance_on_Patient_Information_Sheets_and_Consent_Forms.doc (accessed 5 June 2005).

### Contract to run a trial should include patient-researchers in steering groups

EDITOR—In response to Sackett's editorial on participants in research Laurence writes that the patient information sheets, and a consent form signed by both parties, contain the contract terms for participants in research (previous letter).[1] They may legally do so, but the quality of information sheets for patients is suboptimal,[2] patients' understanding of them is poor, and their purpose misconstrued. The parties to the contract are unequal, and patients' rights and responsibilities are poorly understood. Public understanding of the research process is poor; the purpose of reducing uncertainties about the effects of treatment (both old and new) by the risk limiting method of trials is only hazily appreciated.

The notion of adhering to the trial's objectives for its duration to ensure that robust data are produced demands a jointly drawn-up contract from the outset of the trial. In other words, the contract to run a trial should include knowledgeable or trained patient-researchers as members of the working group, the steering committee, and the data monitoring and ethics committee, working together with health professional members. Sufficient evidence is now available about the value of patients' involvement in research,[3] and models are available to endorse the benefits (and indicate the drawbacks) of this type of working.[4 5]

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Competing interests: None declared.

1 Sackett DL. Participants in research. *BMJ* 2005;330:1164. (21 May.)
2 Lees N, Dixon-Woods M, Young B, Heney D, Thornton H. CaTLET: evaluation of information leaflets for patients entering cancer trials. *Psycho Oncol* 2001;10:266.
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## Surveying the literature from animal experiments

### Systematic review and meta-analysis are important contributions

EDITOR—We agree with Lemon and Dunnett that better methods of surveying the literature on animal experiments are needed,[1] but we do not share their confidence in the utility of non-systematic reviews.

In clinical trials, systematic review and meta-analysis have made important contributions to our understanding of sources of bias, and the quality of clinical trials has improved as a result. We believe that the same approach can be used to increase our understanding of sources of bias in animal experiments, again leading to improvements in study quality.

Not to publish negative results from clinical trials is widely accepted as unethical, because this may lead to the study being unwittingly repeated by other investigators, exposing trial participants to risk of drug side effects with no prospect of benefit. We consider that non-publication of data from animal studies is equally unethical.

It is argued that tight control of experimental conditions minimises variance and allows for small sample sizes, but most animal studies are still hopelessly underpowered. Systematic review has allowed the analysis of sample size in studies of FK506 in animal models of stroke; the observed variance suggests that 65 animals per group would be needed to give an 80% chance of detecting an improvement in outcome of 20%.[2] In fact, the largest study reported 16 animals per group and the average was eight animals per group. Performing underpowered studies is as unethical in animal studies as it is in human studies.

Whatever the merits of animal experiments, for many diseases, including stroke, the benefits seen in animal models have been lost in translation. Finding out the reasons for this discrepancy is a matter of some urgency, and a problem to which all available tools—including systematic review—should be brought to bear.

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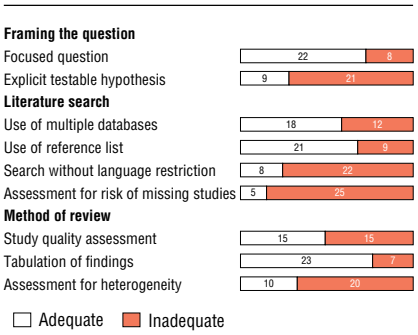
Competing interests: MRM and IR have conducted systematic reviews of animal experiments.

1 Lemon R, Dunnett SB. Surveying the literature from animal experiments. *BMJ* 2005;330:977-8. (30 April.)
2 Macleod MR, O'Collins T, Horky LL, Howells DW, Donnan GA. Systematic review and metaanalysis of the efficacy of FK506 in experimental stroke. *J Cereb Blood Flow Metab* 2005; 25:713-21.

*Details of the other three authors are on bmj.com*

### Avoidance of bias is objective of systematic reviews, not meta-analysis

EDITOR—We assessed the methodological quality of reviews of animal studies identified using a carefully designed search term combination in Medline and Embase (1996-2004). We also examined bibliographies of known reviews and contacted experts. In




Methodological features of reviews of animal research (1996-2004). Data presented as 100% bars with numbers of studies

total, 30 reviews summarised studies in live animals, which measured laboratory variables or examined treatment effects, identified from search of a publicly available resource. These reviews often lacked methodological features (figure), which increased the risk of biased inferences. We therefore concur with Lemon and Dunnett, that better methods of surveying the literature on animal experiments are needed.[1]

However, we do not understand their objection to the use of systematic reviews. Their argument seems to be that it is difficult to combine data from different studies in animal research. Perhaps it needs to be emphasised that meta-analysis is not the objective of a systematic review. The proportion of reviews of animal research that would benefit from meta-analytic techniques is unknown. Among the reviews of animal studies we assessed, 12/30 (40%) used a statistical combination of individual results. The reviews usually did not assess key features in assessing suitability of combining results statistically—for example, exploration of heterogeneity between studies, assessment of study validity, and the risk of missing studies.

These deficiencies interfere with gauging the strength of any inferences. Reviews earn the adjective systematic by use of explicit methods to minimise bias at every step of the reviewing process including literature search, study selection, critical appraisal, and data synthesis. The need for rigour when reviewing animal research is undeniable and systematic review method[2] provides a sound strategy for conducting such reviews.

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1 Lemon R, Dunnett SB. Surveying the literature from animal experiments. *BMJ* 2005; 330: 977-8. (30 April.)
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## *Horizon*: Does the MMR Jab Cause Autism?

### Has the fat lady sung then?

EDITOR—MacAuley, in his review of the BBC *Horizon* programme, Does the MMR Jab Cause Autism?,[1] repeats what I assume was the programme's determination—namely, that there was nothing in the measles, mumps, and rubella (MMR) and autism story, and that many children have suffered unnecessarily from the outcome of the widespread publicity.

MacAuley concludes that parents are more likely to have their children immunised if they see what happens when others are ill. This witnessing of suffering presumably alludes to graphic images of children who have contracted some of the diseases from which they might have been protected had they been vaccinated against them. I suspect that the only serious outcomes from non-vaccination would be through infection with measles. Mumps and rubella in children are unlikely to produce images that would be considered graphic enough for television.

Ironically, he uses his vision of how parents might react by referring only to the vision of reality when disease strikes. He seems to forget that many parents have also witnessed the real experience of seeing children who have had a severe reaction to vaccination, and this of course extends beyond MMR.

He should consider that for many years, the medical profession consistently refuted the idea that the vaccination for diphtheria, tetanus, and pertussis (DTP) could cause brain damage and its consequent disability. In addition he should reflect that government has paid out sums as "awards" to over 1000 children who have been damaged by vaccines.

The world might have moved on, and there are more topical medical controversies, but this story of the MMR vaccination and its problems has not been concluded.

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Competing interests: AC is the father of a vaccine damaged daughter.

1 MacAuley D. Horizon: Does the MMR jab cause autism? *BMJ* 2005;330:1335. (4 June.)

### We cannot win

EDITOR—For measles, mumps, and rubella (MMR) and autism, epidemiology is dismissed.[1] For power lines and cancer, epidemiology is king.[2,3] As MacAuley wrote, many parents might have found such [anecdotal] arguments seductive.

This is the problem. Personal experience speaks loudly. It is difficult to deny the evidence of one's own eyes. The "MMR dissenters" start from the proved fact that MMR causes autism (because they've seen it); the "electromagnetic fields cancer advocates" know power lines cause cancer (because they've seen it). All later evidence is interpreted on the basis of that experience. The rapid responses to the articles in the issue of 4 June will be witness.

As Watts writes in favour of perspective: "In 2002, according to the Child Accident Prevention Trust, more than 36 000 children were hurt in road accidents and around 200 were killed … five cases annually of childhood leukaemia may be associated with power lines."[3] But perspective is precisely what is rejected by personal experience: so we have illogical campaigns to uproot speed cameras, to move pylons, and to give single vaccines.

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Competing interests: None declared.

1 MacAuley D. Horizon: Does the MMR jab cause autism? *BMJ* 2005;330:1335. (4 June.)
2 Draper G, Vincent T, Kroll ME, Swanson J. Childhood cancer in relation to distance from high voltage power lines in England and Wales: case-control study. *BMJ* 2005;330:1290. (4 June.)
3 Watts G. Power to confuse. *BMJ* 2005;330:1293 (4 June.)

## Anger is the weed, hate is the tree

EDITOR—I understand what mountain the new wind is blowing from on reading the editorial by Davies and Delamothe on rooting out weeds in bmj.com's rapid response garden.[1] It will be interesting to observe the smoke coming from placid chimneys and billowing in different directions. Weeds are not candles in the wind.

What is a weed, however? A plant whose virtues have not yet been discovered? A flower in disguise? An unloved flower?

Greater minds than can be paid for by the *BMJ* have not been able to separate the weeds from the flowers.

Personally, I find some of the weeds on bmj.com very interesting and, to me, a garden without weeds is like a church without sinners.

In closing, may I remind our brave editors that weeds tend to grow in any soil, a characteristic conducive to genetic superiority through the abundance of available nutrients.

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Competing interests: None declared.

1 Davies S, Delamothe T. Revitalising rapid responses. *BMJ* 2005;330:1284. (4 June.)