LETTERS

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HRT AND CARDIOVASCULAR EVENTS

Trial does not change results of Cochrane review

We considered whether to include Schierbeck and colleagues' trial in our 2012 Cochrane review of long term hormone therapy for perimenopausal and postmenopausal women.[1 2]

However, it did not meet the inclusion criteria for our review because it had no placebo control group. We also thought that the open label design may have influenced the behaviour of those taking the hormone therapy. Furthermore, we had serious concerns about the use of a composite outcome that was not described in the original study protocol in 1990.

Our review concluded that hormone therapy is not indicated for primary or secondary prevention of cardiovascular disease and that there are insufficient data to assess the risk of long term use in perimenopausal women or postmenopausal women under 50 years of age. These conclusions remain unchanged.

Jane Marjoribanks editor, Cochrane Menstrual Disorders and Subfertility Group
j.marjoribanks@auckland.ac.nz
Cindy Farquhar postgraduate professor of obstetrics and gynaecology
Helen Roberts associate professor of women's health
Anne Lethaby editor, Cochrane Menstrual Disorders and Subfertility Group, University of Auckland, Auckland 1142, New Zealand

Competing interests: None declared.

1 Schierbeck LL, Rejnmark L, Tofteng CL, Stilgren L, Eiken P, Mosekilde L, et al. Effect of hormone replacement therapy on cardiovascular events in recently postmenopausal women: randomised trial. *BMJ* 2012;345:e6409. (9 October.)
2 Marjoribanks J, Farquhar C, Roberts H, Lethaby A. Long term hormone therapy for perimenopausal and postmenopausal women. *Cochrane Database Syst Rev* 2012;7:CD004143.

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Safety couldn't be investigated

There are several concerns about the robustness of Schierbeck and colleagues' findings.[1] The open label design means that the trial could not control for placebo effect, and knowledge of active treatment could have led to differential ascertainment of outcomes.

The composite outcome of mortality, myocardial infarction, and heart failure was not prespecified in the design paper, which does however note the lack of statistical power for effects on breast cancer and coronary heart disease.[2] We are not aware of an effect of hormone therapy on heart failure, which is a curious choice of outcome.

Our concern about outcomes ascertainment is heightened by the low event rates for myocardial infarction compared with those found in the Women's Health Initiative (WHI) data for younger women.[3] The low proportion of myocardial infarctions compared with strokes and heart failure, and the high number of cardiovascular deaths compared with cases, are unexpected. Point estimates from the WHI suggests that oestrogen plus progestin increases the risk of stroke, breast cancer, and venous thromboembolism compared with placebo and might decrease total mortality risk in younger women. Non-significant hazard ratios for myocardial infarction in WHI were 1.29 for women aged 50-59 years and 0.88 for women less than 10 years into the menopause. Hence, only this study's findings for total mortality are consistent with the much larger WHI trial. Finally, combining results from the oestrogen plus progestin and oestrogen alone intervention arms is inappropriate, especially for outcomes such as breast cancer, for which WHI intervention effects were widely divergent.

This study had insufficient power to investigate the safety of menopausal hormone therapy. The findings add further questions about the study design and results.

Jacques E Rossouw medical officer, National Heart, Lung, and Blood Institute, Bethesda, MD, USA
rossouwj@nih.gov
JoAnn E Manson professor of medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA
Andrew M Kaunitz professor of obstetrics and gynecology, University of Florida College of Medicine-Jacksonville, FL, USA
Marcia L Stefanick professor of medicine , Stanford University School of Medicine, Stanford, CA, USA

Competing interests: None declared.

1 Schierbeck LL, Rejnmark L, Tofteng CL, Stilgren L, Eiken P, Mosekilde L, et al. Effect of hormone replacement therapy on cardiovascular events in recently postmenopausal women: randomised trial. *BMJ* 2012;345:e6409. (9 October.)
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Was composite outcome specified in original protocol?

It is unclear whether Schierbeck and colleagues' primary composite outcome was defined in the original protocol.[1] In other words, is their analysis exploratory or confirmatory? The main question is whether the results should lead to changes in clinical practice. Schierbeck has advocated for the safety of hormone therapy,[2], but we believe that the study results should be interpreted with caution and put in the context of previous findings.

In their response Schierbeck and colleagues state that "the outcomes in the present study were secondary outcome measures in the original protocol" and "the primary endpoint of the present study was defined in the protocol as safety measures for cardiovascular events."[3]

A previous publication on the design of the Danish Osteoporosis Prevention Study mentions coronary heart disease (defined as angina and myocardial infarction) but not the composite outcome.[4] The published trial of fracture outcomes uses the term "cardiovascular incidents" but does not describe what this means.[5] Nothing is stated on clinicaltrials.gov about cardiovascular outcomes. The protocol we received from Schierbeck (1995 version, patients enrolled 1990-93) states that myocardial infarction would be assessed as a side effect but not specified as a primary or secondary outcome in the data analysis. The statistical analysis section states that "incidence of ischaemic heart disease" will be evaluated, without describing what this means.

We found no information on the outcome, admission to hospital for heart failure, or the composite outcome in previous publications or the provided protocol. Can the authors please clarify whether the composite outcome of death, admission to hospital for heart failure, and myocardial infarction was defined before the trial started enrolling in 1990 or whether it was defined afterwards?

Jeppe Schroll researcher
Andreas Lundh researcher, Nordic Cochrane Centre, 2200 Copenhagen N, Denmark

Competing interests: None declared.

1 Schierbeck LL, Rejnmark L, Tofteng CL, Stilgren L, Eiken P, Mosekilde L, et al. Effect of hormone replacement therapy on cardiovascular events in recently postmenopausal women: randomised trial. *BMJ* 2012;345:e6409. (9 October.)
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Age hypothesis does not stand

Schierbeck and colleagues suggested that "initiation of hormone replacement therapy in women early after menopause significantly reduces the risk of the combined endpoint of mortality, myocardial infarction, or heart failure."[1] They invoke the age hypothesis—that women in the Women's Health Initiative who were well past the menopause responded differently from symptomatic menopausal women with respect to cardiovascular risk.

All such arguments ignore the double blind placebo controlled studies of such symptomatic women. A synthesis of such studies (including unpublished ones—a process resisted by the manufacturers) was published in the *BMJ* and the *Lancet* and later summarised in the *BMJ*.[2-4] Evidence from a synthesis of some 200 efficacy studies submitted for licensing of hormone therapy products strongly suggests that the risk is the same in both sets of women. Purveyors of the age hypothesis need to take on board all of the evidence, especially from well controlled randomised controlled trials.

Klim McPherson visiting professor of public health epidemiology, New College, Oxford OX1 3BN, UK klim.mcpherson@new.ox.ac.uk

Competing interests: None declared.

1 Schierbeck LL, Rejnmark L, Tofteng CL, Stilgren L, Eiken P, Mosekilde L, et al. Effect of hormone replacement therapy on cardiovascular events in recently postmenopausal women: randomised trial. *BMJ* 2012;345:e6409. (9 October.)
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Authors' reply

Although randomisation is an important part of any trial, we acknowledge that performance and detection bias exist in open label trials,but these biases are less important when endpoints are driven by mortality. Furthermore, no trial of hormone replacement therapy (HRT)—including the Women's Health Initiative—conducted in women with a uterus is completely blinded. [1] Thus, our results are probably as valid as other randomised HRT trials. In the Danish Osteoporosis Prevention Study (DOPS), randomised treatment was stopped in 2002 when it was suggested that HRT had negative effects on cardiovascular health. This should have resulted in a higher detection rate of cardiovascular disease in the subsequent post-randomisation years of follow-up. However, this was not the case.

In the original protocol cardiovascular safety was defined as safety measures. In the present study the primary focus was on cardiovascular outcome; this was defined before data analysis and only hard endpoints that are associated with poor prognosis were included. These were all cause mortality, acute myocardial infarction, and hospital admissions for heart failure because these are also events validated in national Danish registers; thus, this is not a weakness but a strength.[2]

We agree that power is low to detect breast cancer, but power is not a problem in relation to the significant findings driven by all cause mortality. Also, we did analyse the data separately for estradiol plus sequential norethisterone acetate and estradiol alone (labelled as intact uterus and hysterectomy in figs 3-6).

Thanks to McPherson for bringing smaller randomised controlled trials to our attention. We did not intend to discuss the vast HRT data, but these studies are relevant to our discussions.

DOPS provides long term longitudinal randomised trial data in a cohort of the very women who are normally treated, for which no data previously existed. Thus, it directly provides information on the long term effects of HRT in close proximity to menopause, especially with regard to cardiovascular disease, total mortality, and to some degree cancer. Finding significant effects in small groups underscores the clinical relevance for efficacy in reducing cardiovascular disease and total mortality.

Louise Lind Schierbeck registrar, Department of Endocrinology, Hvidovre Hospital, 2650 Hvidovre, Denmark louise.schierbeck@gmail.com
Lars Køber professor, consultant, Department of Cardiology, Rigshospitalet, Copenhagen, Denmark
Jens-Erik Beck Jensen associate professor, consultant, Department of Endocrinology, Hvidovre Hospital, 2650 Hvidovre, Denmark

More extensive answers are at www.bmj.com/content/345/bmj.e6409/rr/612351.

Competing interests: LLS, LK, and J-EBJ are authors of the discussed research article.

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Press release was misleading

Schierbeck and colleagues are correct that their paper made it clear that it was a secondary analysis of a trial done to test a different hypothesis.[1 2]

However, whoever wrote the *BMJ* press release, which includes the following sentence, did not.

"So authors from Denmark carried out a randomised trial over 10 years with additional six years of follow-up to establish whether HRT can reduce cardiovascular risk if it is started early after menopause."[3]

This is misleading and has encouraged naive journalists to write headlines suggesting that the cardiovascular risks of hormone therapy have been proved to have been false. Even Schierbeck and colleagues would surely not claim this.

Jim G Thornton professor of obstetrics and gynaecology, University of Nottingham, Nottingham NG5 1PB, UK jim.thornton@nottingham.ac.u

Competing interests: None declared.

1 Schierbeck LL, Køber L, Beck Jensen JE. [Electronic response to Schierbeck LL, et al. Effect of hormone replacement therapy on cardiovascular events in recently postmenopausal women: randomised trial.] *BMJ* 2012. www.bmj.com/content/345/bmj.e6409/rr/612351.
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CONTENT AREA EXPERTS AS AUTHORS

One disease expert is mandatory

Gøtzsche and Ioannidis recommended that "teams performing systematic reviews should not include content area experts as authors" but must include experts in systematic review.[1] Given the influence of systematic reviews on clinical guidelines and funding, patients need reviewers experienced with the condition and treatment. A general medical education may be adequate in common diseases. Expertise in systematic review methods is essential in diseases with large numbers of trials, but just having a medical degree, even with such expertise, is not adequate for rare serious conditions, for surgical treatments, and for complex interventions.

For example, reviewing therapeutic hypothermia for neonatal hypoxic-ischaemic brain injury requires knowledge of diagnosis of brain injury, cooling methods, and

neurodevelopmental assessment of infants. If treatment aims to prevent "serious disability," it is vital to understand the definition of serious disability in each trial before deciding whether outcomes can be meta-analysed together.

Reviewers of treatment of intraventricular haemorrhage need knowledge of neuroimaging, neurosurgical procedures, and neurodevelopmental outcomes.

Developmental care involves multiple interventions to provide stimulation, reduce stress, and help parental bonding. These interventions may have similar names but be different or have different names but be similar.

Experts in systematic reviewing cannot on their own apply review software to neonatal subjects and produce a review that helps clinicians.

The Cochrane Collaboration has greatly improved treatment decisions for millions of patients, and the founders were right in insisting that at least one reviewer must be an expert in the condition and treatment being reviewed.

Andrew Whitelaw professor of neonatal medicine, Department of Neonatal Neuroscience, University of Bristol, St Michael's Hospital, Bristol BS2 8EG, UK
andrew.whitelaw@bristol.ac.uk

Competing interests: AW has undergone Cochrane systemic review training and has published five Cochrane reviews.

1 Gøtzsche PC, Ioannidis JPA. Content area experts as authors: helpful or harmful for systematic reviews and meta-analyses? *BMJ* 2012;345:e7031. (1 November.)

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VITAMIN D DEFICIENCY

British Paediatric and Adolescent Bone Group's statement

BIOPHOTO ASSOCIATES/SPL

Because of the lack of well designed studies on vitamin D and health,[1] the British Paediatric and Adolescent Bone Group has produced a position statement based on current expert opinion. This statement is supported by the British Society of Paediatric Radiology and child protection and nutrition committees of the Royal College of Paediatrics and Child Health.

There is currently considerable clinical and research interest in vitamin D deficiency. Definitions of a sufficient vitamin D concentration vary across clinical guidelines. This causes confusion and may influence clinical decision making in children and adolescents.

The British Paediatric and Adolescent Bone Group's current opinion is that the definition of vitamin D deficiency should relate only to vitamin D's effect on the skeleton. Deficiency should be a plasma concentration of 25 hydroxyvitamin D of less than 25 nmol/L (10 ng/mL), with insufficiency being 25-50 nmol/L and sufficiency a concentration greater than 50 nmol/L. We generally use these thresholds in practice, although we recognise that the evidence base in children and adolescents is limited.

In infants with unexplained fractures, unless conventional radiography and biochemistry (abnormal blood concentrations of calcium, phosphate, alkaline phosphatase, or parathyroid hormone) provide evidence of rickets, 25 hydroxyvitamin D is not implicated.

It is important that people at risk of vitamin D deficiency take vitamin D supplements, as recommended by the chief medical officers for the UK.[2] These include all pregnant or breastfeeding women and all infants and children from the age of 6 months to 5 years. We also recommend that exclusively breastfed infants receive vitamin D supplements from soon after birth.

Paul Arundel consultant in paediatric metabolic bone disease and secretary, British Paediatric and Adolescent Bone Group, Sheffield Children's NHS Foundation Trust, Sheffield S10 2TH, UK p.arundel@sheffield.ac.uk
S F Ahmed Samson Gemmell chair of child health, School of Medicine, University of Glasgow, Glasgow, UK
J Allgrove consultant paediatric endocrinologist, Great Ormond Street Hospital, London. UK
N J Bishop professor of paediatric bone disease, Academic Unit of Child Health, University of Sheffield, Sheffield, UK
C P Burren consultant paediatric endocrinologist and diabetologist, Bristol Royal Hospital for Children, Bristol, UK
B Jacobs consultant paediatrician, Royal National Orthopaedic Hospital, Stanmore, UK
M Z Mughal consultant in paediatric bone disorders, Royal Manchester Children's Hospital, Manchester, UK
A C Offiah HEFCE clinical senior lecturer, Academic Unit of Child Health, University of Sheffield, Sheffield, UK
N J Shaw consultant paediatric endocrinologist, Birmingham Children's Hospital, Birmingham, UK

Competing interests: None declared.

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HIRSCHSPRUNG'S DISEASE

Identifiable genetic causes

As Arshad and colleagues point out, some patients with Hirschsprung's disease will have a family history.[1] Many patients will also have an identifiable genetic cause.

For example, *RET* mutations may account for up to 41% of non-syndromic Hirschsprung's disease and half of all familial disease. There are further monogenic causes for both non-syndromic and syndromic Hirschsprung's disease. Syndromic causes include neurofibromatosis type 1, Smith-Lemli-Opitz syndrome, and multiple endocrine neoplasia type 2 (caused by *RET* mutations), and around 12% have a genetic cause. These include Down's syndrome, but also several small chromosome deletions.[2] It may be helpful for the family and clinicians to know who and whose children are at risk of Hirschsprung's disease. Your local regional genetics service would be happy to discuss appropriate investigations.

Francis H Sansbury academic clinical fellow/specialty registrar in clinical genetics, Peninsula Clinical Genetics Service, and Department of Molecular Genetics, Royal Devon and Exeter NHS Foundation Trust, Royal Devon and Exeter Hospital (Heavitree), Exeter EX1 2ED, UK fsansbury@nhs.net
Sian Ellard professor of human molecular genetics, University of Exeter Medical School, Royal Devon and Exeter Hospital (Wonford), Exeter EX2 5DW, UK
Charles Shaw-Smith consultant clinical geneticist
Peter Turnpenny consultant clinical geneticist, Peninsula Clinical Genetics Service, and Department of Molecular Genetics, Royal Devon and Exeter NHS Foundation Trust, Royal Devon and Exeter Hospital (Heavitree), Exeter EX1 2ED, UK

Competing interests: None declared.

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Surgery does not always work

Arshad and colleagues highlight that, although rare, late presenting Hirschsprung's disease in children can result in serious morbidity.[1] However, their statement that most patients with Hirschsprung's disease have "near normal anorectal function after surgery" is misleading.

A recent Finnish population based cross sectional study of adults with Hirschsprung's disease found that only 47% had a normal bowel function score,[2] a result that is consistent with English studies using the same scoring system.[3] Furthermore, up to 48% of adults report occasional soiling, with 14% experiencing potentially socially disabling faecal incontinence.[2]

Although most patients with Hirschsprung's disease learn to actively manage their bowels and report adequate quality of life scores, this disease has lifelong implications, both medically and socially.

David Wilkinson speciality registrar in paediatric surgery d.wilkinson1@mac.com
Simon Kenny consultant paediatric surgeon, Alder Hey Children's Hospital NHS Foundation Trust, Liverpool L12 2AP, UK

Competing interests: None declared.

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CAN SUICIDE BE PREVENTED?

Role of mental illness

SANTIAGO CARUSO/GUERRILLA ZOO

Is Kamerow as guilty of lacking specificity and focus as the US surgeon general he criticises?[1] "Psychiatric disorders" may be present in around 90% of people who kill themselves, but what does this actually mean?

Meta-analysis of North American autopsies of patients with psychiatric disorders shows that substance misuse, present in 40%, predominates.[2] However, this includes "harmful use" of substances, including binge drinking, and even simple alcohol intoxication; no sensible person would consider these phenomena psychiatric disorders.

Mental illness, such as depressive disorder (34%) and schizophrenia (4%), is represented,[2] but even these figures warrant closer observation. Depressive disorder is hugely overdiagnosed in life,[3] and this is probably true in death. Recent prescription of antidepressants or retrospective reports of insomnia, lethargy, and low mood from family members do not automatically equate to a true diagnosis of depressive disorder. Yet the pragmatic methodology of psychological autopsy studies dictates precisely that.

In reality, a small minority of people who commit suicide are mentally ill. Most are people encountering difficult life circumstances, to whom suicide seems a logical solution at the time; acts are often carried out impulsively and while intoxicated. This is borne out by the close association between suicide rates over the past century and international economic trends, exemplified by the recent increase in the US during the current recession.[4]

We must not be complacent in managing people who are unwell, but any suicide reduction policy that targets psychiatric or primary care services is bound to fail, as in the US. Successful policies in the UK, such as restriction of analgesic sales,[5] have restricted access to means.

The US national suicide rate will not dramatically fall until radical gun law is introduced. Kamerow may be waiting some time.

Rich Braithwaite consultant psychiatrist, Isle of Wight NHS Trust, St Mary's Hospital, Newport PO30 5TG, UK richard.braithwaite@iow.nhs.uk

Competing interests: None declared.

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CHANGES TO THE GP CONTRACT

Problems date back to 2004

The time for action on the GP contract was in 2004.[1] In exchange for quick and easy cash from the Quality and Outcomes Framework (QOF), the core General Medical Services (GMS) contract was left vulnerable to unilateral imposed change, did not specify core duties, and did not contain inflation proofing.

Clinical commissioning groups have brought no extra work or legal requirements, there is no contractual obligation (yet) to do any of this work, and those who volunteer are either well remunerated or must be enjoying the avalanche of paperwork. The BMA could have simply advised doctors not to engage in deckchair rearranging.

The failure to ensure that unfunded changes to the QOF could not be imposed unilaterally leads back to the decisions made in 2004; the structural soundness of the contract was neglected in the scramble for John Reid's fiver.

It was predicted: "We have got the pigs in the pen, now watch us change the shape of the pen."[2]

The editorial ignores some of the good sides of the proposals,[1] like the long overdue equalisation of funding for core services. This should extend not only to personal medical services, but also to alternative provider medical services and primary care trust medical services, which have enjoyed disproportionate amounts of funding.[3] Same pay for the same work will be a welcome relief for GMS practices.

Hendrik J Beerstecher general practitioner, Canterbury Road Surgery, Sittingbourne ME10 4JA, UK hendrick.beerstecher@nhs.net

Competing interests: HJB is a GP in the only practice in Kent not affiliated with a clinical commissioning group (orphan practice).

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REVALIDATION AND APPRAISAL

Still won't catch poor doctors

Thank you, Nigel Hawkes, for your article on revalidation.[1] The press has claimed that revalidation will safeguard the public from poorly performing doctors by using five yearly checks. As a GP with an interest in education and assessment I share your concern that the public has been misled.

The revalidation process will centre on self analysis by appraisees, and the potential for collusion with appraisers is immense. Annual appraisal in its current format is unreliable and not reproducible, and is therefore unfair to appraisees and the public. Five appraisals will equal one revalidation, but five times nothing is still nothing.

The educational tools we use for formative analysis and reflection are being railroaded into a use for which they were not intended.

There are alternatives. An example might be the external review of randomly selected medical records. Appraisers, not appraisees, need to survey patients and peers. Assessment of knowledge, skills, and attitudes might then be more valid.

Wakeford wrote the following nine years ago[2]: "In its revalidation guidance for doctors the General Medical Council says that one good professional comparison is with airline pilots . . . Imagine two airlines, whose pilots' revalidation arrangements are on the following bases:

- Airline A—flight simulator skills tests, including rarely met but crucial challenges; a thorough medical examination
- Airline B—informal personal development plans, agreed privately with a colleague, maybe of their choice; cabin crew and passenger surveys of the gentleness of their landings and the clarity of their communications; a self declaration of sobriety, health, and honesty.

. . . With which airline would you travel?"

Revalidation will not reliably detect poorly performing doctors. Perhaps those who tell the public that this process is fit for purpose should hesitate before ticking the probity box.

Leslie Lowenthal general practitioner, Downland Practice, Chieveley RG20 8UY, UK leslielowenthal@doctors.org.uk

Competing interests: None declared.

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