letter. I don't know Peter Sturbanch but I did like his column in the Lancet. I also think he is right and although "the concept of cholesterol levels being a risk factor will remain a basic believable one" (your words) this does not imply that all diet propaganda has anything to do with science. There has been a time that diabetes was treated by limited sugar intake; it was just correlation. The world at large now suffers from the cholesterol and margarine lobbies. There is no proof what so ever that changing cholesterol levels changes one's risk for coronary artery disease. I trust that you will admit that at least there is conflicting evidence regarding "killer love"

I got a letter from Henry Blackburn and even he starts to waver. The risk factor concept is just what it says it is: a concept, one ex bored and exhausted for women. It will turn out to be the medical mistake of this century. I hope you and I will live to see it happen.
I don't mind guidelines but they are not presented as such, they are presented as commandments. "Thou shall or thou shall not." They are not presented as advice but as prescriptions, then compare our patients and then interfere with the trustworthiness of our professions. I can give you examples. This is enough. I probably was not careful enough with my answer but you know me. Nobody is more in doubt about what Fritz Perls says than Fritz Perls himself. Thus take me "cum grano salis".

come over & took what pictures. What about our X-mas photograph of this year? By the way I got myself a new camera, the Nikon N90.
With love and affection and all our best wish for your health and happiness in 1994. Your letters are always spiritual manna for me.

Fritz

P.S. Can you arrange a meeting with Henry Blackburn on March 7? For lunch may be.

The epidemiology of errors

Confounded be your strife . . .

(1 Henry VI, act iv)

"A good deal of scientists, many of them in the professional fields of epidemiology and public health, have never learned how to avoid waffling when yes or no are not available and the only correct answer is, I don't know".[1] Lewis Thomas was referring to risk-factor epidemiology. By the misuse of language and logic, observed associations are presented as causal links.[2] Electric blankets are said to increase the risk of breast cancer; coffee drinking in men reduces their risk of developing breast cancer; and so on.

In clinical medicine, strict standards apply for evaluation of therapies, and anything short of randomised double-blind trials is frowned upon as unreliable evidence. By contrast, risk-factor epidemiology relies on case-control or cohort studies, without rigorous standards of design, execution, and interpretation,[3,4] even though such studies are susceptible to at least fifty-six different biases.[5] Observed relative risk (or odds ratios) in such studies are generally so small that even if their confidence intervals do not include unity, they are well within the limits of errors caused by unavoidable biases and confounding, which cannot be rectified by statistical manipulation. How should one remedy this state of affairs—bigger studies, better measurement of risk factors, more complex statistics?

Statistics are no cure for the faulty paradigm of risk-factor epidemiology.[6] Even randomised intervention trials of coronary heart disease prevention, with over one million man-years of data, have failed to convince many impartial observers (called sceptics by the believers) that risk-factor epidemiolgy is on the right track. Phillips and Davey Smith[7] suggest that a major revision of how observational epidemiological studies are designed is now in order: instead of larger samples, more measurements of risk factors would readily compensate for a reduced sample size. But would concern about measurement imprecision solve anything? As the Irish country saying goes, you cannot make a pig grow by weighing him. An example used by Phillips and Davey Smith is the case in point: can repeated administration of dietary questionnaires tell us anything about the aetiology of cancer? The purpose of risk-factor epidemiology is to provide testable hypotheses and not to narrow confidence intervals of spurious associations.

In politically sensitive areas—for example, the alleged harm of passive smoking—poor data are manipulated to reach a foregone conclusion, even if it means arbitrary adjustments, selective meta-analysis, 90% confidence intervals, and one-tailed statistics, to save a relative risk of 1·19 from being insignificant.[8] This may be a special case when corrupt science moves from mandated conclusion back to selected data in order to reach the mandated conclusion.[9] But there is also a general malaise in risk factor epidemiology, the symptoms of which fit the description of pathological science.

In 1953, the Nobelist Irving Langmuir gave a celebrated colloquium on pathological science at General Electric's Knolls Atmoc Power Laboratory; the transcript is available.[10] Characterising "the science of things that aren't so", Langmuir mentioned, among the common denominators, (*a*) the study of effects whose magnitude remains close to the limits of detectability so that many measurements are necessary to establish statistical significance; (*b*) the magnitude of the effect is substantially independent of the intensity of the "cause"; (*c*) the maximum effect is observed with a causative agent of barely detectable intensity; (*d*) there are claims of great accuracy; and (*e*) criticisms of such findings are brushed aside by ad hoc excuses.

Risk-factor epidemiology will need more than better or more frequent measurements to establish itself as a science.

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1 Thomas L. An epidemic of apprehension. *Discover* 1983; November: 78–79.

2 McCormick JS. The abuse of language and logic in epidemiology. *Persp Biol Med* 1992; 35: 186–87.

3 Mayes LC, Horwitz RI, Feinstein AR. A collection of 56 topics with contradictory results in case-control research. *Int J Epidemiol* 1988; 17: 680–85.

4 Angell M. The interpretation of epidemiological studies. *N Engl J Med* 1990; 323: 823–25.

5 Sackett DL. Bias in analytic research. *J Chron Dis* 1979; 32: 51–63.

6 Vandenbroucke JP. Statistical modelling: the old standardisation problem in disguise? *J Epidemiol Commun Med* 1989; 43: 207–08.

7 Phillips AN, Smith GD. The design of prospective epidemiological studies: more subjects or better measurements? *J Clin Epidemiol* 1993; 46: 1203–11.

8 Gori GB. Passive smoking. *Lancet* 1993; 341: 965.

9 Luik JC. Pandora's box: the dangers of politically corrupted science for democratic public policy. *Bostonia* 1993; Winter: 50–60.

10 Langmuir I. Pathological science. *Physics Today* 1989; 42 (10): 36–48.

and disorders that secondarily affect the connective tissue. Each group is described with its history, clinical picture, molecular defects, and genetic background. The various subgroups of the different disorders are clearly outlined. The book also contains chapters on keratin and disorders of keratinisation. The well-established connection between cells of the epidermis and the underlying connective tissue makes this a rational addition.

Heritable disorders of connective tissue can be found in various medical specialties because symptoms and signs can affect several organs, vary in intensity, and occur within every age group. For this reason the book should appeal to many areas of medical practice and science. Readers are, however, likely to vary in their background knowledge. By providing general chapters on biology of connective tissue and comprehensive descriptions of the specific disorders, while at the same time ensuring that the text is clear and easily understood, the authors have achieved a book of great interest to a wide circle of clinicians and scientists.

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Cardiovascular Disease

Risk Factors and Intervention.—*Edited by Neil Poulter, Peter Sever, Simon Thom. Oxford: Radcliffe Medical Press. 1993. Pp 351. £49.50. ISBN 1-870905547.*

Twenty-five years ago, when the "epidemic" of coronary disease was in full swing, Richard Asher begged contributors to medical literature: "please, do not write any more articles about cholesterol and coronary disease and the diet and drugs which are supposed to influence it". Twenty-five years later, we are still afflicted with an epidemic of cholesterol literature—at least we understand the causes.

This volume shares eight authors and most of the subject matter with another volume, nearly twice as thick although only 50 pence dearer, published in Oxford last year. Who reads these books year after year? Believers, unbelievers, or neither? As an uncritical syllabus of the consensus of risk-factor epidemiologists, *Cardiovascular Disease* does not ask questions—it repeats answers. It is too superficial doctrinaire for a real student. A token "unorthodox" position is represented by D J P Barker. Chapters on garlic and (not again) coffee are in, but there is no chapter on alcohol. E Barrett-Connor is the only contributor who dares to question whether all this panic about the prevention of heart disease is worth it, since the alleged decline in mortality from coronary disease in the USA between 1977 and 1987 was accompanied by an 80% increase in the prevalence of the disease and by a 150% increase in the costs of morbidity. She does not, however, speculate on how much anti-smoking policy has contributed to these increases. In Framingham, in middle-aged men, the incidence, prevalence, and mortality of coronary disease in the past 30 years has increased (*Int J Epidemiol* 1989; **3** (suppl): 67), despite a dramatic reduction in "risk factors". Don't buy this book if you want to find out why. Conflicting evidence is not cited or discussed so that the message about "killesterol" remains loud and clear. The chapter on screening concludes that "screening should be for all risk factors", and it is implied, by fanciful calculations, that it is good value for money.

The proof of the pudding, evidence from intervention trials, is glossed over in three pages. Recent critical analyses of all relevant trials (only those by Muldoon and Davey Smith are mentioned) are dismissed as "selective". D J Betteridge grudgingly concedes that prevention trials have not shown a reduction in all-cause mortality, but then, he says, they were not designed to do so and "did not have the statistical power to examine non-cardiac deaths". Even if all the statisticians who worked on these trials were laid end to end, they would not reach a conclusion. Richard Asher, we miss you.

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Selected Books: Oncology

Imaging Strategies in Oncology.—Daniel Vanel, David Stark. London: Martin Dunitz. 1993. Pp 536. £99.50. ISBN 1-853170526.

Clinical Oncology.—Geoffrey R Weiss. Norwalk, Connecticut: Appleton & Lange. 1993. Pp 416. $33. ISBN 0-838513255.

File! Meigles Burchell