Letters

Communicating risks through analogies

Editor—Articles in this theme issue have described approaches that try to enhance the communication of risk by developing communication skills, using decision aids, and simplifying the representation of information. But when clinicians talk about actual risks with individual patients they often use analogies. We asked visitors to bmj.com to tell us some of their analogies.[1] Many of their responses addressed screening and chronic disease—perhaps because they are both particularly fraught with difficulty.

The value of illustrations was reinforced by Barth, a surgeon: he described how he gives patients a digital picture of their scan that includes a risk calculation. This, of course, also indicates the extent of disease.

Many of the examples from readers try to convey the size of a risk. People know, for example, that smoking is a risk but find it difficult to comprehend its magnitude. Mackay relates how Richard Peto (responsible for many of the big studies on the effects of smoking) tosses a coin and slaps it on the back of his hand to illustrate to his audience the (true) 50% risk of being killed from long term tobacco smoking. "It always produces a gasp of surprise."

Clinicians may be faced with conveying very small risks. Markowicz relates how he sometimes engages in the following dialogue:

"Do you know what is the biggest risk you face in connection with this procedure?

"No, doctor. What is it?

"Driving to the test!"

Similarly, Anton compares mobile phones with genetically modified (GM) food. Both have a (probably) small, unquantifiable, and theoretical risk of causing serious health problems. "No one wants to eat GM food, everyone wants a mobile phone." (Bellaby explores some of these discrepancies further (p 725).)[2]

In all screening programmes the accuracy of the screening test is limited by its specificity and sensitivity. Nottingham relates two analogies to try to explain this phenomenon. The first analogy shows how most abnormalities are picked up but a few slip through the net, sometimes with disastrous results:

"Imagine you are a fire fighter called to a burning house. From inside you hear screaming. You manage to rescue x of the y occupants but despite your best efforts z perish. Should you be hailed as a hero or indicted for homicide?"

The second analogy emphasises that by increasing the sensitivity more people without disease will be investigated while by increasing the specificity more people with disease will be missed:

"Convict everyone who is tried by a jury and fewer criminals will walk the streets but some innocent people will get locked up. Move too far the other way and there will be fewer wrongful convictions but some guilty people will get away with it. This doesn't make the lawyers, the judges, or the juries incompetent or negligent: it's an inevitable part of the system. Change the system and things could be better or worse."

Dawson considers abnormal cervical smear test results, particularly women's concerns about why their smear test needs to be repeated sooner than expected. To answer the question, "If I've got cancer why isn't it being treated?" he draws on the analogy of blood pressure measurement.

"If the blood pressure is 'plumb normal' then there's no need to repeat for three years; if it's obviously abnormal then we need to make sure but would expect to treat it. Often the blood pressure is not low enough to consider normal or high enough to investigate and treat. In those cases we arrange another check a little later. Depending on the value then, we either treat or continue closer follow up until we are happy that all is normal."

Other respondents considered chronic diseases. Mukhopadhyay conveys the multiplication of risks in diabetes when several risk factors are present by considering the risks of breaking a limb on leaving a house:

"A healthy person gets out of the house through the door. If you're diabetic, you are jumping from the first floor. If you also have high blood pressure, you're jumping from the second floor … and so on. Finally, if you smoke in addition, you're jumping from the top of a five storey building.

"And, to make people understand the impact of control, I mention that they can come down the stairs by controlling their risk."

Many of Bieber's patients are from professional groups, and he likens the process of improving lifestyle and risk factors to making decisions about a retirement portfolio.

"With an investment counsellor, a person periodically assesses how their assets are invested and what the anticipated yield will be over time. If a shift in assets from 4% yield to 6% yield is easy and available, over 20 years of investing, great profits can be expected. Likewise, healthy decisions (cholesterol control, weight modification, blood pressure control, etc) can improve a patient's chances of having a full 'portfolio' and healthy body when retirement time comes in 20 years. Each small incremental improvement, over 20 years, can offer compounding results statistically."

Finally, two correspondents raise interesting issues about the framing of risk and the limits to certainty.

Arnold writes of a mother who had a baby with spina bifida. "She asked me the risk of a recurrence with a second pregnancy. As I had no idea, I consulted a relevant book. The risk stated was 1 in 10. I told her that she had a 90% chance of having a normal baby. She happily ran out of the surgery, soon became pregnant, and produced an infant with a normal spine.

"I relate this anecdote because I was berated when I recounted this story in a discussion of risk, at a meeting with 'medical consumers.' I was told that I had lied to my patient. Had I?"

Garcia communicates cardiovascular risk to his patients using the analogy of car crashes. He explains what could happen if they do not wear a seat belt, observe the speed limit, or follow traffic lights, etc. It does not mean that they will die in a car crash. Similarly, wearing a seat belt, observing the speed limit, or complying with traffic lights does not mean that they won't. However, among hundreds of people in the first population and hundreds in the second, the number of traffic deaths will definitely be higher in the first group.

Moreover, he adds, "You should know that whatever the behaviour you adopt you will never know what would have happened should you have adopted the other way."

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Competing interests: None declared.

1 Supplemental. How do you communicate risk? bmj.com/cgi/content/full/327/7405/DC1 (accessed 8 September 2003).
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Reliability of PSA testing remains unclear

EDITOR—Gottlieb's news item reports that the reliability of prostate specific antigen (PSA) testing is poor.[1] Necropsy studies have for a long time shown that the prostate of men over 50 harbours cancer in about 30% of cases, but only 8-10% develop a clinical cancer during their lifetime. That histological evidence of cancer is found when six or more biopsy samples are taken from the prostate is therefore not surprising. Screening studies also show that this happens below the classic threshold of a prostate specific antigen concentration of 4 ng/ml.[2]

Most of these cancers are overdiagnosed and would never be symptomatic during lifetime or cause death. This has been estimated repeatedly from screening data.[3 4]

The aim of screening with prostate specific antigen is to reduce mortality and save lives, not to detect histological, non-aggressive cancer, which is to be avoided. Using a cut-off point of 4 ng/ml already allows for great diagnostic anticipation and considerable overdiagnosis.

Lowering this threshold needs to be carefully considered. It will result in more biopsy specimens showing cancer but also greater overdiagnosis. Whether it will increase screening efficacy (mortality reduction) is unclear and remains to be shown. Waiting for the results of ongoing screening randomised studies would be a more reliable option.

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Balancing benefits and harms in health care

Observational data on harm should complement systematic reviews of benefit

EDITOR—The meeting on "rare events" mentioned by Cuervo and Clarke was an opportunity to bridge two worlds: the world of systematic reviews of randomised trials, which investigate benefits of treatment, and the world of pharmacoepidemiology, which mainly investigates the harms of the same treatments.[1] Bridging will not be easy.

On one hand, there are entrenched views about the superiority of randomised evidence, leading to prejudices that there has been no reliable progress except for randomised trials, that case reports are biased (despite evidence to the contrary about adverse effects[2]), or that observational databases are intrinsically problematic. This attitude ignores established differences between discovery and verification, and between investigating intended and non-intended effects of treatments.[3 4]

On the other hand, discussions about epidemiological methods as applied to adverse drug reactions can become so arcane that only a few people still follow.[5] Judging observational research on harm will entail more knowledge of subject matter and will be less easily codified than judgments about randomised trials.

However, participants from both fields wanted to think about incorporating the other in future activities, perhaps by giving young people dual training in both types of research. This should lead to a future where several types of evidence can be entered in a single systematic review to present a true balance of risks and benefits.

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Competing interests: None declared.

1 Cuervo LG, Clarke M. Balancing benefits and harms in health care. *BMJ* 2003;327:65-6. (12 July.)
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Observational data on harm are already included in systematic reviews

EDITOR—The correspondence on the issue of including observational data of harm in systematic reviews surprised us.[1 2] The tone of the prose implied that this might happen in the future, and the limits of randomised controlled trials discussed told readers what anyone working in assessing adverse events knows only too well. Observational data have been included in systematic reviews of possible harms for some time, precisely for the reasons that Johnston illustrates for vaccinations.[2]

An increasing number of potentially damaging allegations of associations between exposure to one or more vaccines and harmful events were made recently. Evidence was scattered, seldom assessed by its methodological quality, and sometimes included in descriptive reviews. We used allegations of harmful events after immunisation with pertussis, measles, mumps, and rubella (MMR), and hepatitis B vaccines to develop methods to identify, assess, and synthesise evidence from studies of different designs, ranging from randomised controlled trials to case-only designs (www.who.int/vaccines-surveillance/ISPP/IssuesofInterest.shtml).[3–5]

Development of quality assessment criteria for such studies was a worthy challenge. Assessment of possible rare and unforeseen adverse events after vaccination is methodologically particularly difficult because independent controls are lacking in most cases. Most of the population is already vaccinated, and those who are not are likely to be unrepresentative of the reference population. Such difficulties can be overcome by including studies with no independent controls (before and after and case crossover designs) as no single study design is likely to answer the study question (table). All available evidence should probably be assessed and included.

Broadening the focus of systematic reviews is not an optional feature that may come about in the future: it's here already.

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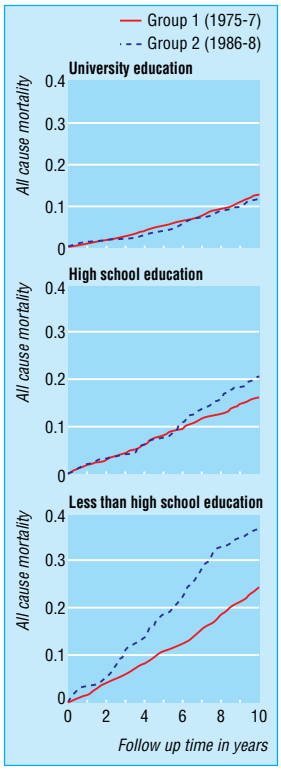
Strengths and weaknesses of studies included in systematic reviews of harmful effects

Method/study design	Strengths	Weaknesses
Case report	Early warning	Bias, differing case definitions, lack of comparators
Passive and active surveillance	Early warning or detection of rare events	Bias, differing case definitions, lack of comparators
Ecological study	Powerful, cheap	Difficulty in interpretation, confounding, bias, differing case definitions
Case crossover and case based studies	No need for independent controls	Lack of wide acceptance, bias, differing case definitions
Multiple time series	Flexible, powerful	Credibility, bias, differing case definitions
Case-control study	Can test hypotheses, especially rare events	Confounding, bias, differing case definitions
Cohort study	Powerful, cheap (if retrospective)	Confounding, bias (especially attrition), differing case definitions
Historical control study	Powerful, cheap	Bias, differing case definitions, difficulty in interpretation, differing case definitions
Randomised and clinically controlled studies	Powerful, minimisation of all biases	Short follow up, limited power, differing case definitions

Increase in mortality in Russia in the 1990s

Time of risk factor assessment is of special importance

EDITOR—Plavinski et al found that in two cohorts in St Petersburg mortality increased over the past decades in men without a university education but remained unchanged in those with one.[1] In a subanalysis, mortality increased substantially over time in consumer groups with both high and low alcohol consumption while differences in mortality between the groups were much smaller. Therefore, the authors conclude, alcohol accounts only partly for the recent rise in Russian mortality.



I doubt that the study design is appropriate to detect differences in alcohol related mortality. Alcohol consumption was asked for only on recruitment, 1975-7 and 1986-8, respectively. This is of special importance for the later cohort since its recruitment coincided with Michail Gorbachev's anti-alcohol campaign, which caused a considerable decline in alcohol consumption in Russia.[2] This decline, however, was shortlived, and alcohol consumption rose again quickly after the campaign's end. Therefore, many people classified as consumers of low quantities of alcohol on recruitment might have consumed considerably higher amounts during follow up and consequently been misclassified.

Alcohol consumption, in contrast to, for example, smoking or hypercholesterolaemia, seems to be, at least for cardiovascular diseases, a risk factor with a close temporal relation to the event.[3] Therefore it is of special importance to assess it continuously in prospective studies.

An additional limitation of the study is that it did not account for drinking patterns, which might be crucial to the effects of alcohol on cardiovascular mortality.[3]

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Authors' reply

EDITOR—The hypothesis that alcohol is a major determinant of the recent mortality fluctuations in Russia is based on circumstantial evidence.[1] Changes in binge drinking patterns are not compatible with changes in mortality.[2] High death rates from cardiovascular disease cannot be explained by misclassification of deaths attributable to acute alcohol poisoning.[3] Our study also failed to support an "alcohol only" hypothesis.

Firstly, the mortality structure did not change in two cohorts: non-natural death was responsible for 11% of all cases in the first cohort and 7% in the second cohort. The proportion of deaths due to cancer remained the same (26.3% and 26%), which argues against sudden increase in alcohol related deaths even misclassified as deaths from cardiovascular disease.

Secondly, "weekend deaths" are believed to be an indicator of alcohol related mortality. In the first cohort among people with the lowest education there was slight excess of mortality on Saturdays, but in the second cohort the excess was on Tuesday and Friday. No weekend excess was evident. The same pattern was observed for participants with a university education who showed no increase in overall mortality.

Thirdly, alcohol is not a major risk factor for mortality in Russian middle aged men,[4] and an increase in mortality was seen in only 5% of frequent heavy drinkers.[5]

Fourthly, another study performed in St Petersburg showed that people with a poor education receive poor hypertension treatment—only 12.5% received adequate drug treatment in contrast to 50% of people with a university education (S L Plavinski and E V Frolova, 12th lipid meeting, Leipzig, 2002). We sincerely believe that other factors should be discussed as the reason for the mortality increase in Russia.

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Effect of NSAIDs on risk of Alzheimer's disease

Confounding factors were not discussed

EDITOR—In their study of the effect of non-steroidal anti-inflammatory drugs (NSAIDs) Etminan et al did not consider possible confounding factors for what is currently only an observed reduction in risk.[1]

What if having osteoarthritis is negatively associated with developing Alzheimer's disease? Presumably some form of osteoarthritis is the likely reason why older people are receiving NSAIDs. Both conditions have genetic and environmental components that may well be mutually exclusive.

That NSAIDs offer some protection against the development of Alzheimer's disease cannot yet be stated with confidence. As Davey Smith and Ebrahim pointed out in an editorial, an association does not show causation, and doctors have been caught out giving poorly based and premature advice (and treatment) before.[2]

A recently published randomised controlled trial did not show any benefit from either naproxen or rofecoxib in preventing the progression of early Alzheimer's disease.[3] We may be no further forward in being able to prevent or treat this condition.

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Author's reply

EDITOR—We agree with Robertson that confounding may explain the potential protective benefit of non-steroidal anti-inflammatory drugs (NSAIDs) in our meta-analysis. We specifically addressed the issue of confounding in the discussion of our paper.

We also agree that the results of our meta-analysis are purely hypothesis generating and that a cause and effect relationship cannot be inferred from our results.

Robertson refers to a recent randomised trial that found no benefit with the use of NSAIDs.[1] This study is a secondary prevention trial in subjects whose Alzheimer's

disease has already been diagnosed, with one year follow up. Our meta-analysis attempts to answer a different question: mainly whether NSAIDs can prevent the development of Alzheimer's disease.

The Alzheimer's disease anti-inflammatory prevention trial (ADAPT) is currently comparing naproxen and celecoxib in the primary prevention of Alzheimer's disease.[2] The results of this large randomised trial may finally shed light on this question.

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1 Aisen PS, Schafer KA, Grundman M, Pfeiffer E, Sano M, Davis KL, et al. Effects of rofecoxib or naproxen vs placebo on Alzheimer disease progression: a randomized controlled trial. *JAMA* 2003;289:2819-26.
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Conclusions on NSAIDs and Alzheimer's disease were overstated

Editor—Etminan et al present a systematic review and meta-analysis on the use of non-steroidal anti-inflammatory drugs (NSAIDs) and the risk of developing subsequent Alzheimer's disease.[1] The trials included in the review were observational studies (six cohort and three case-control). While theoretical mechanisms for protection against Alzheimer's disease exist (prevention of senile plaque formation), the authors correctly point out that, currently, no randomised controlled trials have examined the role of NSAIDs in preventing Alzheimer's disease.

As the Women's Health Initiative has shown,[2] overstating the findings of observational studies is risky. In light of the lack of evidence from randomised controlled trials, the conclusions by Etminan et al that NSAIDs offer some protection against the development of Alzheimer's disease (in the abstract) and that their results show that use of an NSAID lowers the risk of developing Alzheimer's disease (in the discussion section) are overstated. The authors later correctly note that the appropriate dose, duration, and risk/benefit of NSAID use for protection against Alzheimer's disease are unclear (as well as the appropriate population for a prevention strategy).

Pending randomised controlled trials to address these issues, it is best to note that the use of NSAIDs is associated with a decreased risk of Alzheimer's disease.

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1 Etminan M, Gill S, Samii A. Effect of non-steroidal anti-inflammatory drugs on risk of Alzheimer's disease: systematic review and meta-analysis of observational studies. *BMJ* 2003;327:128-31. (19 July.)
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Statistical interpretation can also bias research evidence

Editor—Kaptchuk discussed the effect of interpretive bias on research evidence.[1] Let me add one more example. Studies are designed to determine whether "a statistically significant difference" exists between the outcomes of two alternative treatments. If no difference is discovered the temptation for authors is to conclude that the treatment under investigation is "just as good" as the gold standard. To make such a statement, the study needs to have adequate statistical power, ensuring the chance of a type II error (incorrectly accepting the null hypothesis) is sufficiently small.

Since power can generally be increased by enlarging the sample size, it has become popular for researchers who do not have sufficient power to speculate in a way that makes the actual power meaningless. For example, such a typical speculative statement might read: "While the study failed to have sufficient power to confirm the findings that the drugs were not different, had the sample size been increased from 10 to 180, then the power would have been sufficient to conclude no difference exists." In this way, the researcher implies that it's only a statistical convention is preventing him or her from stating that no difference exists between the two drugs. In reality, had the sample size been so increased, there is no guarantee as to what the researchers may have found.

For those who cannot resist such hypothetical conclusions may I suggest (tongue in cheek) you skip the study, examine one patient, report the results, and speculate that whatever you find could be of greatest statistical significance if only the study had been conducted with more people.

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1 Kaptchuk TJ. Effect of interpretive bias on research evidence. *BMJ* 2003;326:1453-5. (28 June.)

Interpretation of randomised trials is indeed open to debate

Editor—McCormack and Greenhalgh once argued that the interpretation of randomised controlled trials is open to debate.[1] A recent *BMJ* news item featuring an overly favourable comment on the diabetes subtrial of the heart protection study illustrates this perfectly.[2–4] We question the study's relevance to general practice on four points.

Generalisibility—The diabetes subtrial is part of the main study published in 2002. The researchers in the original trial excluded more than two thirds of the original 63 603 patients. Most of them opted out or were deemed not to be reliably compliant for the trial.

Bias—Analysis of the study's design raises the possibility of a non-match between treatment and control group[5]; there is a potentially important difference in the dropout rate.

Merging the boundaries—Both studies look at patients with and without pre-existing cardiovascular disease. This is misleading as patients with established disease have a higher chance of benefit from lipid lowering treatment. We disagree with the impression in the *BMJ* news item that the case for using statins in diabetic patients without cardiovascular disease who have low density lipoprotein cholesterol, <3.0 mmol/l, is made; the 3.1% absolute difference between treatment and placebo group is only marginally significant ($P=0.05\%$).

Presentation of results—Relative risk reduction and composite end points as seen in both studies exaggerate the potential benefit of a proposed treatment and should not be used in serious medical journals.

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Black report remains helpful to government

Editor—Can someone, anyone, give John Reid a copy of the Black report.[1 2]

That tells him all he and Gordon Brown need to do.

The rest of us will gladly help.

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