of datasets for secondary analysis that maintain the confidentiality of subjects. Adequate documentation should accompany such datasets, and the costs of producing the material should be covered by the initial grants—in which case funding agencies would have to provide the necessary finance—or costs should be covered by the secondary analysts.

Clearing houses for shared data would reduce the burden on primary researchers. After providing a documented dataset to the central archive once, researchers need not repeatedly answer requests, as the archive takes on this task. The Economic and Social Research Council has established such an archive,²⁴ and other topic based databanks exist.⁶

Journals also have a role in encouraging the sharing of data. For example, the *American Journal of Public Health* stipulates that data are, in principle, available to the editors and to interested researchers.⁶ If papers submitted to journals had to be accompanied by a disk copy of the data on which they were based then statistical referees could check that the results were not the product of overenthusiastic data torture.²⁵ In this way sharing of data could contribute to improving the quality of published research. The fall in submissions to a journal brave enough to implement this policy would be a useful indicator of its success.

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Organochlorines in the environment and breast cancer

The data so far produced provide reassurance rather than anxiety

Women have been worried recently by press accounts of six comparatively small epidemiological studies suggesting that certain organochlorines in the environment might increase the risk of breast cancer.¹⁻⁶ The studies were concerned with 1,1-dichloro-2,2-bis(*p*-chlorophenyl) ethylene (DDE), the main metabolite of the insecticide DDT and also with the polychlorinated biphenyls (PCBs), a group of compounds used in industry—for example, as electrical insulators. How strong are the grounds for concern?

The results are summarised in the figure. The individual estimates plotted are ratios of the mean concentrations of DDE or of polychlorinated biphenyls in fat or serum in women with breast cancer divided by the mean concentrations in control women without breast cancer. Summary ratios were derived from the weighted averages of the log ratios. For

DDE the summary ratio was 1.11 (99% confidence interval 0.97 to 1.26). In other words, the women with breast cancer had slightly higher concentrations of DDE than controls but the difference was not statistically significant. For the polychlorinated biphenyls the summary ratio was 1.01 (0.92 to 1.10)—indistinguishable from no difference at all.

When they were first published two of the studies—by Falck and colleagues³ and by Wolff and colleagues⁴—created considerable anxiety with respect to DDE, but they were based on only 20 and 58 women with breast cancer respectively. The recent study of Krieger and colleagues⁶ had as many cases as all the previous studies put together and used serum collected an average of 14 years before diagnosis, whereas the other studies used fat or serum collected shortly before or after diagnosis. Krieger and colleagues found no association of DDE with breast cancer.

Widespread use of DDT began in the United States in 1946 and increased until 1959. It then declined steadily until it effectively stopped in 1972.⁷ DDT accumulates in the body, but the reduction in its use caused concentrations in adipose tissue in the general population to fall from about 8 ppm in 1970 to about 2 ppm by 1983.⁷ Polychlorinated biphenyls were first produced commercially in 1929 and since then have been used in many industrial products. They were detected as environmental contaminants in 1966, and in the United States production ceased in 1977. In 1972, 61% of the American population was estimated to have concentrations of polychlorinated biphenyl in adipose tissue above 1 ppm, but this had dropped to 6% by 1983.⁷ In most developed countries patterns of use of both types of organochlorines have resembled those in the United States, but DDT

Ratios of mean concentrations of DDE and polychlorinated biphenyls in cases and controls from six studies. Areas of solid squares are proportional to information in each study

is still widely used in some countries to control mosquitoes.

Temporal trends in breast cancer rates are difficult to relate to changes in exposure to suspected risk factors because the rates are affected by complex variations in the known risk factors for breast cancer such as age at menarche, age at first birth, parity, and age at menopause. In several developed countries, however, breast cancer mortality rates were high before any widespread exposure to DDT or polychlorinated biphenyls, and the rates have not risen strikingly since these chemicals were introduced.

The International Agency for Research on Cancer has classified DDT as "possibly carcinogenic" to humans, largely because it can cause liver cancer in experiments on animals.⁸ Another reason for suggesting that DDT from the environment might cause breast cancer is that DDT is oestrogenic. This is a very theoretical risk; even in large doses DDT is only weakly oestrogenic in animals, and it has not been shown to have oestrogenic effects in women. Oestrogen replacement therapy, which has clear oestrogenic effects, may increase the risk of breast cancer by about 30% after 15 years of use,⁹ so any small oestrogenic effect of DDT in the environment would probably be impossible to detect by epidemiological studies.

Polychlorinated biphenyls have been classified by the International Agency for Research on Cancer as "probably carcinogenic" to humans, with a possible association with cancers of the liver and biliary passages.¹⁰ Polychlorinated biphenyls are sometimes referred to as oestrogenic, but in fact some show antioestrogenic activity, and no oestrogenic effects of polychlorinated biphenyls have been established in women.^{11 12} There is, therefore, no strong reason to expect polychlorinated biphenyls to cause breast cancer rather than any other cancer.

Putting all this evidence together, we conclude that it is unlikely that DDT in the environment increases the risk of breast cancer. However, all published epidemiological evidence comes from the six studies cited—based on only

301 women with breast cancer and 412 women without. The question is so important that it seems justified to examine it further in at least the same number of women again, using specimens collected before the women develop breast cancer. For polychlorinated biphenyls there is no evidence for an association with breast cancer risk, and there seems to be no need to pursue this question further.

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Cycle helmets and the law

Even when the science is clear policy decisions may still be difficult

Any discussions of the law and the use of bicycle helmets will be helped by focusing on three questions. Firstly, how much do cycle helmets affect the risk of injury in a crash? Secondly, how much do laws requiring cyclists to wear helmets affect casualties? And, thirdly, should wearing of bicycle helmets be required by law?

Answers to the first two questions have objective answers that can be sought from specific empirical studies and what is already known about traffic safety.¹ Three unrelated sources of evidence consistently show that cycle helmets reduce risk substantially in a crash. The science of biomechanics shows that a helmet reduces the peak acceleration forces that are associated with injury. Many published epidemiological studies, including that by Maimaris and colleagues in this issue of the *BMJ* and the references it cites,² find that helmets reduce injury and the risk of death. Such studies fall short of the methodological ideal of comparing harm in matched treatment and control populations—so less direct methods must necessarily be used.

For example, examining the ratio of head injuries to non-head injuries in cyclists wearing and not wearing helmets indicates that helmets reduce the risk of head injury by more than half. One important way in which these studies fall short

of the ideal is due to selective recruitment,^{1 3} the tendency of people who wear protection devices to differ in many ways from those who do not. Estimates of effectiveness will be biased if they ignore the reasonable expectation that crashes will be more severe for cyclists who do not wear helmets than for those who do.^{1 3} The third source of evidence is the analogy with motorcycle helmets. The effectiveness of a motorcycle helmet has been determined by comparing outcomes for a driver and a passenger riding the same motorcycle, and thereby having the same crash, but one wearing and the other not wearing a helmet.^{1 4} The large sample sizes available in data from the United States gave a precise estimate of the effectiveness in reducing death of 28% (SE 8%).

Similar effectiveness was found for drivers and passengers and for men and women. Independent examination of the ratio of head to non-head injuries provides results comparable to those in pedal cyclists—suggesting similar effectiveness for bicycle and motorcycle helmets. A universal property of all protection devices is that effectiveness declines as severity increases,^{1 3} so bicycle helmets are less effective for the higher severities that result from crashes with other vehicles.

Even if cycle helmets protect in a crash casualties need not necessarily decline if more cyclists use them. There is