Leukaemia risks and radon

SIR — A correlation has been established between domestic radon exposure and mutation in peripheral T lymphocytes[1]. Some caution must be exercised, however, in interpreting this result as evidence that levels of domestically encountered radon are sufficient to cause leukaemogenic chromosomal alterations. Radon may simply be acting as a surrogate for some other mutagenic factor.

Levels of radon in houses in the United Kingdom, although strongly related to local geology, are also positively influenced by the quality and value of housing stock[2]. Detached houses have higher radon levels than semidetached or terraced houses which, in turn, have higher levels than flats and maisonettes[2]. The possibility that higher domestic radon levels merely reflect higher socioeconomic status is of concern as such status is an independent risk factor for lymphoproliferative disease[3]. Domestic radon as a surrogate for socioeconomic status could also confound estimates of lung-cancer risk due to environmental radiation exposure[4].

Higher radon levels are significantly correlated with the county percentage level of unemployment, percentage of households owning a car, the percentage of households headed by individuals in occupational class I or II as well as the percentage of households headed by individuals in occupational class IV or V (see table). Lower domestic radon levels thus appear to reflect relatively greater socioeconomic deprivation whereas higher levels reflect greater prosperity. The relative risk of lymphoproliferative disease correlates with the same factors that determine domestic radon levels at the county level.

Putative relationships between domestic radon exposure and cancer thus need to be controlled for socioeconomic status and associated factors, at least at the county level[5]. (The correlations may not apply to smaller areas.) Similarly, the causative factors underlying the relationships between higher regional socioeconomic status and leukaemia require closer examination.

SOCIOECONOMIC VARIABLES AND DOMESTIC RADON LEVELS

	Spearman's rank correlation coefficients			
	Radon (arithmetic mean)		Lymphoproliferative disease (relative risk)	
	r_s	P	r_s	P
Unemployment	−0.44	(<0.025)	−0.73	(<0.0005)
Car ownership	0.54	(=0.005)	0.37	(<0.05)
Overcrowding	−0.29	(NS)	−0.25	(NS)
Home ownership	0.11	(NS)	0.04	(NS)
Class I/II	0.42	(<0.05)	0.43	(<0.025)
Class IV/V	−0.41	(<0.05)	−0.44	(<0.025)

Socioeconomic variables from ref. 6; data from the 22 countries examined in ref. 7. Note that radon levels are both positively and negatively correlated with socioeconomic status.

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Do bacteria have sex?

SIR — In eukaryotic organisms, sex is the basis of two fundamental phenomena: first, reproduction, and hence maintenance of the species; and second, genetic recombination, and hence generation of the diversity that allows the species to evolve. These different aspects of sex are linked in a single strategy for the maintenance and evolution of the species.

But what about sex in bacteria (J. Maynard Smith *et al. Nature* **349**, 29–31; 1991)? The word has been used for three different mechanisms of gene interchange in bacteria, namely transformation, conjugation and transduction, none of which is tied to reproduction, and none involving combination of entire genomes to obtain new individuals. Moreover, they all involve only small specialized pieces of DNA that, in the case of plasmids (conjugation), encode most of the machinery for independent replication, and in the case of phage (transduction) can be considered as bacterial viruses rather than part of the bacterial genome. Furthermore, a usual rate of plasmid conjugation is 10^{-5} per generation, which is tiny compared with 100 per cent per generation in eukaryotic sexual recombination. In the eukaryotic world, the most similar phenomenon to these mechanisms of bacterial recombination is viral infection, and it seems improper to talk about viral infection in terms of sex.

Why then use the term sex to describe phenomena of genetic recombination in bacteria? Maybe, as pointed out by 'E. Coli' (*Nature* **349**, 97; 1991), our eukaryotic chauvinism makes us think about bacteria from a strongly anthropocentric point of view.

Although at first it seems to be a useful simplification, talking about sex in bacteria is confusing, as we are mixing together completely different concepts. I suggest that the term sex be used to describe only the well-characterized mechanism of reproduction-linked genetic recombination in eukaryotes, leaving bacteria with their own very different mechanisms of genetic recombination, which are completely unrelated to what we usually call sex.

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What's the score?

SIR — Anyone who has participated in the dating ritual knows that a rendezvous does not necessarily mean a 'score'. As Hackett has aptly pointed out in News and Views[1], our data[2] and those of Peters *et al.*[3] seem to disagree about the initial site of rendezvous for class II histocompatibility molecules and antigenic fragments. But we do not disagree, in principle, about the timing of the 'score'.

In our paper[2], in which we reported colocalization of the molecules involved in antigen processing and presentation in early endocytic compartments, we also presented data (Figs 3 and 4) showing that these molecules remain colocalized in endocytic compartments for as long as 30 minutes. We suggested that, although the relevant molecules can intersect early in the endocytic pathway, the gradual acidification and increasing proteolytic activity accompanying maturation in the endocytic pathway could be required for the complete processing of antigen and release of class II molecules from invariant chain. This would then explain the delayed kinetics of antigen presentation relative to the timing of initial colocalization of the molecules involved.

This point may have been missed by those who did not read beyond the title and abstract of our paper. We have gone