

# news and views

## Genetic engineering with viruses

A SIGNIFICANT development in the field of genetic engineering is described in an article by Drs Noreen and Ken Murray in this week's issue of *Nature* (page 476). They have been able to isolate a mutant of bacteriophage  $\lambda$  which can replace the normally used plasmid in experiments designed to construct new hybrid DNA molecules.

These experiments are very simple in principle and (in brief) involve mixing together in a test tube the donor DNA molecule (the gene to be studied), the receptor (the plasmid) and a 'DNA cutting' enzyme, the restriction enzyme *EcoRI*. The cutting enzyme breaks the circular double stranded plasmid at a single point converting it into a linear molecule. But it does more than this. The enzyme cuts DNA slightly asymmetrically leaving four nucleotides projecting at each end. These 'sticky ends' can pair up by forming Watson-Crick base pairs with similar sticky ends formed on the donor DNA molecule by the cutting enzyme. Thus a linear hybrid plasmid is formed which can circularise. To stabilise the new hybrid plasmid another enzyme, DNA ligase (a 'joining' enzyme), is added to reform the covalent bonds broken by the restriction enzyme. The success of such experiments, which have now been carried out using staphylococcal DNA, *Xenopus laevis* ribosomal genes and *Drosophila* DNA as donor, depends on there being a single site for the restriction enzyme on the plasmid. The problem in phage  $\lambda$  is that there are five sites.

The Murrays' work involves a brilliant series of genetic and biochemical manipulations to construct a mutant phage  $\lambda$  with one or two sites for the restriction enzyme *EcoRI*. Then, in subsequent experiments, restriction fragments

of these various mutant phage  $\lambda$ s were joined together *in vitro* to form hybrid molecules with a deletion in a non-essential part of the phage. These new molecules were isolated as biologically active phage  $\lambda$  after infection of *Escherichia coli*. By this means the single-site mutant was converted to a bacteriophage with a gap in it leaving room for an incoming piece of DNA. The authors were able to insert the genes specifying the biosynthesis of tryptophan in *E. coli* (the *trp D* and *E* genes) into this receptor phage  $\lambda$ . They imply that they hope to extend this work to study the expression of the *trp* genes, for which phage  $\lambda$  is an ideal host (and has significant advantages over a plasmid host), as well as to study the insertion of DNA from higher organisms.

Many readers of *Nature* will know that there is some controversy among scientists about the ethics of performing genetic engineering experiments (see *Nature*, 250, 279; 1974). Part of the concern is about the nature of the particular *E. coli* plasmid used in these experiments which confers on the *E. coli* resistance to the antibiotic tetracycline. Clearly, if one wishes to introduce foreign, and possibly hazardous DNA, it is advantageous to use microorganisms susceptible to antibiotics. Dr K. Murray, in a recent lecture to the Biochemical Society, has pointed out that phage  $\lambda$  is much safer than the plasmid. It is sensitive to antibiotics and only infects some strains of *E. coli*. There is also evidence that the laboratory strains for which  $\lambda$  is infective do not establish themselves in the human gut. It is thus improbable that hybrid molecules (of whatever type) could be pathogenic.

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## Some myths about heritability and IQ

THINGS have gone too far. Estimates of the heritability of IQ reported in *Nature* have assumed a distinctly bimodal distribution. First, Eaves and Jinks<sup>1</sup> decried my data on the heritability of scholastic aptitudes<sup>2</sup> as a failure to detect the high heritability of IQ. In a sample of urban school-age twins, I found that black and economically disadvantaged children from both races seemed to have lower heritabilities for verbal and quantitative aptitudes than white and middle-class children. In some cases heritabilities were not calculable, because the correlations of opposite-sex twins were slightly larger than those of same-sex twins. Now, Schwartz and Schwartz<sup>3</sup> proclaim zero heritability for IQ as the obvious conclusion to be drawn from the same study and from Kamin's iconoclastic efforts (Invited Address, Eastern Psychological Association, Washington DC, March 1973). I beg to differ with both extremes and to argue against any simple conclusions on the issue.

The notion that there is a single answer to the question "What is the heritability of IQ?" is patently false. Heritability estimates vary according to what skills are measured as intelligence, by how they are tested, by the age at which abilities are measured, and by the genetic and environmental composition of the population tested. To claim that there is a single estimate for the genetic contribution to human

abilities is to deny, first, half a century of evidence on differential abilities. Some aspects of intelligence, such as vocabulary, have consistently higher heritability estimates than, say, numerical reasoning. Even if one concluded, with Burt, that general intelligence (*g*) is more important than specific abilities, different tests of *g* will still yield different heritability estimates<sup>3</sup>.

Differences in heritability estimates across abilities and tests are exceeded only by differences across age groups and populations. Bayley Infant Intelligence Test scores for the first two years have been found to have generally lower heritabilities than later scores. Even early heritabilities vary enormously: from 0.2 to 0.4 for siblings (McCall, American Psychological Association, Honolulu, September 1972), 0.75 for some twins (Nichols and Broman, preliminary report of the Collaborative Perinatal Study, 1973), and 0.3 for other twins<sup>6</sup>. Across populations, Nichols<sup>7</sup> reported a significantly lower correlation for black than white siblings on the Stanford-Binet test at age 4. Another twin study (Scarr-Salapatek, Katz, and Barker, *Black and White Twins*, in preparation) found lower heritabilities for adolescent black and white twin pairs on each of four tests of cognitive abilities and for the first principal component from the four tests. These data are presented in Table 1. Not only are