acids that rendered it suitable to interact with the cell membrane, leading to the evolution of a lysis function. Of course, the evolutionary constraints upon the sequence representing both proteins must be very severe and it remains difficult to estimate how much scope there might be for variation in the sequences of the two overlapping functions. And, of course, overlapping expression must mean that all mutants in gene *E* also carry alterations in gene *D* (and *vice versa* within the region of overlap): while some of these mutations may affect one protein but not the other (due to third base degeneracy) it will be interesting in evolutionary terms to determine the general relationship between effects on *D* and *E* function. A possible utilisation of the overlap by the phage as a control is that one might expect ribosomes translating *D* to interfere with access to the initiation codon of *E*; this predicts that mutants causing termination early in *D* might have increased expression of *E*.

The complete sequence of the phage DNA will no doubt reveal whether the strategem of overlapping expression is used elsewhere in it, but the possibility is at least raised by the observation that the products of genes *A*, *B* and *C* have an estimated total molecular weight apparently somewhat greater than could be coded by this region of the genome.

What is the organisation of the genome of the filamentous phages? The genetic map constructed by Lyons and Zinder (*Virology*, **49**, 45; 1972) places the eight genes in a circular array; and with one reversal of order this was confirmed by the restriction maps calibrated with mutant locations in several laboratories (Van den Hondel *et al.*, *Eur. J. Biochem.*, **53**, 559; 1975; Seeburg and Schaller, *J. molec. Biol.*, **92**, 261; 1975; Horiuchi, Vovis and Zinder, *J. molec. Biol.*, **95**, 147; 1975). Proteins have been identified for six of the genes in two series of experiments; the results are in good agreement and suggest a total for the phage protein products of about 206,000 daltons (Model and Zinder, *J. molec. Biol.*, **83**, 231; 1974; Konings, Hulsebos and Van den Hondel, *J. Virol.*, **15**, 570; 1975). In addition, Model and Zinder identified a protein of 12,000 daltons, apparently phage coded but not yet equated with any gene.

Varying estimates have been taken for the size of the filamentous phage genome. Taking an estimate of 6,400 bases, and assuming that the genes *VI* and *VII* whose products are unidentified are about 250 bases each, led Van den Hondel *et al.* (*op. cit.*) to infer the presence of an unaccounted region of some 300 bases, which they located between genes *II* and *V* and suggested might code for the protein of 12,000 daltons. Taking an estimate of 6,800 bases, and assuming that genes *VI* and *VII* are small, led Vovis, Horiushi and Zinder (*J. Virol.*, **16**, 674; 1975) to suggest that there must be a considerable amount of intercistronic material, which they placed between genes *IV* and *II*. The assumption that genes *VI* and *VII* are small is supported by the mapping of known mutations to restriction fragments, but as is clear from the location of unaccounted regions in different sites, there is some latitude in the construction of the map, resulting principally from the uncertainty as to where the ends of genes lie. (The restriction fragments of these phage genomes have not been sized with respect to outside standards but only relative to each other, which makes the construction of a physical map dependent upon the size assumed for the total genome.)

It has generally been assumed that the size of the filamentous phage genome is close to 15% greater than that of φX. This would suggest a length of not more than 6,200 bases, which is only 600 bases greater than the 5,600 bases required to code for the six proteins of identified origin. If there were no intercistronic region, this would leave genes *VI* and *VII* able between them to code for some 22,000 daltons of protein. If an unidentified gene codes for the 12,000 dalton protein, then only 300 bases (or 11,000 daltons of coding capacity) remains for both these genes. In contrast to these calculations, Berkowitz and Day (*J. molec. Biol.*, **102**, 531; 1976) used biophysical means to measure a length for fd DNA of 5,740 (±210) nucleotides. This would at best place a severe restriction on the length of DNA available to represent genes *VI* and *VII*; and if these genes should prove to code for larger protein products, or if the 12,000 dalton protein proves to represent a new gene, there would be the same paradox as seen with φX174, with insufficient DNA to code for the phage products. □

Lymphoid cell interactions in Japan

from Marc Feldmann

A Symposium entitled Cell Interactions in the Initiation and Regulation of the Immune Response, organised by Drs Yamamura and Hamashima, and sponsored by the International Union of Immunological Societies, was held in Kyoto, on September 1–4, 1976.

CELL interactions in the immune response were first recognised in 1966, with the work of Claman and his colleagues[1], and rapidly confirmed and extended by Miller[2], Davies[3], Mitchison[4], and others. At that time only interaction (or collaboration) between thymus derived (T) and bone marrow derived (B) lymphocytes was known, but the evidence of the following 10 years has shown that every immunological phenomenon involves interactions between several distinct cell types and their products. This symposium covered the whole spectrum of modern cellular immunology and only a few of the aspects discussed can be reported here. Since lymphocyte heterogeneity is at the root of all these interactions recent developments in this field will take priority.

The many different surface antigens of lymphocytes have been exploited to purify different lymphocyte populations[5,6]. The Thy-1 antigen which distinguished T from B cells[5] is the most used. The three best characterised Ly (lymphocyte) alloantigens, Ly-1, Ly-2, and Ly-3, have been used by Cantor, Boyse and colleagues to define three subsets of T cells, bearing Ly-1,2,3, Ly-1, or Ly-2,3 antigens, which are present in decreasing proportions in the peripheral lymphoid pool.

H. Cantor (Harvard University) summarised recent work showing that helper T cells and cells involved in the mixed lymphocyte reaction (Ly-1) are distinct from cytotoxic T cells (Ly-2,3)[7]. The fact that suppressor T cells are also in the Ly-2,3 subset[8], which is the smallest (5–10% of peripheral T cells) raised the interesting possibility that suppressor cells and cytotoxic cells may be identical, and the specificity of alloaggressive suppressor cells like killer cells for the H-2K or D regions, rather than the I region was cited to support this view[9]. However, recent studies by Beverley *et al.*[10] indicate that antigen specific suppressor cells (reactive to proteins) may be separated from cyto-

Correction

E. G. RICHARDS in an article in *News and Views* (*Nature*, **263**, 369; 1976) on September 30 entitled 'Complementary mispairs' inadvertently misnamed transitions and transversions on page 369. The word "transversions" on line 21 should be replaced by "transitions" and *vice versa* on line 24.