a) Measures Against the Spread of R-Factors
(Chairman: M. H. Richmond)

The session concerned itself with three main topics:

1. What is the size of the reservoir of resistant bacteria and is it universally distributed across the world?
2. What are the factors which go to make that reservoir?
3. Are there any steps that we could take to reduce the size of the reservoir?

As far as the first point is concerned, there was general agreement that the incidence of resistant bacteria was high throughout the world. But the distribution was not uniform. In certain parts of the human environment, resistant organisms seem to be very common. In others they were less common. For example, there was no doubt that in the hospital community, resistant organisms were more prevalent than in the community. Similarly, in the group of animals that provide food for man and particularly those animals which are raised under intensive rearing conditions, the incidence of resistant bacteria and of R-factors in particular was high.

Even though the incidence of resistant bacteria across the world is uniformly high in hospitals, this does not necessarily mean that the same types of resistant organisms are prevalent in all hospitals or all geographical regions. This point had been stressed in the formal sessions of the symposium. For example, Prof. Mitsuhashi described the properties of resistant bacteria and of plasmids that he had studied in Japan. The pattern of resistance found there was similar but by no means identical
to that found in the United States or in Britain. Similarly, Dr. Acar described how gentamicin-resistant strains of bacteria were relatively common in French hospitals but Falkow and Richmond both pointed out that similar organisms were not encountered on a large scale in the United States or the British Isles. The general conclusion, therefore, is that while resistant organisms certainly are widespread, there is a clonal distribution of such organisms in certain geographical locations. Moreover, the distribution of the clones need not be even. For example, Anderson in his formal presentation showed that strains of Salmonella typhimurium carrying a particular class of plasmids had been encountered across the most of the Middle East. On the other hand, other examples were provided where a single clone of plasmids was restricted to a single hospital. Thus one has a highly complicated situation in which some clones are distributed widely, and some narrowly, and the total result is a uniformly high level of resistance in certain locations but this does not imply that the high level of resistance is caused by the same organism.

The discussion now turned to the question of what causes the emergence of resistant populations. The first question was whether this could be attributed solely to the use of antibiotics. In practice, the discussion led one to believe that antibiotic use was not the sole force acting to produce resistant populations. For example, the incidence of kanamycin-resistant strains of Haemophilus was reported to be high in Paris and yet the drug in question was used relatively little. Conversely, a very large amount of kanamycin is used in the United States but the kanamycin-resistant Haemophilus has not yet been found. Other examples were given by other people. Levy, for example, had sought carefully for the presence of gentamicin-resistant organisms in Boston and has found them relatively uncommon despite considerable use of that particular antibiotic. It seems clear, therefore, that the use of antibiotics is not necessarily the prime cause for the emergence of given resistant populations. However, the evidence that the use of a particular antibiotic does select resistant populations in individual people, is unquestioned and one, therefore, once again has a complicated relationship between the composition of the reservoir and the use of antibiotics.