is about 1 mK—and the assumptions made in extracting a value of the exchange integral J. The experimental technique of Sites *et al.* was first demonstrated earlier this year. A system of three chambers is formed by two concentric bellows and helium-3 is placed in the innermost chamber; the outer two contain helium-4. The sample cell is cooled with a dilution refrigerator to about 25 mK, and the helium-4 is then bled out of the intermediate chamber generating a sufficient force on the helium-3 to solidify it. The temperature is monitored by nuclear magnetic resonance measurements on copper-63. The sample was found to contain less than one part in 10,000 of helium-4, and a similar purity was claimed by Kirk *et al.* for the experiment.

Kirk *et al.* followed custom by plotting the inverse susceptibility against temperature, and obtained intercepts on the negative temperature axis symptomatic of an antiferromagnet. They point out that it is difficult to interpret the data otherwise, for helium-3 has no electronic structure to produce internal electric and magnetic fields, as in other solids. Sites *et al.* draw attention to the view expressed some years ago that the inverse susceptibility plot may lead to an overestimate of the Weiss constant, and they elected to use a different representation of the results to avoid this. In both experiments there is some room for manoeuvre in interpreting the results, but the negative sign of the exchange integral now seems to be beyond serious dispute.

VIRUSES

Trouble for Asparagus

from our Botany Correspondent

GROWERS of asparagus may not be getting full value from their crops because, although they do not know it, the plants are infected with various viruses. A. F. Posnette of East Malling Research Station reports in the latest issue of the *Journal of Horticultural Science* (**44**, 403; 1969) that several well known viruses have been isolated from weak and stunted asparagus plants. These are clearly different from any viruses previously found in asparagus, and furthermore, some affected plants seem to harbour yet other viruses that have still to be identified. It could be that virus infection is depressing yields unknown to the growers.

Research at East Malling is, of course, chiefly concerned with fruit, but asparagus was planted during ecological studies of infection with arabis mosaic virus because root extracts of asparagus are known to be toxic to the vector of this virus, the nematode worm *Xiphinema diversicaudatum*. After growing for two years in soil where strawberry plants had previously been infected with arabis mosaic virus and strawberry latent ringspot virus, some asparagus plants became stunted and their leaves turned yellow prematurely. More and more plants began to show symptoms, until six years after planting there were two large patches of affected plants.

This situation was investigated by testing for the presence of viruses in samples of leaf and stem taken from plants with and without symptoms. An inoculum prepared by grinding samples with phosphate buffer was applied to the leaves of various test plants, including *Chenopodium amaranticolor* and *C. quinoa*, which were watched for symptoms of virus disease. Viruses were identified by gel diffusion tests with appropriate antisera. Stunted asparagus yielded strawberry leaf roll, arabis mosaic and tobacco black ring viruses, which may have been causing the stunting. All plants, whether stunted or not, carried a virus which under the electron microscope resembled the asparagus virus described by Hein in 1960.

About half of the stunted plants were not apparently infected with any of these viruses, although their symptoms were strongly suggestive of a viral origin. Posnette suggests that these plants were infected with a virus or viruses not transmitted in sap to *C. amaranticolor* or *C. quinoa*, so that the tests carried out in this case would not have revealed them.

There have been two previous reports of viruses in asparagus, but those found by Posnette are clearly not the same, being transmitted in the field by nematode worms. Those viruses yet to be identified from asparagus could add considerably to the tally so far. Some may be carried unnoticed and all could be preventing the plants from giving the highest potential yield as a consequence of their effects on metabolism. A thorough virological investigation of asparagus crops might show that there is much room for improvement.

CELLULAR PARTICLES

The Phantom Monosomes

from our Molecular Biology Correspondent

IT seems that anyone who is prepared to comment on the *in vivo* distribution of polysomes, ribosomes and their subunits must always be made to feel like the man who applauds after the first movement of a symphony. If one forbears to cloud the issue by considering also eukaryotic cells, one may believe with Mangiarotti and Schlesinger that 70*S* ribosomes from *E. coli* arise from degradation of polysomes, or with Davis and his co-workers and Varricchio that they are ribosomes which have attached to a messenger (polysomes of one, so to speak), or since this week, with Phillips, Hotham-Iglewski and Franklin (*J. Mol. Biol.*, **45**, 23; 1969) that they do not exist at all.

Phillips *et al.* had already observed (*J. Mol. Biol.*, **40**, 279; 1969) that different polysome profiles were obtained on sedimentation of *E. coli* lysates, according to the cationic species that were present. Thus in solutions of sodium, lithium, caesium or tetraalkylammonium ions, 50*S* and 30*S* subunits were found in addition to polysomes, but 70*S* particles were absent or almost absent. With potassium, ammonium or rubidium ions, on the other hand, there was a large 70*S* component. One of these distributions, Phillips *et al.* argue, must reflect the situation *in vivo*, and the other therefore represents an artefact of some kind.

The second pattern does not vary with the endogenous nuclease content of the *E. coli* strain, and it therefore seems unlikely that the monosomes are a consequence of polysome degradation. There is no satisfactory strain deficient in ribonuclease II, but the augmentation of this enzyme from an extraneous source does not lead to the breakdown of the polysomes. The key to the problem lies in the use of antibiotics, which interfere at different junctures in the protein synthesis cycle: tetracycline, which blocks the formation of an initiation complex by the 30*S*