PLANT PATHOLOGY

Disease Resistance

from a Correspondent

A DISCUSSION meeting on disease resistance in plants was held on December 9 at the Royal Society. Professor P. W. Brian (Cambridge), recalled in his opening remarks that the last meeting organized by the Royal Society on this subject had been in December 1947. The speakers on that occasion had been Professors W. Brown and F. T. Brooks, together with Mr F. C. Bawden. A link between these two meetings was provided by Sir Frederick Bawden as chairman of the afternoon session.

In his introduction Professor R. K. S. Wood (Imperial College, London) had time to say something more particular about the hypersensitive reaction of plants to invasion by bacterial and fungal pathogens and about the phytoalexin response. His former colleague at the Imperial College, Dr B. J. Deverall (Agricultural Research Council at Wye College), took up the story of phytoalexins in more detail. These protective substances are produced by plants in response to challenge by microorganisms and also to chemical injury; the possibility that phytoalexins can explain why most plants are resistant to invasion by most microorganisms accounts for the wide interest aroused by these plant chemicals. Weyerone acid has recently been identified by Dr Deverall and associates as a phytoalexin produced by the broad bean. From work with potato, the plant in which K. O. Müller first demonstrated the phytoalexin response, Dr D. D. Clarke (University of Glasgow) cited evidence for the view that virulent races of the potato-blight fungus inhibit the synthesis of phytoalexin by their host.

Professor A. Kelman and Dr L. Sequeira (University of Wisconsin, Madison), with a variety of examples, made the point that bacterial pathogens are a natural choice for the study of resistance responses by the host. Working with *Pseudomonas solanacearum*, which causes wilt of tobacco, they have shown that prior infiltration of leaves with heat-killed bacterial cells or their protein fractions will protect leaves later challenged by the living pathogen, and that this protective response can move out into untreated leaves. From studies on the virus-yellow complex of sugar beet, Dr G. E. Russell (Plant Breeding Institute, Trumpington) demonstrated the wide spectrum of resistance mechanisms against virus infection, including a useful degree of resistance to colonization of leaves by aphid vectors.

The increasing threat to wheat and barley from yellow-rust epidemics produced for Dr R. C. F. Macer (Rothwell

Plant Breeders Ltd, Lincoln) the most lively discussion of the day, though this did not detract from the promise of F_1 hybrid varieties as a new kind of answer to this problem. The last two papers in the afternoon served further to emphasize the central theme of this meeting, that is, the wide variety of disease-resistance mechanisms. Dr P. W. Talboys (East Malling Research Station) developed in detail his explanation of how the sequence of post-invasion events can account for the various responses of susceptibility, tolerance or resistance in plants invaded by vascular-wilt fungi. Similarly, Dr J. Rishbeth (University of Cambridge) was able to explain some resistance mechanisms of trees, such as the resin response of conifers, that are effective against root-infecting fungi.

The general impression left by this meeting was that few if any of the new developments in research described during the day were of secondary importance; to counter the genetic opportunism of evolving pathogens, plant pathologists would have to exploit to the full all the resistance mechanisms possessed by the plant.

TUMOUR VIROLOGY

Mitosis Required

from our Cell Biology Correspondent

ALTHOUGH nobody has yet suggested a good reason why it should be the case, many tumour virologists maintain that the transformation of cells by both RNA and DNA tumour viruses depends on the cell going through at least one round of division, a quantal mitosis, after it has been infected. The idea has an immaculate pedigree, stemming from the experiments of such people as Green, Todaro and Temin to mention but a few, and as Weiss now points out (*Virology*, **46**, 209; 1971) it ties in with investigations of the differentiation of myoblasts in culture which have led Holtzer and others to conclude that cellular transformation during normal differentiation depends on a quantal mitosis.

Most of the experiments with the RNA tumour viruses, which have been interpreted in favour of the quantal mitosis idea, have involved the use of drugs which inhibit either DNA replication or mitosis. What is notable about the

Variation in Chromosome Pairing in Diploid Wheat

BREAD wheat, *Triticum aestivum*, behaves as a classical allohexaploid ($2n=6x=42$) with regular bivalent formation at meiosis and disomic inheritance, although the three genomes, A, B, and D, are sufficiently homologous for chromosome pairing to occur between them. This homoeologous pairing is prevented by an allele at the *Ph* locus on chromosome 5B. All of the synthetic tetraploid and hexaploid wheats produced from the probable diploid ancestors show homoeologous pairing, indicating that none of the diploid plants used has an allele which prevents this. On the contrary, all plants of *Aegilops speltoides* that have been studied suppress the control by the *Ph* allele. *Aegilops speltoides* is considered the most likely contributor of the B genome. Although it has been assumed that control by the *Ph* allele originated in the polyploid wheats, the origin and establishment of this genetic control of chromosome pairing is still an unsolved problem.

In *Nature New Biology* next week (January 12), Dover and Riley report the first evidence of genetic variation, in a diploid species, which influences homoeologous pairing in polyploid wheat. This variation is present in *Aegilops mutica* which, like *Ae. speltoides*, has previously been shown to suppress the activity of the *Ph* allele. Homoeologous pairing and chiasma frequency were studied in several *T. aestivum* × *Ae. mutica* progenies. The tetraploid hybrids had the A, B, and D

genomes from a homozygous *T. aestivum* and the Mt genome from naturally heterozygous *Ae. mutica*.

Dover and Riley counted the chiasmata in seven hybrid families, and found that the chiasma frequencies for the individual plants fell into four classes with mean chiasma frequencies of 1.51, 4.13, 7.06 and 12.91 chiasmata per cell. In the lowest pairing class homoeologous pairing was prevented and there was a high frequency of univalents. In the highest pairing class bivalents, trivalents and quadrivalents were formed. The segregation into four pairing classes indicates that more than two alleles are involved, possibly two alleles at each of two loci. One family segregated 1:1:1:1, indicating heterozygosity at both loci in the parent plant.

The discovery in *Ae. mutica* of this variation in factors regulating chromosome pairing suggests that similar variation may be present in other diploid species. The effect of such variation in the diploids is not known, but if it is effective in hybrids lacking the *Ph* allele, it provides material for the immediate establishment of regular bivalent formation in the new spontaneous amphiploid, that is, a diploid with low pairing alleles would give a classical allopolyploid. The finding of this variation in a diploid provides evidence that the *Ph* allele need not have arisen at the tetraploid level. If similar variation exists in *Ae. speltoides* it would add weight to the evidence that this was the donor of the B genome of wheat.