crosses with *YBR* and another strain *CH3/HeHa* proved that the structural gene of glucuronidase in *YBR* was not mutated and showed that *YBR* lacks a specific factor essential for the insertion of the enzyme into the endoplasmic reticulum. This factor is also required for the insertion of glucuronidase in *C3H/HeHa* and the gene which specifies it, the *E* gene, is in a different linkage group to the glucuronidase structural gene.

This is the first proof that a nuclear gene can specifically regulate the intracellular localization of an enzyme. This factor could work either by altering the structure of native glucuronidase so that it can fit into the reticulum or, more probably, by providing a specific binding site for the enzyme; it may even be a structural component of the reticulum membrane. If so, the implication is that other enzymes associated with the reticulum are localized by similar nuclear genes and the localization of enzymes in other organelles may also be genetically controlled. Certainly this would explain the highly specific distributions found in practice.

In mitochondria, where the correct assembly of enzymes is critical for co-ordinated function, Woodward and Munkres (1966) found evidence that the precise conformation of malic dehydrogenase, which is specified by a nuclear gene, is determined by interaction with mitochondrial structural protein, which is specified by the organelles DNA. Specification of enzyme binding sites could be one of the major functions of plastid DNA, whereas in organelles which do not contain DNA this could be effected by nuclear genes like the *E* gene discovered by Ganschow and Paigen.

Plant Virus in Vectors

from our Microbiology Correspondent

SINCE 1960 several research groups have demonstrated by electron microscopy the presence of insect-borne plant viruses within their insect vectors. Dr Karl Maramorosch and his colleagues Hirumi and Granados have concentrated their efforts on the wound tumour virus (WTV) and studied its distribution and multiplication in leafhoppers. The location of WTV in a relatively efficient vector, *Agallia constricta*, and in an inefficient vector, *Agalliopsis novella*, is described in two recent publications (*J. Virol.*, **1**, 430; 1967; and *J. Invert. Pathol.*, **9**, 147; 1967), and the observations made are related to the transmitting efficiencies of the leafhoppers. In the inefficient vector, wound tumour virions were distributed systemically in the midgut, musculature, blood, fat bodies, tracheoblasts and ventral ganglia. This is the first direct evidence of plant viruses invading the nervous tissues of insect vectors and corroborates the earlier antigenic analyses of Sinha (*Virology*, **26**, 673; 1965). However, Sinha's experiments did not distinguish between complete viruses, sub-viral constituents and soluble viral antigens.

The accumulation of WTV in all organs of *A. novella* was slight and, significantly, the low titre or complete absence of virions in the salivary glands suggests an immediate reason why this species is a poor vector for this virus. Although systemic infection of these leafhoppers by WTV is clearly indicated, the establishment of a disease condition has not been verified. The authors rightly point out that the killing of

occasional vectors could easily be missed and suggest that it is improbable that such systemic viraemia could remain completely harmless. Invasion of the insect nervous system was examined fully in *A. constricta*, the more efficient transmitter of the disease. Three types of viral formation occurred in nervous tissues: isolated virions (V_1) found predominantly in the ganglion cells; tubular formations (V_2) in which single files of virions lie within membranous envelopes; and aggregate formations (V_3) bounded by limiting membranes. These latter inclusion bodies were distributed mainly in the glial cells, perineurium and tracheoblasts. Wound tumour virus produces an intense cytopathogenic effect on nerve cells and, most notably, the cytoplasm of degenerating ganglion cells was converted into an electron dense matrix containing fibrous material and ribosomes. These matrix areas surrounded by WTV particles closely resemble those *in situ* aggregations of pox-viruses, reo-viruses and polio-viruses which are thought to represent centres of virus synthesis. A reasonable inference from the present study is that the V_1 -type virions associated with polyosomes are early stages of virus development and that the dense matrix areas indicate active sites of WTV multiplication. In contrast, the V_3 -aggregations which are rarely associated with the matrix areas or polyosomes are considered to denote virus storage. The significance of the tubular symmetry in virus development is not known. They have been detected before in non-nervous tissues of the vectors and in host plants while similar structures are common to other insect-borne plant virus systems. Maramorosch's group are at present using leafhopper culture cells to pursue this problem further and their future reports will be awaited with interest.

Hydroxyproline in Cell Walls

from a Correspondent in Cell Biology

HYDROXYPROLINE, an imino-acid which is found in only a few proteins such as animal collagen, also occurs in a protein of plant cell walls. This protein, sometimes called extensin, is linked to the cellulose microfibrils of the wall. One of its properties is to increase the tensile strength of the wall but it also seems to be associated with cell growth. Free hydroxyproline inhibits cell growth at 1 mM, and even does so in the presence of indole acetic acid (IAA)—a plant hormone, which greatly stimulates cell enlargement. A great deal of work published this year attempts to pin-point the site of hydroxyproline inhibition in the hope that this will give a clue to the mechanism of cell growth regulation.

IAA is thought to enhance cell growth through the stimulation of RNA and protein synthesis. Cleland (*Plant Physiol.*, **42**, 1165; 1967), using oat seedling coleoptiles, has shown that free hydroxyproline does not affect these two processes. Instead, he proposes that hydroxyproline blocks the utilization of a particular protein rich in hydroxyproline, possibly by feedback inhibition, needed for wall growth. Earlier, Cleland had shown (*Plant Physiol.*, **42**, 271; 1967) that the effect of hydroxyproline, on growth stimulated by IAA is not immediate. This suggests that a pool of this protein is present and is used up before inhibition takes place. Experiments with cycloheximide and *p*-fluorophenylalanine suggest that the protein is being synthe-