VIROLOGY

Simultaneous Activation of Pentose Phosphate Shunt Enzymes in a Virusinfected Local Lesion Host Plant

In the course of work on the biochemical basis of plant virus localization it has been found that around the necrotic lesions of Nicotiana glutinosa leaves infected with tobacco mosaic virus (TMV) the dehydrogenases are highly activated as shown by the in vivo application of the triphenyl tetrazolium chloride (TTC) technique¹. Because of the possible implication of oxidative metabolism in resistance phenomena, the nature of the enzymes involved was examined. Our attention was directed to the pentose phosphate cycle enzymes as alterations in their activities were recently reported to occur in host tissues infected by fungal pathogens^{2,3}. Isotope investigations also suggested an increased in vivo role of the pentose phosphate cycle in these hosts^{4,5}. Virus diseases have not been investigated from this point of view so far, although the host-virus systems offer distinct advantages; the direct contribution of the pathogen to the enzymatic changes occurring in the host was in this way excluded. In the present work *Nicotiana tabacum* var. White

Burley plants were inoculated mechanically with the para strain of TMV⁶. The infection resulted in the development of necrotic local lesions surrounded by a yellow ring of living tissue. Infiltration with 0.6 per cent TTC led to the rapid appearance of red colour around the lesions, indicating the area of enhanced dehydrogenase activity. From the corresponding tissues of a number of infected leaves not infiltrated with TTC, disks were punched out with a miniature cork borer (2.5 mm. d). The cell-free extract of 120 disks was used for the spectrophotometric assays. An equal number of disks punched from the normal green areas of the same leaves served as controls.

A representative experiment is shown in Fig. 1. It may be seen that in the tissues surrounding the lesions



Fig. 1. Activity of glucose-6-phosphate dehydrogenase in White Burley tobacco leaves infected with the para strain of TMV. •, Extract from tissues surrounding local lesions; \bigcirc , extract from normal green areas of the same leaf. Composition of the system: 2.5 μ M glucose-6-phosphate, 0.25 μ M triphosphopyridine nucleotide, 1 μ M magnesium chloride. Enzyme corresponding to 20 mg fresh weight. Glycyl-glycine buffer pH 7:5. Final volume 1.5 ml. Light path, 0.5 cm

From a biochemical point of view, it is quite remarkable that two members of a cycle are affected simultaneously in the same sense despite the fact that the two enzymes are distinct, soluble proteins. The nature of increased activity (de novo protein synthesis, removal of inhibitors, activation) is under investigation.

The role in host-virus interactions of the phenomenon described here is not fully understood; however, it might be postulated that the enhanced production of reduced triphosphopyridine nucleotide by the dehydrogenases involved plays a part in maintaining a high phenol/quinone ratio, thereby hindering the growth of the lesions⁷. Further investigations along these lines might contribute to the elucidation of the biochemical nature of localized acquired resistance of the tissues surrounding the virus-induced lesions^{8,9}.

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Serological Relationship of Chrysanthemum Virus B to Carnation Latent Virus and Potato Viruses M and S

WHEN leaf extracts from chrysanthemum plants infected with chrysanthemum virus B (CVB) were tested against antisera to 22 different elongated plant viruses, those prepared against carnation latent virus (CLV), potato virus M (PVM) and potato virus S(PVS) gave a positive serological reaction. In reciprocal tests an antiserum prepared against CVB itself reacted positively with partially purified preparations of CLV, PVM and PVS.

These results indicate that CVB should be included in the group of plant viruses hitherto represented by CLV, PVM and PVS, the serological relationships of which have already been established among previous authors1-4.

In Table 1 the average homologous titres of the antisera to CLV, CVB, PVM and PVS respectively are summarized together with their titres against the heterologous viruses of the group.

| | Table 1. VIRUS | PREPARATION | TESTED | |
|--------------------------|---------------------------|---------------------------|-------------------------|------------------------|
| Antiserum to : | CLV | CVB | PVM | PVS |
| CLV CVB PVM PVS | $8192 \\ 256 \\ 16 \\ 64$ | $32 \\ 2048 \\ 16 \\ 128$ | 128 32 2048 32 | 32 64 16 4096 |

The relatively large differences between homologous and heterologous titres suggest a major antigenic fraction specific for each virus together with