Competitiveness Between Genotypes of Raspberry Ringspot Virus is Mainly Determined by RNA-1

(Accepted 17 February 1976)

SUMMARY

Chenopodium quinoa plants were inoculated with pairs of pseudo-recombinant isolates of raspberry ringspot virus that shared one part of their genome but not the other. Results of typing progeny virus obtained from systemically infected leaves indicated that RNA-1 from different virus strains differed in competitiveness, and also in ability to allow the expression of differences in competitiveness between RNA-2 from different strains. Ability of genotypes to dominate was related to the rapidity with which they induced systemic symptoms in singly infected plants.

The RNA genome of raspberry ringspot virus (cryptogram R/1 : 2·4/43 + 1·4/30 (or Σ 2·8/46) : S/8 : S/Ne, nepovirus group – see Gibbs & Harrison, 1976) is in two pieces, RNA-1 and RNA-2, which are both needed for infection (Murant et al. 1972; Harrison, Murant & Mayo, 1972a). When RNA-1 was taken from one strain of the virus and RNA-2 from another strain, pseudo-recombinant isolates were produced that combined some of the characters of each of their parents (Harrison, Murant & Mayo, 1972b). By studying the properties of a range of these pseudo-recombinants, determinants for various biological characteristics were assigned to one or other part of the genome (Harrison et al. 1974). For example, a determinant for transmissibility by vector nematodes is in RNA-2 whereas a determinant for ability to infect Lloyd George raspberry is in RNA-1. These are kinds of properties likely to influence the ecological success of any variant of raspberry ringspot virus, and another such property is the ability of a variant to compete in doubly infected plants with other variants of the same virus. In this paper we describe attempts to locate a genetic determinant or determinants for this property.

In these experiments, Chenopodium quinoa plants were inoculated with saps containing pairs of pseudo-recombinant isolates derived from strains E and LG of raspberry ringspot virus and chosen so that the members of each pair shared one part of their genome but not the other. For example, one pair consisted of an isolate containing RNA-1(E) + RNA-2(E) (here called EE) together with an isolate containing RNA-1(E) + RNA-2(LG) (here called EL). Other C. quinoa plants were inoculated with one or other of the two isolates, and each of these two inocula was also inoculated at a range of dilutions to leaves of C. quinoa or C. amaranticolor plants so that their relative infectivities could be assessed by counting the numbers of local lesions that developed in 7 to 10 days. All the isolates used were obtained as described by Harrison et al. (1974), and were derived from single local lesions in C. quinoa. Plants were dusted with 500-mesh carborundum before inoculation and were kept in a glasshouse at 15 to 25 °C.

Ten to sixteen days after inoculation, when the doubly infected C. quinoa plants and the singly infected controls were fully systemically infected, sap from their stem tips was inoculated at a range of dilutions to a second series of C. quinoa or C. amaranticolor plants. Single-lesion isolates were then made from leaves in which the lesions were well spaced, by inoculating juice from each lesion ground in a drop of 0·06 M-phosphate buffer, pH 7·3, to another
C. quinoa plant. All well separated lesions in any source leaf were used, to avoid selection. The genotype of each single-lesion isolate so obtained was then determined; the kind of RNA-2 was deduced by serological typing and that of RNA-1 by the local and systemic symptoms induced in C. quinoa (Harrison et al. 1974).

Table 1 gives the results of a series of experiments. In every experiment the single-lesion isolates derived from singly infected plants had the expected genotype and these results are not shown in the Table. When plants were doubly infected with EE and EL, both genotypes were found equally often among the single-lesion isolates of progeny virus, but in each of the other three pairs of isolates one genotype predominated over the other. EE predominated over LE, LL predominated over LE, and EL predominated over LL. This occurred even when the dominant genotype was used at a smaller infectivity in the original inoculum than the less dominant one (expt. 3, 4, 6 and 8, Table 1). In these experiments, different single-lesion isolates of the same constitution behaved similarly; for example, different single-lesion isolates of EE and EL were used in expt. 1 and 2 (Table 1) and different single-lesion isolates of LL and EL in expt. 7 and 8. We conclude that different kinds of RNA-1 differ in ability to dominate in this system, and that RNA-1 determines whether or not differences in competitiveness between different kinds of RNA-2 are expressed. Hence the competitiveness of a genotype is mainly determined by RNA-1 but RNA-2 can play a secondary role.

This series of experiments gave no evidence about the nature of the property that determines ability to predominate in the doubly infected C. quinoa plants. However, one might expect that the first genotype to become established in the shoot tip would tend to predominate in the virus population produced later. We therefore determined the time needed for isolates of the different genotypes to induce systemic symptoms in C. quinoa. The results of two experiments made at different times of year (Table 2) show that EL and EE produced symptoms the soonest, followed about a day later by LL, and after a further day and a half by LE. Ability of a genotype to dominate, therefore, is correlated with speed of induction of systemic symptoms, and thus probably with speed of movement to the shoot tip, which seems likely to be an important factor in establishing dominance in the kind of experimental system we have used.

That speed of systemic movement is unlikely to be the only factor involved in dominance is suggested by the results of an earlier experiment in which Chenopodium quinoa plants systemically infected with strain LG were inoculated with strain E (Murant, Taylor & Chambers, 1968). No additional symptoms developed but virus recovered subsequently
Table 2. Days taken for pseudo-recombinant isolates of raspberry ringspot virus to induce systemic symptoms in Chenopodium quinoa

<table>
<thead>
<tr>
<th>Expt.</th>
<th>Inoculum dilution</th>
<th>Virus isolate</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>EE</td>
<td>EL</td>
</tr>
<tr>
<td>1</td>
<td>1/50</td>
<td>5.0*</td>
<td>--</td>
</tr>
<tr>
<td></td>
<td>1/250</td>
<td>5.3</td>
<td>--</td>
</tr>
<tr>
<td></td>
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<td>5.1</td>
<td>--</td>
</tr>
<tr>
<td>2</td>
<td>1/50</td>
<td>5.9</td>
<td>5.9</td>
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<tr>
<td></td>
<td>1/250</td>
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<td>5.7</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>6.1</td>
<td>5.8</td>
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</tbody>
</table>

* Figures are means for four or five plants.

from the shoot tip was more virulent than strain LG, implying that the RNA-I of strain E had become established there (Harrison et al. 1974). In this system, therefore, the RNA-I of strain E seems at least partially to have replaced that of strain LG in plants that were already infected systemically. This provides a further illustration of the greater competitiveness of the RNA-I of strain E as compared with that of strain LG.

K. Hanada is indebted to the Japan Ministry of Education for financial support.

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REFERENCES

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(Received 31 December 1975)