of naturally occurring severe strains of PMV, and 3) potential synergistic interactions with unrelated viruses. Cowpeas can serve as a source of PMV transmitted in all commercial cowpea cultivars and occurs in all plantings. In a mixed infection with the potyvirus BICMV, CMV causes a synergistic disease reaction (11). PMV also is a potyvirus and it also caused a synergistic reaction with CMV in one cowpea cultivar although not as strong a reaction as the combination of CMV and BICMV. Several cowpea lines were identified as resistant to PMV. Virus could not be isolated from inoculated plants, and no natural infection occurred in one of the lines planted in a field test.

LITERATURE CITED
Table 1. Results of typical European Clubroot Differential (ECD) test using a clubroot sample collected from broccoli at Mount Vernon, WA

<table>
<thead>
<tr>
<th>ECD set</th>
<th>Symptom category*</th>
<th>Disease indexb</th>
<th>Reaction typec</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brassica campestris</td>
<td>01 29 0 0 0</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>Brassica napus</td>
<td>06 23 2 1 0</td>
<td>5</td>
<td>?</td>
</tr>
<tr>
<td>Brassica oleracea</td>
<td>10 0 1 8 5</td>
<td>77</td>
<td>+</td>
</tr>
</tbody>
</table>

*0 = No clubs; 1 = small clubs on lateral roots, 2 = small clubs on main root not girdling root, and 3 = large club girdling main taproot at or near soil level.

bDI = Σ (no. of plants in each symptom category × category no.) × 100 / total no. plants

*Reaction type based on DI cutoff point of 33: susceptible = “+” (DI >33); resistant = “−” (DI = 0 or “?” (0 < DI < 33).

Table 2. Verification of uncertain (“?”) virulence genes by reinoculation of European Clubroot Differential (ECD) hosts with spores collected from specific ECD differential hosts

<table>
<thead>
<tr>
<th>ECD set designation</th>
<th>Symptom category*</th>
<th>Disease indexb</th>
<th>Reaction typec</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clubs from ECD cultivar 06 reinoculated on:</td>
<td>06 0 0 7 12</td>
<td>87</td>
<td>+</td>
</tr>
<tr>
<td>Clubs from ECD cultivar 08 reinoculated on:</td>
<td>08 8 0 0 2</td>
<td>20</td>
<td>?</td>
</tr>
</tbody>
</table>

*0 = No clubs; 1 = small clubs on lateral roots, 2 = small clubs on main root not girdling root, and 3 = large club girdling main taproot at or near soil level.

bDI = Σ (no. of plants in each symptom category × category no.) × 100 / total no. plants

*Reaction type based on DI cutoff point of 33: susceptible = “+” (DI >33); resistant = “−” (DI = 0 or “?” (0 < DI < 33).

Table 3. Virulent genes in collections of Plasmodiophora brassicae from West Coast states of the United States defined by the European Clubroot Differential set

<table>
<thead>
<tr>
<th>Host plants and locations of Plasmodiophora brassicae collectionsa</th>
<th>Disease reaction typesb</th>
<th>Pathotype numerical designacionc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chinese cabbage, Puyallup, WA</td>
<td>B. campestris</td>
<td>B. napus</td>
</tr>
<tr>
<td>Collard, Puyallup, WA</td>
<td>01 02 03 04 05</td>
<td>06 07 08 09 10</td>
</tr>
<tr>
<td>Mustard, Puyallup, WA</td>
<td>Chinese cabbage, Fir Island, WA</td>
<td>Cauliflower, Gresham, OR</td>
</tr>
</tbody>
</table>

a All Brassica oleracea L. except Chinese cabbage, B. campestris L., and B. juncea (L.) Coss.

bReaction type based on DI cutoff point of 33: susceptible = “+” (DI >33); resistant = “−” (DI = 0) or “?” (0 < DI < 33).

cECD hosts in each Brassica spp. group were assigned values of 1, 2, 4, 8, and 16, respectively. By adding the values for each susceptible reaction, a unique virulence designation is obtained (3), eg, 16/3/31 represents positives reactions for 05, 06, 07, and 11-15. All uncertain reactions (?) were treated as resistant.

RESULTS

Table 1 illustrates a typical ECD reaction to a clubroot sample (sample F) collected from broccoli in Washington state. Based on a DI cutoff point of 33, ECD 09 gives a resistant reaction indicating no virulence genes present in the pathogen, whereas ECD 07 exhibits a susceptible reaction showing the presence of virulence genes. When spores from clubs of an ECD host with an uncertain (“?”) reaction, eg, ECD 06 and 08 in Table 1, were reinoculated onto appropriate ECD hosts, ECD 06 proved to be susceptible, whereas ECD 08 remained “uncertain” (Table 2).

Table 3 summarizes the virulence after reinoculation of the populations. Collections were remarkably homogeneous with respect to ECD virulence codes that were either 16/02/31 or 16/03/31.

DISCUSSION

Assigning each pathotype a coded response to the ECD hosts based on susceptible reactions requires a clear distinction between resistant (−), and
susceptible (+) reactions. This was often not clear-cut. A DI cutoff point was needed to permit one to establish arbitrarily whether a host was susceptible or resistant based on the disease data. Although no established cutoff point has been agreed upon, a DI of 33 has frequently been used by other workers and was followed in this work.

Numerical pathotype designations must be interpreted carefully. Field collections of resting spores may consist of mixtures of virulent pathotypes (4). One pathotype may be dominant and easily identified; another pathotype occurring at a very low frequency may infect only a few plants of a susceptible ECD host and be overlooked as insignificant. To avoid such a host being mislabeled as resistant, all plants showing questionable reactions were reinoculated with spores from the same infected ECD host so that the frequency of the pathotype or virulence gene was greatly increased to the point where a susceptible reaction could be seen (Tables 1 and 2, ECD 06). Williams (9) and Crute et al (4) have also stressed that any code can be misleading because hiding virulence genes can occur at low levels where infection of susceptible hosts is below the cutoff point, thus being rated resistant.

In cases where the virulence code did not change upon reinoculation, a lack of genetic homogeneity of certain ECD hosts would make adequate pathotype differentiation impossible in a single screening. Susceptible plants in genetically impure differential stocks may account for one or two severely infected plants even after reinoculation (Table 2, ECD 08).

Despite the lack of genotypic homogeneity of certain differentials, the populations of P. brassicae collected in California, Oregon, and Washington show considerable similarity in their virulences (Table 3). Generally, there was little or no virulence in the pathogen populations for the B. campestris group except for the universal susceptible (05), more virulence for the B. napus group, and complete virulence for the B. oleracea group. In preliminary studies, one of us (J. Robak) obtained an ECD code 16/02/31 for spores collected from Chinese cabbage in Puyallup, WA, from a mustard species in Willamette Valley, OR, and from cabbage in Tillamook, OR, and in Mount Vernon, WA (6). A clubroot collection from broccoli grown in Corvallis, OR, initially showed low virulence to the B. napus group (ECD 16/0/31) but upon reinoculation, virulence was determined to be 16/02/31 (Table 3). ECD results with similar virulence patterns have also been reported with collections from British Columbia (8).

In terms of pathotypes or races, race 7 virulent on ECD 11 (Badger Shipper) is present as Williams (9) reported in 1966. This is in contrast to race 6, which is avirulent on Badger Shipper and which is predominant in the eastern United States (7,9). Race 6 may also be present in the western states but could not be detected because genes virulent to ECD 11 were present in all collections. To clearly separate mixtures of pathotypes, single spore isolates would be required.

LITERATURE CITED