A chapter of mine (2) now in the press covers almost all of the
points raised by Browder and Eversmeyer. It even covers more
about Browder and Eversmeyer's own temperature studies than
they themselves now include in their letter to you. I shall not repeat
the chapter here; what needs to be covered now is relatively little.

My thesis was (1) and still is: “What specificity there is, is in
susceptibility. Resistance is unspecified.” Put differently,
susceptibility may be, but is not necessarily, specific; resistance is
always unspecified. If one goes from disease in general to gene-for-
gene disease in particular, the thesis becomes: Susceptibility is
specific, resistance unspecified. I deal in this reply with gene-for-gene
disease only.

Browder and Eversmeyer clearly do not understand my tables.
They strip my 5 × 5 table (1, Table 2.1) down to a 2 × 2 table (their
Table 1) shedding degrees of freedom in the process. Their 2 × 2
table is left with only one degree of freedom. That degree of
freedom indicates interaction, but more degrees are needed to show
the direction of the interaction. Put differently, this 2 × 2 table
indicates specificity, but cannot show where that specificity lies. No
2 × 2 table can distinguish between specific susceptibility and
specific resistance. A distinction between specific susceptibility and
specific resistance was the whole point of my original 5 × 5 table.

My Table 1, given here, extends Browder and Eversmeyer's 2 × 2
Table 1 into a 3 × 3 table, and gives the necessary extra degrees of
freedom. Specific susceptibility and unspecific resistance begin to
glimmer through. A 4 × 4 table, which readers can construct for
themselves, is more emphatic, and my original 5 × 5 table better
still. I hope readers will consult my original tables in place of
Browder and Eversmeyer's pointlessly truncated ones. In my
chapter (2, Section VII-B) I devise a “multiquadratic” check to
supply degrees of freedom in a different way.

Flor, in repeated formulations of his gene-for-gene hypothesis, is
entirely consistent and unequivocal. For each resistance gene in the
host there is a specific complementary gene conditioning
pathogenicity (my italics) in the parasite. Flor's hypothesis is about
specific pathogenicity. There is unfortunately a large school that
surreptitiously contradicts Flor and substitutes a gene conditioning

<table>
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<tr>
<th>Pathogen genotype</th>
<th>R1r1</th>
<th>R1r2 R2r3</th>
<th>R1r1 R2R3</th>
</tr>
</thead>
<tbody>
<tr>
<td>v1v1</td>
<td>2/1</td>
<td>2/1</td>
<td>2/3</td>
</tr>
<tr>
<td>v1v2</td>
<td>2/1</td>
<td>2/2</td>
<td>2/3</td>
</tr>
<tr>
<td>v1v3</td>
<td>2/1</td>
<td>2/2</td>
<td>2/3</td>
</tr>
</tbody>
</table>

*Genes conditioning susceptibility in the host plant and genes conditioning
avirulence in the parasite do not enter Flor's formulation of his gene-for-
gene hypothesis, and were rightly omitted from my 5 × 5 table (1, Table
2.1). They are included here only to conform with Browder and
Eversmeyer's Table 1, but are unnecessary.

avirulence for a gene conditioning pathogenicity in the parasite.
That is, they substitute a gene-for-gene system in which the gene for
resistance in the host is matched by a specific complementary gene
conditioning avirulence in the parasite. Thus, Browder and
Eversmeyer write “Resistance [in Flor's system] was the result of
specific host genotypes and specific parasite genotypes functioning
together” (italics added by me). In short, Browder and Eversmeyer
believe in specific avirulence. I demonstrate (2, Section II-A), I
believe conclusively, that no gene-for-gene system can possibly be
devised on the basis of avirulence in the parasite. Attempts to
launch a concept of specific resistance on the back of specific
avirulence must fail.

To digress into terminology, one repeatedly sees references to
"specific resistance" to disease when specific resistance does not
exist. There is no such thing as specific resistance to disease.

LITERATURE CITED
   Academic Press, Orlando, FL.
2. Vanderplank, J. E. 1986. Specific susceptibility and specific feeding