The Sorting and Analysis of Infection Types from *Triticum aestivum/Puccinia recondita* Interactions

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Accepted for publication 6 May 1981.

Browder and Eversmeyer (1) outlined a procedure for computerized sorting of sets of data consisting of infection types (ITs) from interactions between cultivars of *Triticum aestivum* L. emend. Thell. and cultures of *Puccinia recondita* Rob. ex. Desm. They indicated that their objective was to sort "IT data toward the model shown by Robinson," and to use the sorted data to determine the minimum number of corresponding gene pairs (CGPs) that will explain the variation in the ITs, and to compare known and unknown host genotypes. It is not clear which model they meant since Robinson (4) described two, the Person model and the Person/Habgood model, although the latter was elaborated by Robinson. However, in their abstract they stated: "This sorting resulted in a table in which lines and cultures were arranged with a generally increasing number of low infection types from top to bottom and a generally increasing number of high infection types from left to right, as in the theoretical gene-for-gene model." This pattern is characteristic of the Person model when host genotypes are listed down the side and pathogen genotypes across the top, but not of the Person/Habgood model in which the frequency of either low infection types (LITs) or high infection types (HITs) cycles from low to high, back to low and up to high again, etc.

Browder and Eversmeyer (1) used their sorting procedure for a set of data involving the reaction of 14 lines of wheat to seven cultures of *P. recondita*. Examination of their Table 2, which was obtained following sorting, shows that the number of LITs in each row, going down the rows, was 0, 6, 2, 3, 4, 4, 7, 2, 4, 6, 3, 7, 7, 7. These numbers do not appear to follow the pattern of gradually increasing numbers of LITs suggested, and we do not see how they fit the Person/Habgood model either. The number of HITs in each column was less variable but the number in each column going from left to right was 1, 3, 7, 7, 6, 7, 5. Again there was no obvious pattern. In fact, it can be shown that the sorting procedure described by Browder and Eversmeyer (1) will result in many different sorted tables depending on the order of the host lines and pathogen cultures in the original unsorted table; ie, their Table 2 is only one of many possible tables that could be generated by the sorting procedure. This is the case because the final order of the host lines and pathogen cultures depends largely on the differences among the ITs in the last row and last column of the unsorted data set. These ITs are sorted in descending order and determine the final order of the pathogen cultures and host lines, respectively. This method gives greater weight to small differences among ITs in the last row and last column of the unsorted data, than to the total number of HITs or LITs in any row or column, despite the fact that the identification of CGPs is based on whether ITs are high or low. If a third sorting is required, as described by Browder and Eversmeyer (1), then the last row after the second sorting becomes the key one. However, the order of the ITs in the preceding rows and columns does have some effect, since, when several entries in one row or one column have identical ITs, their order remains the same as it was in the preceding row or column. The following example will illustrate the effect of a deliberate change in the order of the host lines in the unsorted table. If the cultures in Browder and Eversmeyer's Table 1 are numbered from 1 to 7 from left to right, then in their sorted Table 2, the order is 1, 2, 3, 5, 7, 6, 4. If, however, in their unsorted Table 1, Browder and Eversmeyer (1) had placed the susceptible check, Marquis, at the top instead of the bottom and had put the remaining three cultivars at the bottom of the table in alphabetical order, Chiccoro 'S,' Era, and Waldron, the order of the cultures after sorting would have been 1, 2, 7, 6, 5, 3, 4. Similarly, for the lines the order derived from the rearranged table would be 14, 10, 8, 5, 9, 3, 7, 12, 1, 13, 2, 11, 4, 6, compared to their order in Table 2, of 14, 10, 8, 5, 9, 3, 7, 12, 2, 11, 1, 13, 4, 6. Thus, the order of the cultures has changed greatly, and the order of the lines somewhat less. Other reorganizations of the cultures and lines in the unsorted table would result in many different sorted tables. The ineffectiveness of the sorting procedure is also shown in the sorted data of Browder and Eversmeyer (1), in which two lines with almost identical infection types, LR10(TC) and Era, occur six rows apart.

Browder and Eversmeyer (1) stated that they tested their procedure using data from a theoretical model with five CGPs and that, regardless of whether the unsorted data were random or arranged according to the theoretical model, the sorting procedure returned it to the "theoretical order identically." We set up data fitting either the Person or the Person/Habgood model. When the sorting procedure was run on the theoretical data for either model, the order remained unchanged. However, when we randomized the data for either set and then used the sorting procedure, it did not return to the theoretical order in either case. Presumably, by chance Browder and Eversmeyer (1) obtained a randomization with an order such that the sorting procedure returned it to the theoretical form, but we did not. That this can happen can be predicted on the basis of the process involved in the sorting. As we have already pointed out, the result of the sorting depends largely on the differences among the ITs in the last row and first column of the unsorted data. Some of the possible randomizations will be such that the sorting returns the data to the form of the theoretical model, but many will not.

The two gene-for-gene models discussed by Robinson (4) showed the interaction between sets of host and pathogen genotypes that included all of the possible genotypes for the GCPs involved. Any real set of data will almost certainly be derived from groups of host lines and pathogen cultures that do not include all of the possible genotypes. In the absence of complete genetic information for all lines and cultures, we do not see how such data can be sorted to fit a Person/Habgood model. However, it can be sorted quite easily into a form that approaches the Person model, and that simplifies the genetic analysis of the host and pathogen, and the identification of CGPs. All that is necessary is a simple extension of the procedure outlined by Loegering and Burton (3). They first classified their infection types as high or low. The cultivars were then arranged in descending order with those having the most HITs first. Loegering and Burton (3) left the cultures in random order. However, to arrange the data as closely as possible to the gene-for-gene model, the cultures should be placed in ascending order from left to right for number of HITs.

In Browder and Eversmeyer's (1) data there is a large gap in the infection types between 99P, which is high, and all of the others, which can be classified as low. We reorganized their data by the procedure outlined above (Table 1). In several cases, two or more host lines had the same number of LITs and two or more cultures had the same number of HITs. Within these groups the lines or cultures can be placed in an order corresponding to the gene-for-

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0031-949X/81/10101003/$03.00/0

gene model only if the genotypes of both the lines and cultures are known, which is not the case here. For convenience, we placed the lines or cultures with the same number of LITs or HITs in order, with those having the highest LITs first. This has no particular bearing on the analysis and is not essential, but it does tend to put lines or cultures with similar infection types into adjacent rows or columns, respectively. The data were then analyzed by the procedure outlined by Loegering and Burton (3), and also used by Browder and Eversmeyer (1), going down the table row by row. As shown by Loegering and Burton (3), if two host lines and two pathogen cultures interact to give a box of the following type (their type A),

LIT 1	HIT	
		(the LIT may be in any corner)
HIT	HIT	

then one CGP is operating, and the genotypes of the two lines and the two cultures can be specified. Neither the two lines nor the two cultures have to be adjacent. If two host lines and two pathogen cultures interact to give a box of the following type (their type B),

LIT 1	HIT	
		(two LITs on a diagonal to two HITs)
HIT	LIT 2	

then two CGPs are operating, and again the genotypes of both lines and both cultures can be determined. If one of the HITs in box type B is replaced by a LIT (their box type E), then two CGPs are operating, but the genotypes of the line and culture that interact to give the addition LIT cannot be completely determined. Although nine of the hosts used by Browder and Eversmeyer (1) were single-gene, backcross lines, it is clear that they analyzed the data as though all the resistance genes were unknown.

Our analysis based on Table 1 is as follows:

1. LR10(TC)—The underlined LIT with culture 4 shows that LR10(TC) carries a gene for low reaction, presumably *Lr10*, that is not carried by Marquis, and the occurrence of a CGP is shown by the type A box that can be formed. It cannot be determined unequivocally that the LIT with culture 1 is also due to *Lr10*.
2. Era—Era gave the same pattern of ITs as LR10(TC) and probably carries *Lr10*. The slight difference in the LITs (02C vs 03C) could be due to a difference in the genetic backgrounds of Era and LR10(TC).
3. LR2D(PL)—The underlined LIT shows that LR2D(PL) carries a gene for low reaction, presumably *Lr2d*, not carried by Lr10(TC), and the occurrence of the second CGP is shown by the type B box that can be formed. It cannot be proven that the remaining two LITs are caused by *Lr2d*. We are aware that Dyck and Samborski (2) showed that *Lr2c* and *Lr2d* are the same, but to avoid confusion we have used *Lr2d* as was done by Browder and Eversmeyer (1).
4. LR1(TC)—The underlined LIT shows that LR1(TC) carries a gene for low reaction, presumably *Lr1*, that is not present in LR10(TC) or LR2D(PL), and the occurrence of the third CGP is shown by the two type B boxes that can be formed. Browder and Eversmeyer (1) did not identify a CGP involving *Lr1* because they had already identified a CGP for *Lr17* which conditioned an LIT to all three cultures to which *Lr1* conditioned an LIT.
5. LR17(TC)—A comparison of the underlined LIT with the ITs above it shows that LR17(TC) carries a gene for low reaction that is not present in either LR10(TC) or LR2D(PL). However, since LR1(TC) also gives a LIT with culture 3, it cannot be concluded that the LIT for LR17(TC) is due to the interaction of a different CGP. Nevertheless, the reactions to culture 7 show that LR17(TC) carries a gene for low reaction not carried by LR1(TC). Thus, it carries either *Lr1* plus a second gene, presumably *Lr17*, or *Lr17* alone. The fact that LR17(TC) gives a slightly higher reaction to culture 3 than does LR1(TC) (23C vs 01C) makes it unlikely that it carries *Lr1*.
6. LR3(TC)—A comparison of the underlined LIT with the ITs above it, shows that LR3(TC) carries a gene that is not present in LR10(TC), LR2D(PL), LR1(TC) or LR17(TC), and presumably is *Lr3*. The formation of type B boxes with the latter three lines and a type E box with LR10(TC) demonstrates the occurrence of a fourth CGP.
7. LR2A(TC)—A comparison of the underlined LIT with the ITs above it, shows that LR2A(TC) carries a gene that is not present in LR10(TC), LR2D(PL), LR1(TC), LR17(TC), or LR3(TC), and is presumably *Lr2a*. The formation of a type E box with LR2D(PL) and a type B box with each of the other four lines demonstrates the occurrence of a fifth CGP.
8. LR16(TC)—A comparison of the underlined LIT with the ITs above it shows that LR16(TC) carries a gene that is not present in LR10(TC), LR2D(PL), LR1(TC), LR17(TC), or LR3(TC). Since both LR2A(TC) and LR16(TC) give an LIT with culture 6, it cannot be conslusively shown that LR16(TC) does not carry *Lr2a*. However, the reactions to culture 3 show that LR16(TC) carries a gene for low reaction, presumably *Lr16*, that is not present in LR2A(TC). Thus, it carried either *Lr2a* plus a second gene, presumably *Lr16*, or *Lr16* alone.
9. Waldron—A comparison of the underlined LIT with the ITs

TABLE 1. Infection-type data from *Triticum aestivum*:*Puccinia recondita* host-pathogen interaction trials with the *T. aestivum* lines sorted in ascending order for number of low infection types (LITs) from top to bottom and the cultures sorted in ascending order for number of high infection types (HITs) from left to right

Entry number	*T. aestivum* line name	Infection type produced by *P. recondita* culture: 1 UN01-68B	2 UN01-68A	4 UN09-66A	7 65359-01	3 UN17-68A	5 0967-1	6 6B-NA65-9	No. of LITs
14	Marquis	99 P	99 P	99 P	99 P	99 P	99 P	99 P	0
8	LR10(TC)	03 C	99 P	03 C(a,b)*	99 P	99 P	99 P	99 P	2
12	Era	02 C	99 P	02 C	99 P	99 P	99 P	99 P	2
5	LR2D(PL)	03 C	24 C	99 P	03 C(a,b)	99 P	99 P	99 P	3
1	LR1(TC)	01 C	01 C	99 P	99 P	01 C(a,b)	99 P	99 P	3
9	LR17(TC)	03 C	14 C	99 P	23 C	23 C(b)	99 P	99 P	4
3	LR3(TC)	03 C	03 C	03 C	99 P	99 P	03 C(b)	99 P	4
2	LR2A(TC)	01 C	01 C	99 P	01 C	99 P	99 P	14 C(b)	4
10	LR16(TC)	24 N	24 N	34 N	99 P	34 N	34 N	34 N	6
11	Waldron	01 C	01 C	03 C	01 C	99 P	13 C	03 C	6
7	LR24(Agent)	03 C	03 C	03 C	03 C	03 C	03 C	03 C	7
13	Chiccoro 'S'	01 C	01 C	03 C	23 X	01 C	03 C	23 X	7
4	LR9(TC)	01 C	01 C	03 C	01 C	01 C	01 C	01 C	7
6	LR19(TC)	01 C	01 C	02 C	01 C	01 C	01 C	01 C	7
Number of HITs		1	3	5	6	7	7	7	

*The underlined LITs have been used in the analysis. Any two LITs that are followed by the same letter and are not in the same column can be used to form the diagonal of a type B or type E box as described in the text. The two LITs do not need to be in adjacent columns.

above it, shows that Waldron carries a gene that is not present in LR10(TC), LR2D(PL), LR1(TC), LR17(TC), or LR3(TC). However, it cannot be shown that it does not carry *Lr2a* or *Lr16*.

10. LR24(Agent)—Since LR24(Agent) gave an LIT to all seven cultures, it cannot be conclusively demonstrated that it does not carry a combination of two or more of the genes in the lines above it, that would give a LIT to all cultures (eg, *Lr2a* plus *Lr16*). However, differences in the LITs between LR24(Agent) and the other lines suggest that this is not the case. Furthermore, the fact that it gives identical LITs to all cultures suggests that only one gene for low reaction, *Lr24*, is involved. Browder and Eversmeyer (1) stated that the CGP for *Lr24* had been demonstrated. However, the evidence is incomplete, since all cultures gave an LIT on LR24(Agent) and, therefore, there is no evidence for a corresponding gene in the pathogen that gives high pathogenicity on *Lr24*.

11. Since LR24(Agent) and the remaining three lines are identical to the extent that they give LITs to all seven cultures, no further genes can be demonstrated. In particular the lines LR9(TC) and LR19(TC) gave almost identical LITs and cannot be distinguished, even though they are known to carry different genes.

As noted by Browder and Eversmeyer (1), the number of CGPs that can be demonstrated cannot exceed the lesser of the number of lines or the number of cultures used (in this case seven). They demonstrated four CGPs involving *Lr10, Lr2d, Lr17, Lr3* and they claimed to have demonstrated the CGP for *Lr24*, although the evidence is incomplete. Our analysis demonstrated the presence of five CGPs involving *Lr10, Lr2d, Lr1, Lr3,* and *Lr2a*. The presence of CGPs involving *Lr16* and *Lr17* is also reasonably clear. The various differences in the analyses are partly due to differences in the order in which the lines and cultures appeared in their Table 2 and our Table 1. Our results also illustrate the value of the better sorting of the data obtained by our minor modification of the procedure used by Loegering and Burton (3) compared to the method of Browder and Eversmeyer (1).

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