# A Model for the Evolution of Formae Speciales and Races

Y. Tosa

Faculty of Agriculture, Kochi University, Nankoku, Kochi 783, Japan.
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The basis of host-parasite specificity has been discussed by many phytopathologists and other researchers. With the accumulation of experimental data from genetic, cytological, and biochemical studies, some basic concepts of host-parasite specificity have been developed. Day (2) distinguished two types of host-parasite interaction: One determines whether or not a plant is a host for a parasite, and the other determines the gene-for-gene specificity. Ellingboe (3) introduced the term *basic compatibility* to designate a state of harmony between a host and a parasite developed by their interactions over a period of time and assumed that the gene-for-gene relationship was superimposed on basic compatibility. Heath (6) presented a more generalized concept. Specificity determining host species range was termed plant species specificity, and specificity determining cultivar range within a given host species was termed cultivar specificity. In the former, compatibility is specific, and the resistance involved (nonhost resistance) is genetically complex. In the latter, incompatibility is specific, and the resistance involved (cultivar resistance) is controlled by gene-for-gene interactions. Cultivar resistance was considered to be superimposed on basic compatibility resulting from the first type of interaction. This model was called the basic compatibility model of specificity (9).

Although this model has been widely accepted as a basic concept, some experimental data might be interpreted as suggesting that nonhost resistance is controlled by major genes (12), or that plant species specificity is controlled by gene-for-gene interactions (9). To address the apparent contradiction between the concept and the evidence, Heath (9) presented a modified model for the evolution of biotypes (formae speciales in fungi or pathovars in bacteria) and races.

My coworkers and I showed that gene-for-gene interactions played a role in determining the forma specialis-genus specificity in the *Erysiphe graminis* DC.-gramineous plant system (15,18). Heath (9) suggested that our data could be explained by applying the modified model. In this letter I first discuss whether or not her modified model is compatible with our data, and second, present another model that illustrates the evolution of formae speciales and races of biotrophic fungi.

Heath's modified model assumes that parasite biotypes evolved from a common ancestor carrying potential avirulence (AVR) genes by acquiring different species specificity (SSP) genes. In her example, biotype 1 carries SSPW and SSPX to establish basic compatibility with plant species 1, and biotype 2 carries SSPY and SSPZ to establish basic compatibility with plant species 2. If so, these genes should segregate in progenies from a cross between the two biotypes. For simplification, let us assume that the parasite is haploid and that these genes are inherited independently. Then, a fourth part of the $F_1$ population from biotype 1 × biotype 2 carries neither SSPW nor SSPX. When plant species 1 is inoculated with the $F_1$ population, the progeny without SSPW or SSPX should induce nonhost resistance and distort ratios resulting from the segregation of AVR genes. Tosa (14) crossed *E. graminis* f. sp. *tritici* (parasitic on wheat) with *E. graminis* f. sp. *agropyri* (parasitic on wheatgrass), and inoculated wheat cultivars with their $F_1$ cultures. Segregation ratios of avirulent and virulent cultures suggested involvement of four major genes. Each of the four genes corresponded to a single resistance gene in wheat and was considered to be an AVR gene under the control of gene-for-gene interactions (15,18). The distortion of segregation ratios was not detected. Therefore, it is highly unlikely that the modified model is applicable to our system.

What if there are many SSP genes in a forma specialis that have small but additive effects like polygenes? In this case, $F_1$ cultures that carry no SSP genes would occur in a very low ratio and hardly affect the ratios resulting from the segregation of AVR genes. In order to reconcile Heath's (9) modified model with our genetic evidence, SSP genes must be like polygenes.

Here I will argue that the evolution of formae speciales can be explained by applying the original basic compatibility model (6), if the usage of the term *nonhost* is changed. A general definition of nonhost is very difficult to give (7). Niks (11) called a plant species a nonhost to a microbial strain when all members of the plant species were resistant to the strain. This definition implies that a plant species is a nonhost to a forma specialis if all members of the plant species are resistant to the forma specialis (11). Heath (8) argued that plant species that were taxonomically related to host species of a forma specialis in question, but were hosts of other formae speciales, should not be considered as typical nonhosts. However, it has been general usage to call such plant species nonhosts, and Heath (9) has followed this usage. The contradiction between the concept of the basic compatibility model and experimental evidence in fungal systems (9,12) appears to be attributable to the traditional usage of the term *nonhost*. In my opinion, wheat is a host not only of *E. g. tritici* but also of *E. g. agropyri*. *E. g. tritici* establishes basic compatibility with both wheat and wheatgrass. These ideas may be supported by cytological data. Tosa et al (16) found that *E. g. agropyri* penetrated cell walls of wheat leaves in the same manner as *E. g. tritici*, and that the resistance of wheat leaves to *E. g. agropyri* was attributable to the hypersensitive reaction of epidermal cells, a common factor in cultivar resistance. This was the case in wheatgrass leaves inoculated with *E. g. tritici*. Tosa and Sakai (18) concluded that the forma specialis-genus specificity is a case of cultivar specificity, rather than plant species specificity. On the basis of this concept, a hypothetical model was constructed that illustrated the evolution of formae speciales and races (Fig. 1).

An ancestral fungus became a parasite of a range of ancestral plant species by acquiring SSP genes to establish basic compatibility with the plant species. The parasite also carried potential avirulence genes (*Ax*1–*Ax*4 and *Ay*1–*Ay*4) that controlled characters other than avirulence. The host species gradually evolved into several genera, X, Y, and so on. In this process, plant genus X acquired resistance genes, *Rx*1 and *Rx*2, that recognized fungal features or products controlled by *Ax*1 and *Ax*2, respectively. A portion of the parasite population accommodated itself to plant genus X by rendering *Ax*1 and *Ax*2 nonfunctional through random mutations and became forma specialis X. On the other hand, plant genus Y acquired other resistance genes, *Ry*1 and *Ry*2, that corresponded to *Ay*1 and *Ay*2, respectively. Another portion of the parasite population became virulent to plant genus Y by mutating *Ay*1 and *Ay*2 and became forma specialis Y. These processes led to the establish-

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ment of the forma specialis-genus specificity. Further, many species, varieties, and cultivars developed in host genus X. Cultivar $\alpha$ acquired $Rx3$ corresponding to $Ax3$, whereas cultivar $\beta$ acquired $Rx4$ corresponding to $Ax4$. An individual of f. sp. X accommodated itself to cultivar $\alpha$ by mutating $Ax3$ and became race $\alpha$, whereas another accommodated itself to cultivar $\beta$ by mutating $Ax4$ and became race $\beta$. These processes led to the establishment of cultivar specificity between host genus X and f. sp. X. Similar processes occurred between host genus Y and f. sp. Y.

Apparently, this model is based on Heath's (6) original basic compatibility model but is different from the modified model (9). Following Heath's (9) modified model, plant genera X and Y are nonhosts of f. sp. Y and f. sp. X, respectively. Forma specialis X and f. sp. Y carry different SSP genes and establish basic compatibility with plant genus X only and plant genus Y only, respectively. The forma specialis-genus specificity is determined by SSP genes, not by AVR genes. In contrast, my present model assumes that plant genera X and Y are hosts of both f. sp. X and f. sp. Y. These formae speciales carry almost the same set of SSP genes and establish basic compatibility with both plant genera. The forma specialis-genus specificity is determined by AVR genes and their corresponding resistance genes. For example, $Rx1$ and $Rx2$ are genes conditioning the resistance of plant genus X to an inappropriate forma specialis. Forma specialis Y cannot parasitize plant genus X because of the presence of the corresponding avirulence genes, $Ax1$ and $Ax2$, whereas f. sp. X can because of the lack of these AVR genes.

Why must the parasite have evolved into formae speciales that had restricted "host ranges"? Why didn't a biotype evolve that lost $Ax1$, $Ax2$, $Ay1$, and $Ay2$ and therefore parasitize both plant genera? An answer may be obtained from consideration of the original function of AVR genes. Person and Mayo (13) presented a hypothesis that the primary role of AVR genes was to function in support of the parasite's success as an intrinsically viable organism. Flor (5) argued that aggressiveness was associated with AVR genes. If these ideas are true, the loss of AVR genes would result in a decrease of vigor or aggressiveness. Therefore, the biotype parasitic on both plant genera, even if it may arise, could not compete on plant genus X with f. sp. X that carries more AVR genes. Similarly, it could not compete on plant genus Y with f. sp. Y. Consequently, the generalist would disappear as a result of the competition with the specialists.

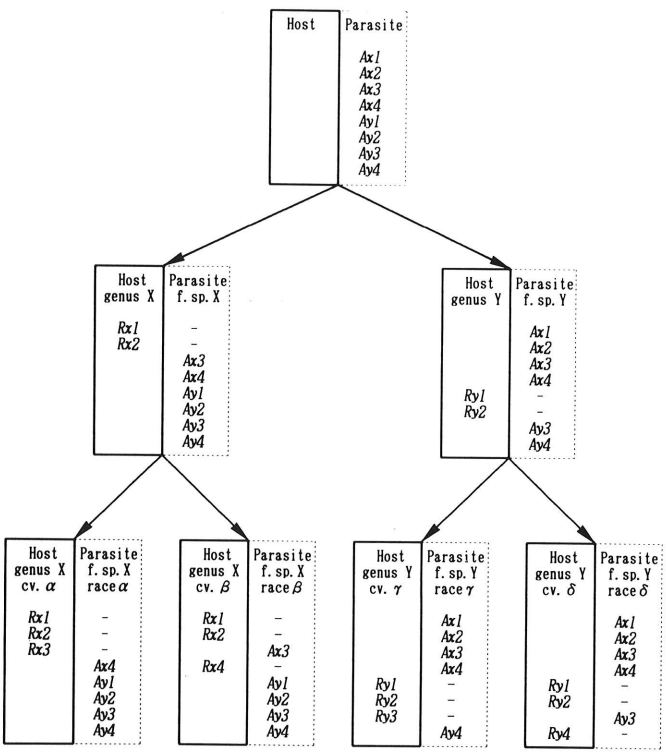


**Fig. 1.** Hypothetical model to illustrate the evolution of formae speciales and races. $Rx1$–$Rx4$ and $Ry1$–$Ry4$ are resistance genes that arose in plant genera X and Y, respectively. $Ax1$–$Ax4$ and $Ay1$–$Ay4$ are avirulence genes corresponding to $Rx1$–$Rx4$ and $Ry1$–$Ry4$, respectively.

Nonfunctional genes resulting from the mutation of AVR genes are recognized as virulence genes allelic to the AVR genes. The generalist that lost four AVR genes, $Ax1$, $Ax2$, $Ay1$, and $Ay2$, is considered to have obtained four virulence genes, $ax1$, $ax2$, $ay1$, and $ay2$; $ax1$ and $ax2$ are unnecessary on plant genus Y, and $ay1$ and $ay2$ are unnecessary on plant genus X. Flor (4) proposed an idea that unnecessary genes for virulence decreased in frequency in parasite populations. Vanderplank (19) referred to the selection against unnecessary genes for virulence as stabilizing selection. Thus, the explanation of the generalist's disappearance implies that stabilizing selection operated in the process of evolution of formae speciales. Wolfe (20–22) noted that, under selection pressures exerted by barley cultivars carrying different resistance genes, the pathogen (*E. g. hordei*) did not accumulate all of the virulence genes necessary to infect those cultivars into a single phenotype. Although this is an observation on the dynamics of races, there is no reason why similar dynamics did not occur in the evolution of formae speciales, if the forma specialis-genus specificity is, as I suggested earlier, qualitatively the same as cultivar specificity.

I do not deny that different plant genera have developed peculiar nonhost resistance after differentiation and have added it to the common portion of nonhost resistance. Also, each forma specialis would have continued to adapt to the additional nonhost resistance of its appropriate plant genus. However, the additional, different portion would be very small, especially between phylogenetically close genera, e.g., wheat and wheatgrass, as suggested by cytological data (16,17). It may not be so small between phylogenetically remote genera, e.g., wheat and oats, which may be expressed as decreased incidence of successful penetration of cell walls in oat leaves inoculated with the wheat mildew fungus (16). However, I assume that the additional nonhost resistance has not been the driving force in the evolution of formae speciales but has played a role only in stabilizing them.

The present model would apply not only to cereal mildews but also to cereal rusts, because formae speciales of the latter systems are also considered to have no qualitative difference from races (10). However, it does not seem to be applicable to formae speciales of *Fusarium* spp. Among host ranges of the formae speciales of *F. oxysporum*, there is little taxonomic consistency (1). Thus, specificity in *Fusarium* spp. at the level of formae speciales is considered to belong to plant species specificity. From the viewpoint of the basic compatibility model the term *forma specialis* seems to be used to designate different levels of parasitic specialization in different pathosystems.

## LITERATURE CITED


1. Barrett, J. A. 1986. Host-parasite interactions and systematics. Pages 1-17 in: Coevolution and Systematics. A. R. Stone and D. L. Hawksworth, eds. Clarendon Press, Oxford.
2. Day, P. R. 1976. Gene functions in host-parasite systems. Pages 65-73 in: Specificity in Plant Disease. R. K. S. Wood and A. Graniti, eds. Plenun Press, New York.
3. Ellingboe, A. H. 1976. Genetics of host-parasite interactions. Pages 761-778 in: Physiological Plant Pathology. R. Heitefuss and P. H. Williams, eds. Springer-Verlag, Berlin.
4. Flor, H. H. 1956. The complementary genic systems in flax and flax rust. Adv. Genet. 8:29-54.
5. Flor, H. H. 1971. Current status of the gene-for-gene concept. Annu. Rev. Phytopathol. 9:275-296.
6. Heath, M. C. 1981. A generalized concept of host-parasite specificity. Phytopathology 71:1121-1123.
7. Heath, M. C. 1981. Nonhost resistance. Pages 201-217 in: Plant Disease Control: Resistance and Susceptibility. R. C. Staples and G. H. Toenniessen, eds. John Wiley & Sons, New York.
8. Heath, M. C. 1985. Implications of nonhost resistance for understanding host-parasite interactions. Pages 25-42 in: Genetic Basis of Biochemical Mechanisms of Plant Disease. J. V. Groth and W. R.

Bushnell, eds. American Phytopathological Society, St. Paul, MN.
9. Heath, M. C. 1991. The role of gene-for-gene interactions in the determination of host species specificity. Phytopathology 81:127-130.
10. Johnson, R. 1976. Genetics of host-parasite interactions. Pages 45-62 in: Specificity in Plant Disease. R. K. S. Wood and A. Graniti, eds. Plenum Press, New York.
11. Niks, R. E. 1987. Nonhost plant species as donors for resistance to pathogens with narrow host range. I. Determination of nonhost status. Euphytica 36:841-852.
12. Niks, R. E. 1988. Nonhost plant species as donors for resistance to pathogens with narrow host range. II. Concepts and evidence on the genetic basis of nonhost resistance. Euphytica 37:89-99.
13. Person, C., and Mayo, G. M. E. 1974. Genetic limitations on models of specific interactions between a host and its parasite. Can. J. Bot. 52:1339-1347.
14. Tosa, Y. 1989. Genetic analysis of the avirulence of wheatgrass powdery mildew fungus on common wheat. Genome 32:913-917.
15. Tosa, Y. 1989. Evidence on wheat for gene-for-gene relationship between formae speciales of *Erysiphe graminis* and genera of gramineous plants. Genome 32:918-924.
16. Tosa, Y., Akiyama, T., and Ogura, H. 1990. Cytological aspects of interactions between formae speciales of *Erysiphe graminis* and genera of gramineous plants, and their evolutionary implications. Can. J. Bot. 68:1249-1253.
17. Tosa, Y., Mise, K., and Shishiyama, J. 1985. Recognition of two formae speciales of *Erysiphe graminis* by wheat cells. Ann. Phytopathol. Soc. Jpn. 51:223-226.
18. Tosa, Y., and Sakai, K. 1990. The genetics of resistance of hexaploid wheat to the wheatgrass powdery mildew fungus. Genome 33:225-230.
19. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York.
20. Wolfe, M. S. 1987. Trying to understand and control powdery mildew. Pages 253-273 in: Populations of Plant Pathogens: Their Dynamics and Genetics. M. S. Wolfe and C. E. Caten, eds. Blackwell Scientific Publications, Oxford.
21. Wolfe, M. S. 1988. Co-evolution in host-parasite relations. Kulturpflanze 36:209-224.
22. Wolfe, M. S., Barrett, J. A., and Slater, S. E. 1983. Pathogen fitness in cereal mildews. Pages 81-100 in: Durable Resistance in Crops. F. Lamberti, J. M. Waller, and N. A. Van der Graaff, eds. Plenum Press, New York.