4病害抵抗性遺伝子系における非病原性遺伝子座の非機会的結合(1)

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Nonrandom Association of Avirulence Loci in Four Disease Resistance Gene System I. Principles of Determination of Nonrandom Association Type and Pattern and Its Confirmation within Host Groups Having the Same Number of Susceptibility Genes

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Abstract

Using the simulation model for a four gene system on host-pathogen relationship, we studied the principles included in determination of types (cross- and noncross-types) of nonrandom associations between avirulence loci in a pathogen population. Analyses were carried out by giving some changes in host genotype frequencies which bring equilibrium conditions in frequencies of pathogen genotypes. Determination of types [cross-type (C) and noncross-type (N)] of nonrandom association was conducted by complicated but definite principles. When four avirulence genes (a, b, c and d) and four corresponding virulence ones ($+^a$, $+^b$, $+^c$ and $+^d$) and 16 genotypes consisting of four avirulence loci were considered, definite characteristics on determination of types of nonrandom associations appeared depending on the number of susceptibility genes [S_0, S_1, S_2, S_3 and S_4 (subscript is the number of susceptibility genes)]. When, for example, a part of a host genotype S_0 (ABCD) was replaced by another host genotype S_2 (for example, AB++), that is, ($S_0 \rightarrow S_2$), nonrandom association patterns, NCCCCC, CNCCCC, CCCNCC, CCNCCC, CCCCNC and CCCCCN, were observed for host genotypes AB++, A+C+, +BC+, A++D, +B+D and ++CD belonging to S_2, respectively. NCCCCC is a nonrandom association type for interactions between avirulence loci, a-b, a-c, a-d, b-c, b-d, and c-d, respectively. For determination of the patterns, there were two cases; in some cases a new host genotype (genotype after the replacement; called receptor, for convenience, in the simulations) gave influence, and in other cases, it was the old host genotype (genotype before replacement; donor in the simulations). In the former cases, combinations of a resistance gene and a susceptibility gene induced a cross-type nonrandom association, and combinations between a resistance gene and another resistance gene and between a susceptibility gene and another susceptibility gene induced noncross-type nonrandom association. In contrast, when a host genotype (S_2) was changed to S_0, the pattern became to be reversed, that is, CNNNNN, NCNNNN, NNNCNN, NNCNNN, NNNNCN and NNNNNC, respectively, in the above example.

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Key words: population genetics, nonrandom association, virulence analysis, host, pathogen, fitness.

INTRODUCTION

In the previous paper, Kiyosawa *et al.*[2] reported the following, on plant diseases showing gene-for-gene relationships. Nonrandom association, which means a deviation from random association of virulence genes expected when mutations occur independently in two avirulence loci in a pathogen population, was found in fungal population collected from resistant varieties having various resistance genes. There were two types of nonrandom association: cross-type and noncross-type. The former means that observed frequencies in genotypes (a+ and +b) having one avirulence gene and one

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virulence gene in two avirulence loci (a and b) are larger than the expected ones. The latter means that observed frequencies of genotypes (ab and ++) with two avirulence genes and two virulence genes are larger than the expected ones. In the collections from resistant varieties, different types of nonrandom associations between different avirulence loci are always found in fields where two or more nonrandom associations were found.

In another report[3], a method approximately to estimate fitness values of virulence genes using the same simulation program was discussed.

In the present paper we will attempt to search for the principle determining nonrandom association types and patterns and to search for the reason of occurrence of two types of nonrandom associations when two or more nonrandom associations were found in a field[2]. Furthermore, the significance of the occurrence of these types of nonrandom association is examined in breeding and/or population genetics.

MATERIALS AND METHODS

The model of the four gene system described in detail by Kiyosawa *et al.*[1] was used for this study, except for the following points.

The program for calculation that was previously made in BASIC system was improved to draw clearly the difference between observed frequencies calculated from fitness values given for each gene and expected frequencies which are expected from random association of the two genes in observed frequencies.

Sixteen genotypes consisting of four genes in the host were considered. As frequencies of these genotypes in consideration of their field susceptibility, Q_1 to Q_{16} were given. These were divided into five groups based on the number of susceptibility genes for simulation experiments. These groups were named as follows:

ABCD, ABC+, AB+D, A+CD, +BCD,
Q_1 Q_2 Q_3 Q_4 Q_5
S_0 S_1

AB++, A+C+, +BC+, A++D, +B+D, ++CD,
Q_6 Q_7 Q_8 Q_9 Q_{10} Q_{11}
S_2

A+++, +B++, ++C+, +++D, ++++.
Q_{12} Q_{13} Q_{14} Q_{15} Q_{16}
S_3 S_4

Here, $Q_n = q_n \cdot s_n$, and $\sum Q_n = 1$ ($n = 1, 2, 3, \ldots, 16$). The q and s are frequency and field susceptibility of each host genotype, respectively. Corresponding to host genotypes, genotypes of pathogen population were numbered as follows.

Genotype	abcd,	abc+,	ab+d,	a+cd,	+bcd,
Number	1	2	3	4	5

ab++,	a+c+,	+bc+,	a++d,	+b+d,	++cd,
6	7	8	9	10	11

a+++,	+b++,	++c+,	+++d,	++++
12	13	14	15	16

Using the method of Kiyosawa *et al.*[1], frequencies of 16 pathogen genotypes for pathogenicity were calculated during 100 or 200 vegetative generations. From the obtained frequencies (=observed in the present paper) the observed and expected values for all possible combinations of two in four avirulence loci were calculated by Wolfe's method[5,6]. For example, for avirulence loci, a and b, frequencies of virulence genes, $+^a$ (hereafter often abbreviated as +) and $+^b$, were calculated from observed frequencies of ab, a+, +b and ++ obtained by the simulation. Expected frequencies were calculated by $(1-a)(1-b)$, $(1-a)\cdot+^b$, $+^a\cdot(1-b)$ and $+^a\cdot+^b$. These frequencies were compared with observed frequencies: if the sum of observed frequencies of ab and ++ is larger than the sum of those expected values, we call it noncross-type (N) and the reverse is called cross-type (C) of nonrandom association as mentioned by Kiyosawa *et al.*[1]

In this and following paper[4], 0.9, 0.8, 0.6, and 0.7 were given as fitness values for vegetative reproduction during infection cycles to four virulence genes, $+^d$, $+^c$, $+^b$ and $+^a$ and the results were compared with cases where 0.75 is the given average fitness values to the four virulence genes in order to know the influence of fitness values given. Below, the former and the latter were called standard (E) conditions and nonstandard (U) conditions.

RESULTS

Proposal of hypotheses on decisive factors of nonrandom association patterns

Simulations began from comparison between simple combinations to know determinative factors for nonrandom associations. At first $S_0 \rightarrow S_1$ and $S_1 \rightarrow S_0$ were examined. The results are shown in Tables 1 and 2. In Table 1, some values (frequencies) were transferred

Table 1. Influence of interaction between two avirulence loci on type of nonrandom associations ($S_0 \rightarrow S_1$)[a]

Donor (Genotype)	Gen.[b]	Receptor Q_2 (ABC+)	Q_3 (AB+D)	Q_4 (A+CD)	Q_5 (+BCD)
Q_1 (ABCD)	10	NNCNCC[c]	NCNCNC	CNNCCN	CCCNNN
	100	NNCNCC	NCNCNC	CNNCCN	CCCNNN

a) $S_0 \rightarrow S_1$ means transfer of a value from S_0 to S_1 or proportion of area in which S_0 variety was changed to S_1 variety.
b) Gen. means generations.
c) N and C are noncross-type and cross-type nonrandom associations.
NNCNCC: Indicating interaction between two avirulence loci in following order; a-b, a-c, a-d, b-c, b-d and c-d.

Table 2. Influence of interaction between two avirulence loci on type of nonrandom associations ($S_1 \rightarrow S_0$)

Receptor (Genotype)	Gen.	Donor Q_2 (ABC+)	Q_3 (AB+D)	Q_4 (A+CD)	Q_5 (+BCD)
Q_1 (ABCD)	10	CCNCNN	CNCNCN	NCCNNC	NNNCCC
	100	CCNCNN	CNCNCN	NCCNNC	NNNCCC

See Table 1 for explanation.

from S_0 (ABCD) to S_1 (ABC+, AB+D, A+CD and +BCD). In these cases, nonrandom association patterns were different between receptors although they came from same donor. This suggests that receptors play an important role in determining the type of nonrandom associations.

In contrast to them, in Table 2, different donors showed different nonrandom association patterns for the same receptor. This suggests that the donor plays an important role in determination of nonrandom association patterns. Tables 1 and 2 show that types of nonrandom associations of individual pairs are quite the

Table 3. Influence of fitness values[a] given in each virulence gene on type of nonrandom associations. ($S_4 \rightarrow S_{0,1,2,3}$) and ($S_{0,1,2,3} \rightarrow S_4$)

(Genotype)		Donor Q_{16} (++++)		Receptor Q_{16} (++++)	
		Observed[b]	Expected	Observed	Expected
S_0	Q_1 (A B C D)	NNNNNN[b]	R NNNNNN D CCCCCC	CCCCCC	R NNNNNN D CCCCCC
S_1	Q_2 (A B C +)	NNC NCC	R NNC NCC D CCCCCC	CCNCNN NNNNNN[a]	R NNNNNN D CCNCNN
	Q_3 (A B +D)	NC NC NC	R NC NC NC D CCCCCC	CNCNCN NNNNNN	R NNNNNN D CNCNCN
	Q_4 (A +C D)	CNNCCN	R CNNCCN D CCCCCC	NCCNNC NNNNNN	R NNNNNN D NCCNNC
	Q_5 (+B C D)	CCCNNN	R CCCNNN D CCCCCC	NNNCCC NNNNNN	R NNNNNN D NNNCCC
S_2	Q_6 (A B ++)	NCCCCC NCCCCN	R NCCCCN D CCCCCC	CNNNNN	R NNNNNN D CNNNNC
	Q_7 (A +C +)	CNCCCC CNCCNC	R CNCCNC D CCCCCC	NCNNNN	R NNNNNN D NCNNCN
	Q_8 (+B C +)	CCCNCC CCNNCC	R CCNNCC D CCCCCC	NNNCNN	R NNNNNN D NNCCNN
	Q_9 (A ++D)	CCNCCC CCNNCC	R CCNNCC D CCCCCC	NNCNNN	R NNNNNN D NNCCNN
	Q_{10} (+B +D)	CCCCNC CNCCNC	R CNCCNC D CCCCCC	NNNNCN	R NNNNNN D NCNNCN
	Q_{11} (++C D)	CCCCCN NCCCCN	R NCCCCN D CCCCCC	NNNNNC	R NNNNNN D CNNNNC
S_3	Q_{12} (A +++)	CCCCCC CCCNNN	R CCCNNN D CCCCCC	NNNNNN	R NNNNNN D NNNCCC
	Q_{13} (+B ++)	CCCCCC CNNCCN	R CNNCCN D CCCCCC	NNNNNN	R NNNNNN D NCCNNC
	Q_{14} (++C +)	CCCCCC NCNCNC	R NCNCNC D CCCCCC	NNNNNN	R NNNNNN D CNCNNC
	Q_{15} (+++D)	CCCCCC NNCNCC	R NNCNCC D CCCCCC	NNNNNN	R NNNNNN D CCNCNN

Fitness values for all genes are 0.75.

____ and ~~~~ mean that the pattern is same as that expected from receptor (R) and donor (D), respectively.

a) When two patterns are written in one place, it indicates that a change of pattern occurred during 100 generations.

b) "Observed" means the results of the simulations and "expected" means results expected according to the hypotheses. R and D in the table indicate results expected from receptors and donors, respectively. (See Table 1 for explanation, also.)

reverse.

Similar simulations were performed on $S_0 \rightarrow S_2$, $S_2 \rightarrow S_0$, $S_0 \rightarrow S_3$, $S_3 \rightarrow S_0$, $S_0 \rightarrow S_4$, and $S_4 \rightarrow S_0$. Nonrandom association patterns obtained were as follows. In the cases of $S_0 \rightarrow S_2$ and $S_2 \rightarrow S_0$, the patterns for genotypes, AB++, A+C+, +BC+, A++D, +B+D and ++CD, were NCCCCC, CNCCCC, CCCNCC, CCNCCC, CCCCNC, and CCCCCN, in the former and CNNNNN, NCNNNN, NNNCNN, NNCNNN, NNNNCN, and NNNNNC, in the latter. In the cases of $S_0 \rightarrow S_3$ and $S_3 \rightarrow S_0$, the patterns for genotypes, A+++, +B++, ++C+ and +++D, are as follows: CCCCCC, CCCCCC, CCCCCC and CCCCCC, in the former case, and NNNNNN, NNNNNN, NNNNNN and NNNNNN, in the latter case.

From these results, at least the following can be considered.

Hypothesis A: In some cases, receptors play an important role, while in others, donors play an important role in determining the type of nonrandom association.

Hypothesis B: When the direction of the transfer of values is different, it creates a large influence on the type of nonrandom association.

The phenomena mentioned above can be explained by the following hypotheses.

Hypothesis C: When receptors play an important role, combinations of a resistance gene and a susceptibility gene induce cross-type nonrandom association of two avirulence loci corresponding to the resistance genes. For example, in receptors, $A+^B$, $C+^D$ or $+^A D$ induces cross-type nonrandom association of two avirulence loci, a-b, c-d, or a-d. In contrast, combinations between resistance genes and between susceptibility genes induce noncross-type nonrandom association.

Hypothesis D: When donors play an important role, combinations of a resistance and a susceptibility gene induce noncross-type nonrandom association between two avirulence loci corresponding to the resistance genes. Combinations between resistance genes and between susceptibility genes induce cross-type nonrandom associations.

These hypotheses were ascertained with the simulations during 200 vegetative generations on $S_0 \leftarrow \rightarrow S_4$ ($S_0 \rightarrow S_4$ and $S_4 \rightarrow S_0$), $S_1 \leftarrow \rightarrow S_4$, $S_2 \leftarrow \rightarrow S_4$ and $S_3 \leftarrow \rightarrow S_4$. The results are shown in Table 3. Often, reaction patterns changed during 200 vegetative generations. In such a case, two patterns are shown in the table. When there is a change in reaction pattern during these generations, the first pattern is that of donor and the second pattern is that of the receptor. Thus, this is thought to indicate that the above hypotheses were the case.

Nonrandom association patterns occurring with a transfer of a value within S group

In the case of $S_1 \leftarrow \rightarrow S_1$ (Table 4), all nonrandom association patterns were determined by the receptor under standard conditions. Under nonstandard conditions, the patterns show donor patterns in early generations and change to receptor patterns in later generations.

In Table 5, the case of $S_2 \leftarrow \rightarrow S_2$ is shown. In this table, U patterns are omitted, when U patterns (pattern under nonstandard conditions) were completely the same as E patterns (patterns under standard conditions). In this case, a slight difference from $S_1 \leftarrow \rightarrow S_1$ was found in the point that nonrandom association patterns could not be explained by one of donor or receptor in some cases, for example, Nos. 9, 18, 22 and 26 in Table 5. In these cases, the part that could not be explained by donor or receptor could be explained by its partner. In the case of Nos. 9, 18, 22 and 26, three are explained by

Table 4. Influence of interaction between two avirulence loci on type of nonrandom associations ($S_1 \leftarrow \rightarrow S_1$)

Donor (Genotype)		Receptor			
		Q_2 (ABC+) [NNCNCC]	Q_3 (AB+D) [NCNCNC]	Q_4 (A+CD) [CNNCCN]	Q_5 (+BCD) [CCCNNN]
Q_2 (ABC+) [CCNCNN]	E[a]		1. NCNCNC	2. CNNCCN	3. CCCNNN
Q_3 (AB+D) [CNCNCN]	E	4. NNCNCC		5. CNNCCN	6. CCCNNN
	U[a]	CNCNCN			
		NNCNCC			
Q_4 (A+CD) [NCCNNC]	E	7. NNCNCC	8. NCNCNC		9. CCCNNN
	U	NCCNNC	NCCNNC		NCCNNC
		NNCNCC	NCNCNC		CCCNNN
Q_5 (+BCD) [NNNCCC]	E	10. NNCNCC	11. NCNCNC	12. CNNCCN	
	U	NNNCCC	NNNCCC		
		NNCNCC	NCNCNC		

a) E: Standard conditions. Pattern when equal fitness value 0.75 was given to all virulence genes. Only the cases in which the different patterns from the nonstandard conditions were obtained were described.
U: Nonstandard conditions. Unequal fitness values, 0.9, 0.8, 0.6 and 0.7, were given to $+^d$, $+^c$, $+^b$ and $+^a$, respectively. ____ and ~~~ indicate that the pattern is same as that expected from the donor and receptor, respectively. (See Table 1 for explanation, also.)

Table 5. Influence of fitness values given to each virulence gene on type of nonrandom associations ($S_2 \leftarrow \rightarrow S_2$)

Donor (Genotype)		Receptor					
		Q_6 (AB++) [NCCCCN]	Q_7 (A+C+) [CNCCNC]	Q_8 (+BC+) [CCNNCC]	Q_9 (A++D) [CCNNCC]	Q_{10} (+B+D) [CNCCNC]	Q_{11} (++CD) [NCCCCN]
Q_6 (AB++) [CNNNNC]	E		1. CNCCNC	2. CCNNCC	3. CCNNCC	4. CNCCNC	5. CCCCCN
Q_7 (A+C+) [NCNNCN]	E	6. NCCCCN		7. CCNNCC	8. CCNNCC	9. CCCCNC	10. NCCCCN
	U	NCCNCN		NCNNCC		NCCNNC	
		NCCCCN		CCNNCC		CCCCNC	
Q_8 (+BC+) [NNCCNN]	E	11. NCCCCN	12. CNCCNC		13. CCNCCC	14. CNCCNC	15. NCCCCN
	U				CNNCCC		
					CCNCCC		
Q_9 (A++D) [NNCCNN]	E	16. NCCCCN	17. CNCCNC	18. CCCNCC		19. CNCCNC	20. NCCCCN
	U	NNCCNN	CNCCNN	NCCNNN		NNCCNN	
		NCCCCN	CNCCNC	CCCNNN		CNCCNC	
Q_{10} (+B+D) [NCNNCN]	E	21. NCCCCN	22. CNCCCC	23. CCNNCC	24. CCNNCC		25. NCCCCN
	U		CNNCCN				
			CNCCCC				
Q_{11} (++CD) [CNNNNC]	E	26. NCCCCC	27. CNCCNC	28. CCNNCC	29. CCNNCC	30. CNCCNC	
	U	NNNNNC	CNNNNC	CNNNNC	CNNNCC	CNNNNC	
		NCNCNC	CNCCNC	CCNNCC	CCNNCC	CNCCNC	

E: Standard conditions. Equal fitness value 0.75 was given to all virulence genes.
U: Nonstandard conditions. Unequal fitness values, 0.9, 0.8, 0.6 and 0.7 were given to $+^d$, $+^c$, $+^b$ and $+^a$, respectively.
Note: Obtained after more than 100 generations.
(See Table 1 for explanation, also.)

Table 6. Influence of interaction between two avirulence loci on type of nonrandom associations ($S_3 \leftarrow \rightarrow S_3$)

Donor (Genotype)		Receptor			
		Q_{12} (A+++) [CCCNNN]	Q_{13} (+B++) [CNNCCN]	Q_{14} (++C+) [NCNCNC]	Q_{15} (+++D) [NNCNCC]
Q_{12} (A+++) [NNNCCC]	E		NNNCCN	NNNCNC	NNNNCC
			CNNCCN	NCNCNC	NNCNCC
	U		NNNCCN	NCNCNC	NNCNCC
					NNNNCC
Q_{13} (+B++) [NCCNNC]	E	NCCNNN		NCNNNC	NNCNNC
		CCCNNN		NCNCNC	NNCNCC
	U	CCCNNN		NCNCNC	NNCNCC
Q_{14}(++C+) [CNCNCN]	E	CNCNNN	CNNNCN		NNCNCN
		CCCNNN	CNNCCN		NNCNCC
	U	CNCNNN			NNCNCC
		CCCNNN			
Q_{15} (+++D) [CCNCNN]	E	CCNNNN	CNNCNN	NCNCNN	
		CCCNNN	CNNCCN	NCNCNC	
	U	CCNNNN			
		CCCNNN			

(See Table 1 for explanation, also.)

the donor and the other three are explained by the receptor. This case was called 3DR pattern. For example, in Nos. 6, 7, and 29, five types are explained by the donor as shown by ~~~~, and a different set of five are explained by the receptor as shown by ____. This pattern was called 5DR pattern. Other patterns are 6D (No. 16), 6R (No. 1), 5D (No. 26), 5R (No. 5), and 4D (No. 18).

Under the standard conditions, when resistance and susceptibility genes are complementary in three or four loci, 6R and 5R are induced, respectively. Under nonstandard conditions, different patterns are found for about half of the combinations. In these cases, 6D or 5D changed to 6R or 5R. In cases where four loci are complementary (on the diagonal line from upper-right to

lower-left in Table 5), 5R patterns under the standard conditions and a tendency to change from donor patterns to receptor under unstandard conditions are found.

Table 6 shows patterns in the case of $S_3 \leftarrow \rightarrow S_3$. In this case, all combinations are complementary in two loci. Changes in patterns with generations were found, rather, in the standard conditions. These changes occur from DR patterns to R patterns.

DISCUSSION

In this paper, about half of the possible combinations of host genotypes in the four gene system were analyzed by simulations. By transferring a value (frequency of host genotype) from one host genotype to another host genotype under equilibrium conditions, the following principles controlling nonrandom associations between avirulence loci were found.

A nonrandom association is induced between avirulence loci in the pathogen population by transferring a value (frequency) of host genotype, with some exceptions. In some cases, host genotype of donor (old variety) determines nonrandom association type (C or N). In other cases, receptor (new variety) determines the nonrandom association type.

With the same given fitness value for all four virulence genes ($+^a$, $+^b$, $+^c$ and $+^d$), nonrandom association pattern at the 200th generation is determined by receptors with an increase in susceptibility genes and by donors with a decrease in susceptibility genes, when genotypes ABCD or ++++ are donors or complementary genes are four. In these cases, S_2 and S_3 groups show a change in nonrandom association patterns from intermediate patterns between donor and receptor or from donor pattern to receptor pattern during the first 100 generations.

Receptor patterns are found when combinations of resistance genes or combinations of susceptibility genes induce noncross-type nonrandom association, and combinations of a resistance gene and a susceptibility gene induce cross-type nonrandom associations. Donor patterns are found, when reverse types are induced; that is, the former combinations induce cross-type and the latter induce noncross-type associations.

In the range of combinations of host genotypes examined in this paper, the number of complementary loci seems to play an important role in determining nonrandom association type and pattern.

In the next paper[3], the results obtained between other S groups will follow.

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和 文 摘 要

清沢茂久・Donna PURBA・Md. Shamsher ALI・沖中 泰・清水 勉・斉藤明彦：4病害抵抗性遺伝子系における非病原性遺伝子座の非機会的結合 I．非機会的結合決定の原理と同一罹病性遺伝子数群内でのその確認

シミュレーションモデルを用いて，4抵抗性遺伝子系における非機会的結合の型やパターンを決定する原理について研究した。非機会的結合には二つの型が見られた。ここで非機会的結合とは次のような場合を言う。菌の二つの遺伝子座に関して，非病原性遺伝子と病原性遺伝子を考えた場合（例えばabと$+^a+^b$），観察値（シミュレーションにより得られた頻度）と，期待値（非病原性遺伝子座間の機会的結合を仮定したときに得られる値）との差に三つの型が見られた。頻度a+と+bが共にabと++より大きい場合[交差型(C)]と，abと++が共にa+と+bより大きい場合[非交差型(N)]と，両者に差が無い場合である。この非機会的結合の型と，四つの非病原性遺伝子座間の六つの組合せについて，この型の組合せパターンは，多くの場合新しい品種の遺伝子型により決定され，時には古い品種の遺伝子型により決定される。新しく栽培される品種（シミュレーションの中での移動値の受容者）により決定される場合には，受容者の抵抗性遺伝子同士の組合せと罹病性遺伝子同士の組合せでは非交差型，罹病性遺伝子と抵抗性遺伝子との組合せでは交差型となる。前の（古い）品種（シミュレーションの中では供与者）により決定される場合には非機会的結合の型は，新しい品種による場合の逆になり，抵抗性-抵抗性と罹病性-罹病性で交差型，抵抗性-罹病性で非交差型となる。ただし，供与者と受容者が同一数の罹病性遺伝子を持つ場合は，各病原性遺伝子に同一相対適応値を与えた場合には逆にならない。このような非機会的結合型は与えた適応値により多少の変化をする。