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# Method for mapping a partial lethal-factor locus on a molecular-marker linkage map of a backcross and doubled-haploid population

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**Abstract** Distorted segregation has been repeatedly observed in various plant species in molecular-marker linkage mapping where distant crosses were made. It may be caused by a partial lethal-factor acting in the filial generations. A method is presented for estimating the recombination values between a partial lethal-factor locus and a linked molecular marker and the relative viability or fertilization ability of zygotes or gametes, respectively affected by the partial lethal factor in backcross (BC) and doubled-haploid (DH) populations using the maximum-likelihood method associated with the expectation maximization (EM) algorithm. The method was applied to segregation data of molecular markers for a population of 150 DH lines developed from the 'Steptoe' × 'Morex' cross in barley. The presence of a partial lethal-factor locus, located on chromosome 4, causing partial selection was suggested. This locus was tightly linked to the ABG500B marker, and the chance of fertilization of female gametes possessing the partial lethal factor was, on average, 59.8% that of a normal one. Two additional partial lethal factors were found on chromosome 5.

**Key words** Distorted segregation · Molecular marker · Recombination value · Maximum-likelihood method · EM algorithm

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## Introduction

In order to obtain higher DNA polymorphism, crosses between wild species and cultivars, between subspecies, and even between species have been used for linkage analyses of crop plants. Distorted segregation of marker genotypes was often encountered in such distant crosses (Wendel and Parks 1984; Torres et al. 1985; McCouch et al. 1988; Paterson et al. 1988, 1990, 1991; Konishi et al. 1990; Heun et al. 1991; Lyttle 1991; Saito et al. 1991; Schon et al. 1991; Zivy et al. 1992; Komatsuda et al. 1993; Causse et al. 1994; Wang et al. 1994; Devaux et al. 1995; Li et al. 1995a,b; Bezant et al. 1996; Harushima et al. 1996; Lin et al. 1996; Cadalen et al. 1997; Li et al. 1997). Distortion may be caused by a variety of factors, such as hybrid sterility, incompatibility, and nuclear cytoplasmic interaction. For simplicity, we call all of these partial lethal factors. The distortion is the result of the elimination of gametes or zygotes which is controlled by a partial lethal factor located in the neighboring region around the marker.

In barley, Tabata (1961) first reported linkage between a partial lethal factor (gametophyte factor) and the morphological marker *wx* (waxy endosperm). Konishi et al. (1990) also reported linkage between a partial lethal factor and isozyme markers. They estimated recombination values and the differential fertilization ability of male gametes using $F_2$ and $F_3$ segregation data. Employing similar methods, linkage analyses between partial lethal factors and morphological or isozyme markers have been reported in rice (Iwata et al. 1964; Nakagahra 1972; Nakagahra et al. 1972; Mori et al. 1973; Nakagahra et al. 1974; Maekawa et al. 1981; Maekawa 1982; Kinoshita and Takamura 1984; Maekawa and Kita 1985; Lin and Ikehashi 1993).

Cheng et al. (1996) recently presented a method for estimating the recombination values between a partial lethal-factor locus and linked molecular markers, as

well as the relative viability or fertilization ability of gametes or zygotes affected by the partial lethal factor, solely by using marker segregation data of an $F_2$ population. The relative viability or fertilization ability of a gamete or zygote with the partial lethal allele as compared with the normal one was also estimated. Three models of gametic or zygotic selection affected by a partial lethal factor, were considered: (1) either male or female gamete selection, (2) both male and female gamete selection, and (3) zygotic selection. In the case of an $F_2$ population, the best model could be determined by the goodness of fit in testing the observed frequencies of the phenotypes to the expected ones under the supposed model. But in the cases of BC and DH populations, the gametic or zygotic selection caused by a partial lethal gene cannot be directly distinguished by the segregation data of markers.

In the present paper we present a method for mapping a partial lethal factor by using co-segregation data in BC and DH populations. The expectation maximization (EM) algorithm (Dempster et al. 1977) was employed to obtain the maximum-likelihood estimates and their standard errors. Among the segregation data of molecular markers in a DH population which were used to construct a barley linkage map by Kleinhofs et al. (1993), data for a group of loci exhibiting distorted segregation on chromosome 4 were used as a concrete example to illustrate our method.

## Materials and methods

Consider a mating of $AALLBB\,(P_1) \times aallbb\,(P_2)$, where: (1) *A*-*a* and *B*-*b* are a pair of molecular markers, and (2) *L*-*l* is a partial lethal-factor locus which is assumed to be located between the markers *A* and *B* on the same chromosome. The order of the three loci is then *A*-*L*-*B*. Let the recombination value between *A* and *L* be $r_1$, and that between *L* and *B* be $r_2$, and let *r* denote the recombination value between markers *A* and *B*. The differential viability of gametes with the genotype *l* or zygotes *ll* was expressed as $t$ $(0 < t < 1)$ relative to that of normal gametes, *L*, or zygotes, *LL* and *Ll*. Because the two map functions of Haldane (1919) and Kosambi (1944) have been used by many researchers in constructing linkage maps, we consider both of these models.

If *L*-*l* were a normal molecular-marker locus with no partial lethal effect for the recessive, the expected gametic frequencies of the eight genotypes, *ALB*, *ALb*, *AlB*, *Alb*, *aLB*, *aLb*, *alB* and *alb*, derived from the $F_1$ (*ALB*/*alb*) individuals for Haldane's model $(r = r_1 + r_2 - 2r_1r_2)$ would be $(1 - r_1)(1 - r_2)/2$, $(1 - r_1)r_2/2$, $r_1r_2/2$, $r_1(1 - r_2)/2$, $r_1(1 - r_2)/2$, $r_1r_2/2$, $(1 - r_1)r_2/2$ and $(1 - r_1)(1 - r_2)/2$, respectively. Those for Kosambi's model $(r = r_1 + r_2 - 2r_{12}$, $r_{12} = 2r_1r_2r)$ would be $(1 - r_1 - r_2 + r_{12})/2$, $(r_2 - r_{12})/2$, $r_{12}/2$, $(r_1 - r_{12})/2$, $(r_1 - r_{12})/2$, $r_{12}/2$, $(r_2 - r_{12})/2$ and $(1 - r_1 - r_2 + r_{12})/2$, respectively. Let *r* be known, then $r_2$ is a function of $r_1$ and *r*, as shown by

| Haldane's model | Kosambi's model |
|---|---|
| $r_2 = (r - r_1)/(1 - 2r_1)$ | $r_2 = (r - r_1)/(1 - 4r_1r)$. |

Table 1 shows the expected frequencies $f_i$ of the eight classes of genotypes in the BC and DH populations with no differential viability. The genotypes of *LL*, *Ll* and *ll* cannot be observed directly, and only the segregation data of the four phenotypic classes with respect to molecular markers *A* and *B* can be obtained. The corresponding expected frequencies, $f_k'$ (k = 1, 2, 3, 4), can be expressed as the sum of the products of functions $f_i(r_1)$ and $g_j(t)$, (i = 1, 2, … , 8; j = 1, 2):

$$f_k' = f_{2k-1}g_1 + f_{2k}g_2 \qquad (k = 1, 2, 3, 4)$$

where $g_1 = 1/(1 + t)$, and $g_2 = t/(1 + t)$.

Theoretically, the observed counts $a_k'$ $(k = 1, 2, 3, 4)$ of phenotype with respect to *A* and *B* is a sum of two quantities, i.e., $a_k' = a_{2k-1} + a_{2k}$, (k = 1, 2, 3, 4), and $a_{2k-1}$ and $a_{2k}$ could be expressed as:

$$a_{2k-1} = a_k' \times f_{2k-1}g_1/f_k'; \quad a_{2k} = a_k' \times f_{2k}g_2/f_k'$$
$$(k = 1, 2, 3, 4). \qquad (1)$$

Let *n* be the total counts of individuals in a BC or DH population, then the log likelihood is:

$$\begin{aligned}\mathbf{L} &= \log\{[f_1(r_1) \times 1/(1 + t)]^{a_1} \times [f_2(r_1) \times t/(1 + t)]^{a_2} \\ &\quad \times \cdots \times [f_7(r_1) \times 1/(1 + t)]^{a_7} \times [f_8(r_1) \times 1/(1 + t)]^{a_8}\} \\ &= \log\{[t^{(a_2 + a_4 + a_6 + a_8)}/(1 + t)]^n \times \prod_{i=1}^{8} [f_i(r_1)]^{ai}\} \\ &= \sum_{i=1}^{8} a_i \log f_i(r_1) + (a_2 + a_4 + a_6 + a_8)\log t - n\log(1 + t) \qquad (2)\end{aligned}$$

**Table 1** Expected genotype frequencies $f_i$ in a backcross (BC) and a doubled-haploid (DH) population[a,b]

| Genotype | | (Haldane's model) | |
|---|---|---|---|
| *BC* | *DH* | *Ll* (*BC*) or *LL*(*DH*) | *ll* |
| *AaBb* | *AABB* | $f_1 = (1 - r_1)(1 - r_1 - r)/(1 - 2r_1)$ | $f_2 = r_1(r - r_1)/(1 - 2r_1)$ |
| *Aabb* | *AAbb* | $f_3 = (1 - r_1)(r - r_1)/(1 - 2r_1)$ | $f_4 = r_1(1 - r_1 - r)/(1 - 2r_1)$ |
| *aaBb* | *aaBB* | $f_5 = f_4$ | $f_6 = f_3$ |
| *aabb* | *aabb* | $f_7 = f_2$ | $f_8 = f_1$ |
| | | (Kosambi's model) | |
| *AaBb* | *AABB* | $f_1 = [1 - r + 2r_1r\,(r - 2) + 2r_1{}^2r]/(1 - 4r_1r)$ | $f_2 = 2r_1(r - r_1)r/(1 - 4r_1r)$ |
| *Aabb* | *AAbb* | $f_3 = [r - r_1 - 2r_1(r - r_1)r]/(1 - 4r_1r)$ | $f_4 = [r_1(1 - 4r_1r) - 2r_1(r - r_1)r]/(1 - 4r_1r)$ |
| *aaBb* | *aaBB* | $f_5 = f_4$ | $f_6 = f_3$ |
| *aabb* | *aabb* | $f_7 = f_2$ | $f_8 = f_1$ |

[a] The recombination values between *A* and *L*, and between *A* and *B* are $r_1$ and *r*, respectively
[b] All frequencies are multiplied by 1/2

and the two equations for score are:

$$S_{r_1} = \partial \mathbf{L}/\partial r_1 = \sum_{i=1}^{8} \{(a_i/f_i) \times (\partial f_i/\partial r_1)\} = 0 \quad (3)$$

$$S_t = \partial \mathbf{L}/\partial t = (a_2 + a_4 + a_6 + a_8)/t - n/(1+t) = 0. \quad (4)$$

It should be noted that equation (3) does not include $t$, indicating that the recombination value $r_1$ can be estimated independently of the estimate of $t$. The maximum-likelihood estimates can be obtained by solving these equations. $S_t = 0$ could be algebraically solved as:

$$t = (a_2 + a_4 + a_6 + a_8)/(a_1 + a_3 + a_5 + a_7). \quad (5)$$

Since $S_{r_1} = 0$ cannot be solved by conventional algebraic methods, iterative calculations are needed to obtain the estimate of $r_1$. In each iteration cycle the estimate was obtained by the bisection method. For finding maximum-likelihood estimates of $r_1$ and $t$ simultaneously, we first give $r_1$ and $t$ arbitrary initial values within the intervals (0, $r$) and (0, 1). In the (p + 1)st expectation step (E-step) of the EM-algorithm, $a_i^{(p+1)}$ can be obtained by formula (1), with the observed counts $a_k'$ and the current estimates of the parameters $r_1^{(p)}$, and $t^{(p)}$. The (p + 1)st maximization step (M-step) then gives the maximum-likelihood estimates of $r_1^{(p+1)}$ $t^{(p+1)}$. These two estimates would converge to their respective limits simultaneously (Wu 1983). When the differences between the values of the estimates in the previous M-step and current one become very small, and less than the pre-determined quantity, then the iteration is stopped and the final estimates $r_1$ and $t$ are obtained.

Under Fisher's model amounts of information (Edwards 1972) are:

$$\mathbf{I}_{r_1} = -\mathbf{E}(\partial^2 \mathbf{L}/\partial r_1^2) = n \sum_{i=1}^{4} [(1/f_i) \times (\partial f_i/\partial r_1)^2] \quad (6)$$

$$\mathbf{I}_t = -\mathbf{E}(\partial^2 \mathbf{L}/\partial t^2) = \mathbf{E}[(a_2 + a_4 + a_6 + a_8)/t^2 - n/(1+t)^2].$$

When we substitute the expected counts for the observed ones, i.e., $nf_k'$ for $a_k'$, then $a_{2k} = a_k' \times f_{2k} g_2 / f_k' = \mathrm{n} \times f_{2k} g_2$, so that $\mathbf{E}(a_2 + a_4 + a_6 + a_8) = nt/(1+t)$; this gives

$$\mathbf{I}_t = -\mathbf{E}(\partial^2 \mathbf{L}^2/\partial t^2) = n/[t(1+t)^2]. \quad (7)$$

In particular, under Haldane's model

$$\mathbf{I}_{r_1} = n/[r_1(1-r_1)] + n/[(1-r_1-r)(r-r_1)] - 4n/(1-2r_1)^2 \quad (8)$$

has a simple form, and the corresponding standard error (SE) is then given by $(\mathbf{I}_t)^{-1/2}$ and $(\mathbf{I}_{r_1})^{-1/2}$.

As described above, the recombination value, $r_1$, between a partial lethal-factor locus and marker $A$, and the differential viability, $t$, as well as their standard errors, can be estimated. The map distance between a marker $A$ and the partial lethal-factor locus, $L$, can be calculated from the estimate of recombination value obtained under the mapping function of Haldane's (1919) or Kosambi's (1944) model. Finally, the partial lethal factor locus can be located on the linkage map.

## Results

Kleinhofs et al. (1993) reported that in mapping barley chromosomes there was a group of molecular markers showing distorted segregation on chromosome 4 in a DH population derived from the cross of 'Steptoe' × 'Morex'. In the present paper we have used the published data of Kleinhofs et al. (1993) to analyze the linkage between the molecular markers and a possible partial lethal-factor locus ($L$) in the neighboring region of chromosome 4. Table 2 shows the distorted segregation of seven molecular markers which were located in the region of chromosome 4 and the results of chi-square tests for the goodness of fit of the observed segregation ratios to the expected ratio of 1 : 1. The possibility of gametic or zygotic selection affected by a partial lethal factor was strongly suggested in this region, implying the existence of a partial lethal-factor locus located close to the ABG500B, ABG498 and WG144 molecular markers.

**Table 2** Distorted segregation of a group of molecular markers on barley chromosome 4 and the results of a chi-square test for the segregation ratio (Data from Kleinhofs et al. 1993)

| Marker name | Segregation (total) Steptoe : Morex | $\chi^2$ (1 : 1) |
|---|---|---|
| iAco2 | 88 : 62 (150) | 4.17* |
| ABG500 B | 91 : 55 (146) | 8.39** |
| ABG498 | 90 : 56 (146) | 7.46** |
| WG114 | 90 : 51 (141) | 10.24** |
| ABG54 | 85 : 57 (142) | 5.13* |
| ABG394 | 89 : 59 (148) | 5.68* |
| ABG366 | 82 : 54 (136) | 5.36* |

*,** Significant at the 5% and 1% levels, respectively

Let the genotypes of Steptoe ($P_1$) and Morex ($P_2$) be $AALLBB$ and $aallbb$, respectively, where $A - a$ is the flanking marker locus ABG500B, $B - b$ one of the other four molecular marker loci, and $L - l$ a partial lethal-factor locus. Tentatively, let $l$ be a partial lethal allele and $L$ the normal one. Because the distances in the linkage map of Kleinhofs et al. (1993) were calculated using the Kosambi mapping function, we estimated the recombination value between the partial lethal factor locus ($L$) and molecular marker $A$ ABG500B ($r_1$) and the differential viability of gametes or zygotes ($t$) under Kosambi's model. The estimates and their standard errors are shown in Table 3.

It is clear that all of the estimated recombination values ($r_1$) for ABG500B ($A$) and the partial lethal factor locus ($L$) were close to zero (Table 3). We calculated the weighted averages of $r_1$ and $t$ estimates over the four cases shown in the table, and obtained $r_1 = 0.002$, and $t = 0.598$. By the Kosambi mapping function the map distance between marker ABG500B and the partial lethal-factor locus ($L$) was calculated to be 0.2 cM. The weighted average of the estimated differential viability of the gametes of genotype $l$ or the zygotes of genotype $ll$ was 59.8% as compared with that of normal ones. In Fig. 1, we located the partial lethal-factor locus ($L$) on chromosome 4 of the barley linkage map constructed by Kleinhofs et al. (1993). The fact that the estimated value of $t$ is less than 1 shows that the donor of the partial lethal factor was the parent 'Morex'; otherwise, the estimated $t$ would be larger than 1. We also found two partial lethal factors located in the region of distorted segregation on chromosome 5. One was 5.2 cM downstream from the marker ABA4,

**Table 3** Estimated recombination values and viabilities (Data from Kleinhofs et al. 1993)

| *A–L–B* | *AABB* | *AAbb* | *aaBB* | *aabb* | $r_1 \pm$ SE | $t \pm$ SE |
|---|---|---|---|---|---|---|
| ABG500B – *L* – ABG498 | 87 | 2 | 1 | 53 | $0.002 \pm 0.003$ | $0.606 \pm 0.105$ |
| WG114 | 87 | 2 | 1 | 49 | $0.002 \pm 0.003$ | $0.561 \pm 0.099$ |
| ABG54 | 83 | 3 | 1 | 52 | $0.001 \pm 0.002$ | $0.616 \pm 0.108$ |
| ABG394 | 86 | 4 | 2 | 53 | $0.002 \pm 0.003$ | $0.610 \pm 0.104$ |
| Weighted mean | | | | | 0.002 | 0.598 |

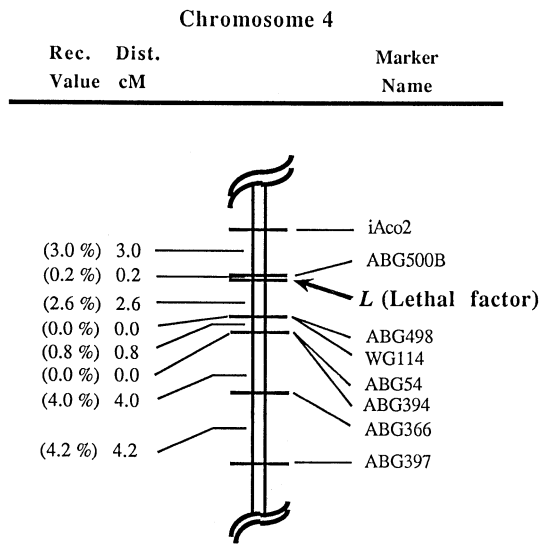


**Fig. 1** A partial linkage map of barley chromosome 4 with a single partial-lethal factor (*L*)

and the other 5.5 cM downstream from the marker BCD98. The estimated values of *t* were 0.528 and 0.533, respectively. The donor was 'Steptoe' for both factors.

## Discussion

When molecular-marker linkage maps are constructed, crosses are usually made between varieties that are distantly related in order to obtain a higher degree of DNA polymorphism, and the consequence of this is that distorted segregation is often encountered. The closer the linkage between a marker and a partial lethal factor, the more conspicuous is the distortion of segregation expected. The magnitude of the chi-square values in testing the observed ratio against the expected one or the upper probability of the chi-square distribution can be used for guessing the position of the partial lethal factor. However, it is only the interval between flanking markers of the partial lethal factor, and not the position, that can be determined. Hence, if the interval is not sufficiently close we cannot locate the factor on a linkage map. In the present study, we showed that in a BC or DH population by using data on the co-segregation of a pair of closely linked markers with distorted segregation we can estimate the location, viability, or relative fertilization ability of a partial lethal factor.

In barley, distorted segregation was reported as early as 1961 by Tabata. Recently in molecular-marker mapping studies distortion has been found in some publications (Heun et al. 1991; Schon et al. 1991; Zivy et al. 1992; Kleinhofs et al. 1993; Devaux et al. 1995; Bezant et al. 1996 ). It is now believed that the distorted segregation at a marker locus in barley is mainly due to the linkage of the marker and a gametophyte gene (denoted as *ga*) located near one another on the same chromosome (Tabata 1961; Konishi et al. 1990), although other factors, such as complementary genes, duplicate genes, chromosomal abnormality, and competitive fertilization between marker genotypes, were presumed to be the cause of the distortion in some reports.

The donor of the partial lethal factor on chromosome 4 was 'Morex'. Devaux et al. (1995) compared the mapping of the barley genome with male (another culture, AC) and female (*Hordeum bulbosum*, Hb) recombination-derived, doubled-haploid populations developed from the cross 'Steptoe' × 'Morex'. They found that segregation distortions of the same loci on chromosome 4 only occurred in the (Hb)-derived DH population (same data as used in our study), and did not occur in the (AC)-derived DH population. Based on their conclusions it is inferred that the partial lethal factor mapped by us is a female gametic selection factor.

In the $F_2$ generation it is possible to decide which of the three models of lethality is most appropriate for the explanation of the observed distortion (Cheng et al. 1996). However, in BC and DH populations it is not possible by the present method to decide which of the three models of lethality is best. In a BC population of the type *Aa aa* distorted segregation may be caused by a gametophytic factor acting in the male gamete or by a zygotic factor. Which of the two is appropriate cannot be determined. The same is true for a DH population. So the mode of action of the partial lethal factor in BC and DH populations needs to be determined experimentally.

Recently, Lorieux et al. (1995) reported how an estimation of recombination value between markers is affected by differential viability at the gamete or zygote level in a backcross population. They also showed how

to estimate the recombination value and relative viability of the gametes or zygotes by maximum likelihood. In their analysis, however, they assumed for simplicity that the markers showing distortion are exactly located on genes affected by gametic or zygotic selection. Contrary to their method, we assumed that the distortion of marker segregation is due to linkage of the marker with a lethal gene, the effect of which is due to the partial or entire selection of a gamete or zygote with the gene. Our results proved theoretically that a recombination value estimated by the ordinary maximum-likelihood method is not affected by distortion in both the BC and DH generations and that the recombination value, $r_1$, can be estimated independently of the fertilization ability ($t$).

In this paper it was assumed that the differential viability of gametes with the genotype $l$ or zygotes $ll$ was expressed as $t$ $(0 < t < 1)$ relative to that of the normal gametes, $L$, or zygotes, $LL$ and $Ll$. In some cases, hybrid sterility or the semi-fertility of heterozygotes is often encountered in distant crosses. A little modification is needed for the application of the present method to such a case. For example, if the differential viability of zygotes with the genotype $Ll$ is expressed as $t$ $(0 < t < 1)$ relative to that of the normal ones, $ll$, in a BC population, then let $g_2 = 1/(1 + t)$, and $g_1 = t/(1 + t)$.

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