Analysis of segregation distortion and its relationship to hybrid barriers in rice

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Abstract

Background: Segregation distortion (SD) is a frequently observed occurrence in mapping populations generated from crosses involving divergent genotypes. In the present study, ten genetic linkage maps constructed from reciprocal F2 and BC1F1 mapping populations derived from the parents Dasanbyeo (indica) and Ilpumbyeo (japonica) were used to identify the distribution, effect, and magnitude of the genetic factors underlying the mechanisms of SD between the two subspecies.

Results: SD loci detected in the present study were affected by male function, female function, and zygotic selection. The most pronounced SD loci were mapped to chromosome 3 (transmitted through male gametes), chromosome 5 (transmitted through male gametes), and chromosome 6 (transmitted through female gametes). The level of SD in BC1F1 populations which defined by chi-square value independence multiple tests was relatively low in comparison to F2 populations. Dasanbyeo alleles were transmitted at a higher frequency in both F2 and BC1F1 populations, suggesting indica alleles are strongly favored in inter-subspecific crosses in rice. SD loci in the present study corresponded to previously reported loci for reproductive barriers. In addition, new SD loci were detected on chromosomes 2 and 12.

Conclusion: The identification of the distribution of SD and the effect of genetic factors causing SD in genetic mapping populations provides an opportunity to survey the whole genome for new SD loci and their relationships to reproductive barriers. This provides a basis for future research on the elucidation of the genetic mechanisms underlying SD in rice, and will be useful in molecular breeding programs.

Keywords: Segregation distortion; Linkage map; Reproductive barrier

Background

Segregation distortion (SD) is defined as a deviation of the observed genotypic frequency from expected Mendelian segregation ratios. Genetic elements that cause SD are increasingly being recognized as a powerful evolutionary force (Sandler and Novitski 1957), and it is thought that SD is due to a number of causes including genetic, physiological, and/or environmental factors (Liu et al. 2008; Matsushita et al. 2003; Wang et al. 2009; Xu et al. 1997).

Several molecular mechanisms of SD have been reported in plants (Fishman et al. 2008; Fishman et al. 2001; Fishman and Willis, 2005; Harushima et al. 2002; Koide et al. 2008a; Koide et al. 2008b; Koide et al. 2011).

As reproductive isolation is a result of multiple genes acting at various stages throughout the life history of an organism, further dissection of SD regions in different populations and cross combinations is of key importance for breeding programs. In rice, previous studies detected loci linked with SD in both single and multiple crosses (Harushima et al. 2001, 2002; Wang et al. 2009). Use of abundant DNA markers allowed development of high-density linkage maps for rice, and these were used to survey the whole genome for SD loci (Causse et al. 1994; Harushima et al. 1996; Yano et al. 1998). Most of the barriers were mapped at different loci and demonstrated to have formed after japonica–indica differentiation (Harushima et al. 2001, 2002).

Since varietal differentiation in O. sativa is complex, data obtained from a single cross are not representative of the full range of differentiating Asian rice (Harushima et al. 2002). Thus, Harushima et al. (2002) used multiple...
crosses to investigate the diverse variation of reproductive barriers in rice. In that experiment, 33 reproductive barriers were identified, 15 of which affected the gametophyte and 18 of which altered viability of the zygote. However, as a probable consequence of the cross combinations used, the experiment was unable to clearly distinguish between female and male gametophytic selection-originated SDs. Therefore, the use of different mapping populations would be likely to reveal distorted loci or causes of SD that were not identified previously.

In the present study, two reciprocal F2 and eight BC1F1 populations developed from two Korean popular rice cultivars, Ilpumbryo (*japonica*) and Dasanbyeo (*indica*), were used to investigate SD. Nuclear genetic or cytoplasmic factors influencing SD rates were inferred from the distortion patterns of DNA markers in reciprocal F2 populations. Gametophytic and zygotic selection as mechanisms underlying SD were inferred from distorted marker patterns in BC1F1 populations conferring female- or male-segregating populations. Information regarding the loci and other factors responsible for SD in rice are important for the selection of breeding cultivars, and could also be beneficial for the development of molecular breeding programs. In this study, we aimed to map the genomic regions showing SD and to characterize whether gametic- or zygotic selection caused SD at each locus.

Results

Construction of linkage maps in two F2 and eight BC1F1 populations

Total map length, average distance between adjacent markers, and the linkage map coverage of the genome constructed from reciprocal F2 and BC1F1 populations was variable in the different mapping populations (Table 1). Physical locations of both end markers for each chromosome and linkage map coverage of the genome constructed from these populations in details were presented in Additional file 1: Table S1. The total map length in the Ilpumbryo/Dasanbyeo (ID) population was 1391.1cM, with an average distance of 13.3cM between adjacent markers. In the reciprocal cross (DI), total map length and average distance were 1462.7cM and 13.9cM, respectively. With a few exceptions, map lengths in female-segregating populations (BC1F1 populations in which F1 was used as a female parent) were generally longer than those in male-segregating population (BC1F1 populations in which F1 was used as a male parent) (Table 1). Reduction in map length was extreme when Ilpumbryo as a maternal parent was crossed with F1 as a pollen parent. The map lengths of ID//I and DI//D were 1424.1cM and 1514.8cM, respectively, while those of I//ID and I//DI were 525.8cM and 787.3cM, respectively (Table 1). This indicates that the frequency of recombination between marker pairs in I//ID and I//DI is lower than those in ID//I and DI//D.

SD of DNA markers in two reciprocal F2 populations

On the whole, considering the single test performed at every marker locus, significant (acceptance level: 0.05) deviation with respect to expected Mendelian ratio in two F2 populations was found at a high number of distorted markers on almost all chromosomes (Table 2).

Overall, 45 (43.7%) and 41 (40.2%) loci were significantly distorted from the expected Mendelian segregation ratios in the ID and DI populations, respectively.

| Population | Generation | Number of tested loci | Mapped loci | Total map length (cM) | Average distance btw markers (cM) | Map coverage(%) |
|------------|------------|-----------------------|-------------|-----------------------|----------------------------------|----------------|
| ID/D       | F2         | 107                   | 103         | 1391.1                | 13.3                             | 91.0           |
| D/I        | F2         | 107                   | 102         | 1462.7                | 13.9                             | 96.0           |
| DI/DI      | BC1F1      | 144                   | 139         | 1495.6                | 11.4                             | 80.2           |
| D/DI       | BC1F1      | 144                   | 142         | 1119.2                | 8.8                              | 77.6           |
| ID/I       | BC1F1      | 144                   | 142         | 1424.1                | 11.7                             | 75.3           |
| I/ID       | BC1F1      | 144                   | 132         | 525.8                 | 5.6                              | 34.6           |
| ID/D       | BC1F1      | 144                   | 140         | 1434.7                | 12.2                             | 73.0           |
| D/D        | BC1F1      | 144                   | 139         | 1043.2                | 8.2                              | 69.2           |
| DI/I       | BC1F1      | 144                   | 140         | 1514.8                | 11.7                             | 89.2           |
| I/D        | BC1F1      | 144                   | 135         | 787.3                 | 6.5                              | 51.4           |

*map coverage in genetic and physical maps was based on the reference of Nipponbare/Kasalath map (Harushima et al. 1998) and IRGSP builds (http://www.rgp.dna.affrc.go.jp/), respectively.

ID = F2 of Ilpumbryo x Dasanbyeo, DI = F2 of Dasanbyeo x Ilpumbryo. 
ID/D = Ilpumbryo/Dasanbyeo/ID/I = Ilpumbryo/Dasanbyeo/Ilpumbryo. 
D/DI = Dasanbyeo/Ilpumbryo/Diganbyeo, D/DI = Dasanbyeo/Ilpumbryo/Ilpumbryo. 
D/I = I/D = Dasanbyeo/Ilpumbryo, ID/I = Ilpumbryo/Dasanbyeo/Ilpumbryo. 
D/I = Dasanbyeo/Ilpumbryo/Dasanbyeo, and D/I = Dasanbyeo/Ilpumbryo/Ilpumbryo.
| Pop. | Mapped loci | Loci of SD | Number of regions of SD | Chromosomal regions of SD | Favored genotype (ratio, %) | Ilpumbyeo (II) | Heterozygote (ID or DI) | Dasanbyeo (DD) |
|------|-------------|------------|-------------------------|---------------------------|-----------------------------|----------------|------------------------|----------------|
| ID   | 103         | 45 (43.7%) | 27 (26.2%)              | Chr.1 (1), Chr.3 (2), Chr.4 (1), Chr.5 (1), Chr.6 (1), Chr.9 (1), Chr.12 (3) | -                            | 11 (24.4%) | 4 (14.8%)             | 34 (75.6%) | 23 (85.2%)         |
| DI   | 102         | 41 (40.2%) | 20 (19.6%)              | Chr.1 (1), Chr.3 (1), Chr.4 (1), Chr.5 (1), Chr.6 (1), Chr.12 (3) | 1 (2.5%)                      | 14 (34.1%) | 4 (20%)               | 26 (63.4%) | 16 (80%)           |
| ID/D | 140         | 7 (5.0%)   | 0                       | -                         | -                            | 3 (42.9%) | -                     | 4 (57.1%) | -                 |
| ID/I | 142         | 28 (19.7%) | 7 (4.9%)                | Chr.6 (1)                 | 18 (64.3%)                    | 5 (71.4%) | 2 (28.6%)             | -             | -                 |
| DI/D | 139         | 23 (15.8%) | 5 (3.6%)                | Chr.1 (1), Chr.6 (1), Chr.12 (1) | -                            | 10 (43.5%) | 1 (20%)               | 13 (56.5%) | 4 (80%)            |
| DI/I | 140         | 11 (7.9%)  | 7 (5%)                  | Chr.6 (1)                 | 5 (45.5%)                     | 6 (54.5%) | 7 (100%)              | -             | -                 |
| I/ID | 132         | 21 (15.9%) | 11 (8.3%)               | Chr.1 (1), Chr.5 (1), Chr.8 (1), Chr.11 (1) | 21 (100%)                    | 11 (100%) | -                     | -             | -                 |
| I/DI | 135         | 25 (18.5%) | 2 (2.2%)                | Chr.12 (1)                | 1 (4.0%)                      | 2 (100%) | -                     | -             | -                 |
| D/ID | 139         | 39 (28.1%) | 11 (7.9%)               | Chr.1 (1), Chr.3 (1), Chr.5 (1), Chr.8 (1), Chr.12 (1) | -                            | 2 (5.1%) | -                     | 37 (94.9%) | 11 (100%)         |
| D/DI | 142         | 44 (31.0%) | 18 (12.7%)              | Chr.2 (1), Chr.3 (1), Chr.5 (2), Chr.8 (1), Chr.12 (1) | -                            | 5 (11.4%) | -                     | 39 (88.6%) | 12 (100%)         |

*Refer to Table 1 for abbreviations.

bObserved segregation to Mendelian expectation was tested using a Chi-square value by single testing (randomly selected single marker).

cObserved segregation to Mendelian expectation was tested using a Chi-square value by multiple testing (sequential Bonferroni correction of P-value) across loci separately within each cross.

dSD is abbreviation of segregation distortion.
(Table 2). With this acceptance level (alpha 0.05), it can be suggested that 5% of the non-Mendelian segregation were caused by chance. However, to detect false $H_0$ upon applying the sequential Bonferroni method, the number of possible SD was extremely reduced. After applying Bonferroni method to segregation data, significant results in 27 out of 45 SD loci in ID population were obtained, which means that 26.2% of the total number of segregation analyzed were non-Mendelian. On the other hand, we obtained significant results in 20 out 41 SD loci in DI population, which means that the SD level decreased from 40.2% to 19.6% (Table 2). Most of the distorted markers in both F2 populations were skewed towards Dasanbyeo alleles, indicating that indica alleles were transmitted at higher frequency than japonica alleles.

**Nuclear and cytoplasmic effects on SD in reciprocal F2 populations**

It can be inferred that nuclear genetic factors are responsible for SD if markers are similarly distorted in both reciprocal F2 populations. Conversely, a cytoplasmic effect can be inferred if the markers are distorted in only one of the reciprocal F2 populations. A total of 18 markers were distorted in both F2 populations (Additional file 2: Table S2), and were thus caused by nuclear genetic factors. Eleven markers deviated from the expected Mendelian segregation ratio in only one of the two reciprocal F2 populations (Additional file 2: Table S2), indicating a cytoplasmic effect. Of these 11 markers, 9 showed SD only in the ID population and 2 showed SD only in the DI population. Favored marker genotypes differed between the ID (Ilpumbeyo cytoplasm) and DI populations (Dasanbyeo cytoplasm); specifically, seven of the 9 SD markers (Additional file 2: Table S2) in the ID population favored Dasanbyeo genotypes and the remaining two loci favored heterozygous genotypes, while two SD loci in the DI population favored Dasanbyeo genotypes (Additional file 2: Table S2 and Additional file 3: Figure S1).

**Effect of gametophytic and zygotic factors on SD in reciprocal BC1F1 populations**

Number of regions of SD and the chromosomal regions associated with SD from eight BC1F1 populations are presented in Table 2 and Table 3, respectively. To determine whether the SD loci detected in the F2 populations originated from female function (embryo-sac effect), male function (pollen effect), or post-fertilization selection among zygotes (hereafter, zygotic selection), genotype ratios were compared in pairs of reciprocal backcross to each parent. The significant SD detected in the BC1F1 populations only when the F1 was used as a female parent is indicative of SD due to the female effect since there was no segregation in male parent. By contrast, SD detected in the BC1F1 population only when the F1 male was used as a parent is indicative of the male effect since there was no segregation in the female parent. However, when markers showed SD in both backcrosses, SD is indicative of zygotic selection. Our data thus suggest three mechanisms that lead to SD, as follows: SD through female function, SD through male function, and SD through zygotic selection.

**SD through female function**

On the basis of SD patterns observed in BC1F1 conferring female-segregating populations (F1 plants were used as female parents), two chromosomal regions (chromosomes 6 and 12) harbored SD loci putatively influenced by female function, hereafter termed female-specific SD loci or fSD (Table 3). Of these, female-function influence on SD was most pronounced in chromosome 6, as indicated by segregation distortion of the marker loci in three of the female-segregating populations (ID//I, DI//D, D1/I), but not in the reciprocal backcrosses (I//ID, D//DI, I//DI). Thus, a region encompassing the markers S06018 and S06031 on chromosome 6 was subject to significant transmission bias in all female-segregating populations with the exception of ID//D (Table 3). Moreover, in the ID/I and DI/I populations, the marker distortion affect extended to the neighboring region (S06040B, S06053, and S06065), resulting in the presence of severely distorted markers across a large portion of chromosome 6 (Additional file 4: Figure S2). In contrast, inheritance of these markers was undistorted in the male-segregating population, implying that the SD was caused by female-specific function only and that pollen grain competition is not responsible for SD detected on chromosome 6. Our finding is in accordance with the earlier studies about SD in japonica-indica hybrids conducted by Lin et al. (1992). In their study, when pollen from F1 hybrids were used for backcrossing, no segregation distortion was found for marker gene loci on chromosome 6 and thus, they suggested that SD of markers on chromosome 6 is caused through partial female gamete abortion in indica-japonica hybrids. In addition, SD through female function on chromosome 6 could also be caused and explained by reducing of spikelet fertility since the certain SD regions was concurred with $qSF6.1$ (S06031-S06040B) and $qSF6.2$ (S06040B-S06053) loci responsible for hybrid fertility genes in our previous study (Reflinur et al. 2012).

Dasanbyeo alleles were over-represented at all distorted markers on chromosome 6 in the three populations, as determined by the skew direction. This indicates that Dasanbyeo embryo sacs were preferred during fertilization by both Dasanbyeo and Ilpumbeyo pollen, thus causing the distortion of certain regions on chromosome 6. The same chromosome 6 markers segregated normally in the
| Chr | Marker | Position (Mbp) | %DD (ID) | %DD (DI) | %DD (ID/DI) | %DD (D//ID) | %DD (D//DI) | %II (ID/I) | %II (D//I) | %II (I//ID) | %II (I//DI) | Mechanism |
|-----|--------|---------------|----------|----------|-------------|------------|------------|-------------|------------|-------------|------------|-----------|
| 1   | S01022 | 4.38          | 0.36**DD | 0.34**DD | 0.78**DD    | 0.71**DD   |            |             |            |             |            | Zygotic selection |
| 1   | S01038 | 7.46          | 0.34**DD | 0.36**DD | 0.75**DD    | 0.73**DD   |            |             |            |             |            | Zygotic selection |
| 1   | S01157B| 39.8          |           |           |             |            |             | 0.83**DD   |            |             |            | mSD       |
| 1   | S01160 | 40.80         |           |           |             |            |             | 0.84**DD   |            |             |            | mSD       |
| 1   | S01181B| 43.20         |           |           |             |            |             | 0.84**DD   |            |             |            | mSD       |
| 2   | S02126 | 29.91         |           |           |             |            | 0.68**DD   |             |            |             |            | mSD       |
| 2   | S02135 | 31.48         |           |           |             |            | 0.69**DD   |             |            |             |            | mSD       |
| 3   | S03027 | 5.71          | 0.38**DD | 0.34**DD | 0.79**DD    | 0.83**DD   |            |             |            |             |            | mSD       |
| 3   | S03041 | 8.90          | 0.38**DD | 0.41**DD | 0.79**DD    | 0.83**DD   |            |             |            |             |            | mSD       |
| 3   | S03046 | 10.14         | 0.41**DD | 0.41**DD | 0.79**DD    | 0.91**DD   |            |             |            |             |            | mSD       |
| 3   | S03048 | 10.75         | 0.41**DD | 0.40**DD | 0.83**DD    | 0.89**DD   |            |             |            |             |            | mSD       |
| 3   | S03065 | 14.43         |           |           |             |            | 0.83**DD   | 0.94**DD   |            |             |            | mSD       |
| 3   | S03130 | 29.83         | 0.32**DD |           |             |            |             |             |            |             |            | Cytoplasm effect |
| 3   | S03136 | 30.11         | 0.33**DD |           |             |            |             |             |            |             |            | Cytoplasm effect |
| 4   | S04113 | 32.61         | 0.22**H  | 0.21**H  |             |            |             |             |            |             |            | Nuclear effect |
| 4   | S04120 | 33.60         | 0.14**H  | 0.16**H  |             |            |             |             |            |             |            | Nuclear effect |
| 5   | S05004B| 0.29          | 0.41**DD | 0.33**DD | 0.73**DD    | 0.78**DD   |            |             |            |             |            | mSD       |
| 5   | S05009 | 0.84          | 0.39**DD | 0.26**H  | 0.73**DD    | 0.83**DD   |            |             |            |             |            | mSD       |
| 5   | S05029 | 3.42          | 0.34**DD | 0.35**DD | 0.75**DD    | 0.81**DD   |            |             |            |             |            | mSD       |
| 5   | S05030A| 3.66          | 0.33**DD | 0.33**DD | 0.77**DD    | 0.80**DD   |            |             |            |             |            | mSD       |
| 5   | S05030B| 3.66          | 0.33**DD | 0.33**DD | 0.77**DD    | 0.77**DD   |            |             |            |             |            | mSD       |
| 5   | S05032 | 4.29          | 0.34**DD |           |             | 0.74**DD   | 0.84**DD   |             |            |             |            | mSD + Cytoplasm effect |
| 5   | S05036 | 4.71          | 0.35**DD |           |             | 0.74**DD   | 0.80**DD   |             |            |             |            | mSD + Cytoplasm effect |
| 5   | S05045 | 6.97          |           |           |             | 0.73**DD   | 0.80**DD   |             |            |             |            | mSD       |
| 5   | S05064 | 16.99         |           |           |             | 0.73**DD   | 0.84**DD   |             |            |             |            | mSD       |
| 5   | S05077A| 20.10         |           |           |             | 0.72**DD   | 0.80**DD   |             |            |             |            | mSD       |
| 6   | S06018 | 4.74          | 0.36**DD | 0.37**DD | 0.68**DD    | 0.34**H    | 0.26**H    |             |            |             |            | fSD       |
| 6   | S06031 | 5.68          | 0.36**DD | 0.36**DD | 0.67**DD    | 0.30**H    | 0.25**H    |             |            |             |            | fSD       |
| 6   | S06040 | 7.83          |           |           |             | 0.26**H    | 0.19**H    |             |            |             |            | fSD       |
| 6   | S06053 | 8.83          |           |           |             | 0.22**H    | 0.22**H    |             |            |             |            | fSD       |
| 6   | S06065A| 14.53         |           |           |             | 0.29**H    | 0.23**H    |             |            |             |            | fSD       |
| 8   | S08060 | 17.27         |           |           |             | 0.25**H    | 0.33**H    | 0.84**DD   |            |             |            | mSD       |
| 8   | S08066 | 18.91         |           |           |             | 0.23**H    | 0.31**H    | 0.80**DD   |            |             |            | mSD       |
| 8   | S08075 | 20.65         |           |           |             | 0.23**H    | 0.30**H    | 0.86**DD   |            |             |            | mSD       |
| 8   | S08080B| 21.33         |           |           |             | 0.23**H    | 0.27**H    | 0.84**DD   |            |             |            | mSD       |
| 8   | S08090 | 23.08         |           |           |             | 0.19**H    | 0.28**H    | 0.84**DD   |            |             |            | mSD       |
| 9   | S09065 | 17.91         |           |           |             | 0.19**H    |           |             |            |             |            | Cytoplasm effect |
| 9   | S09075A| 19.58         |           |           |             | 0.20**H    |           |             |            |             |            | Cytoplasm effect |
| 11  | S11004A| 1.08          |           |           |             |            |             | 0.88**DD   |            |             |            | mSD       |
| 11  | S11006 | 1.27          |           |           |             |            |             | 0.88**DD   |            |             |            | mSD       |
| 12  | S12005 | 0.33          | 0.90**DD | 0.89**DD |             |             |             |             |            |             |            | Nuclear effect |

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Table 3 Characterization of segregation distortion loci underlying the transmission of gametes through female, male and zygotic selections (Continued)

| Locus | Chromosome | Frequency (DD) | Frequency (HI) | Frequency (DHI) | Frequency (DI) | Frequency (IHI) | Frequency (I) | Segregation Distortion |
|-------|------------|----------------|----------------|----------------|----------------|----------------|----------------|------------------------|
| 12 S12009A | 12 | 0.63 | 0.39 | 0.43 | 0.78 | 0.71 | 0.18 | mSD |
| 12 S12011B | 12 | 1.88 | 0.40 | 0.38 | 0.75 | 0.69 | 0.16 | mSD |
| 12 S12030 | 12 | 3.84 | 0.26 | 0.26 | 0.78 | 0.71 | 0.18 | mSD |
| 12 S12039B | 12 | 5.57 | 0.31 | 0.31 | 0.80 | 0.73 | 0.23 | mSD |
| 12 S12055B | 12 | 15.57 | 0.35 | 0.35 | 0.80 | 0.73 | 0.23 | mSD |
| 12 S12066 | 12 | 19.44 | 0.33 | 0.33 | 0.80 | 0.73 | 0.23 | mSD |
| 12 S12071 | 12 | 19.66 | - | - | 0.24 | 0.24 | 0.23 | fSD |
| 12 S12091 | 12 | 23.65 | - | - | 0.24 | 0.24 | 0.23 | fSD |
| 12 S12097B | 12 | 25.00 | 0.41 | 0.41 | 0.80 | 0.73 | 0.23 | fSD |

*indicates chromosomal regions showing segregation distortion (SD) at least in one population.

**genotypic ratios were tested against the expected Mendelian expectation to determine significant of SD (χ² with 2 df for F₂, 1 df for backcrosses: **indicating locus showed significant deviation from Mendelian segregation ratio (Bonferroni corrected). DD and II are abbreviation for genotype frequency (%) of Dasanbyeo and Ilpumbryo, respectively. The direction of skewness is followed in bracket where DD for Dasanbyeo homozygous, and H for heterozygous genotypes. (-) indicating the primers were not tested in certain populations.

SD through male function

Seven SD loci putatively influenced by male function, hereafter denoted as male-specific SD loci or mSD, were detected on distorted regions of chromosomes 1, 2, 3, 5, 8, 11 and 12. The regions harboring the most severe distortion patterns were on chromosomes 3, 5 and 8 (Table 3). Markers showing SD on chromosome 3 were distorted in two of the four male-segregating backcross populations, D//ID and D//DI. The skew direction in both D//ID and D//DI was towards Dasanbyeo alleles. Both skew directions imply that Dasanbyeo pollens were preferred to Ilpumbryo pollens when under competition. Similarly, SD observed on chromosome 5 at the loci S05004B, S05009, S05029, S05030A, and S05030B, were significant in D//ID and D//DI maps but not in their reciprocal backcross populations ID//D and DI//D. The fact that mSD on chromosome 3 and 5 was not observed in either I//ID or I//DI populations reflecting that preferential fertilization might be possible explanation for mSD on certain chromosome. In this case, male gametophytes with D genotype interacted with maternal D genotype loci and preferentially fertilized. Theory suggests that the meiotic drive elements such as gametophytic competition results in preferential fertilization or abortion of gamete or zygote, are the main influence factors of SD in indica-japonica species.
indicating that there might be no cytoplasmic effect on the expression of pollen fertility. However, it is likely that detected SD loci were explained by the fertilities. In particular D//DI population, the SD region extended further, spreading almost to the distal end of chromosome 5 as also observed in I//ID map (S05032, S05036, S05045, S05064, and S05077A) indicating the severity of the pollen competition effect (Additional file 4: Figure S2). However, the role of pollen competition effect on SD should be further clarified through in depth investigation of pollen fertility in BC1F1 progeny. As in chromosome 3, distorted regions on chromosome 5 showed a preference for Dasanbyeo alleles. Dasanbyeo was over-represented in D//ID and D//DI, whereas heterozygous was over-represented in I//ID, indicating in both cases that Dasanbyeo pollens were preferred over Ilpumbyeo pollens when under competition.

Male-function influence on SD in chromosomes 8 and 12 was indicated by segregation distortion of the marker loci in three of the male-segregating populations (D//ID, D//DI, I//ID), but not in the reciprocal backcrosses (ID//I, DI//D, ID//I). Thus, a region encompassing the markers S08060 to S08090 on chromosome 8 and that of the markers S12009A and S12011 on chromosome 12 was subject to significant transmission bias in all male-segregating populations with the exception of I//ID (Table 3). However, unlike the case of mSD on chromosome 3, 5 and 12, Ilpumbyeo alleles were over-represented at all distorted markers on chromosome 8 in the three populations, as determined by the skew direction. Several inconsistencies of mSD loci were found on chromosome 1 and 11 which were only distorted in one out of four male-segregating populations. These regions were detected in I//ID population and the effect of distortion at these loci was skewed towards Ilpumbyeo alleles, implying that maternal I preferred male gametophyte with I genotype under pollen competition. Despite the fact that either F1 plants developed from ID or DI crosses consist of Ilpumbyeo and Dasanbyeo pollen grains, when the two F1 plants were used as male to fertilize the same female parents (Ilpumbyeo) to produce BC1F1 plants, their effect on distortion was different that indicated by undistorted segregation of these regions in I//DI population. Thus, further study is needed to elucidate molecular mechanism of SD in these regions.

**SD through zygotic selection**

In addition to SD occurring as a consequence of female function and male function, zygotic selection was also observed to cause SD in our backcrossing populations. Zygotic selection causing biased transmission would be explained by the significant transmission ratio distortion in both reciprocal backcrosses. Regions exhibiting SD from zygotic selection only were located on the small part of chromosome 1 which indicated by the distortion of S01022 and S01038 markers in the D//ID and DI//D populations (Table 3). Zygotic selection in this region is due to either inviability of indica/japonica hybrid or related to spikelet fertility. Although earlier we were able to get around 100 lines for BC1F1 whose F1 plants were used as female (embryosac source) and male (pollen sources) parents, the viable plants were very poor. Many BC1F1 plants were died in seedlings stage reflecting hybrid weakness phenomenon in japonica/indica crosses and the number of lines survived in the field was decreased. Hence, we assumed that zygotic selection in this region is due to abortion of genotype ID or DI reflected
by hybrid weakness reducing number of plants survived in the field both in D//ID (52 lines) and DI//D (69 lines). Another possible explanation that zygotic selection affected SD in this region is related to spikelet fertility. Mean value of spikelet fertility of lines showing heterozygous genotypes was lower than those lines carrying homozygous genotypes (data not shown).

Discussion

SD across linkage maps

Several gametophytic and zygotic barriers causing deviation of allele frequencies from Mendelian ratios have been reported in inter-specific rice crosses (Harushima et al. 1996; Harushima et al. 2002; Koide et al. 2008b; Wang et al. 2005, 2009). In the present study, a similar level of SD was identified in two reciprocal F2 cross populations, with both nuclear genetic and cytoplasmic factors causing severe SD. This concurs with several prior studies that reported that both nuclear factors (Goloenko et al. 2002; He et al. 2001; Liu and Qu 2008; Wang et al. 2009) and cytoplasmic factors (Faris et al. 1998; Goloenko et al. 2002; Manabe et al. 1999; Wang et al. 2009) play a key role in SD.

In our two F2 populations, nuclear genetic factors from Dasanbyeo (indica) had stronger effects on SD than those from Ilpumbyeo (japonica) since most of the distorted loci were skewed in favor of the indica alleles (Table 2, Additional file 2: Table S2 and Additional file 3: Figure S1). These results are consistent with previous observations that SD in segregated populations generated from indica/japonica hybrids favored the indica parent (Harushima et al. 2002; Wang et al. 2009; Xu et al. 1997). This suggests that indica alleles may be expected to predominate in every inter-subspecific indica/japonica cross in rice even though the chromosomal regions containing the clusters of distorted markers are not the same. Differences in clusters of distorted markers among populations and/or crossing directions (Harushima et al. 2002; Xu et al. 1997) should be the results of different mechanism of SD.

SD in F2 populations possessing indica cytoplasm strongly favored Dasanbyeo (indica) alleles, as did F2 populations possessing japonica cytoplasm (Additional file 2: Table S2). This was somewhat consistent with previous reports that distortion in segregated populations possessing indica cytoplasm favors indica alleles, while populations possessing japonica cytoplasm did not harbor distortion, favoring a specific parental allele (He et al. 2001; Peng et al. 2006; Wang et al. 2009).

Among chromosomal regions showing SD, chromosomes 3, 5, 6 and 12 exhibited the most severely distorted markers in one and/or both F2 populations. Moreover, the marker distortion in these chromosomal regions extended across a large portion of the chromosome, and to the whole arm of the chromosome in some cases (Additional file 3: Figure S1). In F2 populations, the type of selection (gametic and/or zygotic) responsible for SD can be determined by using maximum-likelihood models proposed by previous studies (Lorieux et al. 1995; Pham et al. 1990; Wang et al. 2005). The assumptions of the proposed models are theoretically simplified for only one or two SD loci and male gametophytic genes involving SD. However, the mechanisms producing the reproductive barriers observed in inter- and intra-specific crosses in rice are complex and variable within different populations (Wu et al. 2010). Thus, in the current study, the type of selection affecting SD was investigated by genotyping a common set of markers in a set of all possible reciprocal BC1F1 populations, which contains female-segregating and male-segregating populations. Biologically, SD could result from selection among gametes and/or zygoties, when an allele at a locus diminishes gametic or zygotic fitness, linked loci will deviate from the expected Mendelian segregation ratio (Alheit et al. 2011). Therefore in our study, the number of markers used in reciprocal BC1F1 populations should be increased in order to obtain an improved estimate of biological SD. BC1F1 genotyping is an effective approach for the investigation of SD since this hybridization strategy was able to identify female- and male-function effects and zygotic-selection effects, as well as the effects of cytoplasm and/or cyto-nuclear interaction, on SD.

Our data showed that the level of SD in BC1F1 populations was generally low relative to that in F2 populations, indicating that the cause of SD was simplified into male or female function in each BC1F1 population. Nevertheless, Dasanbyeo alleles were transmitted in higher frequencies than Ilpumbyeo alleles in both F2 and BC1F1 populations; this concurs with previous studies suggesting that indica alleles are strongly favored in inter-specific crosses of rice (Harushima et al. 2002; Wang et al. 2009; Xu et al. 1997). The level of SD among our BC1F1 populations also varied greatly. SD frequency was higher in male-segregating populations than in female-segregating populations, suggesting that the SD observed in our study is strongly driven by male function or pollen competition rather than female function (Table 2).

The SD loci in F2 populations did not always concur with SD loci in BC1F1 populations; for example, although significant SD was detected at S05064 in BC1F1 populations, no SD at S05064 was detected in either of the F2 populations. The Dasanbyeo allele at S05064 was favored in D/ID and I/ID, but the Ilpumbyeo allele was favored in I/ID. This conflicting preference for parental alleles depending on the male or female gametes might nullify the potential SD in F2 populations. Similarly, several SD loci in BC1F1 populations were normally segregated in F2 populations. Conversely, all the SD loci found in F2 populations exhibited SD in BC1F1 populations,
suggesting that the cause of SD loci in F2 populations can be explained by female and/or male function and/or zygotic selection.

It is reported that the linkage distance between markers varies at some chromosomal regions in different crosses and types of population, but the order of markers remains highly conserved (Antonio et al. 1996). In our study, in general, a reduction in the length of the genetic map was observed in male-segregating populations relative to female-segregating populations (Table 1). This suggests that there may have been more recombination in female gametes and thus, recombination frequency between marker pairs in male-segregating populations was lower than those in female-segregating populations. However, the population sizes in this study may not be large enough to draw a conclusion regarding the comparison of recombination frequencies between markers.

**Relationships of SD regions to hybrid barriers in rice**

In order to determine the relationship between SD loci and hybrid barriers in rice, comparative analysis was conducted based on physical mapping of each SD locus to the Nipponbare Pseudomolecule assembly annotated by IRGSP Build5 (http://www.rgp.dna.affrc.go.jp/). The chromosomal regions of substantial SD loci found in this study were compared to the regions with quantitative trait loci (QTLs) underlying hybrid sterility/fertility and gametophyte genes reported in previous studies on rice. Chromosomal regions with SD loci in this study were mostly related to, or corresponded with, chromosomal regions showing hybrid barriers (gametophyte or sterility gene) regions (Table 4).

Our comparisons revealed interesting regions corresponding to severely distorted loci on chromosomes 3, 5, and 6. The SD chromosomal region spanning 4.74–14.53 Mbp on chromosome 6 that was influenced by female function overlapped with hybrid barriers (Table 4). Hybrid barrier loci overlapping this region were as follows: cim and Cif, which are responsible for the cross incompatibility reaction in the male and the female, respectively (Matsubara and Khin-Thidar 2003); SI, which is responsible for gametelneination in O. rufipogon (Koide et al. 2008b); gametophyte genes ga-1 and ga-5 (Kinoshita 1995); S5, a locus with a key effect on embryo sac fertility (Chen et al., 2008; Yang et al. 2012); esa1, which affects embryo sac abortion (Liu et al. 2001); S6, which affects sex-independent SD affecting male and female gametogenesis (Koide et al. 2008a), L6,a locus affecting pollen fertility (He and Xu 2000); qSF6.2, which affects spikelet fertility (Reflinur et al. 2012); and the S8 locus for embryo sac sterility (Wan et al. 1993). These loci suggest that the chromosomal region affecting reproductive barriers, especially those influenced by female factors, is mostly conserved on chromosome 6.

The most severe SD region affected by male function was detected along chromosome 3 (4.3–24.3 Mbp). This chromosomal region overlapped the following loci: the gametophytic genes ga-2 and ga-3 (Xu et al. 1997); qHPS-3, which is responsible for pollen sterility (Chen et al. 2006); ga-2 and ga-3 (Xu et al. 1997); qHPS-3 (Chen et al. 2006); qSF6.2 (Reflinur et al. 2012); and qHPS-3 (Chen et al. 2006).

**Table 4 Comparison of segregation distortion loci with hybrid barriers from previous studies**

*Abbreviations are the same as in Table 3.*

| Chr | Selection type | Marker (range) | Position (Mbp) | Cross | Previous studies shared common regions |
|-----|----------------|----------------|----------------|-------|----------------------------------------|
| 1   | zygotic        | S01011 ~ S01038 | 4.38 ~ 7.46    | D/ID, DI/DI | ga-9 (Xu et al. 1997); f1 (Wang et al. 1998); qSF1 (Reflinur et al. 2012) |
| 1   | mSD            | S01157B ~ S01181B | 39.8 ~ 43.2    | I/ID  | GB (Harushima et al. 2001) |
| 2   | mSD            | S02126 ~ S02135  | 29.91 ~ 31.48  | D/DI  | |
| 2   | mSD            | S03027 ~ S03065  | 5.71 ~ 14.43   | D/ID, D/DII | |
| 3   | mSD            | S05004B ~ S05030B | 0.29 ~ 3.66    | D/ID, D/DI | ga-2 (Wang et al. 2006); f5 (Wang et al. 1998); S33 (Jing et al. 2007); L3b and S3b (He and Xu 2000); He et al. 1999; q33.1 (Marri et al. 2005); qSF3.1 and qSF3.2 (Reflinur et al. 2012); |
| 5   | mSD            | S05032 ~ S05077A  | 4.29 ~ 20.10   | D/DI, I/ID | qSF5.2 (Reflinur et al. 2012); |
| 6   | fSD            | S06018 ~ S06065A  | 4.74 ~ 14.53   | ID/I, DI/DI, DI/I | ga-1, ga-4 and ga-9 (Kinoshita 1995); S5 (Chen et al. 2008); S6 (Koide et al. 2008a); esa-1 (Liu et al. 2001); L6 (He and Xu 2000); S8 (Wan et al. 1993); qSF6.2 (Reflinur et al. 2012); |
| 8   | mSD            | S08060 ~ S08090   | 17.27 ~ 23.08  | I/ID  | S27 (Sobrizal and Yoshimura 2001) |
| 12  | mSD            | S12009A ~ S12011B | 0.63 ~ 1.88    | D/ID, DI/DI, I/DI | qSF12.2 (Reflinur et al. 2012); lwr12.1 (Li et al. 2004) |
| 12  | fSD            | S12066 ~ S12097B  | 19.44 ~ 25.00  | D/DI  | |

*aAbbreviations are the same as in Table 3.*

*bAbbreviations are the same as in Table 2.*
Backcross populations were developed from F1 as the female parent (1-4). Loci exhibiting deviation from et al. Rice is the male progeny (DI) of the S3b (Wang et al. 2006) (Kubo et al. 2000), which affect pollen sterility; qSF3.2, f3 (Wang et al. 1998), and progeny (ID) indica (Wang et al. 1998); (Marri et al. 2005); and variety, Dasanbyeo, (Reflinur et al. 2012).

Populations developed from Korean elite which affects hybrid pollen and eight and S24 populations derived from Nipponbare Pseudomolecule) and chromosome 12 (region for ISD at region of 19.44–25.00 Mbp). These findings suggest that the key factors on these chromosomal regions might be new gametophyte regions that influence SD.

One zygotic-selection-affected SD regions on chromosomes 1 overlapped with hybrid barrier regions that were identified in previous studies (Table 4). SD loci on chromosome 1 corresponded to gametophyte barrier, ga-9 (Xu et al. 1997), f3 (Wang et al. 1998), and qSF1 (Reflinur et al. 2012). Our finding that zygotic selection affects SD on chromosomes 1 conflicts with previous research from Harushima et al. (2002), which found that SD on these chromosomal regions was caused by gametophyte barrier. This may be due to the different genetic backgrounds of the backcross populations used in the previous study and in our study. In addition, no reciprocal backcrosses were performed in the previous study to confirm the action of zygotic selection on SD.

Conclusions
Taken together, based on the SD analysis in reciprocal F2 populations, we summarized that both nuclear genetic and cytoplasm factors from Dasanbyeo (indica) had stronger effects on SD than those from Ilpumbyeo (japonica). The gametophytic (female and male gamete function) and zygotic selection as mechanisms underlying SD could be explained by the distorted marker patterns using early generation of reciprocal backcross populations. The identification of SD distribution and the effect of genetic factors causing SD that were identified in the present study will provide not only a basis for future research on the elucidation of the molecular mechanisms underlying SD in rice, but also information beneficial for breeding strategies.

Methods
Plant materials
Ten reciprocal crosses consisting of two F2 and eight BC1F1 populations developed from Korean elite japonica variety, Ilpumbyeo, and Korean indica variety, Dasanbyeo, were used for genetic studies on SD analysis in interspecific crosses of rice. A total of 210 F2 progeny (ID) derived from the cross of Ilpumbyeo (female parent) and Dasanbyeo (male parent) and 199 F2 progeny (DI) of the reciprocal cross were used. Eight reciprocal BC1F1 populations were subsequently developed to distinguish female meiotic drive from male-specific sources of distortion, and gametic mechanisms from differential selection against zygote, as described in detail in Figure 2. The numbers of

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progeny from the BC1F1 crosses used for mapping and for SD analysis were as follows: 88 from ID/I, 25 from I/ID, 69 from DI//D, 69 from D//DI, 71 from ID//DI, 52 from D//ID, 68 from DI//L, and 27 from I//DI population. All plant materials were planted during rice-growing season 2009, at the experimental farm of Seoul National University, Suwon, Korea. Thirty-day-old seedlings were transplanted to irrigated field condition at one seedling per hill with a 30 x 15 cm of spacing between seedlings. Field management was carried out following normal agronomic practices. Fertilizers were properly applied at the rate of 100 kg N ha−1, 80 kg P ha−1, and 80 kg K ha−1 (100-80-80 kg/ha N-P-K).

Molecular marker analysis
DNA was extracted from young leaf blades according to the method of Causse et al. (1994), DNA samples from the F2 and BC1F1 populations were genotyped using 107 and 144 STS primers, respectively. These STS markers are dispersed throughout the rice genome and the primers used were designed by the Crop Molecular Breeding Lab, Seoul National University (Chin et al. 2007). Polymerase chain reaction (PCR) analysis and visualization of amplicons in the F2 and BC1F1 populations was performed as described previously (Reflinur et al. 2012).

Data analysis
Linkage map construction in F2 and BC1F1 populations was performed with Mapmaker/EXP 3.0 program. A LOD threshold of 3.0 was used for declaring linkage (Lander et al. 1987; Lincoln et al. 1992), and the Kosambi function was used to convert recombinant value to the genetic distances between the markers (Kosambi 1944). For the segregation data of each marker, deviations from the Mendelian ratios (1:2:1 ratio for F2 populations or 1:1 ratio for BC1F1 populations) were tested using chi-square analysis. A non-parametric technique using sequential Bonferroni method (Rice 1989) was applied to the segregation data of each marker in order to avoid type-I error deriving from the large number of tests.

Additional files

Additional file 1: Table S1. Physical locations of both end markers for each chromosome and linkage map coverage of the genome in two reciprocal F2 and eight BC1F1 populations.

Additional file 2: Table S2. Chi-square test for SD of markers and genetic factors influencing distorted markers in two reciprocal F2 populations generated from Ilpumbyeo and Dasanbyeo parents.

Additional file 3: Figure S1. Genotype frequencies of STS markers along chromosomes 3, 5, 6, and 10 in two reciprocal F2 populations generated from Ilpumbyeo and Dasanbyeo parents.

Additional file 4: Figure S2. Chromosomal location of pronounced SD loci observed in genetic linkage maps in the two reciprocal F2 and eight BC1F1 populations.
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