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Selective Embryo Abortion Hypothesis Revisited – A Molecular Approach

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Abstract: Many plant species abort a large fraction of their embryos. It has often been suggested that embryos of genotypes that would perform worse later in life are preferentially aborted. Such selective embryo abortion would lead to investment of resources only in the offspring with the highest potential fitness. Many studies have shown that otherwise viable embryos are aborted. However, only few manipulative studies have indeed shown a correlation between the level of abortion and offspring quality and these studies have been challenged for their experimental design. Molecular techniques open new opportunities to study selective embryo abortion. Non-random abortion at the level of molecular markers can be observed as a deviation from Mendelian segregation: over- or under-representation of markers in the offspring. Subsequently, the over- or under-represented markers can be related to offspring quality later in life. We reviewed the literature on the genetic maps of intraspecific crosses of wild plant species and the selection of cultivated species. The level of non-Mendelian segregation we found in these maps is high. On average, 11.5% of the tested markers in the genetic maps of wild species and 14.6% in the cultivated ones, show a departure from Mendelian segregation. From six studies, providing sufficient data, it was calculated that in 68% of loci segregating in non-Mendelian fashion post-fertilization selection is involved. We propose that the deviation from Mendelian segregation can be partly explained by selective embryo abortion. We describe an experimental design that allows for attributing selective embryo abortion to the non-Mendelian segregation that is found in a genetic map.

Key words: Selective embryo abortion, non-Mendelian segregation, distorted segregation, genetic maps, seed–ovule ratios, seed set, fruit set.

Introduction

Selective embryo abortion (SEA) is the phenomenon that some genotypes are aborted more frequently than others. The Selective Embryo Abortion hypothesis proposes that the fitness of a female plant can be increased by the selective abortion of genotypes with a potential low quality later in life such that an increase in the level of abortion leads to an increase in offspring quality. In evolutionary theory the hypothesis is important in relation to explanations for low seed to ovule ratios and the optimal allocation of resources to male and female reproduction in hermaphroditic plants.

The SEA hypothesis is based on the following premises: i) plants abort a substantial part of the embryos, ii) otherwise viable embryos are aborted, iii) the probability of being aborted depends on the genotype of the embryo. In contrast to ample theoretical attention given to the SEA hypothesis (e.g., Kozłowski and Stearns, 1989[56]; Latta, 1995[61]; Burd, 1998[16]), there are only a few experimental studies in which all conditions, mentioned above, were considered. The SEA hypothesis received a lot of attention in the 1980s (Stephenson, 1981[105]; Wilson and Burley, 1983[122]; Casper, 1988[19]; Lee, 1988[63]; Andersson, 1990[1]; Andersson, 1993[2]) but, probably due to difficulties with the interpretation of the results of experiments in which the abortion level was manipulated, empirical research on the topic has drastically reduced. Most of these problems can now be overcome using molecular techniques. In this paper we review the work on this hypothesis and discuss the possibilities and difficulties that are presented by the use of molecular methods to shed new light on the topic.

In crossings, SEA shows up as a deviation from Mendelian segregation for some molecular markers. We will review data on the genetic maps of plants to judge whether there is a potential for SEA to be detected by means of marker segregation analysis. However, SEA is not the only mechanism leading to deviation from Mendelian segregation. We will discuss how we may distinguish SEA from other mechanisms that include, e.g., meiotic drive, gametophytic selection, and seedling death.

Mechanisms of Embryo Abortion

Two explanations have been proposed for the selective abortion of particular genotypes: 1. Maternal control, 2. Embryo competition.

Since, potentially, SEA may be maternally controlled, it has often been discussed as an aspect of female choice together with pre-fertilization processes, such as selective inhibition of pollen germination and pollen tube growth (Wilson and Burley, 1983[122]; Marshall and Folsom, 1991[73]). Theoretically, in an-

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giosperms maternal control can be through the endosperm, the tissue that nourishes the embryos, because it contains two copies of the maternal and one copy of the paternal genes, also abortion may be related to an interaction of the maternal and paternal genome. However, Marshall and Folsom (1991[73]) concluded in their review on mate choice in plants that there is little evidence to prove that specific maternal mechanisms produce sorting among compatible donors. The problem with the assumption of maternal control is how the link with offspring quality later in life is brought about, in other words: how can the mother plant "know" which embryos will give the highest fitness contribution? Moreover, it is technically extremely difficult to experimentally test this hypothesis. If changes in the maternal tissue of the seed (nucellus and integuments) precede changes in embryo and endosperm development, this could point to maternal control. If the order of changes is the reverse, this would point to embryo competition (Marshall and Folsom, 1991[73] and refs. therein). As yet there is very little evidence to decide for either of the two possibilities.

More generally accepted is the idea that SEA is brought about through competition among embryos. Some embryos may be better competitors for resources than others, either because they present a larger sink or they may even release chemical substances which are most probably indole compounds that inhibit the sucrose uptake of siblings (Mohan Raju et al., 1996[80]; Krishnamurthy et al., 1997[57]; Arathi et al., 1999[3]). In this scenario, maternal "recognition" of the embryos is not necessary. The mother plant can influence offspring quality indirectly by controlling the level of resources and thus setting the selective arena for the embryos. It has even been suggested that endosperm reduces embryo competition since it is more frequently observed in the species with multiovulated ovaries compared to those with uniovulated ones, and it is found more often in the species with multiovulated species that experience less abortion (Uma Shaanker et al., 1996[111]).

However, from an evolutionary ecological perspective, the mechanism leading to SEA is not as interesting as the fact whether or not it can increase offspring quality. The increase of offspring quality through SEA would mean that embryo abortion is potentially adaptive. The correlation between the abortion level and offspring quality is, under the assumption of embryo competition, brought about by genes that control e.g., basic metabolic processes that are important, both during embryo development and during later life, or by genes that have pleiotropic effects. Goldberg et al. (1989[34]) summarize in their review: "More than 90% of the 15000 diverse mRNAs present in mid-maturation stage embryos are represented in both cotyledon stage and fully differentiated, mature embryos. Most of these mRNAs are also present in post-germination cotyledons and in the mature plant leaf." The fact that most of the genes that are expressed in embryonic stage are also expressed later in life, gives ample opportunity for embryo abortion to have an effect on offspring quality later in life.

Most evidence for the fact that abortion depends on the genotype of the embryo comes from studies on inbreeding depression. If selfed embryos have a higher chance of being aborted than outcrossed ones (Montalvo, 1992[81]; Gibbs and Sassaki, 1998[33]), this suggests that recessive deleterious or lethal alleles may influence competitive strength of embryos. Embryonic viability is often assessed in relation to early-acting inbreeding depression. On the basis of information on seed production after selfing and outcrossing, the number of so-called lethal equivalents is estimated. A lethal equivalent is a lethal gene or a number of deleterious genes that make up one lethal gene. According to Lynch and Walsh (1998[69]), the number of lethal equivalents per gamete affecting early embryonic survival varies approximately from 1.7 to 5.0 for conifers and from 0.4 to 0.91 for short-lived angiosperms. However, for considering SEA it is essential to make a distinction between lethal and deleterious alleles. If abortion results from recessive lethal alleles, both the level of abortion and the direction of selection are fixed. The embryos, which possess lethal alleles, will die irrespective of the conditions they encounter during development. In such cases it is not likely that the level of inbreeding depression during seed set will be correlated with the level of inbreeding depression later in life. This may explain the absence of such a correlation in the studies of Husband and Schemske (1995[40]) or Koelewijn et al. (1999[54]). On the other hand, if the alleles on which embryo abortions depend are deleterious, embryo abortion may be selective and depend on the conditions the embryo encounters. Remington and O'Malley (2000[91]) studied early acting inbreeding depression in loblolly pine *(Pinus taeda)* using information from a genetic map. They estimated that in this species 19 loci have moderately deleterious or lethal embryonic effects. Moreover, most of the alleles reducing viability are recessive and for 3 loci overdominance was found. There is also another study (Melser et al., 1997[75]) suggesting that embryo abortion may not be a result of action of recessive deleterious alleles. On the basis of comparing seed production after selfing and outcrossing in *E. vulgare* (after sufficient amount of pollen was applied), they found that some individual plants aborted more selfed embryos and others more outcrossed ones. Melser et al. (1997[75]) concluded that in *E. vulgare* the effects of the deleterious alleles are mostly additive.

One can imagine that embryo abortion may be influenced by a number of (mildly) deleterious alleles that each by themselves have only a small effect and are therefore not easily purged from a population.

In the remaining part of the paper we will first present the more traditional phenotypic approach to study SEA and then we will discuss how molecular techniques can be used to study the same research questions.

Phenotypic Approach

The level of abortion

Flowering plants commonly produce more ovules than seeds. In many angiosperm species ovules may not develop into seeds due to pollen limitation (Wolfe, 1983[123]; Zimmerman and Pyke, 1988[126]; and see Burd, 1994[15] for a review) or because they are involved in self-incompatibility mechanisms (Waser and Price, 1991[118]; Seavey and Carter, 1996[98]). However, even after successful fertilization, a considerable proportion of the ovules fail to produce seed in many species. Wiens (1984[119]) estimated that seed–ovule ratio equals, on average, about 85% for annuals and 50% for perennials. Wiens' data are based on developing fruits. If ovules in undeveloped fruits are also included, the seed ovule ratios may be even lower. Dissections of ovaries showed that a large fraction of embryos are aborted, for example in *Prunus cerasus* (Bradbury, 1929[12]), *Oxalis magnifica* (Guth and Weller, 1986[36]) and *Epilobium angustifolium* (Wiens et al., 1987[120]). Some species show extremely high abortion rates. In *Dedeckera eurekensis,* the seed–ovule ratio equals 2.5%, although about 90% of the ovaries initiate growth, indicating that fertilization took place (Wiens et al., 1989[121]). In *Asclepias speciosa* approximately only 3.8% of the ovaries develop into mature fruits, although 82.4% of them were fertilized (Bookman, 1984[10]).

Gymnosperms also abort many seeds. In *Pinus sylvestris* on average 30% seeds are aborted (Karkkainen et al., 1999[49]). The level of embryo abortion is, however, higher due to polyembryony. The most common form is simple polyembryony with independent fertilizations of more than one archegonium within the same ovule of which usually only one develops into a seed (Sorensen, 1982[103]; Willson and Burley, 1983[122]; Haig, 1992[37] and references there in).

In some cases it has been argued that embryos are not viable because of high genetic load, as in the *D. eurekensis* example (Wiens et al., 1989[121]). However, as we will discuss later, even viable embryos are often aborted at a very high rate.

Are potentially viable embryos aborted and does abortion increase offspring quality?

In some species with linearly arranged ovules, developing embryos in the basal end of the ovary are more likely to abort. Nakamura (1988[83]) described a successful *in vitro* culture of embryos from the basal end in *Phaseolus vulgaris.* In *Dalberia sisso,* Ganeshaiah and Uma Shaanker (1988[31]) cut off two distal seeds and implanted the remaining pod in agar to complete maturation of the rest of the seeds. This treatment resulted in an abortion rate in the basal end of the pod as low as in the distal end of intact, control pods. Both Nakamura (1988[83]) and Ganeshaiah and Uma Shaanker (1988[31]) did not relate abortion to offspring quality.

To our knowledge, only four papers present evidence that abortion can increase offspring quality. These experiments compared offspring quality after natural abortion and after random thinning of the ovaries. In contrast to the first, the latter is not selective. Species of the family of the Boraginaceae always produce four ovules in each flower arranged in equal positions, in a square. Although pollen is not limiting seed production, on average there are fewer than four seeds per flower found in many species of the Boraginaceae (e.g., *Cynoglossum officinale* – Jong and Klinkhamer, 1989[48]; *Echium vulgare* – Klinkhamer et al., 1994[53] and *Cryptantha flava* (Casper, 1988[19]). In *Cr. flava* (Casper, 1988[19]) and in *Cy. officinale* (Melser et al., 2001[76]) the random destruction of three ovules in a flower resulted in doubling of the chance of maturation for the remaining ovule, compared to a control treatment with all ovules intact. This shows that in the control treatment a large fraction of the aborted embryos was potentially viable. In *Cr. flava* seeds from the control group with natural abortion showed higher emergence and survival during two years of growth (Casper, 1988[19]). Melser and Klinkhamer (2001[76]) found that natural abortion resulted in higher offspring survival in *Cy. officinale.* In *Lotus corniculatus,* offspring produced after natural embryo abortion showed better germination, produced more leaves, and later in life more inflorescences, flowers and matured more seeds compared to the treatment after random destruction of ovules (Stephenson and Winsor, 1986[106]). In a study on patterns of seed abortion in *P. coccineus,* Rocha and Stephenson (1991[96]) found that ovules at the basal end of the ovary are more likely to abort, due to the fact that they lag behind in development because they were pollinated later, which may result in reduced nutrient availability. Destroying the ovules on the stylar end increased the probability of seed maturation on the basal end. The progeny that resulted from this treatment was significantly less successful compared to the control treatment with regard to germination time, vegetative growth, flowering time and number of flowers.

Is abortion dependent on embryo genotype?

Stephenson (1981[105]) and Lee (1988[63]) show in their reviews that in many plant species the chance for an embryo of being aborted depends on factors like time of initiation, position within ovary, resource availability and pollen source. Even if the level of embryo abortion is high and viable embryos are aborted, abortions do not necessarily depend on the genotype of the embryo and may not lead to selection. Both single pollen donor and mixed pollen donor experiments have been used to study the relationship between abortion rate and genotype.

The easiest way to detect selection is when each flower of a plant receives pollen from a single pollen donor only. One can then compare the siring success of different potential fathers directly, by counting the seeds in the flowers, without the use of genetic markers. With this approach, it is unlikely that pollen tube competition influences differences among fathers. The disadvantage of single pollen donor experiments is that competition among the embryos within a flower cannot be detected.

Bertin (1982[8]) studied the self-incompatible trumpet creeper *(Campsis radicans)* and applied pollen of different fathers and found that the pollen donors that were favoured by particular recipients were usually those whose pollinations resulted in fruit with many and large seeds. Although prezygotic mechanims were not all properly excluded, the author concluded that fruit abortion seems to have been more important in donor selectivity than prezygotic phenomena.

Most single pollen donor experiments aim at comparing abortion after self- and outcross pollination, or comparing outcross pollination with close and distant donors. Such comparisons are interesting, especially because Husband and Schemske (1996[41]) showed that embryo development is one of the most important life stages in which inbreeding depression can act.

For the self-compatible *Aquilegia caerulea* Montalvo (1992[81]) found that the abortion rates for selfing were, on average, 38% higher compared to abortion for outcrossing, while there were no significant differences in fertilization rate for both pollination types. For *E. vulgare,* Melser et al. (1997[75]) found that in some individuals self-pollen was relatively more successful compared to outcrossed pollen, while in others the outcrossed pollen was more successful. Pollen donors did not differ in pollen viability, pollen germination and pollen tube growth. Therefore, Melser et al. (1997[75]) concluded that differences in siring success of different pollen donors were most likely caused by selection among embryos.

Gibbs and Sassaki (1998[33]) found for *Dalbergia miscolobium* in the field that 30.0% of crossed flowers and 3.6% of selfed flowers developed mature fruits. This difference was mainly caused by abortion of selfed embryos because, in the ovules dissected 4–6 days after pollination, embryos were found in similar frequency and condition for both treatments.

Marshall and Whittaker (1989[74]) studied effects of identity of a pollen donor on offspring quality in *Raphanus sativus.* They found significant paternal effect on the number of leaves and weight of offspring after eight weeks of growing in a greenhouse. The effects of pollen donor were more pronounced if maternal plants were grown in water stress conditions. Their results suggest that the processes that sort among potential fathers during pollination, fertilization and seed filling may improve offspring quality.

Multiple donor experiments, where a mixture of pollen from different genotypes is applied to a single flower, combined with paternity analysis, can also provide information about SEA. The advantage of multiple donor experiments is that selection among pollen donors within flowers can be detected. The disadvantage is that, if it is not possible to analyse aborted embryos for their paternity, an appropriate method has to be found to separate the effects of pollen tube competition from SEA. Marshall and Ellstrand (1988[72]) carried out a multiple donor experiment on *Raphanus sativus* under stress conditions. Early water stress can affect both fertilization and early seed abortion. In contrast, late water stress can only influence seed abortion. The contribution to the progeny of the three pollen donors differed from the control in the late stress treatment but not after early stress. Apparently, only late abortions provide the opportunity to select in this case.

Attributing the abortion rate to the origin of pollen in some gymnosperms is even easier since they have poorly developed prezygotic selection mechanisms (Willson and Burley, 1983[122]). For example, Karkkainen et al. (1999[49]) determined the abortion rate in *Pinus sylvestris* as a proportion of empty seeds, because seed coat formation in this species is an effect of pollination. They found that frequency of abortion increases with the proportion of self pollen applied to the flowers. The proportion of empty seeds ranges from 23% after outcrossing to 76% after selfing.

Limitations of phenotypic approach

Missing information about the selection among genetically different offspring sired by the same father. Pollination experiments can show that selective abortion exists only if siring success of different fathers is compared, either after single donor pollination or after mixed donor pollination combined paternity analysis. Moreover, it is necessary to eliminate that prezygotic mechanisms that may play a role. The big disadvantage of this approach is that a part of post-fertilization selection, which may occur among genetically different offspring of the same father, cannot be observed. Only molecular techniques can provide data on that.

Crushing ovules does not always reduce abortion levels. One can explain Casper's (1988[19]) and Melser and Klinkhamer's (2001[76]) results by assuming that the resources not used by the destroyed ovules are allocated to the remaining ovules within the same flower, thereby increasing the chance for maturation. If the experimental treatment is applied to only a part of the flowers, it is possible that resources that would be used by crushed ovules are divided among all ovules of the plant and not only among those that remained in the hand-thinned flowers. In such a case, the difference among the treatments would be small and could go undetected. Perhaps this may at least partly explain the negative results found in two studies on *Anchusa officinalis* (Andersson, 1990[1]) and *Achillea ptarmica* (Andersson, 1993[2]). The difficulties in the interpretation of results from the experiments discussed above can be avoided if a single treatment is applied to a whole plant and the same genotypes are used in different treatments (Melser et al., 2001[76]).

Decreased offspring quality after random crushing of ovules may be an artefact. In experiments based on ovule destruction, inferior offspring do not necessarily result from genetic differences but may be caused by subtle effects of the mechanical damage itself. Casper (1988[19]) cautions: “Prematurely removing some reproductive structures might upset initial source–sink relationships and thus plant–resource levels, adversely affecting seed quality. In addition, forcing a flower to distribute resources to an ovule that it normally would not mature might itself result in an inferior seed.” Moreover, developmental irregularities of the flower can influence the competitive strength of the embryo.

An experiment, as described above, is therefore not sufficient to prove that SEA occurs. The best way to show that SEA can increase offspring quality in ovule destruction experiments is to collect genetic evidence as well. We will therefore discuss in the remaining of the paper how molecular data can be used to overcome the problems caused by the traditional approach to the SEA hypothesis.

Genotypic Approach

If embryo abortion is selective, certain alleles will be under- or over-represented in the offspring, compared to Mendelian segregation. The upswing in molecular methods in the last decade has led to easy access to abundant molecular markers in almost every organism (e.g., AFLP). Such molecular markers might be a powerful tool to detect and assess the adaptive value of SEA. Using molecular markers avoids the limitation of pollination experiments because selection among offspring of a single pollen donor can be detected. Even if the plant is self-pollinated, selection among embryos may be observed in the loci for which the parent plant was heterozygous. So far, selection among the offspring of a single father has largely been ignored. This may have caused an under-estimation of the level of SEA.

The second advantage of using molecular markers to test the SEA hypothesis is that the presence or absence of alleles that are under- or over-represented in the offspring, compared to Mendelian segregation, can be related to offspring performance in later life. This would be a much better way of assessing the selective advantage of embryo abortion compared to traditional methods, because no manipulations of flowers or plants (e.g., destroying ovules) are needed and because selection can be directly linked to the genotype of the offspring.

Selection among embryos can be presented at the level of DNA as a deficiency or excess of certain genotypes among the offspring that successfully went through seed maturation, compared to expected Mendelian segregation. We reviewed genetic maps of plants in order to determine the potential for SEA. If the percentage of molecular markers showing non-Mendelian segregation found in genetic maps of plants is as low as expected due to chance alone, we have to conclude that SEA is not an important process. This argument, however, cannot be reversed. If many markers show non-Mendelian segregation, that could be due to SEA but other selective mechanisms cannot be excluded. For instance, meiotic drive and gametophytic selection can also lead to non-Mendelian segregation (Appendix). The difficulty in distinguishing the cause of non-Mendelian segregation is a disadvantage of this method. An appropriate experimental design should be used to study segregation in plants, with different treatments leading to differences in the level of abortion, as will be discussed later.

Is non-Mendelian segregation common in plants?

Data about non-Mendelian segregation in plants can be found in genetic maps. In almost all genetic maps of plants we have reviewed, authors refer to a statistically significant departure from Mendelian segregation as *distorted segregation,* although they usually do not present any evidence for the presence of segregation distorter genes *sensu* Lyttle (1991[70]). Lyttle defines segregation distorters as genetic elements that exhibit meiotic drive. That is why, when we consider a statistically significant departure from Mendelian segregation, we will use the more neutral term: non-Mendelian segregation.

It is common practice to test, by means of a chi square test at a 5% significance level, whether or not segregation of a certain marker deviates from the expected ratio. If all markers are inherited independently, 5% of all markers should show non-Mendelian segregation, if no selection occurs. However, it is extremely difficult to determine the expected fraction of markers showing non-Mendelian segregation under the null hypothesis, that no selection occurs. Firstly, non-Mendelian segregation can be over- or under-estimated when judged from the number of loci with a significant non-Mendelian segregation because, in a distorted region of the genetic map, the density of mapped molecular markers may differ from the average. Secondly, an unknown percentage of DNA markers is located in non-functional regions (e.g., not or loosely linked to functional regions). For such markers, only non-Mendelian segregation due to chance is expected. Nevertheless, Tables **1** and **2** provide useful information because, averaged over all species, the first problem should disappear as we have no reason to assume that the density of molecular markers is higher or lower in the region where selection occurs. The second problem can only lead to an under-estimation of selection. Unfortunately, we do not know the quantitative importance of this.

We searched for genetic maps based on intraspecific crosses of cultivated and wild species. Wild species were defined in the broadest sense possible. The basic criterion we used was that artificial selection and inbreeding were minimal. We expected that in genetic maps of cultivated species non-Mendelian segregation is found more often because mapping populations are often derived from crosses between different inbred line varieties or come from distinct geographical areas (e.g., Loarce et al., 1996[68]; Jenczewski et al., 1997[43]; Liu et al., 1997[67]; Qi et al., 1998[89]). It may happen that genes from one inbred line/variety do not function properly when combined with genes from another inbred line/variety.

Results of the literature survey are presented in Tables **1** and **2**. The percentage of markers showing non-Mendelian segregation differs significantly from 5% for the 59 analysed species ($t = 9.143$; $df = 58$; $p < 0.001$). It ranges from 0.1 – 40.82% (average: 14.6) for cultivars (Table **1**) and from 0 – 41.0% (average: 11.5) for wild species (Table **2**).

The difference in the average percentage of markers showing non-Mendelian segregation between cultivated and wild species is not significant ($F = 1.099$; $df = 1,57$; $p = 0.299$).

Distinguishing between biological phenomena and technical problems

Sometimes it is argued that sampling error or irreproducibility of the techniques can be responsible for a high percentage of molecular markers showing non-Mendelian segregation. Here we will consider the importance of those problems.

Inconsistent PCR amplification can cause irreproducibility of the method and hence a detection of apparently higher non-Mendelian segregation. RAPD is known as a technique that does not always give fully reproducible results (Jones et al., 1997[45]). We therefore compared the level of non-Mendelian segregation detected in genetic maps using three techniques: AFLP, RAPD and RFLP. None of the techniques gave significantly higher level of non-Mendelian segregation (paired samples test results for: RAPD vs. RFLP: $df = 10$, $p = 0.433$; RFLP vs. AFLP: $df = 4$, $p = 0.222$; RAPD vs. AFLP: $df = 4$, $p = 0.386$), although PAGE gels used in AFLP give much higher resolution than agarose gels used commonly in RAPDs. Note, however, that the tests are based on a small number of comparisons. The constant warning (e.g., Jones et al., 1997[45]) that RAPDs are not fully reproducible may have caused a severe selection against markers giving non-Mendelian segregation previously used in mapping. Many authors using RAPD markers for the construction of a genetic map, only include markers which are efficiently amplified and exhibit unambiguous polymorphism. Jenczewski et al. (1997[43]) write: "When such precautions are taken, RAPD does not induce higher levels of distortion than restriction fragment length polymorphisim (RFLP)." Discarding markers before use in mapping, although to a smaller extent, may have happened in these other techniques as well. Tables **1** and **2** may therefore underestimate the level of non-Mendelian segregation.

Other sources of artefacts can be homoplasy, which is the amplification of two fragments of the same length from non-allelic regions, low resolution of agarose-gels, and co-migrating and overlapping polymorphic fragments. However, we expect these explanations to have only a minor influence on the level of non-Mendelian segregation. Rieseberg (1996[95]) tested the homology of 220 RAPD co-migrating fragments in three close-

Table 1 Percentage of non-Mendelian segregation found in genetic maps of cultivated species[$]

Species	Segregating population		No. of polymorphic markers	% of markers showing nMS at 0.05	No. of loci showing nMS for each type of marker								References
					RAPD		RFLP		AFLP		Other[#]		
	Nature	No.			Total	% nMS	Total	% nMS	Total	% nMS	Total	% nMS	
Allium cepa[*]	F3	58	128	14.84[b]	14	0.00	112	16.96			2	0.00	King et al., 1998[52]
Brassica napus	F2	90	82	29			82	29					Cloutier et al., 1997[22]
Carica papaya[*]	F2	200–253	62	20.97[a]	61	21.31[a]					1	0.00	Sondur et al., 1996[104]
Cucumis melo	F2	112	5	20.00							5	20.00	Katzir et al., 1996[50]
Cucumis melo	BC	66	228	14.47	6	0.00			221	14.93	1	0.00	Wang et al., 1997[116]
Cucumis melo[*]	F2	218	110	12.73	68	16.18	36	2.78			6	33.33	Baudracco-Arnas and Pitrat, 1996[5]
Festuca pratensis[*]	F2	56	70	37.14[a]			70	37.14[a]					Chen et al., 1998[20]
Gossypium hirsutum[*]	F2.F3	96	138	8.70			138	8.70					Shappley et al., 1998[99]
Glycine max[*]	F2	65	85	2.35							85	1.18	Wang et al., 1998[115]
Hordeum vulgare	F9 RIL	103	568	8.45					568	6.45			Qi et al., 1998[89]
Hordeum vulgare	F8 RIL	167	113	4.42[b]	32	9.37[b]	78	2.56[b]			3	0.00[b]	Teulat et al., 1998[108]
Hordeum vulgare[*]	F2	134 or 40	306	7.52[a]			22	9.09[a]	284	7.39[a]			Richter et al., 1998[93]
Malus domestica[*]	F1	96	10	0.10							10	0.10	Gianfranceschi et al., 1998[32]
Malus pumila[*]	F1	152	290	11.38[a]	133	7.52[a]	124	14.52[a]	4	0.00[a]	29	10.34	Maliepaard et al., 1998[71]
Medicago tornata	F2	80	33	39.39	32	40.62					1	0.00	Jenczewski et al., 1997[43]
Medicago truncatula	F2	122	49	40.82	48	39.58					1	100.00	Jenczewski et al., 1997[43]
Oryza sativa	F5 BIL	98	245	4.9[a,b]			245	4.9[a,b]					Lin et al. 1998[65]
Oryza sativa	BC	122	118	12.50			57	[c]	61	[c]			Tan et al., 1998[107]
Oryza sativa[*]	3way cross	230	191	8.38[a]			191	8.38[a]					Liu et al., 1997[67]
Oryza sativa[*]	F2	178	71	12.68[a]			71	12.68[a]					Price and Tomos, 1997[88]
Phaseolus vulgaris	F8 RIL	72–75	599	17.53	489[a]	[c]	100[a]	[c]			10[a]	[c]	Freyre et al., 1998[30]
Pisum sativum	F2 RIL	139	259	8.11	248	8.06	9	11.11			2	0.00	Laucou et al., 1998[62]
Prunus persica[*]	F2	63	270	1.85	92	1.09	50	8.00	115	0.00	13	0.00	Dirlewanger et al., 1998[25]
Secale cereale[*]	F2	258	91	10.99			88	9.09			3	66.67	Korzun et al., 1998[55]
Secale cereale[*]	F2	54	104	20.19	12	0.00[a]	77	19.48[a]					Loarce et al., 1996[68]
Setaria italica[*]	F2	138	160	21.25[a]			160	21.25[a]					Wang et al., 1998[117]
Sorghum bicolor[*]	F5 RIL	110	155	5.81[a,b]			155	5.81[a,b]					Dufour et al., 1997[26]
Sorghum bicolor[*]	F5 RIL	91	129	24.03[a,b]			129	24.48[a,b]					Dufour et al., 1997[26]
Theobroma cacao[*]	BC	131	138	4.35[a]	104	3.85[a]	32	6.25[a]			2	0.00[a]	Crouzillat et al., 1996[24]
Triticum turgidum[*]	F7 RIL	65	259	18.53			244	[c]			15	[c]	Blanco et al., 1998[9]
Vigna unguiculata[*]	F8 RIL	94	181	14.36[a]	133	11.28	19	15.79	25	24.00	4	0.00	Menendez et al., 1997[77]
Zea mays	F2[d]	112	201	14.43	140	17.14	161	3.11					Beaumont et al., 1996[6]
Zea mays[*]	F3:4	80	106	8.49[a]			106	8.49[a]					Tuberosa et al., 1998[110]

[$] We have searched for the genetic map of cultivated species using the keywords: "genetic map" or "linkage map" and "plant"in the journal "Theoretical and Applied Genetics" from volume 93 (year 1996) till volume 97 (year 1998). We used Winspirs 2.0 to search in the Current Contents database. The search resulted in 222 records. Data from 33 out of 222 papers could be included in the table. A paper was included if the number of loci with significant non-Mendelian segregation for the genetic map could be calculated. Partial genetic maps and maps based on doubled haploids or intraspecific cross were not included.

Many doubled haploid lines are derived from the pollen of one parent. Analysing these lines yields the segregation directly without the necessity of crossing. After pollen germination and regeneration of haploid plants, chromosome doubling occurs spontaneously or it is induced chemically (by colchicine). The plants grow and then the material is sampled for DNA analysis. During the germination of pollen and while the plants are growing in *in vitro* culture, selective mortality may occur. This mortality might explain the relatively high levels of non-Mendelian segregation found in the doubled haploid offspring (Xu et al., 1997[125]). Since the offspring did not develop from embryos, the genetic maps based on double haploid populations were not reviewed in this study. We did not include maps derived from interspecific and intergeneric crosses because, in such wide crosses, chromosome pairing and other phenomena – that are not related to selective embryo abortion – may play roles.

nMS: non-Mendelian segregation, BC: backcross, RIL: recombinant inbred lines, BIL: backcross inbred lines;

[a] % of markers showing nMS only in the map (the number of loci that showed nMS and that are not linked in the genetic map could not be retrived from the article),

[b] only markers showing nMS given for the probability level $p < 0.01$ were presented,

[c] the type of markers that showed nMS could not be indentified in a paper,

[d] two mapping populations combined together,

[#] this category of markers may have included the following markers: isoenzymes, minisatellites, microsatellites, IGS, SCAR, CAPS, PCR markers, rDNA, STS, morphological and biochemical markers.

[*] indication of a genetic map that could be used for analysis of distribution of loci with non-Mendelian segregation along linkage groups. This analysis is described in a chapter: "distinguishing between biological phenomena and technical problems".

Table 2 Percentage of non-Mendelian segregation found in genetic maps of wild species[$$]

Species	Segregating population		No. of poly-morphic markers	% of markers showing nMS at 0.05	No. of loci showing nMS for each type of marker								References
					RAPD		RFLP		AFLP		Other[#]		
	Nature	No.			Total	% nMS	Total	% nMS	Total	% nMS	Total	% nMS	
Acacia magnum	F1	108	153	3.92			153	3.92					Butcher et al., 2000[14]
Acacia magnum	F1	123	169	5.33			169	5.33					Butcher et al., 2000[14]
Aegilops tauschii *	F2	56	546	24.18			447	27.96[a]	53	11.32[a]			Boyko et al., 1999[11]
Arabidopsis thaliana	BC	93	47	10.64[a]	31	[c]	2	[c]			14	[c]	Kuittinen et al., 1997[60]
Cryptomeria japonica *	F2	73	164	23.78	33	21.21	128	24.22			3	33.33	Mukai et al., 1995[82]
Hordeum chilense	F2	100	463	32.61					463	32.61			Vaz Patto et al., 2001[113]
Hordeum vulgare ssp. spontaneum	F2	233	321	41.00					308	41.56	13	23.08	Vanhala – unpublished data
Lotus japonicus	F2	50	15	0.00[a]	12	0.00					3	0.00	Jaing and Gressloff, 1997[42]
Picea abies	F1 – M.	17	14	0.00	14	0.00							Skov, 1998[101]
Picea abies	F1 – M.	17	11	0.00	11	0.00							Skov, 1998[101]
Picea abies	F1 – M.	80	145	3.45	145	3.45							Skov and Wellendorf, 1998[100]
Picea abies *	F1 – M.	72	447	5.60					366	6.01	81	3.70	Paglia et al., 1998[84]
Pinus edulis	F1 – M.	40	542	6.09					542	6.09			Travis et al., 1998[109]
Pinus pinaster	F1 – M.	124 or 34	473	0.00[b]	437	0.00					35	0.00	Plomion et al., 1995[86]
Pinus pinaster	F2 – M.	126	120	0.00[b]	120	0.00[b]							Plomion et al., 1996[87]
Pinus pinaster *	F2 – M.	200	378	6.35	127	5.51			239	7.11	61	0.00	Costa et al., 2000[23]
Pinus radiata	F1 – M.	198	202	38.12	198	37.88					4	50.00	Kuang et al., 1998[59]
Pinus strobus *	F1 – M.	72	103	2.91	97	3.09					6	0.00	Echt and Nelson, 1997[27]
Pinus sylvestris	F1	29	226	8.40					226	8.40			Lerceteau and Szmidt, 1999[64]
Populus deltoides	BC	93	523	6.50					523	6.50			Wu et al., 2000[124]
Psedotsuga menziesii *	F2	48	217	6.45	27	[c]	188	[c]			2	[c]	Jermstad et al., 1998[44]
Psedotsuga menziesii *	F1 – M.	80	247	18.22	247	18.22							Krutovskii et al., 1998[58]
Psedotsuga menziesii *	F1 – M.	80	261	10.34	261	10.34							Krutovskii et al., 1998[58]
Quercus robur	F1	94	307	19.87[a]	271	[c]					36	[c]	Barreneche et al., 1998[4]
Senecio jacobaea *	F1	140	77	15.58					77	15.58			Vrieling – unpublished
Zizania palustris	F2	172	126	10.32			126	10.32					Kennard et al., 1999[51]

Abbreviations as in Table **1**, M.: megagametophytes;

[$$] We searched for genetic maps of wild species by means of Winspirs 2.0 in the Current Contents database until August 2000. We used the same keywords as in the search for genetic maps of cultivated species, but in all available journals. The search resulted in 1275 records. We scanned the abstracts of all articles to find genetic maps of intraspecific crosses of wild plant species. The criteria of incuding the data from a genetic map into this review were the same as for cultivated species.

ly related species of sunflowers and found that 91% of fragments are homologous. This means that artefacts like homoplasy and wrong scoring due to low resolution of agarose gels may be responsible for only 9% of co-migrating fragments. However, this number would be much lower if individuals from the same mapping population derived from an intraspecific cross were compared.

Moreover, artefacts mentioned above cannot explain why non-Mendelian segregation often occurs in clustered loci. We screened the reviewed genetic maps for the distribution of markers showing non-Mendelian segregation. On the basis of the data from 30 maps (these maps are indicated in Tables **1** and **2**) we found that 56% of 633 loci segregating in non-Mendelian fashion formed clusters of two or more markers.

A strong argument for the fact that the non-Mendelian segregation is not found by chance or sampling error is the repeatability of finding skewed markers in the same species in many crosses, with a different set of parents, or in the same chromosomal regions. Xu et al. (1997[125]) mapped chromosomes of rice using many types of crosses: inter-subspecific crosses, doubled haploid and recombinant inbred lines. They detected a non-Mendelian segregation by means of RFLP in all types of crosses, ranging from 17% for one of the inter-subspecific crosses, to 70% for one of the doubled haploid populations. 227 distorted markers were clustered in 17 chromosomal regions, and nine of these regions were associated with segregation distortion in more that one population. Repeatability of non-Mendelian segregation in the same region of genetic

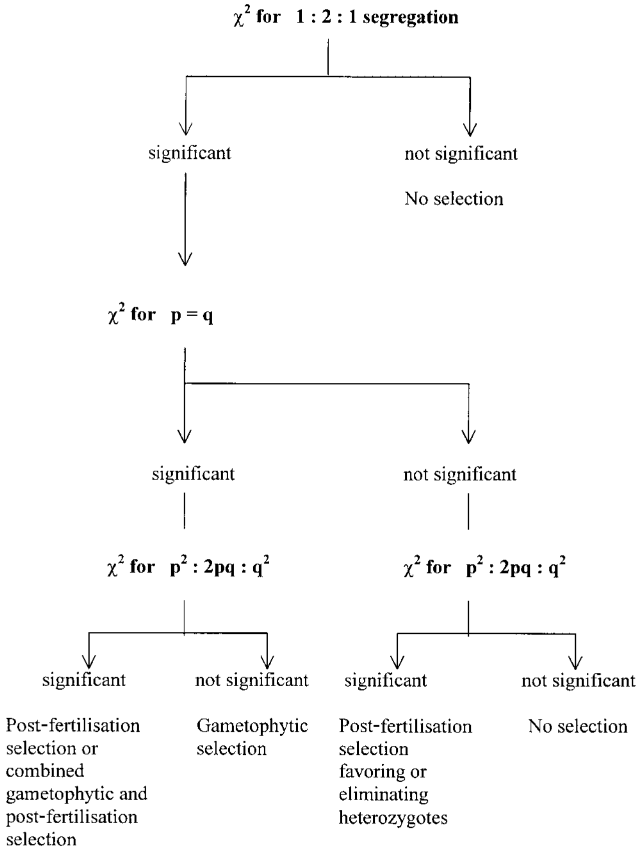

Fig. 1 Key for determining whether gametophytic or post-fertilisation selection takes place in a segregating F_2 population analysed with co-dominant markers (based on Pham, 1990[85]).

maps derived from different crosses of the same species was also reported by Price and Tomos (1997[88]).

How to distinguish between SEA and other biological explanations for non-Mendelian segregation?

There is a long list of biological explanation for non-Mendelian segregation in plants, it includes, apart from SEA, meiotic drive, gametophytic selection, selective germination and seedling death, B chromosomes, cytoplasmic inheritance, endophytes and chromosomal rearrangements (Appendix). Therefore, it is very difficult to separate SEA and other causes of non-Mendelian segregation. Below we will present three ways allowing for such discrimination.

Genotyping embryos

Direct evidence for SEA may be presented by molecular marker segregation analysis of aborting embryos in comparison to mature seeds. Rigney (1995[94]) performed a successful paternity analysis for aborting embryos by means of the MDH allozyme marker. Rigney (1995[94]) removed embryos that were being aborted from a plant and analysed their paternity in *Erythronium grandiflorum.* Selfed embryos were more likely to abort than outcrossed ones. Moreover, the progeny fertilized by nearby donors are aborted more often than those sired by distant donors.

The difficulty with isolation of embryos and the very small amount of material may limit the feasibility of this method. PCR-based techniques, like microsatellites, can be a better alternative to allozyme analysis since they require much a smaller amount of plant material. Hufford et al. (2000[39]) have shown that aborting embryos of *Platypodium elegans* can be successfully genotyped by means of microsatellites. Reusch (2000[92]) also used microsatellites to genotype developing embryos in *Zostera marina.* However, isolation of embryos at the stage when they are large enough for analysis makes it impossible to investigate effects of very early stages of abortion.

Analysing segregation patterns

When one cannot analyse the aborting embryos for their paternity, it is rather difficult to judge what was the cause of observed non-Mendelian segregation that is already detected in a map. An attempt to separate different causes has been made by Pham et al. (1990[85]), who determined whether selection before or after fertilization took place on the basis of segregation analysis of isozyme loci in an F_2 generation in several crosses of rice. They used successive χ^2 tests for 18 loci in which non-Mendelian segregation was found. Firstly, the equifrequency of alleles (p, q) was tested. Secondly, a χ^2 test was made to test if the distribution of genotype frequencies fits $p^2 : 2pq : q^2$ (based on the observed allele frequencies p and q) (see Fig. **1**). Since, for most of the tested skewed loci, the frequency of alleles was not equal and genotype frequencies fitted the $p^2 : 2pq : q^2$ distribution, Pham et al. (1990[85]) concluded that prefertilization (gametophytic) selection was responsible for non-Mendelian segregation. However, for other loci, evidence for post-fertilization selection was found. Guiderdoni (1991[35]) reported similar results for crosses between different varieties of rice.

We performed such an analysis for the reviewed genetic maps that reported segregation data for the F_2 generation for co-dominant markers. We found segregation data for such markers only in 6 genetic maps (Mukai et al., 1995[82]; Baudracco-Arnas and Pitrat, 1996[5]; Katzir et al., 1996[50]; Chen et al., 1998[20]; Korzun et al., 1998[55]; Vanhala, unpublished data). Chi square analysis of 56 loci revealed that, in 31 loci, post-fertilization selection affecting heterozygotes took place, while in 7 loci, zygotic or a combination of selection before and after fertilization occurred. In 15 loci gametophytic selection was detected. In 3 loci the stage of selection could not be determined by means of subsequent χ^2 tests. This analysis shows that post-fertilization selection occurs in the majority of cases (68%) involved in non-Mendelian segregation.

Another method to track down the cause of non-Mendelian segregation is the analysis of segregation of molecular markers in reciprocal crosses. Korzun et al. (1998[55]) performed such crosses in rye *(Secale cereale)* and found, in one cross, 7 loci showing non-Mendelian segregation, while in the other cross such a skewed segregation was found for 9 loci. Only 2 of those loci were common for both crosses and they are potential loci in which post-fertilization selection could take place. Asymmetry of segregation data in reciprocal crosses could be caused by post-fertilization selection due to an interaction between nuclear and cytoplasmic genes which is different, depending on which plant is used as a female in a cross, or gametophytic selection affecting either male or female function of one of the parents. The distinction between pre- and post-fertilization selection in the latter case could be made if reciprocal backcrosses to both parents are performed. Faris et al. (1998[28]) compared non-Mendelian segregation in 4 such crosses in *Aegilops tauschii.* They attributed nearly the whole observed non-Mendelian segregation on chromosome 5D to gametophytic selection affecting male function, however, they could not exclude nuclear–cytoplasmic interaction in one region on that chromosome.

Possible experimental design to test SEA hypothesis

Attributing non-Mendelian segregation, in crosses used to make genetic maps, to one of the explanations given in the appendix can be done in some cases if an experiment is carefully planned or additional research is conducted. Some of the explanations (other than SEA) for non-Mendelian segregation can be ruled out by additional studies such as: chromosome counting and observation of pollen germination (Appendix). The best way to separate the effect of SEA from other explanations listed in appendix would be to find an experimental treatment with which the level of abortion is manipulated without influencing other processes. Nutrient stress would be a good candidate: it is known to influence abortion levels while there are no reports that it influences e.g., meiotic drive. If the deviation from Mendelian segregation for certain molecular markers is positively correlated with the level of embryo abortion this would imply that, indeed, embryo abortion is selective. Using the same genotype (clone) in all treatments could further strengthen the argument because meiotic drive, B chromosomes, cytoplasmic inheritance and chromosomal rearrangements can be excluded, since the same nuclear genes (chromosomes) are in the same cytoplasmic environments in all nutrient treatments. If clones are grown in the same environment, they could possibly also contain the same endophytes.

An alternative experiment would be to compare the segregation of molecular markers in the offspring coming from two treatments performed on self-pollinated clonal replicates of one genotype of a plant. The first treatment would have the ovules randomly crushed, while the control treatment would not be manipulated. If selective abortion is playing a role, then it is expected that more loci in the offspring of control plants would show non-Mendelian segregation.

The advantages of these two experimental designs are that:

1. non-Mendelian segregation can be attributed to SEA,
2. it can be established if SEA leads to higher offspring quality, and linked to the genotype of the offspring,
3. at least for the control treatment, SEA can be studied in unmanipulated plants,
4. non-Mendelian segregation can also be studied among the offspring of a single father,
5. it is possible to find markers for SEA that can be used on other genotypes,
6. major loci controlling SEA can be detected.

Conclusions

The traditional experimental phenotypic approach to test the SEA hypothesis has the disadvantage that the treatment itself can be a source of artefacts. That is why the evidence that SEA increases offspring quality is still weak. The molecular genotypic approach links SEA to the presence and absence of particular alleles, which is why offspring quality can be related to specific alleles and, therefore, manipulations, such as crushing ovules, are not necessary.

The level of non-Mendelian segregation found in the published genetic maps suggests that there is ample opportunity to detect SEA. An appropriate experimental design, which we propose in this paper, would not only detect SEA and provide the possibility to relate this to offspring quality, but it would also provide more information about the genetic mechanisms controlling this process.

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Appendix

Biological explanations for non-Mendelian segregation in genetic maps of plants. Meiotic drive. Lyttle (1991[70]) defines meiotic drive as "mechanics of meiotic division that cause one member of a pair of heterozygous alleles or heteromorphic chromosomes to be transmitted to progeny in excess of the expected Mendelian proportion of 50%". A number of meiotic drive systems are described in detail for animals. However, little is known about meiotic drive in plants. In most flowering plants, megasporogenesis may lead to meiotic drive because of an obvious asymmetry of meiotic division: only one of the four haploid cells develops into a functional egg and this cell may contain preferentially transmitted alleles or chromosomes. An example of such a process is the preferential transmission of chromosomal knobs (large clusters of repetitive DNA) on chromosome 10 into viable megaspores in maize (Buckler et al., 1999[13]). Another example of meiotic drive, although of interspecific origin, is the preferential transmission of alien chromosomes. Such chromosomes, common in Triticeae, are called "cuckoo" chromosomes (Miller, 1983[78]). Finch et al. (1984[29]) described the effects of one chromosome coming from *Aegilops sharonensis* in wheat *(Triticum aestivum)* plants. Such monosomic plants have abnormal female and male meiosis, only meiospores containing the alien chromosome develop into normal gametophytes. Only such a cytological analysis combined with mapping would allow for attributing non-Mendelian segregation found in the map to meiotic drive.

Gametophytic selection includes all phenomena that cause differential success of pollen from different donors or pollen from the same donor but bearing different alleles. Gametophytic selection may occur, for example, during pollen germination and pollen tube growth. Germination of pollen *in vitro* is a standard method used to assess both its viability and pollen tube growth. However, only in a very few cases are such tests combined with data on segregation of molecular markers (Lin et al., 1992[65]; Sari-Gorla et al., 1992[97]). Often, gametophytic selection is assumed to occur on the basis of allele frequencies in the offspring for the loci in which non-Mendelian segregation was found. Under-representation of one of the alleles is then attributed to gametophytic selection in one of the parents (see e.g., Wagner et al., 1992[114]). Such studies neglect the fact that post-fertilization selection affecting homozygotes also influences allele frequency.

Selective germination and seedling death. Kuang et al. (1998[59]) linked non-Mendelian segregation to seedling death in *Pinus radiata.* A comparison of the segregation of RAPD markers was made for megagametophytes, for surviving seedlings and those that died within the first month after germination in order to find markers for which segregation was significantly skewed in opposite directions in both groups. A null allele of one locus was over-represented in dead seedlings while it was strongly under-represented in the seedlings that were still alive. The authors suggested that an allele closely linked to this null allele is responsible for the seedlings' death. Moreover, a segregation analysis at the same locus for unsown seeds showed that the null allele was over-represented in this stage. Kuang et al. (1998[59]) gave two possible explanations: selection favouring this allele prior to germination or a sampling error. If the allele responsible for seedling death is indeed favoured during embryo maturation, this would present a case opposite to that is predicted by the SEA hypothesis.

The elimination of selective seed germination and seedling death as the explanation for non-Mendelian segregation found in the map can be done if seed that did not germinate and dead seedlings are included into the segregation analysis.

B chromosomes are supernumerary chromosomes that are not essential for growth and reproduction of organisms. They have been described in more than 1000 species of plants (see Jones and Rees, 1982[47] for a review). B chromosomes can be distinguished from normal (A) chromosomes because they are usually smaller and consist of large amounts of heterochromatin. Their number may be variable even within the same individuals due to nondisjunction in anaphase of mitosis. B chromosomes are inherited in a non-Mendelian fashion which, according to Jones (1991[46]), can be caused by their nondisjunction in female or/and male meiosis, nondisjunction in mitosis during development of the male gametophyte, or preferential fertilization by B-containing sperm. B chromosomes usually consist of repetitive DNA and some of such repeats were found to code for ribosomal RNA (Camacho et al., 2000[17] and references therein). Theoretically, markers of B chromosomes may appear in genetic maps as single, unlinked markers or separate linkage groups. A way to avoid this possibility is careful selection of parents without B chromosomes (by chromosome counting), for the mapping population.

Cytoplasmic inheritance via plastids and mitochondria can show up as non-Mendelian segregation in molecular markers in the case of biparental inheritance of organelles. In angiosperms, inheritance of plastids is predominantly maternal and in gymnosperms – paternal. However, cases of biparental inheritance of those organelles were also described. Examples are alfalfa *(Medicago sativa),* evening primrose *(Oenothera)* and *Pelargonium* cultivars (Mogensen, 1996[79]). Ten out of 68 angiosperm species and 3 out of 11 gymnosperms listed by Reboud and Zeyl (1994[90]) are classified as having biparental inheritance of plastids. A similar picture arises from a review of Smith (1988[102]). Nineteen out of 60 angiosperms showed at least occasional biparental inheritance. Less examples (only four species) are available for biparental inheritance of mitochondria for two reasons. Firstly, this phenomenon has not received much attention (Reboud and Zeyl, 1994[90]). Secondly, it may occur less often. Species with biparental inheritance of plastids may have strictly maternal transmission of mitochondria (Mogensen, 1996[79]).

Endophytes. A diversity of organisms, like bacteria and fungi, are known to live inside and among plant tissues (Carroll, 1988[18]; Clay, 1988[21]; Hallmann et al., 1997[38]). The DNA from endophytes may be extracted together with plant DNA and eventually give the same effect as contamination. Cytoplasmic inheritance and endophytes can potentially be observed as distorted unlinked markers. However, molecular markers for organelle DNA will never be linked to markers for nuclear genes and, if there are two polymorphic markers for organelle DNA, they will be 100% linked to each other because of a lack of recombination. Molecular markers for eucaryotic endophyte DNA may appear in a map (resulting in more groups than chromosomes). However, they will never be linked to the markers that are known to be developed for plants e.g., morphological markers.

Chromosome rearrangements, such as translocation and duplication, are often suggested causes of non-Mendelian segregation found in genetic maps (e.g., Vaillancourt and Slinkard, 1992[112]). However, genetic mapping alone is not sufficient to link non-Mendelian segregation directly to translocation. Belay and Merker (1998[7]) analysed inheritance of the translocation on chromosome 6B in tetraploid wheat *(Triticum turgidum)* by cytogenetics. They observed C-banding patterns in the F_2 generation derived from two crosses, both with one parent homozygous for a translocation and one parent homozygous for the lack of translocation. In both crosses, non-Mendelian segregation was observed. Homozygotes containing 2 chromosomes without translocation were over-represented.

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