Balanced male sterile-lethal systems for hybrid barley production
by Mohammad Mokhlesur Rahman

A thesis submitted to the Graduate Faculty in partial fulfillment of the requirement for the degree of DOCTOR OF PHILOSOPHY in Crop and Soil Science
Montana State University
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Abstract:
Linkage relationships between nine previously located lethal mutants and male sterile genes were determined. Nine lethal mutants not previously located were described. All nine mutants were inherited as monogenic recessives and temporary symbols, alb,,a through alb,,i were assigned. Linkage relationships between these mutants and identified male sterile, genes were determined and five mutants were assigned to the chromosomes. The effects of heterozygous and homozygous loci of 20 lethal mutants on yield and yield components were studied. Nine of the 20 population studied exhibited one locus heterosis (positive or negative advantages of Aa genotypes as compared to AA genotypes) for yield per plant or one or more of the components of yield.

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MOHAMMAD MOKHLESUR RAHMAN

A thesis submitted to the Graduate Faculty in partial fulfillment of the requirement for the degree

of

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ABSTRACT

Linkage relationships between nine previously located lethal mutants and male sterile genes were determined. Nine lethal mutants not previously located were described. All nine mutants were inherited as monogenic recessives and temporary symbols, alb,,a through alb,,i were assigned. Linkage relationships between these mutants and identified male sterile genes were determined and five mutants were assigned to the chromosomes. The effects of heterozygous and homozygous loci of 20 lethal mutants on yield and yield components were studied. Nine of the 20 population studied exhibited one locus heterosis (positive or negative advantages of Aa genotypes as compared to AA genotypes) for yield per plant or one or more of the components of yield.

INTRODUCTION

The successful commercial production of hybrid barley, *Hordeum vulgare* L., depends upon a reliable source of male sterile individuals to serve as the female parents. Recent proposals for production of hybrid barley by Wiebe (1960), Ramage (1965) and Eslick (1970) utilizing genetic male sterility emphasize the desirability of locating many genes to facilitate such approaches. Each hybrid system requires a unique genetic mechanism, gene action, and linkage relationship.

The possible use of seedling lethality for elimination of undesired genotypes in the hybrid barley systems suggested this study.

The pusposes of this study were to determine the linkage relationship between located seedling lethal and male sterile genes, and to assign previously unlocated lethal genes to their respective chromosomes and determine their linkage relationship with male sterile genes.

Since several proposed systems result in F_1 commercial production with all or a portion of the plants heterozygous for seedling lethality available stocks were used to determine the effects of the heterozygous lethal condition on yield components and yield.

REVIEW OF LITERATURE

Male Sterility:

Male sterility in barley, *Hordeum vulgare* L., was first reported by Suneson in 1940, since then 335 different male sterile stocks of spring barley have been listed (Hockett et al., 1968; Hockett and Eslick, 1968, and Hockett, 1972). Twentythree have been determined to be non-allelic and the remaining need to be tested for allelism, except those that are derived through backcrossing.

Nuclear genes govern the genetic male sterility in tomato, *Lycopersicon esculentum* Mill., (Larson and Paur, 1948), sunflower, *Helianthus annuus* L., (Putt and Heiser, 1966), corn, *Zea mays* L., (Beadle, 1932), and wheat, *Triticum aestivum* L., (Athwal et al., 1967). Genetic male sterility in barley is controlled by single recessive nuclear genes, *ms* (Suneson, 1940; Hockett et al., 1968; and Hockett and Eslick, 1968). However, more complex cases of male sterility inheritance have been reported. Allison and Fisher (1964) reported that male sterility in cotton, *Gossypium hirsutum* L., was controlled by a single dominant gene, while in sunflower male sterility was governed by duplicate recessive genes (Putt and Heiser, 1966).

Cytoplasmic male sterility in different crops has been discussed by a number of investigators, but its existance in barley was first reported by Schooler in 1967. The behavior of this cytoplasmic male sterility was different from that of genetic male sterility and the fertility seemed to be controlled by two genes (Foster and Schooler,

1970); whereas the fertility restoration in genetic male sterility of barley is controlled by a dominant allele, _Ms_ (Suneson, 1940; and Hockett and Eslick, 1968).

Hybrid Barley Systems:

Heterosis in corn, sugarbeet, _Beta vulgaris_ L., sorghum, _Sorghum vulgare_ Pers., and onion, _Allium cepa_ L., has stimulated the development of hybrid barley. Since barley, by nature, is a highly self-pollinated species, time consuming hand emasculation precludes any possibility of commercial hybrid barley seed production without genetic or cytoplasmic sterility.

Barley researchers have proposed several schemes. The most important among them are: Utilization of stocks carrying informational gene or genes closely linked with natural or chemical roguing mechanisms for undesirable genotypes, and genetic and/or cytoplasmic male sterility systems for development of the desired populations.

Following the discovery of the DDT (Dichloro-dipheno-trichloro-ethane) resistance gene in barley (Hays, 1959), Wiebe (1960) proposed a scheme for hybrid barley using _ms-ddt_ linkage which requires adequate isolation and one or more sprayings with DDT in the early seedling stage. Male sterility as well as DDT resistance are recessive and can be incorporated by back-crossing into a desired variety that could be used as the female in hybrid seed production. The parental genotype would be _ms-ddt/Ms-Ddt_, which upon selfing would produce 1 _ms-ddt/ms-ddt_, male sterile and resistant to DDT : 2 _ms-ddt/Ms-Ddt_, fertile and

susceptible to DDT : 1 Ms-Ddt/Ms-Ddt, fertile and susceptible to DDT. Application of DDT would kill all but the genotype ms-ddt/ms-ddt, resistant to DDT. Seeds on the male sterile plants would come from the pollen furnished by the unsprayed rows of the genotype, Ms-Ddt/ms-ddt.

To maintain the seed stock, seeds obtained from the male sterile plants, the genotypes of which would be Ms-Ddt/ms-ddt and ms-ddt/ms-ddt, would be sown in rows. Every alternate row would then be sprayed with DDT to obtain pure stands of male sterile plants and ensure enough pollen supply from the unsprayed rows. Seed harvested from the sprayed rows would be 1 ms-ddt/ms-ddt : 1 ms-ddt/Ms-Ddt. Repeating this cycle would ensure stock maintainance indefinately.

To produce hybrid seed, however, seeds from the sprayed rows would be planted in alternate rows or blocks with the desired male. The rows from the previously sprayed seeds would be sprayed again to obtain pure stands of male sterile plants, ms-ddt/ms-ddt. Seeds harvested from these sprayed rows would be hybrid. The closest linkage between a male sterile and ddt genes, so far as reported, was seven cross-over units between ddt and ms16 on chromosome 7 (Wiebe, 1964). Extensive hand roguing would be required to purify the female stock in the crossing block.

Ramage in 1963 first advocated the use of balanced tertiary trisomics (BTT) for hybrid barley seed production. The basic approach of this system was to establish a stock in which the recessive male sterile

allele, ms would be carried on the normal diploid chromosome complement. The dominant allele, Ms would be carried on the translocated extra chromosome closely coupled with the interchange break-point (Ramage, 1965). Since the extra chromosome is usually not transmitted through pollen, BTT, upon selfing, would produce about 70 percent diploids, 30 percent BTT's and less than one percent primary trisomics.

The first commercial hybrid barley, 'Hembar', was developed utilizing the BTT system. Since this hybrid system lacks any genetic mechanism for the separation of diploids from trisomics or vice versa, hand roguing of diploids for parental stock maintenance is necessary and hand roguing or competition for removal of trisomics from diploid females in crossing blocks to produce pure diploid stand is relied upon.

To avoid hand roguing and to ensure sufficient pollen production by BTT's, Ramage (1965) suggested a modification of Wiebe's (1960) scheme, where two phytocides instead of one would be used. The dominant gene, A, would be resistant to phytocide "A". The recessive gene b would be resistant to phytocide "B". The dominant phytocide genes, A and B would be carried on the extra translocated chromosome of a BTT closely linked with a Ms gene. Trisomics would be resistant to phytocide "A" but susceptible to phytocide "B", while the diploids would be susceptible to phytocide "A" but resistant to phytocide "B".

To maintain the seed stock and produce the hybrid, two isolated blocks would be required. A field planted with trisomic seeds would be

sprayed with phytocide "A" to eliminate the diploids to maintain pure trisomic seed stock. The seeds from this sprayed trisomics would be planted in another block where application of phytocide "B" would ensure pure stands of the diploid. These diploids would serve as female parents for hybrid seed production.

An alternative method to this scheme would be to use sprayed trisomics as the male to pollinate an adjacent block planted with the seeds from the trisomic and sprayed with phytocide "B" to leave only male sterile diploids. The crossed seed harvested would serve as seed for diploid female seed for the hybrid seed production field without phytocide application. Vigorous trisomics as pollinators would be necessary.

To reduce the cost of production of hybrid seed Wiebe and Ramage (1970) suggested the use of an albino, *a*, gene in the BTT system as a sorting mechanism. This method is very similar to the system proposed by Ramage in 1965 except the phytocide is replaced by a lethal or albino gene, *a*.

This BTT with the seedling lethal albino, *a*, system requires establishment of a very close coupling of a male sterile allele, *ms*, and the recessive allele, *a*, of a seedling lethal or albino.

To produce this BTT-male sterile-albino stock, BTT-*Ms*-*A**/*ms*-*a*/*ms*-*a*, an initial stock of *ms*-*a*/*ms*-*A* would be produced through selection from a cross between *ms*-*A*/*ms*-*A* and *Ms*-*A*/*Ms*-*a*. This *ms*-*a*/*ms*-*A* stock in turn

* Indicates coupling of *Ms* and *A* on the extra translocated chromosome.

would be crossed with a homozygous translocation where Ms and A are either closely linked with the break-point or on the interstitial segment. Through selfing and selection the BTT-Ms-A*/ms-a/ms-a genotype will be recovered. Selfing of the BTT-Ms-A*/ms-a/ms-a would produce 30 percent BTT, the parental type and 70 percent diploid. The diploid will die due to homozygous lethality. Assuming no crossing over repeated selfing of BTT in isolation would prepetuate this stock indefinitely.

In the second phase, the BTT-Ms-A*/ms-a/ms-a used as a male would be crossed onto the ms-A/ms-a genotype. Assuming no transmission of BTT-Ms-A* through the pollen, the homozygous albino seedlings would die leaving ms-A/ms-a which would be crossed with an appropriate male, Ms-A/Ms-A. Seeds harvested from this cross would be true hybrid seed for commercial production.

Eslick and Hockett (1967) suggested the use of a non-chromosomal aberation breeding method not requiring the use of translocations or trisomics. They proposed the use of male steriles in conjunction with dominant pre-flowering selective genes. Translocation data established ms10 and ms14 as being very close to the centromere of chromosome 1 (Eslick, 1970); there is no reported crossing over between these two male sterile genes, except in one case where in a very small population 2.0 ± 2.0 percent crossing over was observed (Eslick, Hockett and Kushnak, 1972). The general approach of this scheme was such that ms10 and ms14 would be in repulsion phase, ms10-Ms14/Ms10-ms14 to serve as

parental stock. Since there is practically no recombination between ms10 and ms14 the stock could be maintained indefinitely if grown in isolation.

To produce hybrid seed separation of homozygous male sterile seeds is needed. A pre-flowering selective gene, such as blue aleurone, Bl2 on chromosome 1 and Bl on chromosome 4 might be utilized (Eslick, 1970). These genes are complementary and at least one dominant allele at each locus is needed to express blue aleurone (Myler and Stanford, 1942). Eslick (1970) reported that ms10, ms14 and bl2 were very close to the centromere of chromosome 1. To establish the system chromosome 4 would have the homozygous dominant alleles, Bl Bl. Chromosome 1 would be heterozygous, Bl2 bl2 and the parental genotype would be ms10-Ms14-Bl2/Ms10-ms14-bl2. Upon selfing the segregation would be 1 ms10-Ms14-Bl2/ms10-Ms14-Bl2, male sterile and blue seeds: 2 ms10-Ms14-Bl2/Ms10-ms14-bl2, fertile and blue seeds, : 1 Ms10-ms14-bl2/Ms10-ms14-bl2, male sterile and white seeds. In isolation with no recombination this stock would be self maintaining. About 25 percent of these seeds, i.e. white seeds, could be color sorted and utilized as female stock for hybrid seed production.

To separate the white seeds from the blue ones mechanically incorporation of the naked caryopsis, n, gene on chromosome 1 would be desirable, if not essential.

Alternately, Eslick (1970) also proposed the use of a balanced

male-sterile-recessive-lethal stock where one male sterile gene very closely linked with a lethal gene could be used. The parental genotype would be ms-A/Ms-a. One-fourth of the population, homozygous for fertility and lethality would die, leaving the heterozygous parental type and homozygous male sterile genotype. With no recombination, this stock would be self maintaining if grown in isolation.

For hybrid seed production, as for the balanced male sterile system, separation of the homozygous male sterile seeds from the parental type is necessary. This may be accomplished with any pre-flowering selective gene closely coupled with the male sterile and albino seedling lethal genes. The parental genotype would be ms-A-x/Ms-a-X*. In F_2 the segregation might be expected to be 1 ms-A-x/ms-A-x, male sterile, viable and short : 2 ms-A-x/Ms-a-X, fertile, viable and tall, : 1 Ms-a-X/Ms-a-X, fertile, lethal and tall. Homozygous fertile would be eliminated naturally due to homozygous lethality. The progeny of the parental type would be harvested in bulk and would maintain the stock by crossing or selfing in isolation. If the tall plants were cut previous to flowering only the short plant, ms-A-x/ms-A-x would remain to serve as female in the crossing block for hybrid seed production.

Other suggested systems have involved the use of male sterile genes with restorer cytoplasm (Hermsen, 1965; Hockett and Eslick, 1970; and Pfeifer, 1972) and restoration of fertility of genetic male steriles

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* Hypothetical gene for plant height or other pre-flowering selective gene.

through the application of gibberellic acid (Kasembe, 1967).

Linkage:

Ten different translocations involving chromosome 1, 3, 4, 5, and 6 established male sterile, ms10 and male sterile, ms14 to be very close to the centromere of chromosome 1 (Eslick, 1970). Considering the double cross-overs from F_3 progeny rows Eslick, Rahman and Crowell (1971) gave a preliminary report that the gene sequence of male sterile, ms10, short awn, lk2, naked caryopsis, n, and albino seedling, a_{c2} was lk2-n-ms10-a_{c2} on chromosome 1; ms10 being very close to the centromere (Eslick, 1970). The three point recombination values confirmed the gene order: lk2-n-ms10 (Eslick, Hockett, and Kushnak, 1972). Using the recombination value between various genes belonging to linkage groups III and VII Takahashi and Hayashi (1958) summarized the gene orders of lax head, l, virescent seedling, y_c, brachytic br, lk2, n, & a_{c2} in the sequence of lk2-n-l-a_{c2}-y_c-br, with the conclusion that these two linkage groups belonged to the same chromosome. This contention was supported by the translocation studies of Kramer, Veyl and Hanson (1954).

Results from crosses involving 24 translocations and 9 individual genes (Persson, 1969a,b) established the gene order to be br-f_c-y_c-a_{c2}; a_{c2} being about 5 to 10 cross-over units from the centromere and the recombination value between a_{c2} and n was about 11 percent. Tsuchiya (1972) utilizing telocentric chromosomes tentatively proposed the position of the centromere of chromosome 1 to be between a_{c2} & n; a_{c2}

being on the short arm and *n* on the long arm (Persson, 1969b).

A brief summary of linkage values pertinent to this study are presented in Table 1.

Linkage was detected between Betzes male sterile, *ms23b* and *se* with recombination values of 11.3 ± 3.7 percent (Hockett unpublished data) and 6.6 ± 3.4 percent (Eslick unpublished data). Jarvi (1970) located *ms23b* on chromosome 1 and *se* was linked with *ms23b*. McProud (1971) observed *ms23b* to be independent of *n* and *lk2*.

Eslick (1970) observed no recombination between long awned outer glume, *e*, and male sterile, *ms2*, in T2-7d balanced tertiary trisomics; and complete linkage between *ms2* and translocation break-points of T2-4a and T2-6a. However, when 13 translocations involving chromosome 2 were tested against *ms2* nil to 2 percent recombination values between *ms2* and *e* were observed (Ramage, 1966); this close linkage placed *e* and *ms2* very close to the centromere of chromosome 2 (Ramage and Burnham, 1962). Since the recombination values between male sterile, *ms3* and *e* was 4.6 ± 2.0 percent (Kasha and Walker, 1960) *ms3* should be close to the centromere of chromosome 2. Robertson, Wiebe, and Shands (1955) proposed the gene ordering on chromosome 2 of orange seedling, *or*, chlorina plant color, *f*, virescent seedling, *y*, and virescent seedling, y_x, which was determined to be a multiple allele of *y* (Robertson, Wiebe, and Shands, 1955), as $or - y \overset{Yx}{-} f - e$. Subsequently, using the recombination percentages between various genes, Robertson *et. al.* (1965) and

Table 1. A brief summary of linkage data

Character	Percent recombination	Authority
Genes on Chromosome 1		
Normal vs. male sterile, Ms10 ms10 in relation to:		
Covered vs. naked caryopsis, N n	7.2 ± .4	Eslick, Hockett and Kushnak, 1972
Covered vs. naked caryopsis, N n	12.7 ± 1.6	Jarvi, 1970
Long vs. short awn, Lk2 lk2	14.7 ± .7	Eslick, Hockett and Kushnak, 1972
Normal vs. albino seedling, A_{c2} a_{c2} in relation to:		
Covered vs. naked caryopsis, N n	27.2 ± 2.0	Robertson, 1937
Covered vs. naked caryopsis, N n	11.3 ± 1.4	Persson, 1969b
Covered vs. naked caryopsis, N n	7.3 ± 1.6	Takahashi and Hayashi, 1958
Green vs. virescent seedling, Yc yc	28.1 ± 1.6	Haus, 1958
Long vs. short awn, Lk2 lk2	36.1 ±	Takahashi and Hayashi, 1958
Normal vs. waxy endosperm, Wx wx	46.8 ± 2.3	Tabata, 1957
Normal vs. waxy endosperm, Wx wx	43.8 ± 1.3	Tabata, 1961
Green vs. virescent seedling, Yc yc in relation to:		
Long vs. short awn, Lk2 lk2	37.9 ±	Takahashi and Hayashi, 1958
Covered vs. naked caryopsis, N n	34.5 ± 4.1	Persson, 1969b
Covered vs. naked caryopsis, N n	31.5 ± 3.9	Takahashi and Hayashi, 1958
Long vs. short awn, Lk2 lk2 in relation to:		
Covered vs. naked caryopsis, N n	8.7 ± .6	Takahashi and Hayashi, 1958
Covered vs. naked caryopsis, N n	7.9 ± .3	Eslick, Hockett and Kushnak, 1972
Covered vs. naked caryopsis, N n	8.7 ±	Konishi, 1972
Covered vs. naked caryopsis, N n	8.8 ±	Takahashi *et al.*, 1953

Table 1. Cont.

Character	Percent recombination	Authority
	Genes on Chromosome 2	
Normal vs. male sterile, Ms2 ms2 in relation to:		
Normal vs. glossy sheath, Gs5 gs5	9.1 ± 0.7	McProud, 1971
Two row vs. six row, V v	33.5 ± 2.1	McProud, 1971
Normal vs. long awned outer glume, E e	23.0 ± 6.0	Austenson, 1948
Normal vs. chlorina, F f	27.0 ± 6.9	Austenson, 1948
Normal vs. glossy sheath, Gs5 gs5 in relation to:		
Two row vs. six row, V v	32.0 ± 2.5	Rasmusson and Lambert, 1965
Two row vs. six row, V v	24.2 ± 1.3	Takahashi, Hayashi & Moriya, 1971
Two row vs. six row, V v	26.0 ± 1.4	McProud, 1971
Normal vs. long awned outer glume, E e in relation to:		
Normal vs. white streak 4, Wst4 wst4	6.5 ± 1.0	Takahashi and Moriya, 1969
Two row vs. six row, V v	31.0 ± 2.2	Rasmusson and Lambert, 1965
Two row vs. six row, V v	22.9 ± 4.8	Kasha and Burnham, 1965
Two row vs. six row, V v	26.5 ± 2.0	Woodward, 1957
Two row vs. six row, V v	33.3 ±	Kasha and Walker, 1960
Normal vs. glossy sheath, Gs5 gs5	2.5 ± 0.8	Rasmusson and Lambert, 1965
Normal vs. albino seedling, A2 a2	18.4 ± 1.2	Robertson, 1967
Normal vs. white streak 4, Wst4 wst4	6.5 ± 1.0	Takahashi, Hayashi, & Moriya, 1971
Normal vs chlorina, F f	2.6 ± 0.4	Robertson, et. al., 1944

Table 1. Cont.

Character	Percent recombination	Authority
Genes on Chromosome 2		
Normal vs. orange seedling, Or or in relation to:		
Normal vs. chlorina, F f	8.7 ± 0.4	Robertson and Coleman, 1940
Normal vs. long awned outer glume, E e	14.8 ± 0.9	Robertson, et. al., 1944
Normal vs. virescant seedling, Y y	13.3 ± 0.6	Robertson and Coleman, 1940
Normal vs. virescent seedling, Y y	15.0 ±	Robertson and Coleman, 1942
Normal vs. albino seedling, A2 a2	5.5 ±	Robertson, 1967
Two row vs. six row, V v in relation to:		
Normal vs. white streak 4, Wst4 wst4	34.3 ±	Takahashi, Hayashi & Moriya, 1971
Green vs. albino seedling, A2 a2 in relation to:		
Normal vs. chlorina seedling, F f	13.0 ± 0.7	Robertson, 1967
Normal vs. long awned outer glume, E e	18.3 ± 1.2	Robertson, 1967
Normal vs. chlorina seedling, F f in relation to:		
Normal vs. virescent seedling, Y y	0.8 ±	Robertson and Coleman, 1940
Position of genes in relation to centromere:		
Normal vs. orange seedling, Or or	15.0 ±*	Ramage, 1966
Normal vs. orange seedling, Or or	13.0 ± 3.9*	Ramage, Burnham & Hagberg, 1961
Normal vs. chlorina, F f	8.0 ± 1.7*	Ramage, Burnham & Hagberg, 1961
Two row vs. six row, V v	28.0 ± 5.4*	Ramage, Burnham & Hagberg, 1961
Two row vs. six row, V v	28.0 ±*	Ramage, 1966

Table 1. Cont.

Character	Percent recombination	Authority
	Genes on Chromosome 2	
Position of genes in relation to centromere:		
Normal vs. long awned outer glume, E e	10.7 ± 3.4*	Ramage, Burnham & Hagberg, 1961
Normal vs. long awned outer glume, E e	11.7 ± 2.6*	Persson, 1969a
Normal vs. male sterile, Ms2 ms2	2.0 ±*	Ramage, 1966
	Genes on Chromosome 3	
Normal vs. Uzu dwarf, Uz uz in relation to:		
Normal vs. Xantha seedling, Xs xs	29.5 ± 3.9	Kasha and Burnham, 1965
Normal vs. Xantha seedling, Xs xs	23.8 ± 3.5	Kasha and Burnham, 1965
Normal vs. Xantha seedling, Xs xs	31.9 ±	Takahashi, 1972
Normal vs. Xantha seedling, Xs xs	29.3 ±	Takahashi, 1972
Normal vs. Xantha seedling, Xs xs	25.0 ±	Takahashi, 1972
Green vs. albino seedling, Ac ac	14.4 ± 1.0	Takahashi and Hayashi, 1953
Green vs. albino seedling, Ac ac	10.7 ±	Takahashi and Yamamoto, 1951
Green vs. albino seedling, Ac ac	14.2 ± 3.2	Takahashi and Hayashi, 1959
Green vs. white seedling, An an	19.0 ± 0.1	Takahashi and Hayashi, 1958
Green vs. white seedling, An an	13.1 ± 2.1	Kasha and Burnham, 1965
Green vs. white seedling, An an in relation to:		
Normal vs. xantha seedling, Xc xc	9.0 ±	Robertson, 1929 & 1937
Normal vs. xantha seedling, Xs xs	15.5 ± 0.8	Robertson, 1937
Normal vs. xantha seedling, Xs xs	16.0 ±	Kasha and Walker, 1960

Table 1. Cont.

Character	Percent recombination	Authority
Genes on Chromosome 3		
Green vs. white seedling, An an in relation to:		
Normal vs. xantha seedling, Xs xs	15.0 ± 2.4	Kasha and Burnham, 1965
Position of genes in relation to centromere:		
Normal vs. uzu dwarf, Uz uz	14.0 ± 3.0*	Ramage, Burnham & Hagberg, 1961
Normal vs. uzu dwarf, Uz uz	14.0 ± 1.9*	Ramage, Burnham & Hagberg, 1961
Normal vs. uzu dwarf, Uz uz	9.7 ± 2.2*	Persson, 1969b
Green vs. albino seedling, Ac ac	3.0 ± 1.4*	Ramage, Burnham & Hagberg, 1961
Green vs. albino seedling, Ac ac	4.6 ± 1.0*	Persson, 1969b
Green vs. albino seedling, Ac ac	5.7 ± 0.9*	Persson, 1969b
Green vs. white seedling, An an	18.4 ± 3.6*	Persson, 1969a
Green vs. white seedling, An an	12.3 ± 1.3*	Persson, 1969a
Green vs. white seedling, An an	15.1 ± 2.1*	Persson, 1969a
Green vs. white seedling, An an	17.0 ± 2.3*	Ramage, Burnham & Hagberg, 1961
Green vs. white seedling, An an	13.0 ± 1.5*	Ramage, Burnham & Hagberg, 1961
Green vs. white seedling, An an	12.0 ± 3.2*	Ramage, Burnham & Hagberg, 1961
Genes on Chromosome 7		
Rough vs. smooth awn, R r in relation to:		
Green vs. cream seedling, Cm cm	2.3 ± 0.3	Robertson, 1967
Green vs. cream seedling, Cm cm	closely linked	Kramer and Blander, 1961
Normal vs. fragile stem, Fs fs	44.0 ± 3.3	Ramage and Suneson, 1961
Normal vs. male sterile, Ms11 ms11	17.3 ± 4.5	Walker et. al., 1958
Normal vs. variegated 3, Va3 va3	4.3 ± 0.9	Walker et. al., 1963

Table 1. Cont.

Character	Percent recombination	Authority
	Genes on Chromosome 7	
Long vs. short haired rachilla, *S s* in relation to:		
Normal vs. variegated 3, *Va3 va3*	28.8 ± 8.3	Walker *et. al.*, 1963
Green vs. cream seedling, *Cm cm*	24.4 ± 0.3	Robertson, 1967
Rough vs. smooth awn, *R r*	25.1 ± 1.6	Takahashi and Hayashi, 1966
Rough vs. smooth awn, *R r*	18.2 ± 4.5	Wells, 1958
Rough vs. smooth awn, *R r*	28.0 ± 2.2	Ramage and Suneson, 1961
Rough vs. smooth awn, *R r*	28.0 ±	Yesuda and Takahashi, 1961
Rough vs. smooth awn, *R r*	26.5 ± 3.7	Woodward, 1957
Green vs. white strip, *Wst2 wst2*	15.6 ±	Takahashi and Moriya, 1964
Normal vs. fragile stem, Fs fs	24.0 ± 3.8	Ramage and Suneson, 1961
Normal vs. male sterile, *Ms16 ms16* in relation to:		
Resistance vs. susceptible to Ddt, *Ddt ddt*	7.0 ±	Wiebe, 1964

* Translocation Data

Robertson (1967) suggested the gene sequence was rattail, rt-or-ms3 y-f-e-ms2. The recombination values between ms2 and f (Austenson, 1948) and between f and a2 (Robertson 1967) placed y between ms2 and a2.

Results with telocentric chromosomes suggested the possible gene order to be ms3-f-centromere-gs5-e-ms2-wst4-tr; ms3 and f on the short arm, while gs5, e, ms2, wst4 and tr were on the long arm (Tsuchiya, 1972).

Assignment of male steriles, ms5, ms16, ms19, ms23b and ms24v to the respective chromosome involved T1-6c, T1-7a, T2-4a, T2-6a, T3-5b, T3-7a and T4-5a translocation tester stocks (Hockett & Eslick, 1970). From the information based on F_3 family segregation ms5, ms16, ms19, ms23b and ms24v were assigned to chromosomes 3, 7, 7, 1, and 4, respectively. Male steriles, ms5, ms19 and ms24v were near the centromere while ms16 and ms23b were not. The location of ms16 agreed with Hayes and Rana (1966) who found ms16 to be linked with the ddt gene on chromosome 7.

From studies of three chlorophyll mutants Robertson (1929 and 1937) proposed the gene order of albino seedling a_c, xantha seedling, x_c and white seedling, a_n as a_c-x_c-a_n on chromosome 3. Persson (1969a) confirmed this sequence; a_n was calculated to be 15 to 20 cross-over units from the centromere, and a_n and x_c were placed on the long arm of chromosome 3.

Robertson (1967) located white streak, wst on chromosome 3, and

considering the recombination values between wst and a_c-x_c- a_n and xantha seedling, x_s, he proposed the gene order: a_c-x_c-wst-a_n-x_s. In order to locate albino lemma, al, Takahashi and Hayashi (1958) conducted a three-point linkage study which included uzu dwarf plant, uz. Their proposed gene sequence was, uz-a_c-al-a_n. Kasha and Walker (1958) suggested the gene order, including absent lower spikelet, als to be, als-uz-ys-a_c-a_n.

Jarvi and Eslick (1967) observed linkage between ms24v and translocation break-points of T2-4a and T4-5a, and independence with T1-6c, T1-7a, T2-6a, T3-5b, and T3-7c break-points. Using F_3 data from crosses between ms24v and translocation tester stocks, T1-6c, T1-7a, T2-6a, T3-5b and T3-7c ms24v was assigned to chromosome 4 and probably close to the centromere (Hockett and Eslick, 1970).

An F_3 linkage test showed that male sterile, msl, was on the short arm of chromosome 5 (Ramage 1961). Eighteen translocations involving chromosome 5 and four individual genes: third outer glume, trd, black lemma and pericarp, B, white seedling, a_t, and a male sterile, msl; positioned msl very close to the centromere of chromosome 5 (Ramage, 1961, and 1963); the proposed gene order in the short arm was trd-B-a_t-msl-centromere. The genes trd and B were about 15 recombination units apart and the genes B and a_t were about 30 recombination units apart, while msl and a_t showed independence (Ramage and Lehman, 1964). Contrary to Ramage (1963), Tsuchiya (1972) on the basis of telocentric trisomics suggested that B, trd and a_t were on the long

arm instead of the short arm; and the gene order was: centromere-a_t-B-trd. Cream seedling, cm2 on chromosome 5 was observed to be linked with partially sterile ovule, os, with a recombination percentage of about 7 (Nilan and Moh, 1955).

Ramage and Burnham (1962) reported that orange lemma, o, and early heading, ec were close to the centromere of chromosome 6 having 0.5 ± 0.3 percent recombination (Ramage, 1962). Uniculm, uc2, was also determined to be very closely linked with o with a recombination percentage of 0.6 ± 0.1 (Shands, 1962); thus placing uc2 in the same region along with ec. Xantha seedling, x_n, was about 14 cross-over units away from o (Robertson, 1967).

Seven different translocations involving chromosome 7 placed male sterile, ms16 and ms19 on chromosome 7; ms19 being near to the centromere, while ms16 was distal (Hockett and Eslick, 1970). F_3 linkage data from crosses of 5 translocations involving chromosome 7 and 3 individual genes; fragile stem, fs, smooth awn, r, and short haired rachilla, s; fs showed 23.0 ± 1.2 percent recombination with the centromere (Ramage, 1963, and Ramage and Burnham, 1962); the gene order was fs-s-r. One linkage study involving white stripe, wst2, smooth awn, r, and short haired rachilla, s, suggested gene sequence to be wst2-s-r (Takahashi and Moriya, 1964.)

Kramer and Blander (1961) using T1-7a, T1-7b and T3-7a assigned cm to chromosome 7. This finding agrees with Robertson (1933) who placed

cm (his previous designation was a_b) in the linkage group V. The seedling mutant, cm was closely linked with smooth awn, r and the recombination value was 3.0 ± 2.0 percent (Kramer & Blander, 1961). The recombination percentages between cm and r; and between cm and short haired rachilla, s were 24.4 ± 0.3 and 2.3 ± 0.3, respectively, (Robertson, 1967).

Chlorophyll-deficient mutants:

The appearance of chlorophyll-deficient seedlings is common in self-pollinated corn (Wetz and Goodsell, 1929), in barley (Robertson, 1932) and in many other species. These chlorophyll-deficient genes, when homozygous may or may not cause death of seedlings. Assuming their detrimental effect on yield, plant breeders have been eliminating them from the population. A number of investigators attempted to determine the effects of these recessives upon yield when they occur in the heterozygous condition. Wetz and Goodsell (1929) found no definite relationship between the number of defects present and yield. Nevertheless, when the characters studied were divided into three classes: (seed defects, seedling chlorophyll defects and plant defects), the totals under each of these classes as well as the total for all the defects were negatively correlated with yield. Their experiment, however, was not designed to study the effects of any particular recessive in the heterozygous condition; but rather to show the general tendency of varieties with one recessive character to be more productive or less

productive than varieties with many recessive characters.

Robertson (1932) studied the effects of a single lethal gene in four barley varieties. He found no significant difference between the homozygous viable and heterozygous genotype for all the agronomic characters studied. However, when the lethality was associated with endosperm deficiency a significant difference in grain yield was observed.

In soybeans Wetz and Stewart (1927) and in sorghum, Karper (1930) observed differences between the homozygous and heterozygous genotypes, but the differences were not significant.

Mangelsdorf (1928) reported that homozygous (De De) corn plants were taller than heterozygous (De de) genotypes during early stages of growth, but the differences were overcome by the heterozygotes in the later stages of development.

Doll (1966) studied the effects of 49 chlorophyll-mutants in the two-rowed spring barley, Carlsberg II. Some of these mutants in heterozygous condition had little or no effect on the number of kernels per plant, while others showed noticable detrimental effects on the same character. Only one heterozygous chlorophyll mutant, 52-X, had significantly more kernesl per plant than the corresponding homozygous counterpart. This significant difference, however, was attributed to the interaction of another recessive mutant gene which was observed to be linked with the chlorophyll-mutant gene in the repulsion phase.

Contrary to the findings of Doll (1966), Robertson (1932), and others, Gustafsson, Nybom and von Wettstein (1950) observed superior performances of the heterozygotes of three spontaneously occurring chlorophyll mutants, albino-7, xantha-3 and albinoxantha in a pure line of the Golden vareity All three mutants in single heterozygous condition out-perform their homozygous counterparts when grown under higher plant density and heavy nitrogen fertilization. The dihybrid (albino-7 x xantha-3) heterozygotes exhibited similar performances under similar conditions. Similar results were also obtained by Gustafsson (1947). Robertson and Austin (1935) reported similar results except in one case where the double heterozygotes for a_{c3} and x_c were inferior to the homozygous counterparts. The two lethal mutants, a_c and x_c in single heterozygote condition as well as in double heterozygote condition was consistently superior for the characters studied.

MATERIALS AND METHODS

Seed of all the mutants used in this experiment were provided by Prof. R. F. Eslick, Plant and Soil Science Department, Montana State University, Bozeman, Montana. The mutational events, assignment to chromosome when known, inheritance and the cultivar in which these mutants were originally found are described in Tables 2, 3, 4 and 5.

During the summer of 1968 each of these mutants, the male sterile tester series and an msl msl orange lemma (oo) stock were planted at the Agricultural Experiment Station, Bozeman. Only male sterile (ms/ms) plants were used as females. To avoid contamination, the male sterile spikes, to be used as females in crossing, were bagged with glassine bags when they were about to emerge from the boot. After complete emergence of these heads the bags were removed to make appropriate pollinations and rebagged immediately.

Each previously located heterozygote lethal, (Table 3) was crossed to male sterile (ms/ms) plants whose genetic male sterile gene had been previously assigned to a chromosome (Table 2). Each heterozygote lethal mutant not previously located (Table 4) was crossed onto each identified male sterile (ms/ms) and the orange lemma (o/o) stock (Table 2). In both cases three or four different males were used in making crosses to increase the probability of having at least one male heterozygous for the lethal gene. To identify crosses and parents each was marked with proper identification tags. At maturity crossed seeds and each male were harvested individually.

Table 2. Description of the spontaneous, monogenic, recessive male sterile genes and orange lemma gene used to determine the linkage with seedling lethal mutants.

Gene symbol	Cultivar	Located on chromosome	Authority
ms10 ms10	Compana	1	Eslick and Hockett, 1967; Hockett and Eslick, 1968.
ms14 ms14	Unitan	1	Eslick and Hockett, 1967; Hockett and Eslick, 1968.
ms23b ms23b	Betzes	1	Hockett and Eslick, 1968; Hockett and Eslick, 1970.
ms2 ms2	Trebi	2	Austenson, 1948; Hockett and Eslick, 1970; and Eslick, 1970.
ms5 ms5	Carlsberg II	3	Hockett and Eslick, 1970.
ms24v ms24v	Betzes	4	Jarvi and Eslick, 1967.
ms1 ms1	Betzes	5	Suneson, 1940; Ramage, Burnham and Hagberg, 1961.
Orange lemma (oo)	Titan	6	Myler and Stanford, 1942; and Ramage et. al., 1961.
ms16 ms16	Betzes	7	Hockett and Eslick, 1968 & 1970.
ms19 ms19	CI 4961	7	Hockett and Eslick, 1968 & 1970.

Table 3. Description of the previously located spontaneous, seedling lethal, monogenic recessive mutant genes used to determine their linkage with male sterile genes.

Gene symbol	Cultivar	Located on Chromosome	Phenotype	Authority
$\underline{a_{c2}}\ \underline{a_{c2}}$	Coast II	1	Albino	Robertson and Deming, 1930, and Robertson, 1937.
$\underline{y_c}\ \underline{y_c}$	Coast III	1	Albino	Robertson and Demming, 1930; Robertson, Demming and Koonce, 1932.
$\underline{a2}\ \underline{a2}$	Coast	2	Albino	Robertson, 1967.
$\underline{y}\ \underline{y}$	Faust	2	Shows yellow colour	Robertson and Coleman, 1942.
$\underline{x_s}\ \underline{x_s}$	Smyrna	3	Yellow colour varies with temperature	Robertson, 1929.
$\underline{a_c}\ \underline{a_c}$	Colsses I	3	Albino	Robertson, 1929.

Table 3. Cont.

Gene symbol	Cultivar	Located on Chromosome	Phenotype	Authority
$\underline{a_t}$ $\underline{a_t}$	Trebi	5	Albino	Robertson, 1929
$\underline{cm_2}$ $\underline{cm_2}$	Black-hulless	5	Pale yellow	Nilan and Moh, 1955.
$\underline{cm}$ $\underline{cm}$	Black-hulless	7	Cream colour	Robertson, 1932; Kramer and Blander, 1961.

Table 4. Description of spontaneous seedling lethal mutant genes not previously located.

Gene symbol	Cultivar	Phenotype	Previous designation
alb,,a alb,,a	Erbet	White leaf with yellow tip. The yellow colour belonged to 3C RHS Yellow Colour chart.*	Erbet x_{66} x_{66}
alb,,b alb,,b	Carlsberg II	White	Carlsberg II x_{66} x_{66}
alb,,c alb,,c	Titan	White, seldom papyraceous at the margin	Titan a_{48} a_{48}
alb,,d alb,,d	Erbet	White leaf with yellow tip. The yellow colour belonged to the 3C RHS Yellow Group Chart.*	Early Betzes x_{66} x_{66}
alb,,e alb,,e	Titan	White	Titan a_{65} a_{65}
alb,,f alb,,f	Shabet	White	Shabet a_{66} a_{66}
alb,,g alb,,g	Titan	White, seldom with papyraceous margin.	Belonee short Titan aa
alb,,h alb,,h	Compana X Mars	White	T2-6a-aa
alb,,i alb,,i	Titan	Ivory white leaf with yellow green tip. The yellow green colour belonged to 144C of RHS Colour Chart.*	Shonufertoontan aa

* R.H.S. Colour Chart. The Royal Horticultural Society, London.

Table 5. Determination of inheritance of the seedling lethal mutants not previously located.

Lethal genes	Number of Progeny		Total	Chi-square*	Probability*	Inheritance
	Green (A-)	Albino (aa)				
alb,,a alb,,a	280	99	379	0.25	0.75-0.50	Monogenic
alb,,b alb,,b	276	89	365	0.07	0.97-0.95	Monogenic
alb,,c alb,,c	304	88	392	1.36	0.25-0.10	Monogenic
alb,,d alb,,d	280	106	386	1.24	0.50-0.25	Monogenic
alb,,e alb,,e	276	86	362	0.29	0.75-0.55	Monogenic
alb,,f alb,,f	270	108	378	2.56	0.25-0.10	Monogenic
alb,,g alb,,g	293	90	383	0.44	0.75-0.50	Monogenic
alb,,h alb,,h	300	80	380	3.16	0.10-0.05	Monogenic
alb,,i alb,,i	248	77	325	0.29	0.75-0.50	Monogenic

* For goodness of fit 3 green:1 albino

During the winter of 1968 - 69 the males were checked for heterozygosity for the lethal genes in the greenhouse. To obtain seed for the F_2 generation, 20 F_1 seeds involving only those from heterozygous males were planted in the greenhouse in 1968-69. Each F_1 plant was harvested and checked for heterozygosity for the lethal gene.

Whenever possible 400 seeds from the segregating F_2 seed population were planted in rows 30 cm. apart in the field during the spring of 1969 at a rate of 25 seeds per three meter row. From these segregating rows, two sets of samples consisting of one spike from each F_2 plant were harvested. Male sterile plants were discarded.

Whenever a heterozygous male was not found to be involved in the crosses made in 1968, new crosses were made in 1969 following the same procedure as for 1968. The F_1 and F_2 seeds were handled in the same manner in 1970 as 1969.

During the summer of 1970 and 1971, F_2 spikes were planted in hills on 60 cm. centres and classified for F_3 phenotypes to obtain F_2 genotypes. The hills homozygous for Ms/Ms and Alb,,/Alb,, and the hills segregating for male sterility, ms/ms, but homozygous for Alb,,/Alb,, were marked with appropriate coloured flags as soon as they could be identified. Segregation data for lethal and male sterile genes, as well as for other pertinent markers, whenever possible, were recorded.

Suspected linkage between two genes was determined by Chi-square test. Significant deviations from the expected independent ratios, as

indicated by interaction Chi-square tests (Appendix Table 1) were used as indicators of linkage. The recombination percentages were calculated from the F_3 population data by the maximum likelihood method. This calculation was facilitated by the formulas and tables presented by Allard (1956).

To verify F_3 data, 50 seeds of possible cross-over types from fertile plants of Ms-A/Ms-a and Ms-A/ms-A hills were planted in two three meter rows 30 cm. apart in 1971 and 1972. Segregation for lethal and male sterile genes and other markers were recorded and the F_3 data corrected.

To evaluate the effects of heterozygous lethals on the yield of grain, the following characteristics of mature plants were measured. (1) number of spike bearing tillers, (2) Total number of seeds per plant and (3) weight of seeds per plant. A randomized complete block design with four replications of two row plots was used. The seeds heterozygous for the lethal mutants (Table 3 and 4) were planted in rows 30 cm. apart and spaced 30 cm. within the rows. Each row was three meters long.

To provide equal competition seedlings of a genetically marked genotype were transplanted where seedlings died due to lethality or failed to establish.

At maturity, each plant was harvested individually except the marked genotype that was planted later to provide equal competition.

Twenty seeds from each plant were sown in the greenhouse to differentiate the homozygous from heterozygous plants so that the effects of the heterozygous lethal condition could be evaluated.

Data were analyzed following the analysis of variance and chi-square methods of Steel and Torrie (1960). Homozygous and heterozygous plants for each plot were averaged and the two means considered as splits of variety plots.

RESULTS AND DISCUSSION

Linkage:

Chromosome 1:

Translocation data established male sterile, ms10 and male sterile, ms14 in the centric region of chromosome 1 (Eslick, 1970). From a small population ms10 and ms14 showed 2.0 ± 2.0 percent recombination (Eslick, Hockett and Kushnak, 1972). In 1970, ms10, in the tester 'Shonupana', and ms14, in the tester 'Unitan', showed linkage with albino seedling, a_{c2} with recombination values of 17.3 ± 2.4 percent and 6.3 ± 1.5 percent, respectively, (Table 6). For 1971, the recombination value between a_{c2} and ms10, in the tester 'Compana', was 7.8 ± 1.0 percent (Table 6). This discrepancy of having two different recombination values for the same genes (ms10 and a_{c2}) may be due to different genetic make-up of these two different testers and/or the environmental effects on recombination in two different years. Combining the data for 1970 and 1971 the recombination value between ms10 and a_{c2} was determined to be 10.7 ± 1.0 percent and between ms14 and a_{c2} was 6.3 ± 1.5 percent (Table 6).

To determine the gene order of ms10, n & a_{c2} a three-point linkage study was conducted. The initial cross was n-ms10-A_{c2}/n-ms10-A_{c2} x N-Ms10-A_{c2}/N-Ms10-a_{c2} . From F_3 hills the F_2 genotypes of the hybrid, n-ms10-A_{c2}/N-Ms10-a_{c2} were determined. The possible gene orders of the F_1, gametes present and the observed frequency are presented in Table 7. The probability of having a higher frequency of double cross-over gametes as compared to single cross-over gametes is remote. Assuming a lower

Table 6. Linkage intensities of male sterile genes and previously assigned seedling lethal genes. Expected ratio: 1 MsMsAA : 2 MsmsAA : 2 MsMsAa : 4 MsmsAa.

Cross	Chromosome tested	Number of F_2 progeny: AA Ms Ms	AA Ms ms	Aa Ms Ms	Aa Ms ms	Total	Interaction: Chi-square	Interaction: Probability	Percent recombination and S. E.
ms10-A_{c2} / Ms10-a_{c2}	1	9	13	18	119	159	13.6	<0.005	17.3 ± 2.4 *
ms10-A_{c2} / Ms10-a_{c2}	1	1	16	31	279	327	16.4	<0.005	7.8 ± 1.0 *
ms10-A_{c2}** / Ms10-a_{c2}	1	10	29	49	398	486	40.92	<0.005	10.7 ± 1.0 *
ms14-A_{c2} / Ms14-a_{c2}	1	0	4	11	107	122	12.2	<0.005	6.3 ± 1.5 *
ms23b-A_{c2} / Ms23b-a_{c2}	1	56	100	86	164	406	0.2	0.75-0.50	Independent
ms10-Y_c / Ms10-y_c	1	9	18	22	60	109	0.6	0.50-0.25	36.0 ± 5.7
ms23b-Y_c / Ms23b-y_c	1	41	32	50	67	190	2.3	0.25-0.10	Independent
ms14-Y_c / Ms14-y_c	1	15	43	37	83	178	0.4	0.75-0.50	Independent
ms2-A_2 / Ms2-a_2	2	1	7	13	102	123	8.9	<0.005	9.4 ± 1.9 *
ms2-Y / Ms2-y	2	7	21	1	112	141	16.5	<0.005	13.9 ± 2.2 *
ms5-A_c / Ms5-a_c	3	1	5	4	214	224	36.08	<0.005	2.4 ± 0.7 *
ms5-X_s / Ms5-x_s	3	1	7	10	50	68	1.4	0.25-0.10	15.3 ± 3.5*

Table 6. Cont.

Cross	Chromosome tested	Number of F_2 progeny: AA Ms Ms	AA Ms ms	Aa Ms Ms	Aa Ms ms	Total	Interaction Chi-square	Interaction Probability	Percent recombination and S. E.
ms1-A_t / Ms1-a_t	5	26	59	35	62	182	0.7	0.50-0.25	Distorted
ms1-Cm2 / Ms1-cm2	5	37	70	79	137	323	0.2	0.75-0.50	Independent
ms16-Cm / Ms16-cm	7	31	48	53	94	226	0.2	0.75-0.50	Independent *
ms19-Cm / Ms19-cm	7	21	57	33	96	207	0.5	0.50-0.25	Independent *

* F_3 genotypes confirmed from F_4 data.

** Combined data for this cross, heterogeniety Chi-square was significant.

Table 7. Data for gene ordering naked caryopsis, n, male sterile ms10 and the seedling lethal albino, a_{c2} on chromosome 1.

F_2 Genotypes	Single cross-over gamete present	Cross-over type to produce gamete* — Possible gene orders of F_1: $\frac{NMs10a_{c2}}{nms10A_{c2}}$	$\frac{Na_{c2}Ms10}{nA_{c2}ms10}$	$\frac{a_{c2}NMs10}{A_{c2}nms10}$	Observed frequency
NNMs10Ms10$A_{c2}A_{c2}$					3
NNMs10Ms10$A_{c2}a_{c2}$	NMs10A_{c2}	S	D	S	14
NNMs10ms10$A_{c2}A_{c2}$					10
NNMs10ms10$A_{c2}a_{c2}$	Nms10A_{c2}	S	S	D	39
NnMs10Ms10$A_{c2}A_{c2}$					6
NnMs10Ms10$A_{c2}a_{c2}$	nMs10A_{c2}	D	S	S	2
NnMs10ms10$A_{c2}A_{c2}$	NMs10A_{c2}	S	D	S	4
NnMs10ms10$A_{c2}a_{c2}$					69
nnMs10Ms10$A_{c2}A_{c2}$					0
nnMs10Ms10$A_{c2}a_{c2}$					1
nnMs10ms10$A_{c2}A_{c2}$	nMs10A_{c2}	D	S	S	1
nnMs10ms10$A_{c2}a_{c2}$	nMs10a_{c2}	S	S	D	10
					159

* S = single cross-over gamete
D = double cross-over gamete

frequency of double cross-over gametes and comparing the observed frequency to the type of crossing over required to obtain the gametes within the three possible gene orders, the most probable gene order is n-ms10-a_{c2}.

Considering the recombination values and gene order reported by Eslick, Rahman and Crowell (1971); Eslick, Hockett and Kushnak (1972); Takahashi and Hayashi (1958); and Persson (1969), the probable gene sequence is postulated in Fig. 1.

Persson's (1969b) recombination value between n and a_{c2} closely agrees with Takahashi and Hayashi (1958), but does not agree with Robertson's (1937). The seeming discrepancy is in accordance with the known genetic phenomenon that recombination values determined for two distant genes is nearly always underestimated due to double crossovers. The recombination values between y_c and lk2 (Takahashi and Hayashi, 1958); between y_c and n (Persson, 1969b); between a_{c2} and y_c (Haus, 1958); between a_{c2} and wx (Tabata, 1961), however, confirms the position of a_{c2} between n and y_c. This position of a_{c2} is in complete agreement with Takahashi and Hayashi (1958) and others.

Male sterile, ms23b was observed to be independent of a_{c2} (Table 6), but linked with alb,,d having a recombination value of 7.4 $\pm$ 1.2 percent (Table 8). Since ms23b has been assigned to chromosome 1 (Jarvi 1970) and since alb,,d was linked with ms23b, alb,,d should be on chromosome 1. Seedling lethal mutant, alb,,d was independent of

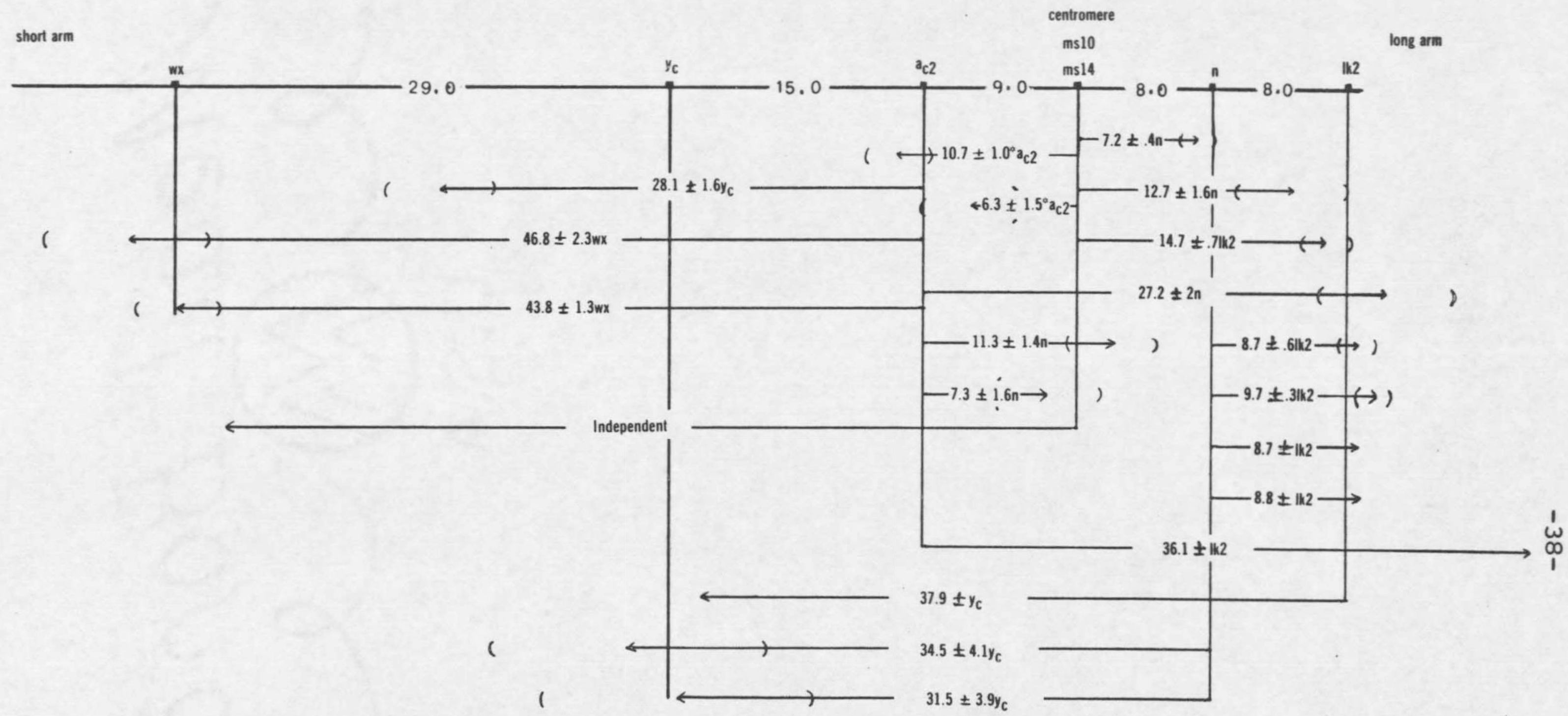

Fig.1. Proposed linkage map of chromosome 1

* Data contributed by this paper

( ) ± 2 standard error

Table 8. Linkage intensities involving male sterile marker genes and the seedling lethal mutant alb,,d, determined from F_3 data.

Expected Ratio: 1 MsMs Alb,,d Alb,,d: 2 Msms Alb,,d Alb,,d: 2 MsMs Alb,,d alb,,d: 4 Msms Alb,,d alb,,d

Cross	Chromosome tested	Number of Hills: Alb,,d/Alb,,d Ms Ms	Alb,,d/Alb,,d Ms ms	Alb,,d/alb,,d Ms Ms	Alb,,d/alb,,d Ms ms	Total	Interaction Chi-Square	Interaction Probability	Percent recombination and S. E.
ms10 Alb,,d / Ms10 alb,,d	1	10	10	15	40	75	2.79	0.10-0.05	Independent
ms14 Alb,,d / Ms14 alb,,d	1	27	23	34	34	118	1.67	0.20-0.10	Distorted
ms23b Alb,,d / Ms23b alb,,d	1	8	19	0	219	246	46.13	<0.005	7.4 ± 1.2*
ms2 Alb,,d / Ms2 alb,,d	2	34	48	51	95	228	1.21	0.50-0.25	Independent
ms5 Alb,,d / Ms5 alb,,d	3	7	16	20	58	101	0.49	0.50-0.25	Independent
ms24v Alb,,d / Ms24v alb,,d	4	16	30	35	51	132	0.43	0.75-0.50	Independent
ms1 Alb,,d / Ms1 alb,,d	5	10	29	32	58	129	0.78	0.50-0.25	Independent
ms16 Alb,,d / Ms16 alb,,d	7	17	31	35	60	143	0.04	0.97-0.95	Independent
ms19 Alb,,d / Ms19 alb,,d	7	15	18	15	65	103	0.88	0.50-0.25	Independent

* F_3 data confirmed from F_4 data

markers on the other chromosomes used in this experiment (Tables 8 and 10). To the author's knowledge no linkage between ms23b and any other marker has yet been reported, except with shrunken endosperm, se, 11.3 ± 3.7 percent (Hockett unpublished data) and 6.6 ± 3.4 percent (Eslick unpublished data). McProud (1971) observed gs3 to be independent of naked caryopsis, n and short awn, lk2. Lack of more linkage information precludes the possibility of positioning ms23b and alb,,d on chromosome 1.

Virescent seedling, y_c was independent of ms10, ms14, and ms23b male sterile genes on chromosome 1 used in this experiment (Table 6).

Chromosome 2:

Male sterile, ms2 was linked with white seedling, a2, and virescent seedling, y, with recombination percentages of 9.4 ± 1.9 and 13.9 ± 2.2, respectively (Table 6). Crosses involving segregation of y-Ms2-E/Y-ms2-e genotypes in F_2 showed no recombination between long awned outer glume, e, and male sterile, ms2. The segregation of the F_2 population is presented in Table 9.

Table 9. F_2 segregation of viable phenotypes from the cross, e-ms2-Y/e-ms2-Y x E-Ms2-y/E-ms2-Y.

Genotype	Frequency
Y—Ms2—E—	51
Y—ms2ms2 E—	0
Y—Ms2—ee	0
Y—ms2ms2ee	88
Total:	139

Table 10. Linkage intensities involving the orange lemma marker gene and seedling lethal mutants, alb,,a; alb,,b; alb,,c alb,,d and alb,,f determined from F_3 data.

Cross	Chromo-some tested	Number of Hills						Total	Interaction Chi-square	Prob-ability	Percent recombination and S. E.
		Alb,, Alb,,		/	Alb,, alb,,						
		OO	Oo	oo	OO	Oo	oo				
oo Alb,,a / OO alb,,a	6	11	15	8	17	26	15	92	0.45	0.25-0.10	Independent
oo Alb,,b / OO alb,,b	6	20	30	10	35	72	39	206	2.80	0.10-0.05	Independent
oo Alb,,c / OO alb,,c	6	12	16	7	14	30	10	89	1.50	0.25-0.10	Independent
oo Alb,,d / OO alb,,d	6	21	33	14	26	69	28	191	2.51	0.25-0.10	Independent
oo Alb,,f / OO alb,,f	6	25	42	18	40	69	38	232	1.77	0.50-0.25	Independent

The observed lack of recombination between e and ms2 agrees with Eslick (1970) and Ramage (1966), but not with Austenson (1948). Nevertheless, the large associated standard error (23.6 ± 6.0 percent) of Austenson does not exclude the possibility that these two genes are closely linked.

The slight disagreement with Ramage's results (1966) may be due to the fact that ms2 and e are perhaps both near the centric region (Ramage and Burnham, 1963) where recombination is expected to be greatly reduced or nil. Austenson (1948), however, conceded that his recombination values between e and ms2, and between e and six row, v did not coincide with his observed cross-over percentage between v and ms2.

The linkage map on the basis of recombination percentages (Table 1) and the gene sequences as reported by Robertson, Wiebe and Shands (1955); Robertson (1967); Robertson et. al. (1965); and Tsuchiya (1971 and 1972) may be presented as: or-a2-y-f-centromere-gs5-e-ms2-wst4-v.

The recombination values between f and ms2 (Austenson, 1948) and between f and a2 (Robertson, 1967) placed y between ms2 and a2 on the short arm of chromosome 2 (Tsuchiya, 1971). The present recombination values between ms2 and a2 places a2 between or and ms2. Tsuchiya (1972) assigned male sterile, ms3, green seedling, lg, and chlorina, f, to the short arm of chromosome 2 and glossy sheath, gs5, long awned outer glume, e and male sterile, ms2 to the long arm by the use of telotrisomics.

The four translocations, T1-2a, T2-4a, T2-4b and T2-6a whose break-points were close to the centromere of chromosome 2 placed orange seedling, or, 15 cross-over unit away from the centromere on the short arm of chromosome 2 (Ramage 1966); while T2-3f translocation placed six row, v, 28 recombination units away from the centromere (Ramage, 1966) on the long arm (Persson, 1969a).

The recombination percentages between e and translocation break-points involving T2-4a, T2-6a and T2-6b were observed to be 11.0 ± 2.3, 10.0 ± 3.4 and 11.7 ± 2.6, respectively (Ramage, Burnham and Hagberg, 1961; and Persson, 1969a).

Considering the translocations and other linkage data (Table 1) the proposed gene order is presented in Fig. 2. Robertson (1967) placed a2 to the right of or on the short arm of chromosome 2. Robertson's a2 was found in a Swedish variety and was originally assigned by Nilsson-Ehle (1922; cited by Robertson 1967) and Hallqvist (1925; cited by Robertson 1967). The mutant tested (Table 1) was a2, previously a1, and was a spontaneous mutant in the cultivar, Coast. Hence these two mutants, so called a2 may not be the same. Moreover, Robertson (1967) noted that the stock number of his material did not agree with the stock number of Nilsson-Ehle (1922) and Hallqvist (1925). However, his result agreed rather closely with Hallqvist's finding which showed a linkage of 10.2 between albino-3 and chlorina. He therefore proposed that possibly albino-1, later designed as a2, was the albino-3 of Hallqvist.

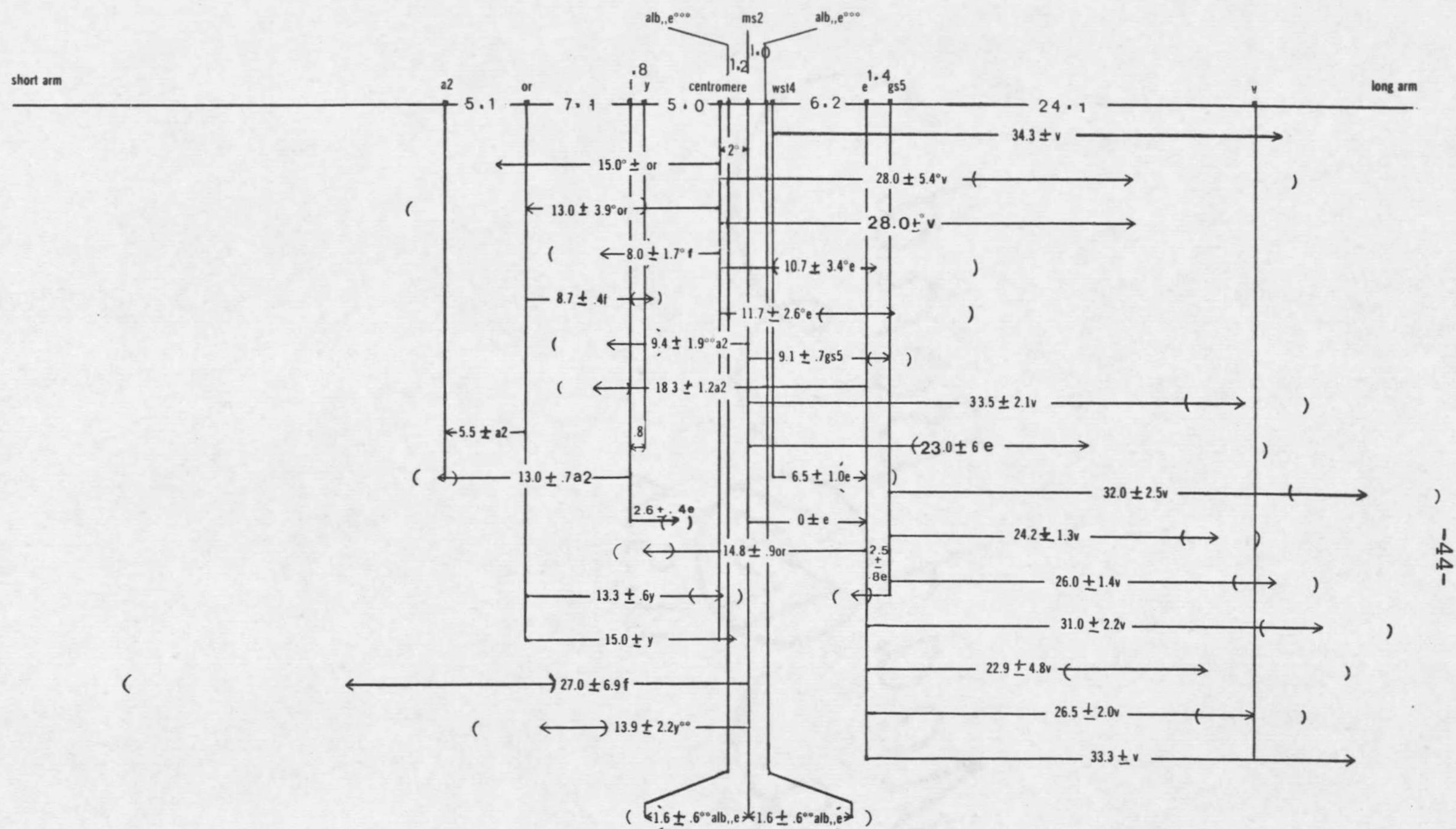

Fig.2. Proposed linkage map of chromosme 2

* Translocation data. ** Data contributed by this paper

*** Suggested alternate postions of alb,,e. ( )± 2 standard errors

The *A2 a2* seed stock used and reported in Table 6 was obtained from D. W. Robertson as *Al al* in 'Coast' and subsequently *Al al* designation was changed to *A2 a2*.

All the male sterile genes involved as markers in this experiment segregated independently in relation to *alb,,e* except male sterile, *ms2* (Table 11). The seedling mutant, *alb,,e* is linked with *ms2* with the recombination value of 1.6 ± 0.6 percent (Table 11). Since *ms2* has been located close to the centromere of chromosome 2 (Ramage and Burnham, 1966), *alb,,e* is assigned to chromosome 2. Since no other marker was involved in locating *alb,,e*, its locus could not be ascertained. A three-point linkage study among *gs5*, *ms2* and white stripe, *wst4* may position *alb,,e* appropriately.

Chromosome 3:

Male sterile, *ms5* was linked with white seedling, a_c and xantha seedling, x_s with recombination percentages of 2.4 ± 0.7 and 15.3 ± 3.5, respectively (Table 6).

The linkage data as reported by Robertson (1927, 1937 and 1967) Persson (1969a,b); Takahashi and Hayashi (1958); and Kasha and Walker (1958) put the genes in the order of, uz-a_c-x_c-a_n-x_s.

The previously reported recombination values and gene sequences by Takahashi and Hayashi (1958 and 1959); Robertson (1967); Kasha and Burnham (1965); and others placed a_c on the long arm of chromosome 3 (Tsuchiya and Haus, 1971) and between *yst2* and x_c. Hockett and Eslick (1970) placed *ms5* in the centric region of chromosome 3. Kasha and

Table 11. Linkage intensities involving male sterile marker genes and the seedling lethal mutant, alb,,e, determined from F_3 data.
Expected ratio: 1 MsMs Alb,,e Alb,,e : 2 Msms Alb,,e Alb,,e : 2 MsMs Alb,,e alb,,e : 4 Msms Alb,,e alb,,e.

Cross	Chromosome tested	Number of Hills				Total	Interaction Chi-Square	Probability	Percent recombination and S. E.
		Alb,,e/Alb,,e		Alb,,e/alb,,e					
		Ms Ms	Ms ms	Ms Ms	Ms ms				
ms10 Alb,,e / Ms10 alb,,e	1	13	20	17	46	96	0.28	0.70-0.50	Independent
ms23b Alb,,e / Ms23b alb,,e	1	8	22	27	29	86	3.97	0.10-0.05	Independent
ms2 Alb,,e / Ms2 alb,,e	2	0	1	2	127	130	28.20	<0.005	1.6 ± 0.6*
ms5 Alb,,e / Ms5 alb,,e	3	6	15	17	37	75	0.17	0.70-0.50	Independent
ms24v Alb,,e / Ms24v alb,,e	4	20	28	20	45	113	1.81	0.20-0.10	Independent
ms1 Alb,,e / Ms1 alb,,e	5	11	13	12	17	53	0.57	0.50-0.25	Independent
ms16 Alb,,e / Ms16 alb,,e	7	4	17	12	28	61	1.83	0.20-0.10	Independent
ms19 Alb,,e / Ms19 alb,,e	7	10	18	24	51	103	0.13	0.70-0.50	Independent

* F_3 genotypes confirmed from F_4 data.

Walker (1960) with translocation data proposed the position of a_n on the long arm.

The genes uz and a_n have been placed on the short arm and long arm respectively, and 9.7 ± 2.2 and 18.4 ± 3.6 percent cross-over units from the centromere, respectively, (Persson, 1969a,b). Takahashi and Yamamoto (1951), however, reported 15.8 percent recombinations between uz and a_n. This recombination percentage between uz and a_n (15.8 percent) seems very low when compared with the recombination values between centromere and uz and a_n. An inversion in one of the stocks used may have reduced the recombination percentage. (Burnham, 1962)

From F_3 data alb,,b was observed to be linked with male sterile, ms5, and independent of all other male sterile genes and orange lemma (oo) gene (Tables 9 and 12). Seedling lethal mutant gene, alb,,b can be assigned to chromosome 3 since it is linked with ms5 which is on chromosome 3. The recombination values between ms5 and a_c (2.4 ± 0.7 percent) and between ms5 and alb,,b (2.9 ± 0.6) suggest that these two genes are very close to ms5 and could be the same gene. An allelism test is needed for further clarification. Since no other marker in relation to alb,,b was studied, the alb,,b locus on chromosome 3 could not be determined. Alternate positions of alb,,b have been suggested. Considering the translocation data and the present recombination values the suggested gene order is presented in Fig. 3.

Table 12. Linkage intensities involving male sterile genes and the seedling lethal mutant alb,,b, determined from F_3 data.
Expected ratio: 1MsMsAlb,,bAlb,,b : 2 MsmsAlb,,bAlb,,b : 2 MsMsAlb,,balb,,b : 4 MsmsAlb,,balb,,b

Cross	Chromosome tested	Number of Hills: Alb,,b/Alb,,b		Number of Hills: Alb,,b/alb,,b		Total	Interaction Chi-Square	Probability	Percent recombination and S. E.
		Ms Ms	Ms ms	Ms Ms	Ms ms				
ms10 Alb,,b / Ms10 alb,,b	1	16	39	25	56	136	0.12	0.80-0.70	Independent
ms23b Alb,,b / Ms23b alb,,b	1	18	20	21	51	110	3.83	0.10-0.05	Independent
ms2 Alb,,b / Ms2 alb,,b	2	17	28	42	61	148	0.21	0.75-0.50	Independent
ms5 Alb,,b / Ms5 alb,,b	3	4	3	2	215	224	51.58	<0.005	2.9 ± 0.6*
ms24v Alb,,b / Ms24v alb,,b	4	14	26	31	76	147	0.56	0.50-0.25	Independent
ms1 Alb,,b / Ms1 alb,,b	5	5	13	16	35	69	0.47	0.50-0.25	Independent
ms16 Alb,,b / Ms16 alb,,b	7	18	18	25	62	123	4.70	0.05-0.02	Independent
ms19 Alb,,b / Ms19 alb,,b	7	15	17	20	55	107	3.94	0.05-0.02	Independent

* F_3 genotype confirmed from F_4 data.

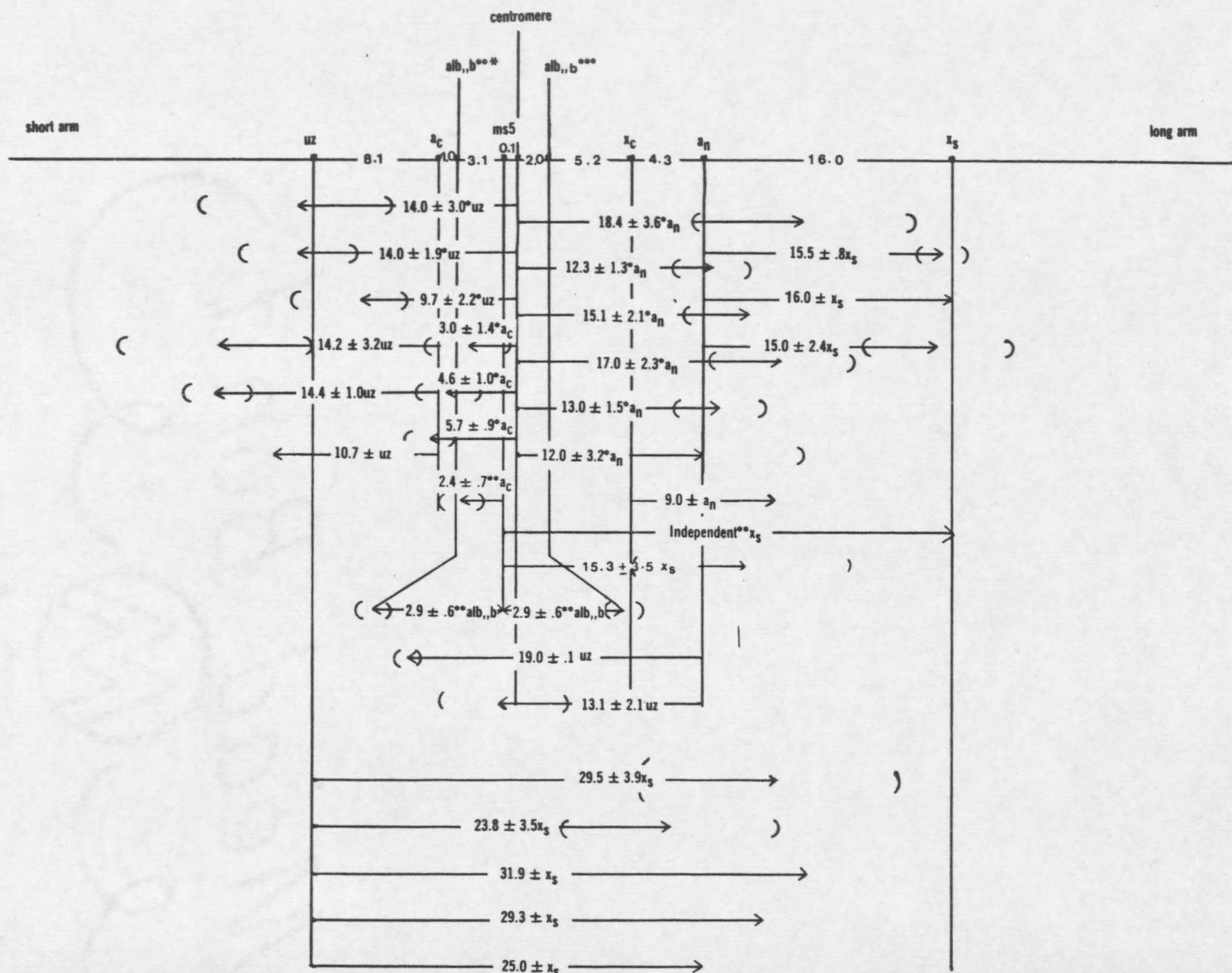

Fig. 3. Proposed linkage map of chromosome 3. * Translocation data. ** Data contributed by this paper. *** Suggested alternate positions of alb,,b. ( ) ± 2 standard errors.

Chromosome 4:

No mutants were associated with genes on chromosome 4.

Chromosome 5:

The cream seedling mutant, cm2 and albino seedling, a_t were independent of male sterile, msl (Table 6). Ramage (1962) also observed independence between msl and a_t. Cream seedling, cm2 could not be positioned in relation to other markers because no marker genes were studied. Linkage studies with other markers are needed.

Albino seedling, alb,,a segregated independently from all male sterile and the orange lemma, (oo) marker genes used in this experiment except msl (Table 10 and 13). The recombination value between msl and alb,,a was determined to be 1.9 ± 0.6 percent (Table 13). The linkage between alb,,a and msl places alb,,a on chromosome 5.

To obtain msl and alb,,a in coupling and to determine the gene order a three-point linkage study involving msl, alb,,a and golden Compana, yh2, was conducted. The genotypes, phenotypes, expected ratio due to independence, observed F_2 phenotypes, test cross ratios and possible gene orders are presented in Appendix Table 2.

An F_1 of the genotype msl-yh2-Alb,,a/Msl-Yh2-alb,,a was grown and classified in F_2 for phenotype except for plants homozygous for alb,,a alb,,a. The data are given in Table 14.

The three male sterile plants were the subject of further study. They were crossed with the F_1 genotype, msl-yh2-Alb,,a/Msl-Yh2-alb,,a.

Table 13 Linkage intensities involving male sterile genes and the seedling lethal mutant, alb,,a, determined from F_3 data.
Expected ratio: 1 MsMsAlb,,aAlb,,a : 2 MsmsAlb,,aAlb,,a : 2 MsMsAlb,,aalb,,a : 4 MsmsAlb,,aalb,,a

Cross	Chromosome tested	Number of Hills: Alb,,a/Alb,,a ms Ms	Alb,,a/Alb,,a Ms ms	Alb,,a/alb,,a Ms Ms	Alb,,a/alb,,a Ms ms	Total	Interaction Chi-Square	Prob-ability	Percent recombination and S. E.
ms10 Alb,,a / Ms10 alb,,a	1	23	54	38	99	214	0.07	0.50-0.25	Independent
ms23b Alb,,a / Ms23b alb,,a	1	20	49	28	69	166	0.04	0.50-0.25	Independent
ms2 Alb,,a / Ms2 alb,,a	2	13	14	33	29	89	0.87	0.50-0.25	Distorted
ms5 Alb,,a / Ms5 alb,,a	3	28	37	56	114	235	1.52	0.30-0.20	Independent
ms24v Alb,,a / Ms24v alb,,a	4	9	7	17	23	56	1.81	0.20-0.10	Independent
ms1 Alb,,a / Ms1 alb,,a	5	0	3	1	99	103	20.13	<0.005	1.9 ± 1.2*
ms16 Alb,,a / Ms16 alb,,a	7	25	47	32	64	168	0.06	0.90-0.80	Distorted
ms19 Alb,,a / Ms19 alb,,a	7	21	37	32	63	153	0.10	0.80-0.70	Independent

* F_3 genotype confirmed from F_4 data.

Table 14. Observed phenotypic frequency in F_2 population of a cross, msl-yh2-Alb,,a/msl-yh2-Alb,,a x Msl-Yh2-alb,,a/msl-Yh2-Alb,,a.

Phenotypes*	Genotype	Observed frequency F_2
Green, fertile, green	Yh2---Msl---Alb,,a---	540
Green, male sterile, green*	Yh2---mslmsl Alb,,a---	3
Golden, fertile, green	yh2yh2-Msl---Alb,,a---	0
Golden, male sterile, green*	yh2yh2-mslmsl-Alb,,a---	300
Albino	------ ------ alb,,a alb,,a	---

* Green vs. golden foliage colour, fertilize vs. male sterile, and green vs. albino seedlings, respectively.

The F_1's of this cross on all three plants segretated for golden, albinos, and green and golden male sterile (Table 15).

Table 15. Phenotypic frequency of recombinant types of a test cross, Yh2-msl-Alb,,a/---msl--- x msl-yh2-Alb,,a/Msl-Yh2-alb,,a.

Phenotypes	Genotype	Frequency
Green, fertile, green*	Yh2---Msl---Alb,,a---	60
Green, male sterile, green*	Yh2---mslmsl Alb,,a---	73
Golden, fertile, green	yh2yh2-Msl---Alb,,a---	2
Golden, male sterile, green*	yh2yh2-mslmsl-Alb,,a---	63
Albino	------ ------ alb,,a alb,,a	--

* Expected parental phenotypes

An examination of Appendix Table 2 will show that there are only four genotypes that are viable, green and male sterile. Only one of these, msl-Yh2-alb,,a/msl-yh2-Alb,,a will segregate for the phenotypes observed in the test-cross. The reciprocal cross-over genotype would be Msl-yh2-Alb,,a/msl-yh2-Alb,,a with the phenotype being fertile, golden, green. None of this class were observed in the F_2 population, but would be expected to occur with only half the frequency of the green, male sterile, green phenotype. Considering that the single

cross-over x parental genotype would be more frequent than the double cross-over x parental genotype then the Yh2-ms1-alb,,a gene order can probably be eliminated from consideration, leaving ms1-yh2-alb,,a and ms1-alb,,a-yh2 gene order for consideration, (Appendix Table 2). Single cross-over gametes of Alb,,a alb,,a are not phenotypically detectable in the F_2 studied. Cross-over gametes of Yh2-yh2 and of Ms1 ms1 can be detected and would occur in equal frequency (Appendix Table 2). Cross-over gametes of Ms1 ms1 did occur whereas none occurred for Yh2 yh2. It may be concluded that Ms1 ms1 is distal to Yh2-yh2 and Alb,,a alb,,a.

In Table 15 are presented the phenotype frequencies from the test cross of the recombinant types.

Considering only cross-over gametes combining with parental gametes the cross-over gametes from the female of this cross are not distinguishable from the parental phenotypes. The two cross-over gametes, yh2 yh2 or Ms1 Ms1, from the male of this cross are distinguishable as golden fertile plants, whereas the Alb,,a alb,,a cross-over gamete is not distinguishable from the parental classes. It is quite likely that the two golden-fertile plants observed (Table 15) are the result of the cross-over gamete involving Ms1ms1 (Ms1-yh2-Alb,,a) rather than the cross-over gamete (Ms1-yh2-alb,,a) involving the Yh2yh2 locus.

All data would tend to confirm the gene order ms1-yh2-alb,,a or ms1-alb,,a-yh2. Further tests are needed to resolve the order of yh2 & alb,,a.

Male sterile, msl is very close to the centromere of chromosome 5 (Hockett and Eslick, 1970) and albino seedling lethal mutant, alb,,a, is 1.9 ± 0.6 cross-over units from msl. Considering other linkage data (Table 1) alternative positions of yh2 and alb,,a loci have been suggested in Fig. 4.

Chromosome 6:

No mutants were associated with genes on chromosome 6.

Chromosome 7:

Complete independence with all genetic markers except male sterile msl6 placed alb,,c on chromosome 7 (Table 16). Based on the distorted ratio the recombination value was determined to be 2.6 ± 5.8 percent. Since no other marker was used the position of alb,,c locus on chromosome 7 could not be ascertained.

Seedling lethal mutant, alb,,f segregated independently in relation to all genetic markers (Table 17). Crosses with alb,,f resulted in high proportions of distorted ratios, either the albino gene, the male sterile gene or both (Appendix Table 1).

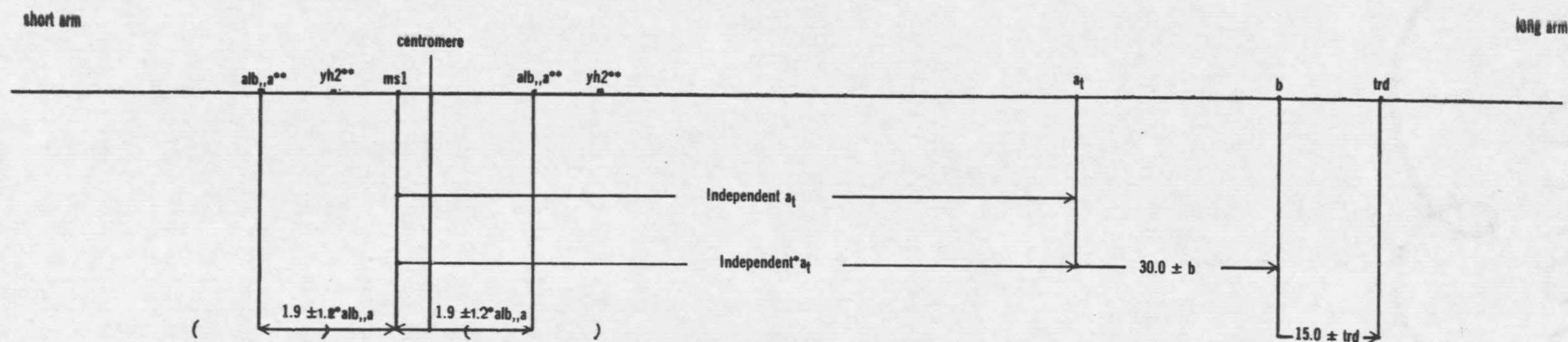

Fig.4. Proposed linkage map of chromosome 5.

* Data contributed by this paper.

** Suggested alternate positions of alb,,a.

( ) ± 2 standard errors.

Table 16. Linkage intensities involving male sterile genes and the seedling lethal mutant, alb,,c, determined from F_3 data.
Expected ratio: 1 MsMs Alb,,c Alb,,c : 2 Msms Alb,,c Alb,,c : 2 MsMs Alb,,c alb,,c : 4 Msms Alb,,c alb,,c

Cross	Chromosome tested	Number of Hills				Total	Interaction Chi-Square	Probability	Percent recombination and S. E.
		Alb,,c/Alb,,c		Alb,,c/alb,,c					
		Ms Ms	Ms ms	Ms Ms	Ms ms				
ms10 Alb,,c / Ms10 alb,,c	1	5	9	10	26	50	0.03	0.90-0.75	Independent
ms14 Alb,,c / Ms14 alb,,c	1	15	37	40	68	160	1.06	0.50-0.10	Independent
ms23b Alb,,c / Ms23b alb,,c	1	12	18	12	31	73	1.15	0.50-0.10	Independent
ms2 Alb,,c / Ms2 alb,,c	2	11	21	8	16	56	0.01	0.95-0.90	Distorted
ms5 Alb,,c / Ms5 alb,,c	3	15	29	21	47	112	0.12	0.80-0.70	Independent
ms24v Alb,,c / Ms24v alb,,c	4	58	88	33	64	243	3.08	0.10-0.05	Independent
ms1 Alb,,c / Ms1 alb,,c	5	9	20	17	40	86	0.02	0.90-0.75	Independent
ms16 Alb,,c / Ms16 alb,,c	7	1	3	18	30	52	35.90	<0.005	2.6 ± 5.8*
ms19 Alb,,c / Ms19 alb,,c	7	10	31	30	42	113	3.55	0.10-0.05	Independent

* F_3 genotypes confirmed from F_4 data.

Table 17. Linkage intensities involving male sterile genes and the seedling lethal mutant alb,,f, determined from F_3 data.
Expected ratio: 1 MsMs Alb,,f Alb,,f : 2 Msms Alb,,f Alb,,f : 2 MsMs Alb,,f alb,,f : 4 Msms Alb,,f alb,,f.

Cross	Chromosome tested	Number of Hills: Alb,,f/Alb,,f Ms Ms	Alb,,f/Alb,,f Ms ms	Alb,,f/alb,,f Ms Ms	Alb,,f/alb,,f Ms ms	Total	Interaction Chi-Square	Probability	Percent recombination and S. E.
ms10 Alb,,f / Ms10 alb,,f	1	26	33	46	46	151	0.06	0.90-0.80	Distorted
ms23b Alb,,f / Ms23b alb,,f	1	31	54	31	75	191	1.11	0.50-0.25	Distorted
ms2 Alb,,f / Ms2 alb,,f	2	62	36	98	46	242	0.71	0.50-0.25	Distorted
ms5 Alb,,f / Ms5 alb,,f	3	25	29	27	57	138	3.77	0.10-0.05	Independent
ms24v Alb,,f / Ms24v alb,,f	4	4	3	10	16	33	0.30	0.50-0.25	Independent
ms1 Alb,,f / Ms1 alb,,f	5	25	14	19	15	73	8.21	<0.005	Distorted
ms16 Alb,,f / Ms16 alb,,f	7	30	54	39	124	247	3.43	0.10-0.05	Independent
ms19 Alb,,f / Ms19 alb,,f	7	72	57	69	60	258	8.96	<0.005	Distorted

Chromosome Unknown:

White seedling, alb,,g segregated independently from all genetic markers used except ms23b (Table 18). Though Chi-squares were highly significant the segregation ratio did not indicate a significant deviation in the direction expected with linkage between alb,,g and ms23b.

Failure at first to obtain seed precluded the linkage study between alb,,g and orange lemma in the F_3. Results from the F_2 population are presented in Table 19. In case of complete linkage the segregation is expected to be 2 Alb,,g——O——: 1 Alb,,g——oo; while in independence the segregation would be 9 Alb,,g——O——: 3 Alb,,g——oo. A part of the total population having alb,,g alb,,g in homozygous condition would die due to homozygous lethality. Had alb,,g alb,,g survived the expected size of this population would have been 104, thus making the total 415 instead of the 311 observed (Table 19). Though the Chi-square value was highly significant the application of the maximum likelihood method for linkage calculation did not yield recombination values different from independence. Due to lethality a part of the population was lost, a study of the F_3 population will be necessary to determine the presence or absence of linkage between alb,,g and o.

Seedling lethal mutant alb,,h segregated independently from male sterile genes on chromosomes 1, 3, 4, 5 and 7. The seedling mutant, alb,,i exhibited distorted ratios with all male sterile genes except

Table 18. Linkage intensities involving male sterile marker genes and the albino seedling lethal mutant, alb,,g, determined from F_3 data.
Expected ratio: 1 MsMs Alb,,g Alb,,g : 2 Msms Alb,,g Alb,,g : 1 MsMs Alb,,g alb,,g : 4 Msms Alb,,g alb,,g.

Cross	Chromosome tested	Number of Hills: Alb,,g/Alb,,g		Number of Hills: Alb,,g/alb,,g		Total	Interaction Chi-Square	Probability	Percent recombination and S. E.
		Ms Ms	Ms ms	Ms Ms	Ms ms				
ms10 Alb,,g / Ms10 alb,,g	1	32	70	66	109	277	1.11	0.50-0.25	Independent
ms23b Alb,,g / Ms23b alb,,g	1	44	36	41	68	189	10.70	<0.005	Distorted
ms2 Alb,,g / Ms2 alb,,g	2	9	23	17	52	101	0.17	0.50-0.25	Independent
ms5 Alb,,g / Ms5 alb,,g	3	20	42	33	76	171	0.06	0.97-0.95	Independent
ms24v Alb,,g / Ms24v alb,,g	4	19	47	28	95	189	0.58	0.50-0.25	Independent
ms1 Alb,,g / Ms1 alb,,g	5	11	20	9	20	60	0.15	0.75-0.50	Independent
ms16 Alb,,g / Ms16 alb,,g	7	33	58	46	91	228	0.25	0.75-0.50	Independent
ms19 Alb,,g / Ms19 alb,,g	7	31	41	39	70	181	1.59	0.25-0.10	Independent

Table 19. Linkage intensity involving orange lemma marker gene and the seedling lethal mutant, alb,,g, determined from F_2 data.

Cross	Chromosome tested	Number of plants Alb,,g/ —— O ——	oo	Total	Linkage Chi-Square	Probability	Percent recombination and S. E.
oo Alb,,g / OO alb,,g	6	262	49	311	14.17	.005	

ms23b where independence was observed (Table 21 and Appendix Table 1). Insufficient seeds precluded the linkage study with other genetic markers on the other chromosomes. Linkage studies with the remaining chromosome markers may help to assign these seedling lethal albino genes to appropriate chromosomes.

In summary (Table 22) five of the nine seedling lethal mutants, alb,,a alb,,b alb,,c alb,,d and alb,,e not previously assigned to any chromosome, were observed to be linked with male sterile genes, ms1, ms5, ms16, ms23b and ms2, respectively. Utilizing the recombination values these mutants are assigned to their respective chromosomes and whenever possible tentative loci in the linkage maps are suggested.

Seedling lethal mutants, alb,,f and alb,,g exhibited distorted ratios to inflate the linkage Chi-square values, though no linkage could be detected by maximum likelihood method. Insufficient seeds precluded the linkage study of alb,,h and alb,,i with all genetic markers used in this experiment.

Table 20. Linkage intensities involving male sterile marker genes and the seedling lethal mutant, alb,,h, determined from F_3 data.
Expected ratio: 1 MsMs Alb,,h Alb,,h : 2 Msms Alb,,h Alb,,h : 2 MsMs Alb,,h alb,,h : 4 Msms Alb,,h alb,,h.

Cross	Chromosome tested	Number of Hills: Alb,,h/Alb,,h Ms Ms	Number of Hills: Alb,,h/Alb,,h Ms ms	Number of Hills: Alb,,h/alb,,h Ms Ms	Number of Hills: Alb,,h/alb,,h Ms ms	Total	Interaction Chi-Square	Interaction Probability	Percent Recombination and S. E.
ms10 Alb,,h / Ms10 alb,,h	1	15	29	37	54	135	0.60	0.50-0.25	Independent
ms14 Alb,,h / Ms14 alb,,h	1	7	17	20	41	85	0.07	0.97-0.95	Independent
ms23b Alb,,h / Ms23b alb,,h	1	15	38	35	59	147	1.25	0.50-0.25	Independent
ms5 Alb,,h / Ms5 alb,,h	3	12	22	23	50	107	0.16	0.75-0.50	Independent
ms24v Alb,,h / Ms24v alb,,h	4	32	45	39	97	213	3.81	0.10-0.05	Independent
ms1 Alb,,h / Ms1 alb,,h	5	23	34	40	72	169	0.38	0.75-0.50	Independent
ms16 Alb,,h / Ms16 alb,,h	7	6	6	4	7	23	1.34	0.30-0.20	Independent
ms19 Alb,,h / Ms19 alb,,h	7	23	51	42	81	197	0.21	0.75-0.50	Independent

Table 21. Linkage intensities involving male sterile genes and the seedling lethal mutant, alb,,i, determined from F_3 data.
Expected ratio: 1 MsMs Alb,,i Alb,,i : 2 Msms Alb,,i Alb,,i : 2 MsMs Alb,,i alb,,i : 4 Msms Alb,,i alb,,i.

Cross	Chromosome tested	Number of Hills: Alb,,i/Alb,,i Ms Ms	Alb,,i/Alb,,i Ms ms	Alb,,i/alb,,i Ms Ms	Alb,,i/alb,,i Ms ms	Total	Interaction Chi-Square	Probability	Percent recombination and S. E.
ms10 Alb,,i / Ms10 alb,,i	1	6	20	16	10	52	6.91	0.01-0.005	Distorted
ms14 Alb,,i / Ms14 alb,,i	1	13	30	47	42	132	6.81	0.01-0.005	Distorted
ms23b Alb,,i / Ms23b alb,,i	1	11	16	22	25	74	0.16	0.50-0.25	Independent
ms5 Alb,,i / Ms5 alb,,i	3	26	24	37	30	117	0.38	0.50-0.25	Distorted
ms16 Alb,,i / Ms16 alb,,i	7	18	27	34	37	116	0.36	0.50-0.25	Distorted
ms19 Alb,,i / Ms19 alb,,i	7	18	35	38	36	127	2.84	0.10-0.05	Distorted

Table 22. Summary of recombination of unidentified seedling lethals with known marker genes.

Gene tested	Symbol	Shown to be independent of chromosome marker on chromosome	Showed distorted ratio with chromosome markers on chromosome	Linkage with chromosome marker	Associated with genes on chromosome	Percent recombination and S.E.
Erbet	alb,,a	1,3,4,6 and 7	2 and 7	ms1 ms1	5	1.9 ± 1.2
Carlsberg II	alb,,b	1,2,4,5,6 and 7		ms5 ms5	3	2.9 ± 0.6
Titan	alb,,c	1,3,5 and 6	7,2,4 and 7	ms16 ms16	7	2.6 ± 5.8
Erbet	alb,,d	1,2,3,4,5 and 7	1 and 7	ms23b ms23b	1	7.4 ± 1.2
Titan	alb,,e	1,3,4,5 and 7		ms2 ms2	2	1.2 ± 0.5
Shabet	alb,,f	3,4,6 and 7	1,2,5 and 7			
Titan	alb,,g	1,2,3,4 and 7	1 and 5			
Compana x Mars	alb,,h	1,3,4,5 and 7				
Titan	alb,,i	1 and 6	1,3 and 7			

Yield of the Heterozygotes:

The seed yield of cereal crops is dependent upon the yielding potentials of individual plants, which in turn is a function of: (1) number of spikes per plant, (2) number of seeds per spike, and (3) weight of a seed, and (4) number of plants per unit area. The first three of these were studied.

The analysis of variance of number of spikes per plant, number of seeds per spike, number of seeds per plant, weight of 1000 seeds, and seed yield per plant are presented in Table 23.

Among varieties, differences for number of spikes per plant, number of seeds per spike and number of seeds per plant, weight of 1000 seeds and seed yield per plant were observed. Since the varieties involved in this experiment are genotypically different from one another the observed differences in yielding potentials are in accordance with expectation.

The genotype, $\underline{A_{c2}A_{c2}}$ had a greater 1000 seed weight and more seed yield per plant than did the genotype $\underline{A_{c2}a_{c2}}$ (Table 24). Since the differences may be associated with mean values means and differences are given in Appendix Tables 3, 4, 5, 6 and 7. A lesser number of seeds per spike, though non-significant, may have given the homozygotes the expected larger seed resulting in the increased yield.

The heterozygous genotype, $\underline{A_c a_c}$, exhibited an increase in number of seeds per spike resulting in more seeds per plant and a higher per plant seed yield than from the homozygous, $\underline{A_c A_c}$ genotype (Table 24).

The heterozygous genotype $\underline{A_c a_c}$ had more seeds per spike than did the homozygous genotype. Seedling mutant Alb,,a alb,,a showed statistical non-significance for all characters except number of seeds per plant and seed yield per plant (Table 23). In the Cm cm population the heterozygotes yielded better per plant, than the homozygotes whereas the homozygotes out-yielded the heterozygotes in the population Alb,,a alb,,a (Table 24). All the yield contributing factors of the Cm cm population favoured the heterozygotes whereas all the yield contributing factors of the Alb,,a alb,,a population, except weight per 1000 seeds favoured the homozygotes.

The Alb,,c alb,,c population showed significant differences for number of spikes per plant and number of seeds per spike (Table 23). The number of spikes per plant of the homozygote was superior to the heterozygote, while the number of seeds per spike for the homozygote were fewer (Table 24). The increase in yield components were not in one particular direction, i.e. not either in favour of homozygote or heterozygote, but multi-directional, which compensated one for the other, and thus causing the differences in yield per plant to be non-significant (Table 23).

In the Alb,,e alb,,e population significant differences in number of spikes per plant, number of seeds per spike and number of seeds per plant were detected (Table 23). The heterozygotes were superior to the homozygotes (Table 24).

Only the number of seeds per spike, seeds per plant and yield per plant was observed to be significant in Alb,,g alb,,g populations (Table 23). The heterozygote produced more seeds per spike resulting in more seeds per plant than the homozygote (Table 24) and in turn resulting in a higher seed yield per plant. The uni-directional contribution resulted in a significant increase in the seed yield per plant in favour of the heterozygotes. The observed performance of this mutant is very similar to the response of the Alb,,e alb,,e populations, although the differences are not as great.

The Alb,,i alb,,i population showed highly significant differences in number of spikes per plant and seed weight (Table 23). The heterozygous genotype had fewer spikes per plant but greater weight per seed (Table 24). This multi-directional response of the yield contributing factors might have compensated one another, thus causing seed yield per plant to be non-significant for the two genotypes.

In summary (Table 24), nine of the twenty populations studied exhibited one locus heterosis (positive or negative advantages of the Aa genotype as compared to the AA genotype) for yield per plant or one or more of the components of yield. Single character heterosis for number of seeds per spike, $A_c a_c$ and weight per 1000 seeds, $A_{c2} a_{c2}$, were observed to contribute to heterosis for yield per plant. Two character heterosis, number of spikes per plant, and number of seeds per spike, were observed to result in increased yield per plant in Alb,,a alb,,a,

and Alb,,e alb,,e and Alb,,g alb,,g populations. The three yield components contributed to heterosis for per plant yield in populations of Cm cm. Component compensation, number of spikes per plant and number of seeds per plant in Alb,,c alb,,c and number of spikes per plant and weight per 1000 seed in Alb,,i alb,,i, resulted in no heterosis for yield in these two lines. It is interesting that in the five instances of significance for number of seeds per spike all show positive single locus (Aa) heterosis.

Table 23. Analysis of variance mean squares for number of spiked tillers per plant, number of seeds per spike, number of seeds per plant, weight of 1000 seeds in grams and seed yield per plant in grams.

Source of Variation	d.f.	Number of spikes per plant	Number of seeds per spike	Number of seeds per plant	Weight of 1000 seeds (grams)	Seed yield per plant (grams)
Replications	3	18.49	97.61	96897.34**	42.64	145.87**
Varieties	19	457.55**	833.22**	149326.60**	125.44**	165.77**
Error (1)	57	13.32	42.59	22524.21	34.67	20.19
Genotypes within variety (20)						
Cm_2 cm_2	1	4.06	2.75	4845.21	0.12	5.87
Y_c y_c	1	0.00	3.93	3815.01	1.13	1.93
A_{c2} a_{c2}	1	0.17	2.21	398.75	45.13*	24.71*
Y y	1	0.68	16.58	11986.93	0.13	9.20
A_2 a_2	1	0.10	2.61	1926.65	0.50	4.06
Y_x y_x	1	5.08	0.00	9398.89	0.50	8.57
A_c a_c	1	0.01	135.30**	26450.00**	0.13	45.22**
X_c x_c	1	5.56	22.37*	1884.68	0.00	0.94
A_t a_t	1	0.27	0.02	473.39	10.13	1.84
X_n x_n	1	0.37	0.14	49.90	6.13	1.65
Cm cm	1	2.31	8.67	9186.90	4.50	21.39*
Alb,,a alb,,a	1	15.82	6.39	18259.60*	3.13	34.82*
Alb,,b alb,,b	1	0.00	3.95	1465.84	10.13	0.21
Alb,,c alb,,c	1	21.32*	25.78*	279.54	6.13	2.22
Alb,,d alb,,d	1	9.05	2.00	634.93	18.00	8.22
Alb,,e alb,,e	1	40.05**	34.70*	132169.97**	0.50	160.02**
Alb,,f alb,,f	1	9.94	4.19	295.61	0.13	0.43
Alb,,g alb,,g	1	6.26	21.71*	36956.64**	0.13	42.46**
Alb,,h alb,,h	1	3.93	6.29	1997.12	3.13	0.84
Alb,,i alb,,i	1	50.25**	0.17	10891.40	60.50**	2.26
Error (2)	60	4.26	5.16	3587.48	7.70	5.33
Total	159					

* Significant at 5% level. ** Significant at 1% level.

Table 24. Summary of comparison of yield and its components from homozygous and heterozygous genotypes for twenty spring barley varieties.

Genotypes	Difference between homozygous (AA) & heterozygous (Aa) genotypes				
	Number of spikes /plant	Number of seeds /spike	Number of seeds /plant	Weight/1000 seed (grams)	Yield /plant, (grams)
Cm_2 Cm_2 vs Cm_2 cm_2	1.42	1.17	49.22	-0.25	1.72
Y_c Y_c vs Y_c y_c	-0.02	1.40	43.67	-0.75	0.98
A_{c2} A_{c2} vs A_{c2} a_{c2}	0.30	-1.05	-14.12	4.75*	3.52*
Y Y vs Y y	0.58	2.88	77.27	-0.25	2.15
A_2 A_2 vs A_2 a_2	-0.22	-1.14	-31.03	-0.05	-1.43
Y_x Y_x vs Y_x y_x	-1.59	0.03	-68.55	0.50	-2.07
A_c A_c vs A_c a_c	-0.08	-8.23**	-115.00**	0.25	-4.76**
X_c X_c vs X_c x_c	1.66	-3.35*	30.69	0.00	0.69
A_t A_t vs A_t a_t	-0.36	0.10	-15.39	2.25	-0.96
X_n X_n vs X_n x_n	0.43	-0.26	5.00	1.75	0.71
Cm Cm vs Cm cm	-1.07	-0.08	-67.77	-1.50	-3.27*
Alb,,a Alb,,a vs Alb,,a alb,,a	2.81	1.79	95.55*	-1.25	4.18**
Alb,,b Alb,,b vs Alb,,b alb,,b	0.04	0.59	-27.07	2.25	-0.32
Alb,,c Alb,,c vs Alb,,c alb,,c	3.26*	-3.59*	-11.95	1.75	-1.05
Alb,,d Alb,,d vs Alb,,d alb,,d	2.12	-1.00	-17.81	3.00	2.03
Alb,,e Alb,,e vs Alb,,e alb,,e	-4.47**	-4.16*	-257.07**	0.50	-8.94**
Alb,,f Alb,,f vs Alb,,f alb,,f	5.01	-1.45	-12.16	0.25	-0.47
Alb,,g Alb,,g vs Alb,,g alb,,g	-1.77	-3.29*	-135.93**	0.25	-4.60**
Alb,,h Alb,,h vs Alb,,h alb,,h	1.40	-1.78	-31.60	1.25	-0.65
Alb,,i Alb,,i vs Alb,,i alb,,i	5.01**	0.29	73.79	-5.50**	1.07

SUMMARY AND CONCLUSION

A collection of 18 different spontaneously occurring seedling lethal mutants, of which nine were not previously identified or assigned to any chromosome, were studied to determine their linkage relationship with 10 different identified genetic markers, at least one on each of the seven chromosomes.

This study has shown that the nine seedling lethal mutants, not previously studied, are monogenically inherited and cause lethality in the homozygous recessive condition. Temporary symbols, *alb,,a* through *alb,,i* have been assigned to these nine mutants.

Male steriles, *ms10* and *ms14*, short awn, lk_2, naked caryopsis, *n*, and albino seedling, a_{c2}, were previously assigned to chromosome 1. The present linkage study suggests the gene order to be: lk_2-*n*-*ms10* *ms14*-a_{c2}. White seedling, *alb,,d* is located on chromosome 1, but its location on chromosome 1 could not be determined. Lack of recombination between long awned outer glume, *e* and male sterile, *ms2* confirms that these two genes are very closely linked. The close linkage between *alb,,e* and *ms2* places *alb,,e* on chromosome 2. Male sterile, *ms5* is linked with white seedling, x_s, and closely linked with alb,,b; thus *alb,,b* is placed on chromosome 3. Lack of appropriate genetic markers precluded the determination of *alb,,b* and *alb,,e* loci. Nevertheless, in the linkage maps alternate loci of these two mutants have been suggested.

Cream seedling *cm2* and a_t are independent of *ms1*, but the *alb,,a*

mutant is tightly linked with msl; thus alb,,a is assigned to chromosome 5.

A three-point linkage study involving yh2, msl and alb,,a suggests that msl is distal to yh2 and alb,,a; and the probable gene order may either be: msl-yh2-alb,,a or msl-alb,,a-yh2. Alternate loci of alb,,a and yh2 are suggested on the proposed linkage map.

The seedling mutant, alb,,c is tentatively placed on chromosome 7. The seedling mutants alb,,f, alb,,g could not be assigned to any chromosome because of distorted ratios. Lack of seed precluded linkage studies with alb,,h and alb,,i and all the genetic markers used in this experiment and hence these two genes remained unassigned to any chromosome.

The effect of heterozygous and homozygous loci of 20 lethal mutants on yield components were studied. For the purpose of discussion the AA genotypes and aa genotypes were considered to be equal for the yield components (character) evaluated. A significant deviation of a character in the Aa from the AA genotype was then considered to be positive or negative one locus heterosis. Nine of the 20 mutants showed one locus heterosis for yield per plant or one or more of the yield components. Single character heterosis is exhibited by the mutants, A_{c2} a_{c2}, X_c x_c and Alb,,a alb,,a, while A_c a_c, Alb,,c alb,,c, Alb,,g alb,,g and Alb,,i alb,,i showed two character heterosis. The two-character heterosis of the seedling mutants, A_c a_c and Alb,,g alb,,g

is positive while Alb,,c alb,,d and Alb,,i alb,,i exhibit both positive and negative. The seedling elthal mutant, Alb,,e alb,,e showed three-character positive heterosis.

A pre-flowering selective gene or genes tightly linked with either alb,,a and ms1; alb,,b and ms5, alb,,d and ms23b or alb,,e and ms2 is needed to produce hybrid barley seed. However, in the absence of such a closely linked pre-flowering selective gene or genes these tight linkages may be used in balanced tertiary trisomics for hybrid seed production. Although conclusions from one year's study of spaced plants can only be tentative, it seems logical to point out that the three-yield-component heterosis exhibited by Alb,,e alb,,e might be a better choice for hybrid seed production.

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APPENDIX

Appendix Table 1. Chi-square values of the ratios tested to determine possible linkage between male sterile and seedling lethal mutant genes.

Cross	Ratio Tested			Interaction chi-square	Conclusion
	1 MsMs AA:2 Msms AA 2 MsMs Aa:4 Msms Aa	2 Msms:1 MsMs	2 Aa:1 AA		
ms10 vs Ac_2	59.94**	19.13**	27.19**	13.62**	Linked
ms10 vs Ac_2	201.58**	77.52**	107.62**	16.44**	Linked
ms10 vs Ac_2	279.21**	98.22**	140.07**	40.92**	Linked
ms14 vs Ac_2	94.24**	32.46**	49.59**	12.19**	Linked
ms23b vs Ac_2	5.36	4.72	0.49	0.15	Ind.
ms10 vs Yc	5.33	1.17	3.59	0.57	Ind.
ms14 vs Yc	1.80	1.35	0.03	0.42	Ind.
ms23b vs Yc	26.24**	20.62**	3.31	2.31	Ind.
ms2 vs A2	75.38**	26.67**	39.84**	8.87**	Linked
ms2 vs Y	76.98**	48.90**	11.61**	16.47**	Linked
ms5 vs Ac	237.81**	102.53**	99.20**	36.08**	Linked
ms5 vs X_s	24.73**	9.01**	14.23**	1.49	Linked
ms1 vs A_t	15.22**	0.00	14.52**	0.7	Distorted
ms1 vs Cm2	1.09	0.96	0.00	0.13	Ind.
ms16 vs Cm	2.04	1.50	0.27	0.27	Ind.
ms19 vs Cm	6.65	4.89	1.75	0.00	Ind.
ms10 vs Alb,,a	2.97	2.23	0.67	0.07	Ind.
ms23b vs Alb,,a	6.56	1.46	5.05	0.04	Ind.
ms2 vs Alb,,a	14.32**	13.49**	0.36	0.87	Distorted
ms5 vs Alb,,a	5.72	0.61	3.40	1.52	Ind.
ms24v vs Alb,,a	6.71	4.33	0.57	1.81	Ind.
ms1 vs Alb,,a	111.57**	48.55**	42.89**	20.13**	Linked
ms16 vs Alb,,a	6.93	0.02	6.85*	0.06	Distorted
ms19 vs Alb,,a	1.67	0.12	1.45	0.10	Ind.

Appendix Table 1. Cont.

Cross	Ratio Tested: 1 MsMs AA:2 Msms AA 2 MsMs Aa:4 MsMs Aa	Ratio Tested: 2 Msms:1 MsMs	Ratio Tested: 2 Aa:1 AA	Interaction chi-square	Conclusion
ms10 vs Alb,,b	3.83	0.62	3.09	0.12	Ind.
ms23b vs Alb,,b	4.10	0.22	0.07	3.83	Ind.
ms2 vs Alb,,b	3.61	2.83	0.57	0.21	Ind.
ms5 vs Alb,,b	241.29**	94.73**	91.98**	51.58**	Linked
ms23b vs Alb,,b	3.51	0.48	2.47	0.56	Ind.
ms1 vs Alb,,b	2.35	0.26	1.62	0.47	Ind.
ms16 vs Alb,,b	5.75	0.14	0.91	4.70	Ind.
ms19 vs Alb,,b	4.51	0.01	0.56	3.94	Ind.
ms10 vs Alb,,c	0.90	0.24	0.63	0.03	Ind.
ms14 vs Alb,,c	1.18	0.07	0.04	1.06	Ind.
ms23b vs Alb,,c	3.13	0.00	1.98	1.15	Ind.
ms2 vs Alb,,c	14.13**	0.02	14.28**	0.01	Distorted
ms5 vs Alb,,c	1.96	0.06	1.78	0.12	Ind.
ms24v vs Alb,,c	83.07**	1.75	78.24**	3.08	Distorted
ms1 vs Alb,,c	0.38	0.36	0.00	0.02	Ind.
ms16 vs Alb,,c	51.51**	0.23	15.38**	35.90**	Linked
ms19 vs Alb,,c	4.21	0.21	0.45	3.55	Ind.
ms10 vs Alb,,d	4.49	0.00	1.70	2.79	Ind.
ms14 vs Alb,,d	23.90**	17.90**	4.33	1.67	Distorted
ms23b vs Alb,,d	201.63**	100.17**	55.33**	46.13**	Linked
ms2 vs Alb,,d	3.50	1.59	0.70	1.21	Ind.
ms5 vs Alb,,d	7.53	1.98	5.06	0.49	Ind.
ms24v vs Alb,,d	2.22	1.66	0.13	0.43	Ind.
ms1 vs Alb,,d	1.36	0.03	0.55	0.78	Ind.
ms16 vs Alb,,d	0.62	0.58	0.00	0.04	Ind.
ms19 vs Alb,,d	6.73	0.81	0.07	5.85	Ind.

Appendix Table 1. Cont.

Cross	Ratio Tested: 1 MsMs AA:2 Msms AA 2 MsMs Aa:4 MsMs Aa	2 Msms:1 MsMs	2 Aa:1 AA	Interaction chi-square	Conclusion
ms10 vs Alb,,e	1.26	0.18	0.04	0.28	Ind.
ms23b vs Alb,,e	6.15	2.09	0.09	3.97	Ind.
ms2 vs Alb,,e	149.37**	59.14**	62.03**	28.20**	Linked
ms5 vs Alb,,e	1.37	0.24	0.96	0.17	Ind.
ms24v vs Alb,,e	6.28	0.21	4.26	1.81	Ind.
ms1 vs Alb,,e	6.38	2.41	3.40	0.57	Ind.
ms16 vs Alb,,e	2.06	1.38	0.03	1.83	Ind.
ms19 vs Alb,,e	1.87	0.00	1.74	0.13	Ind.
ms10 vs Alb,,f	16.38**	13.99**	2.23	0.06	Distorted
ms23b vs Alb,,f	11.89*	0.06	10.72**	1.11	Distorted
ms2 vs Alb,,f	123.34**	117.04**	5.59	0.71	Distorted
ms5 vs Alb,,f	6.92	1.17	2.08	3.77	Ind.
ms24v vs Alb,,f	3.68	1.21	2.17	0.30	Ind.
ms1 vs Alb,,f	45.31**	23.84**	13.26**	8.21**	Distorted
ms16 vs Alb,,f	7.09	2.73	6.93	3.43	Ind.
ms19 vs Alb,,f	93.96**	52.75**	32.25**	8.96**	Distorted
ms10 vs Alb,,g	3.15	0.52	1.51	1.12	Ind.
ms23b vs Alb,,g	29.10**	11.52**	6.88*	10.70**	Distorted
ms2 vs Alb,,g	2.90	2.61	0.12	0.17	Ind.
ms5 vs Alb,,g	1.13	0.42	0.65	0.06	Ind.
ms24v vs Alb,,g	6.88	6.09	0.21	0.58	Ind.
ms1 vs Alb,,g	9.22	0.00	9.07**	0.15	Ind.
ms16 vs Alb,,g	4.86	0.17	4.44	0.25	Ind.
ms19 vs Alb,,g	7.29	2.32	3.38	1.59	Ind.

Appendix Table 1. Cont.

Cross	Ratio Tested: 1 MsMs AA:2 Msms AA 2 MsMs Aa:4 MsMs Aa	Ratio Tested: 2 Msms:1 MsMs	Ratio Tested: 2 Aa:1 AA	Interaction chi-square	Conclusion
ms10 vs Alb,,h	2.26	1.63	0.03	0.60	Ind.
ms14 vs Alb,,h	1.15	0.09	0.99	0.07	Ind.
ms23b vs Alb,,h	1.76	0.03	0.48	1.25	Ind.
ms5 vs Alb,,h	0.28	0.01	0.11	0.16	Ind.
ms24v vs Alb,,h	4.57	0.00	0.76	3.81	Ind.
ms1 vs Alb,,h	1.56	1.18	0.00	0.38	Ind.
ms16 vs Alb,,h	6.06	1.06	3.66	1.34	Ind.
ms19 vs Alb,,h	1.80	0.00	1.59	0.21	Ind.
ms10 vs Alb,,i	15.30**	1.89	6.50	6.91*	Distorted
ms14 vs Alb,,i	15.57**	8.73**	0.03	6.81*	Distorted
ms23b vs Alb,,i	4.71	4.22	0.33	0.16	Ind.
ms5 vs Alb,,i	27.10**	22.07**	4.65	0.38	Distorted
ms16 vs Alb,,i	8.80	6.89*	1.55	0.36	Distorted
ms19 vs Alb,,i	13.48**	6.61	4.03	2.84	Distorted

* probability <0.01

** probability <0.005

Appendix Table 2. F_2 genotypes, phenotypes, frequency due to independence, possible gene orders of F_1's of a cross, yh2-msl-Alb,,a/yh2-msl-Alb,,a x Yh2-msl-Alb,,a/Yh2-Msl-alb,,a and the number of gametes, either parental or cross-over, required to produce the genotypes in F_2.

F_2 Genotypes†	F_2 Pehnotype	Independence frequency in F_2	Possible gene orders of F_1 plants								
			yh2-msl-Alb,,a / Yh2-Msl-alb,,a			msl-yh2-Alb,,a / Msl-Yh2-alb,,a			msl-Alb,,a-yh2 / Msl-alb,,a-Yh2		
			P	SC	DC	P	SC	DC	P	SC	DC*
Yh2-Msl-Alb,,a / Yh2-Msl-Alb,,a	Green, fertile, green	1	-	2	-	-	2	-	-	-	2
Yh2-Msl-alb,,a** / Yh2-Msl-Alb,,a	Green, fertile, green	2	1	1	-	1	1	-	1	-	1
Yh2-Msl-alb,,a** / Yh2-Msl-alb,,a**	Green, fertile, albino	1 dead	2	-	-	2	-	-	2	-	-
Yh2-Msl-Alb,,a / Yh2-msl-Alb,,a	Green, fertile, green	2	-	2	-	-	1	1	-	1	1
Yh2-Msl-alb,,a** / Yh2-msl-Alb,,a	Green, fertile, green	4	1	1	-	1	-	1	1	-	1
Yh2-Msl-alb,,a** / Yh2-msl-alb,,a	Green, fertile, albino	2 dead	1	-	1	1	1	-	1	1	-
Yh2-Msl-Alb,,a / yh2-Msl-Alb,,a	Green, fertile, green	2	-	1	1	-	2	-	-	1	1
Yh2-Msl-alb,,a** / yh2-Msl-Alb,,a	Green, fertile, green	4	1	-	1	1	1	-	1	1	-
Yh2-Msl-alb,,a** / yh2-Msl-alb,,a	Green, fertile, albino	2 dead	1	1	-	1	-	1	1	1	-
Yh2-Msl-Alb,,a / yh2-msl-Alb,,a**	Green, fertile, green	4	1	1	-	1	1	-	-	1	1
yh2-msl-Alb,,a** / Yh2-Msl-alb,,a**	Green, fertile, green	8	2	-	-	2	-	-	2	-	-
Yh2-Msl-alb,,a** / yh2-msl-alb,,a	Green, fertile, albino	4 dead	1	1	-	1	1	-	-	-	2
Yh2-msl-Alb,,a / Yh2-msl-Alb,,a	Green, male sterile, green	1	-	2	-	-	-	2	-	2	-

Appendix Table 2. Cont.

F_2 Genotypes†	F_2 Phenotype	Independence frequency in F_2	Possible gene orders of F_1 plants								
			yh2-msl-Alb,,a / Yh2-Msl-alb,,a			msl-yh2-Alb,,a / Msl-Yh2-alb,,a			msl-Alb,,a-yh2 / Msl-alb,,a-Yh2		
			P	SC	DC	P	SC	DC	P	SC	DC*
Yh2-msl-alb,,a / Yh2-msl-Alb,,a	Green, male sterile, green	2	-	1	1	-	1	1	-	2	-
Yh2-msl-alb,,a / Yh2-msl-alb,,a	Green, male sterile, albino	1 dead	-	-	2	-	2	-	-	2	-
Yh2-msl-Alb,,a / yh2-msl-Alb,,a**	Green, male sterile, green	2	1	1	-	1	-	1	1	1	-
Yh2-msl-alb,,a / yh2-msl-Alb,,a**	Green, male sterile, green	4***	1	-	1	1	1	-	1	1	-
Yh2-msl-alb,,a / yh2-msl-alb,,a	Green, male sterile, albino	2 dead	-	1	1	-	2	-	-	1	1
yh2-Msl-Alb,,a / yh2-Msl-Alb,,a	Golden, fertile, green	1	-	-	2	-	2	-	-	2	-
yh2-Msl-alb,,a / yh2-Msl-Alb,,a	Golden, fertile, green	2	-	1	1	-	1	1	-	2	-
yh2-Msl-alb,,a / yh2-Msl-alb,,a	Golden, fertile, albino	1 dead	-	2	-	-	-	2	-	2	-
yh2-Msl-Alb,,a / yh2-msl-Alb,,a**	Golden, fertile, green	2***	1	-	1	1	1	-	1	1	-
yh2-Msl-alb,,a / yh2-msl-Alb,,a**	Golden, fertile, green	4	1	1	-	1	-	1	1	1	-
yh2-Msl-alb,,a / yh2-msl-alb,,a	Golden, fertile, albino	2 dead	-	2	-	-	1	1	-	1	1
yh2-msl-Alb,,a** / yh2-msl-Alb,,a**	Golden, male sterile, green	1	2	-	-	2	-	-	2	-	-
yh2-msl-alb,,a / yh2-msl-Alb,,a**	Golden, male sterile green	2	1	1	-	1	1	-	1	-	1
yh2-msl-alb,,a / yh2-msl-alb,,a	Golden, male sterile, albino	1 dead	-	2	-	-	2	-	-	-	2

Appendix Table 2. Cont.

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* P = Parental gamete
SC = Single cross-over gamete
DC = Double cross-over gamete
** Parental gamete
***Observed or expected cross-over genotypes
† The gene order may not necessarily be in the sequence written

Appendix
Table 3. Comparison of means of homozygous and heterozygous genotypes of twenty spring barley varieties for 1000 seed weight in grams.

Genotypes	Weight of 1000 seeds in grams		
	Homozygous AA	Heterozygous Aa	Difference AA-Aa
Cm_2 Cm_2 vs Cm_2 cm_2	43.50	43.75	-0.25
Y_c Y_c vs Y_c y_c	39.00	39.75	-0.75
A_{c2} A_{c2} vs A_{c2} a_{c2}	46.00	41.25	4.75*
Y Y vs Y y	32.00	32.25	-0.25
A_2 A_2 vs A_2 a_2	38.25	38.75	-0.50
Y_x Y_x vs Y_x y_x	35.25	34.75	0.50
A_c A_c vs A_c a_c	41.25	41.00	0.25
X_c X_c vs X_c x_c	39.00	39.00	0.00
A_t A_t vs A_t a_t	39.00	41.25	-2.25
X_n X_n vs X_n x_n	39.25	37.50	1.75
Cm Cm vs Cm cm	36.25	37.75	-1.50
Alb,,a Alb,,a vs Alb,,a alb,,a	46.50	47.75	-1.25
Alb,,b Alb,,b vs Alb,,b alb,,b	48.50	46.25	2.25
Alb,,c Alb,,c vs Alb,,c alb,,c	39.75	38.00	1.75
Alb,,d Alb,,d vs Alb,,d alb,,d	38.50	35.50	3.00
Alb,,e Alb,,e vs Alb,,e alb,,e	35.75	35.25	0.50
Alb,,f Alb,,f vs Alb,,f alb,,f	45.00	44.75	0.25
Alb,,g Alb,,g vs Alb,,g alb,,g	37.50	37.25	0.25
Alb,,h Alb,,h vs Alb,,h alb,,h	41.25	40.00	1.25
Alb,,i Alb,,i vs Alb,,i alb,,i	38.50	44.00	-5.50**

* Significant at 5% level.
** Significant at 1% level.

Appendix
Table 4. Comparison of means of homozygous and heterozygous genotypes of twenty spring barley varieties for number of seeds per spike.

Genotypes	Number of seeds per spike Homozygous AA	Heterozygous Aa	Difference AA-Aa
Cm_2 Cm_2 vs Cm_2 cm_2	23.31	22.14	1.17
Y_c Y_c vs Y_c y_c	36.74	35.34	1.40
A_{c2} A_{c2} vs A_{c2} a_{c2}	31.93	32.98	-1.05
Y Y vs Y y	40.79	37.91	2.88
A_2 A_2 vs A_2 a_2	28.75	29.89	-1.14
Y_x Y_x vs Y_x y_x	40.03	40.00	0.03
A_c A_c vs A_c a_c	40.63	48.86	-8.23**
X_c X_c vs X_c x_c	43.56	46.91	-3.35*
A_t A_t vs A_t a_t	39.19	39.09	0.10
X_n X_n vs X_n x_n	38.56	38.82	-0.26
Cm Cm vs Cm cm	36.00	36.08	-0.08
Alb,,a Alb,,a vs Alb,,a alb,,a	18.53	16.74	1.79
Alb,,b Alb,,b vs Alb,,b alb,,b	29.99	29.40	0.59
Alb,,c Alb,,c vs Alb,,c alb,,c	26.48	30.07	-3.59*
Alb,,d Alb,,d vs Alb,,d alb,,d	18.47	19.47	-1.00
Alb,,e Alb,,e vs Alb,,e alb,,e	33.72	37.88	-4.16*
Alb,,f Alb,,f vs Alb,,f alb,,f	13.37	14.82	-1.45
Alb,,g Alb,,g vs Alb,,g alb,,g	37.34	40.63	-3.29*
Alb,,h Alb,,h vs Alb,,h alb,,h	16.34	18.12	-1.78
Alb,,i Alb,,i vs Alb,,i alb,,i	14.49	14.20	0.29

* Significant at 5% level.
** Significant at 1% level.

Appendix
Table 5. Comparison of means of homozygous and heterozygous genotypes of twenty spring barley varieties for seed yield per plant.

Genotypes	Seed yield in grams		
	Homozygous AA	Heterozygous Aa	Difference AA-Aa
Cm_2 Cm_2 vs Cm_2 cm_2	23.94	22.22	1.72
Y_c Y_c vs Y_c y_c	28.80	27.82	0.98
A_{c2} A_{c2} vs A_{c2} a_{c2}	30.99	27.47	3.52*
Y Y vs Y y	24.81	22.66	2.15
A_2 A_2 vs A_2 a_2	23.56	24.99	-1.43
Y_x Y_x vs Y_x y_x	25.58	27.65	-2.07
A_c A_c vs A_c a_c	23.16	27.92	-4.76**
X_c X_c vs X_c x_c	25.81	25.12	0.69
A_t A_t vs A_t a_t	30.14	31.10	-0.96
X_n X_n vs X_n x_n	25.42	24.71	0.71
Cm Cm vs Cm cm	17.17	20.44	-3.27*
Alb,,a Alb,,a vs Alb,,a alb,,a	24.28	20.10	4.18*
Alb,,b Alb,,b vs Alb,,b alb,,b	28.76	29.08	-0.32
Alb,,c Alb,,c vs Alb,,c alb,,c	27.86	28.91	-1.05
Alb,,d Alb,,d vs Alb,,d alb,,d	33.96	31.93	2.03
Alb,,e Alb,,e vs Alb,,e alb,,e	23.91	32.85	-8.94**
Alb,,f Alb,,f vs Alb,,f alb,,f	16.79	17.26	-0.47
Alb,,g Alb,,g vs Alb,,g alb,,g	28.00	32.60	-4.60**
Alb,,h Alb,,h vs Alb,,h alb,,h	23.32	23.97	-0.65
Alb,,i Alb,,i vs Alb,,i alb,,i	16.24	15.17	1.07

* Significant at 5% level.
** Significant at 1% level.

Appendix
Table 6. Comparison of means of homozygous and heterozygous genotypes of twenty spring barley varieties for number of spikes per plant.

Genotypes	Number of spikes per plant Homozygous AA	Heterozygous Aa	Difference AA-Aa
$Cm_2\ Cm_2$ vs $Cm_2\ cm_2$	24.12	22.70	1.42
$Y_c\ Y_c$ vs $Y_c\ y_c$	19.77	19.79	-0.02
$A_{c2}\ A_{c2}$ vs $A_{c2}\ a_{c2}$	21.17	20.87	0.30
Y Y vs Y y	18.99	18.41	0.53
$A_2\ A_2$ vs $A_2\ a_2$	21.29	21.51	-0.22
$Y_x\ Y_x$ vs $Y_x\ y_x$	17.97	19.56	-1.59
$A_c\ A_c$ vs $A_c\ a_c$	13.77	13.85	-0.08
$X_c\ X_c$ vs $X_c\ x_c$	15.23	13.57	1.66
$A_t\ A_t$ vs $A_t\ a_t$	18.88	19.24	-0.36
$X_n\ X_n$ vs $X_n\ x_n$	17.50	17.07	0.43
Cm Cm vs Cm cm	13.67	14.74	-1.07
Alb,,a Alb,,a vs Alb,,a alb,,a	27.90	25.09	2.81
Alb,,b Alb,,b vs Alb,,b alb,,b	21.62	21.58	0.04
Alb,,c Alb,,c vs Alb,,c alb,,c	26.56	23.30	3.26*
Alb,,d Alb,,d vs Alb,,d alb,,d	48.30	46.18	2.12
Alb,,e Alb,,e vs Alb,,e alb,,e	19.73	24.20	-4.47**
Alb,,f Alb,,f vs Alb,,f alb,,f	28.67	23.66	5.01
Alb,,g Alb,,g vs Alb,,g alb,,g	19.29	21.06	-1.77
Alb,,h Alb,,h vs Alb,,h alb,,h	34.42	33.02	1.40
Alb,,i Alb,,i vs Alb,,i alb,,i	28.67	23.66	5.01**

* Significant at 5% level.
** Significant at 1% level.

Appendix
Table 7. Comparison of means of homozygous and heterozygous genotypes of twenty spring barley varieties for number of seeds per plant.

Genotypes	Number of seeds per plant		
	Homozygous AA	Heterozygous AA	Difference AA-Aa
$Cm_2\ Cm_2$ vs $Cm_2\ cm_2$	552.15	502.93	49.22
$Y_c\ Y_c$ vs $Y_c\ y_c$	738.12	694.45	43.67
$A_{c2}\ A_{c2}$ vs $A_{c2}\ a_{c2}$	676.32	690.44	-14.12
Y Y vs Y y	772.68	695.41	77.27
$A_2\ A_2$ vs $A_2\ a_2$	611.80	642.83	-31.03
$Y_x\ Y_x$ vs $Y_x\ y_x$	713.40	781.95	-68.55
$A_c\ A_c$ vs $A_c\ a_c$	558.05	673.05	-115.00**
$X_c\ X_c$ vs $X_c\ x_c$	671.55	640.86	30.69
$A_t\ A_t$ vs $A_t\ a_t$	737.90	753.29	-15.39
$X_n\ X_n$ vs $X_n\ x_n$	671.26	666.26	5.00
Cm Cm vs Cm cm	486.81	554.58	-67.77
Alb,,a Alb,,a vs Alb,,a alb,,a	514.60	419.05	95.55*
Alb,,b Alb,,b vs Alb,,b alb,,b	608.81	635.88	-27.07
Alb,,c Alb,,c vs Alb,,c alb,,c	694.17	706.12	-11.95
Alb,,d Alb,,d vs Alb,,d alb,,d	874.95	892.76	-17.81
Alb,,e Alb,,e vs Alb,,e alb,,e	662.44	919.51	-257.07**
Alb,,f Alb,,f vs Alb,,f alb,,f	368.12	380.28	-12.16
Alb,,g Alb,,g vs Alb,,g alb,,g	731.54	867.47	-135.93**
Alb,,h Alb,,h vs Alb,,h alb,,h	561.59	593.19	-31.60
Alb,,i Alb,,i vs Alb,,i alb,,i	411.35	337.56	73.79

* Significant at 5% level.
** Significant at 1% level.

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