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CYTOLOGICAL LOCATION OF A LETHAL OVULE GENE (*lo2*) IN MAIZE (*Zea mays* L.)[1]

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1. INTRODUCTION

VAN HORN and NELSON (*4*) found a plant of the genotype *Wx/wx* which when self-pollinated produced a semi-sterile ear with 75% of waxy (*wx/wx*) kernels instead of the expected 25%. When plants grown from the starchy (*Wx/-*) kernels were selfed, some had the normal 25% of waxy kernels whereas other plants gave high percentages of waxy, ranging from 53% to 75.5%. Six *Wx/* — plants were used as the female parent in crosses with a *wx/wx* stock. Four of these gave extremely high percentages of waxy kernels (91.1 — 95.3%) and two had approximately 50% waxy. All plants with aberrant percentages of waxy kernels produced semi-sterile ears. Van Horn and Nelson concluded that the *Wx* chromosome carried a recessive lethal ovule factor (*lo2*) producing inviable female gametophytes.

The same investigators showed that the percentage of waxy kernels on ears from test crossed *Wx/wx* female parents was 93.6%. On the assumption that *lo2* female gametes are rarely, if ever, capable of functioning, NELSON (*2*) calculated that *lo2* is six map units distal to waxy.

VAN HORN and NELSON (*4*) also showed the *lo2* female gametophyte may be functional in a low percentage of the ovules. Nineteen *Wx/wx* heterozygotes derived from egg parents of *lo Wx/Lo wx* constitution were self-pollinated. Seventeen had about 25% waxy kernels as expected, but two plants had high percentages of waxy (51.2% and 54.8%). These two plants, therefore, possessed the *lo2* factor which must have been transmitted through the ovules in the preceding generation.

Since *lo2* pollen grains are functional, the lethal ovule phenotype was believed to result from a recessive mutation rather than a deletion.

RHOADES *et alii* (*3*) demonstrated that the B chromosome of maize induce

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breaks in knob-bearing chromosomes of the regular complement and there is a consequent loss of the acentric portion of the chromosome arm distal to the breakpoint (high-loss phenomenon).

In the studies of deficiencies in 9S generated by the high-loss phenomenon, two overlapping deletions were available which made possible the cytological mapping of a lethal ovule locus, presumably allelic to *lo*2.

2. MATERIAL AND METHODS

The high-loss strain used in this experiment had several B chromosomes and chromosome 9 carried a large knob terminating the short arm (9S). Marker genes on this arm included the dominant *Yg*2 (green seedling and plant), *C* (anthocyanin pigment in aleurone) and *Wx* (I-KI gives blue staining starch in endosperm and pollen grains) alleles in sequential order with *Yg*2 close to the knob. The knobbed chromosome 9 was subject to loss. Pollen from the high-loss strain was applied to the silks of yg^2 (yellow green seedling and plant), *C* (colorless aleurone), *wx* (I-KI gives red staining starch in endosperm and pollen grains) tester plants. The fertilization of an egg cell with recessive *yg*2 by a sperm deficient for the *Yg*2 allele produces a yellow green sporophyte. Consequently, all of the exceptional yellow green plants have a deficient chromosome 9 which had undergone breakage.

3. RESULTS AND DISCUSSION

The yellow green exception 264-1 had no pollen abortion, but when used as the female parent in a cross with a *c wx* tester, gave a semi-sterile ear with 24 *c Wx* (14.3%) kernels and 143 *c wx*. Since all kernels were colorless, the *C* locus, cytologically located at about 0.75 in 9S, as well as the *Yg* marker, was deleted. The deficient chromosome carrying the dominant *Wx* allele was transmitted either in very reduced frequencies or not at all in female gametes.

When plant 264-1 was used to pollinate four *c wx* tester plants, 96 of the 996 progeny kernels were *c Wx*. Since terminal deficiencies large enough to include the *C* locus are not male transmissible (*1*), the 9.8% of *Wx* kernels were assumed to represent recombinants between the *Wx* locus and the breakpoint.

To test whether or not the deficient chromosome was female transmissible, 17 of the 24 *c Wx* kernels were planted as family number 648 and the 10 surviving plants were used as male and female parents in test crosses. The results are presented in Table 1.

Three of the 10 plants had low frequencies of *Wx* kernels when used as female and male parents in test crosses. All three plants, 648-2, -5, and -6 had semi-sterile ears and the genetic evidence indicates that they carry the deficient chromosome. Furthermore, plant 648-6 was analyzed cytologically and the presence of the deficiency was confirmed. Consequently, the deficient chromosome 9 was transmitted through the female gametophyte with a low frequency.

Another yellow green exception with no pollen abortion (677-4) gave an average of 12.8% *Wx* kernels when used as the male in test crosses. Since this value is very close to the comparable figure obtained with plant 264-1, the two exceptional plants presumably have deficient chromosomes with similar breakpoints. However, plant 677-4 produced an ear with 87 *c Wx* (46.8%) and 99 *c wx* kernels, and no detectable ovule abortion.

Thus, one can conclude that plant 677-4 contains the wildtype allele at the *lo*2 locus allowing a normal or nearly normal female transmission of the deficient chromosome, whereas this locus is distal to the breakpoint in plant 264-1. Absence of the *Lo*2 locus by deletion greatly reduces female transmission of the deficient chromosome, just as the mutation to the *lo*2 allele found by VAN HORN and

TABLE 1 . Reciprocal test cross data with Df(C) → Wx/N c wx individuals derived from plant 264-1*

	Df(C) Wx/N c wx (♀) X c wx/c wx (♂)			c wx/c wx (♀) X Df(C) Wx/N c wx (♂)		
	Number of Kernels		%	Number of Kernels		%
Family	c Wx	c wx	Wx	c Wx	c wx	Wx
648-1	95	88	52.0	212	153	58.0
648-2	16	109	12.8	11	88	11.2
648-3	88	94	48.3	80	84	48.8
648-4	121	117	50.8	66	64	50.0
648-5	11	86	11.3	33	288	10.3
648-6	8	76	9.5	18	168	9.6
648-7	122	137	47.5	109	121	47.4
648-12	148	156	48.6	98	102	49.0
648-13	94	87	52.0	171	183	48.0
648-16	126	130	49.2	112	104	52.0

* Df(C) - deficient chromosome including C locus;
N - normal chromosome

NELSON (*4*) decreased the recovery rate of a marker in the affected chromosome.

That the *Lo*2 locus has in fact been deleted in plant 264-1 is reinforced by the similarity between these data and the results of VAN HORN and NELSON (*4*). Female semi-sterility and very low frequencies of transmission were observed in both cases. The percentage of crossing over (6.0%) between *Wx* and *lo*2 loci observed by VAN HORN and NELSON differs slightly from the 9.8% value but crossover values are known to vary substantially in different stocks.

Cytological analyses were carried out in plants 264-1 and 677-4 to determine the breakpoints of the deficiencies (Figure 1) in 9S.

As expected from the genetic tests, the breakpoints in 9S for the two *yg*2 exceptions are very similar. The terminal deficiency in plant 264-1 occurred at approximately 0.67 while in plant 677-4 it was at 0.70. Consequently, the best estimate of the physical location of the *lo*2 is at position 0.68 which places it in the seventh or more probably in the eighth chromomere from the end of the short arm.

4. RESUMO

Em milho, um gene recessivo, localizado no braço curto do cromossomo 9, causa a morte do óvulo (*lo*2), produzindo gametófitos femininos inviáveis. Essa descoberta foi feita por meio de testes genéticos.

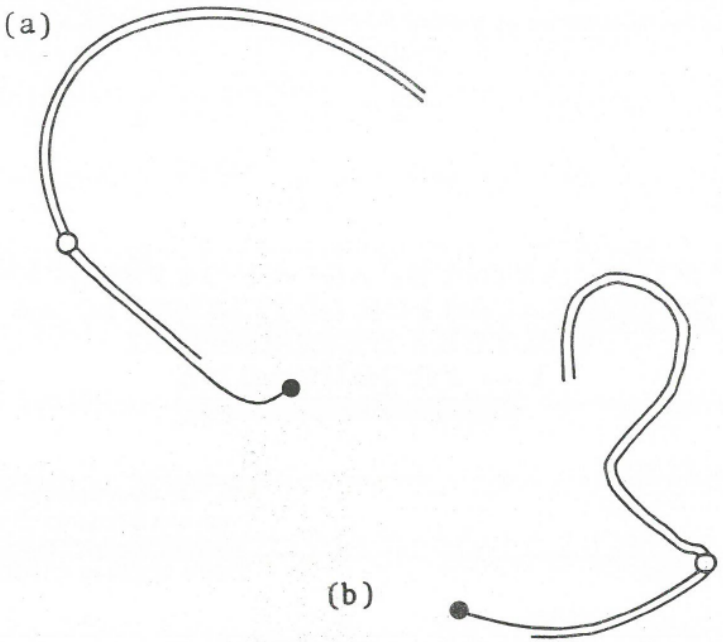

FIGURE 1 - Camera lucida drawings of pachytene preparations of heterozygous deficient chromosome 9 from plants 264-1 (a) and 677-4 (b).

Mediante o uso de deficiências cromossômicas que se sobrepõem, tornou-se possível mapear citologicamente, com precisão, o *locus* para letalidade do óvulo (*lo2*) na posição 0,68 do braço curto do referido cromossomo 9, o que corresponde, provavelmente, ao 8.º cromômero a contar da extremidade do braço.

5. REFERENCES

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