INHERITANCE OF BEET NECROTIC YELLOW VEIN VIRUS (BNYVV) SYSTEMIC INFECTION IN CROSSES BETWEEN SUGARBEET AND *BETA MACROCARPA*

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ABSTRACT

Beet necrotic yellow vein virus (BNYVV), the cause of rhizomania, rarely infects sugarbeet (*Beta vulgaris* L.) systemically. Conversely, from mechanical inoculation BNYVV almost always systemically infects *B. vulgaris* subsp. *macrocarpa* (*B. mac*) line that grows as a weedy annual in the Imperial Valley of California. This *B. mac* has been used for many years in the virology programs at Salinas as an indicator host for virus assays. *B. mac* shows other reactions to viruses that are of interest. When infected young, *Beet yellows*, *Beet mosaic*, and *Beet curly top viruses* kill *B. mac*. Other "nonbeet" viruses, e.g., *Lettuce mosaic virus*, readily produce systemic infection in *B. mac* but not in sugarbeet. It was of interest to determine the genetic basis of these different host-plant reactions. *B. mac* is a very easy bolting annual and highly self-fertile and successful crosses were achieved only when sugarbeet was used as the female. Color patterns and annualism were used as markers to positively identify F_1 hybrids. The very limited number of F_1 plants tested had the virus reaction of sugarbeet or were intermediate. The F_2 suggested that BNYVV systemic infection was conditioned by a homozygous recessive factor but the lack of fit may have been caused by escapes and lethal and sublethal mutant plants and to incomplete expressivity. F_3 population and F_3 line patterns also suggested recessive inheritance, but again ratios appeared disturbed. Most F_3 plants produced from F_2 plants with systemic infection to BNYVV were susceptible to systemic infection and there was no evidence for seed transmission. Evaluation of segregating populations is continuing with the intent to produce a biennial line with the virus reactions of *B. mac* and to determine if different genes for host reaction are involved for each virus or if one recessive factor is predisposing *B. mac* to be widely susceptible to systemic infection by numerous viruses.

INTRODUCTION

For about 70 years, an accession of *Beta macrocarpa* Guss. from the Imperial Valley of California has been used in the sugarbeet virology programs of the USDA-ARS at Riverside and Salinas, California. It was found to be particularly useful as a local lesion host following mechanical inoculation for many viruses. During its use by Drs. C.W. Bennett, J.E. Duffus, H.-Y. Liu, et al. it was found to

have a number of unique traits that were different from sugarbeet. In addition to being highly susceptible to most if not all viruses that infect sugarbeet, it was found also to be a systemic host to some nonbeet viruses, e.g., *Lettuce mosaic virus* (Duffus, unpublished). During its routine use in laboratory and greenhouse research at Salinas and elsewhere, *B. macrocarpa* from the Imperial Valley (from now on called affectionately "Bmac") was found to almost always be susceptible to systemic infection following mechanical inoculation with *Beet necrotic yellows vein virus* (BNYVV) (Putz et al., 1990), the cause of rhizomania. Conversely, sugarbeet is rarely systemically infected following natural or local lesion inoculations (Schlösser, 1984; Hillmann & Schlösser, 1986). On Bmac, BNYVV local lesions are generally chlorotic and visually it appears that the virus establishes localized systemic infection that spreads to the vascular system. Soon thereafter, classical BNYV symptoms (Tamada & Baba, 1973) show on all new leaves.

B.macrocarpa that occurs in the Imperial Valley is believed to be an introduced plant from Europe (Bartsch & Ellstrand, 1999; McFarlane, 1975). Local lore suggests that it was introduced from the Azores by Portuguese immigrants in the early 1900s (Lewellen, unpublished). In the Imperial Valley it is a common and troublesome weed in sugarbeet production. Common selective herbicides used on sugarbeet will not remove Bmac, so it must be controlled mechanically and manually, though in the seedling stage at thinning time it is very difficult to distinguish from sugarbeet. Following sugarbeet sowing into dry beds, Bmac emerges in the fall with the sugarbeet crop. It grows rapidly through the mild winter, bolts under nearly day length neutral conditions, and sets copious amounts of seed prior to sugarbeet harvest.

Naturally occurring hybrids descending from crosses between sugarbeet and *B. macrocarpa* were reported by McFarlane (1975). Using allozyme diversity, Bartsch & Ellstrand (1999) found substantial evidence for hybridization and introgression of *B.vulgaris* alleles into one *B.macrocarpa* accession from the Imperial Valley and suggested gene flow between these species was possible.

It is the very easy bolting nature of Bmac that limits its use in the laboratory as a virus indicator plant. It works ideally in the winter, but in days longer than 12 hours, it bolts very quickly, flowers, sets seed, and dies, limiting its summertime usefulness. For this reason, in the early 1980s, Dr. J.E. Duffus asked me to develop for him an equivalent genotype for virus research but in a nonbolting, biennial background that would remain in the desired rosette stage indefinitely. The genetics of the development of systemic infection from BNYVV local lesions also was an interesting research question. We also wondered if such a biennial beet could be used directly in greenhouse and field plantings to indicate the presence of rhizomania (BNYVV) in soil tests.

A research project was initiated: (i) to determine the inheritance of systemic infection in *B.macrocarpa* by BNYVV from mechanical inoculation; (ii) to determine the specificity of this tendency of *B.macrocarpa* to develop systemic infection for other viruses, and (iii) to develop a biennial, true breeding sugarbeet-like line with the virus reactions of Bmac as a host plant for future virology research.

MATERIALS AND METHODS

The *B.macrocarpa* accession WB157 that had been selected from the Imperial Valley of California in the 1930s was used. This accession has been increased from a few plants many times over the course of its use at Salinas. It appears to be highly uniform and homogeneous. Research in Japan (Abe & Tsuda, 1988) and Salinas (Lewellen, unpublished) showed it to be a highly self-fertile diploid. Similar *B.macrocarpa* material was also used in the investigations of McFarlane (1975) and Bartsch & Ellstrand (1999). At Salinas for this study, it was found necessary to use Bmac and its F_1's with sugarbeet as the male parent and cross it to self-sterile or male-sterile sugarbeet to get the desired crosses. Actual F_1 hybrids were positively identified by the hypocotyl color seedling marker, annualism, and/or growth characteristics. Several sugarbeet lines were used, but self-sterile, green hypocotyl line C37 (Lewellen et al., 1985) was the preferred parent. A line called 747 that is similar to C37 but self-fertile (S^f) and segregates for genetic-male-sterility was used also in the initial investigation.

Plant material was mechanically inoculated with BNYVV in the two-six true-leaf stage. BNYVV was obtained from local lesions on *Chenopodium quinoa* or from systemically infected Bmac or sugarbeet x Bmac plants. Standard mechanical inoculation procedures were used (Grassi et al., 1989; Liu et al., 2003). Serological tests for BNYVV by ELISA were done as described by Wisler et al. (1994, 1999). Following mechanical inoculation, the individual plants were scored for the occurrence of local lesions on inoculated leaves. Only plants that showed one or more local lesions were included in the subsequent counts for systemic vs. nonsystemic infection. A plant was counted as being systemically infected when it developed leaves with typical BNYV symptoms (Tamada & Baba, 1973).

Seed of the parents and their progeny was sown into sterilized sand flats. Following counts for hypocotyls color, seedlings were transplanted to 10 cm pots filled with sterilized greenhouse soil mix. Following growth for 2-4 weeks in heated greenhouses, plants were mechanically inoculated with BNYVV. A pre- and post-conditioning treatment in subdued light or shade was usually used in conjunction with inoculation. Reaction to BNYVV was then allowed to develop in greenhouses maintained above 15°C.

Breeding material was induced to uniformly bolt in overwintered steckling nurseries or vernalization rooms maintained at 6°C. After induction, plant materials were grown in pots in greenhouses under supplemental light. Plants to be crossed were matched for stage of development and paired under paper bags prior to anthesis. Because of the extremely short stature of Bmac plants, pot height was adjusted so that pollen would cascade through the open flowers of the female plant and supplemental agitation was used to disseminate the pollen.

RESULTS AND DISCUSSION

The results are presented in a series of tables (Tables 1 - 5). Table 1 shows the initial test of possible parental lines and *B.macrocarpa* accessions for reaction to BNYVV following mechanical inoculation. Whereas none of the sugarbeet lines

developed systemic infection, *B.macrocarpa* accessions from Imperial Valley did. Accession WB192 from the Canary Islands also was systemically infected whereas WB22 from Spain was not.

Table 1. Evaluation of Beta macrocarpa and sugarbeet for systemic infection of BNYVV following mechanical inoculation.

Sugarbeet		Number of Plants Non-systemic	Systemic
C37	breeding line	12	0
500	inbred line	12	0
600	inbred line	12	0
747	population	12	0
Beta macrocarpa			
WB157	Bmac, Bennett's IV Acc., 2n=18	0	23
WB25	Bmac,IV Acc., 2n=18	0	11
WB192	Canary Island Acc., 2n=36	0	12
WB22	Spain Acc. (PI 198405)	12	0

In 1988, the first successful sugarbeet x Bmac F_2 and BC_1F_1 [(sugarbeet x (sugarbeet x Bmac)] lines were evaluated. Whereas all but one Bmac plant showed systemic infection and none of the sugarbeet parents did, the F_2 fit ($P > 0.5$) a 3 nonsystemic: 1 systemic ratio and the testcrosses to sugarbeet did not have any systemically infected plants (Table 2). In 1989, tests of randomly generated F_3 and BC_1F_2 lines were evaluated (Table 2).

Segregation among F_3 lines fit ($P > 0.25$) a distribution of 1 homozygous nonsystemic: 2 segregating: 1 homozygous systemic ratio. Likewise the BC_1F_2 lines fit a pattern of 1 homozygous nonsystemic: 1 segregating ratio. These results fit the expectations for the segregation of one gene where the allele for nonsystemic reaction is dominant to the recessive allele for systemic infection. In each of these cases, though, there was a tendency for too many nonsystemic plants and too few systemically infected plants. In another set of BC_1F_2 testcross lines in which only nonbolted (biennial) plants were selected for selfing, there were poor fits to the tested 1:1 ratio (Table 2). From these BC_1F_2 and F_3 lines, BC_1F_3 and F_4 lines were randomly generated. In general these lines did not fit the expected ratio for the segregation of one gene with two alleles.

At this point, this research was interrupted and these plant materials were eventually discarded. In part, this was due to the poor fit to the tested ratios, but mostly it was due to the high frequency of abnormal plants that segregated within these lines and the final loss of systemic infection under stringent selection for biennials. In addition to seedlings that perished from chlorophyll deficient mutants, there were many abnormal and unusual growth and leaf traits. Similar distorted genetic ratios and abnormalities had been reported by Abe & Tusda (1988) and Abe et al. (1987).

Table 2. Tests in 1988 & 1989 of parents and their progeny for segregation of systemic infection to BNYVV following mechanical inoculation and fit to the segregation of one gene where nonsystemic is dominant to systemic.

1988		Number of Plants		
Parents		Non-systemic	Systemic	χ^2 (3:1)
Bmac	(*Beta macrocarpa*)	1	27	---
C37		16	0	---
747		24	0	---
F_2 Line				
7203	747 x Bmac	37	10	P > .50
BC_1F_1 Testcrosses				
7204	747 x F_1	64	0	---
7201	C37 x F_1	20	0	---
1989				
Parents				
Bmac		3	69	---
747		24	0	---
C37		24	0	---
Segregation among F_3 lines (expect 1:2:1)				
		Number of Lines		
		Non-sys	Segregate	Systemic	χ^2
F_3	747 x Bmac	10	13	5	P > .25
Segregation among BC_1F_2 lines (expect 1:1)					
BC_1F_2	747 x F_1	17	12	---	P > .25
BC_1F_2	C37 x F_1	11	8	---	P > .25
Segregation among BC_1F_2 lines from biennial plants					
BC_1F_2	747 x F_1	5	0	---	P > .01*
BC_1F_2	C37 x F_1	6	2	---	P > .10

Starting in 1999, a new set of crosses between sugarbeet as the female parent and Bmac were made. Of these crosses, only the results involving C37 sugarbeet will be given in detail. In 2001, individual F_2 lines and BC_1F_1 testcrosses were evaluated for reaction to BNYVV following mechanical inoculation. The segregation of these lines is given in Table 3. With only two exceptions all Bmac plants became systemically infected and no C37 plants developed systemic infection. Nine F_2 lines from individual F_1 plants were tested and all of these fit a 3 nonsystemic: 1 systemic ratio. As in the 1988-89 tests, these results again suggested that systemic infection is recessive to nonsystemic infection and conditioned by one gene. The total F_2 population though had a poor fit and had too few systemically infected plants, as did most of the individual F_2 lines.

In 2002, F_3 lines and populations were tested for reaction to BNYVV (Table 4). For an F_3 bulk produced from F_2 plants that had been selected for systemic infection, 20 out of 23 plants were systemically infected. In a 2003 test similar counts were obtained. This helped substantiate that systemic infection is

essentially entirely under genetic control. Because this F_3 was produced in the greenhouse without pollen protection, the three nonsytemically infected F_3 plants could have resulted from outcrosses to other flowering plants in the greenhouse. In this and other tests with this F_3 bulk with seed produced on systemically infected plants, no non-inoculated plants developed systemic infection to BNYVV. This further supports the evidence that BNYVV is not seed-borne (Asher, 1993). Prior attempts at Salinas to obtain seed on systemically infected plants from natural field infection had not been successful.

Table 3. Tests in 2001 of parents and their progeny for segregation of systemic infection to BNYVV following mechanical inoculation and fit to the segregation of one gene where nonsystemic is dominant to systemic.

	Number of Plants					
	Hypocotyl Color			Systemic Infection		
Parents	Red	Green	χ^2(3:1)	Non-sys	Systemic	χ^2(3:1)
Bmac	33	0	---	2	14	---
C37	0	76	---	23	0	---
F_2 Lines						
0201- 1 C37 x Bmac	38	9	P >.25	26	6	P >.25
- 2	37	8	P >.25	11	4	P >.75
- 4	27	12	P >.25	11	3	P >.75
- 5	32	16	P >.10	14	1	P >.10
- 8	22	4	P >.25	16	3	P >.25
-10	21	6	P >.50	17	2	P >.10
-11	22	5	P >.25	19	2	P >.10
-12	27	9	P >.99	18	6	P >.99
-14	43	7	P >.05	18	5	P >.50
Total	269	76	P >.25	150	32	P >.01*
BC_1F_1 testcrosses			χ^2 (1:1)			
0211- 1 C37 x F_1	8	8	P >.99	12	0	---
- 4	11	7	P >.25	11	0	---
- 5	13	7	P >.10	12	0	---
-10	9	7	P >.50	11	0	---
-12	9	14	P >.25	12	0	---
-14	12	14	P >.50	11	0	---
Total	62	57	P >.50	69	0	---

In the 2002 tests (Table 4), the F_3 population between C37 x Bmac that had been generated from randomly selected and increased F_2 plants would not fit the normal segregation expected for one gene. Nor would the F_3 lines produced from randomly selected F_2 plants. For the segregation of a single gene where systemic infection is conditioned by the homozygous recessive, segregation within these F_3 lines should either be homozygous nonsystemic, segregate 3: 1, or be homozygous systemically infected. Two of the 10 F_3 lines appeared to be homozygous nonsystemic but none appeared to be homozygous systemic. As in the earlier study, whereas the F_2 segregation generally fit the ratio for one gene, the F_3 lines did not. These F_3 results again seem to contradict the F_2 results. Likely, this again is demonstrating that crosses between sugarbeet x B. macrocarpa have distorted segregation patterns (Abe & Tsuda, 1988; Abe et al., 1987).

Table 4. Tests in 2002 of parents and their progeny for reaction to systemic infection by BNYVV following mechanical inoculation.

Parents		Number of Plants	
		Non-systemic	Systemic
Bmac	Beta macrocarpa	0	60
C37	sugarbeet	48	0
F_3 from bulked F_2 plants			
1201	C37 x Bmac	45	3
1202	C78 x Bmac	38	9
1205	500 x Bmac	120	0
F_3 bulk from systemically infected F_2 plants			
1210	C37 x Bmac	3	20
F_3 lines from randomly selected F_2 plants			
1201-101	C37 x Bmac	48	0
-102		17	15
-103		37	11
-104		10	2
-105		23	0
-106		46	2
-107		20	4
-109		25	22
-110		9	14
-111		27	5

As part of the 2002 tests, individual parental and F_3 plants were tested for BNYVV by ELISA. Plants from the F_3 lines were systematically chosen based upon their scored visual reaction to BNYVV for systemic infection. These plants were either positive or negative for visual systemic symptoms. Leaf and fibrous root samples were then measured for ELISA value. The results of these ELISA tests are shown in Table 5. For Bmac, both the root and leaf tissue were highly positive for BNYVV ELISA values with the roots and leaves being 8.6 and 7.4 times the healthy mean, respectively. For C37, both the root and leaf tissue had ELISA values equal to the healthy check (Table 5). For the F_3 lines between C37 x Bmac, several patterns occurred for individual plants. All plants in some lines and individual plants in other lines had the parental phenotypes, that is, the

reactions of C37 or Bmac. In addition, there was a new phenotype where systemic symptoms were not expressed in the leaves but the root tissue was highly positive for BNYVV (ELISA values). This suggested that for these plants, unlike C37, BNYVV moved from the local lesion infection and became systemic in the root tissue but not in the leaf tissue. That is, the virus seemed to be transported to the root, but not back into the shoot. For the plants with systemic reaction type like Bmac, the virus moved from the local lesions into the root and then back into the shoot to form typical BNYVV symptoms in the leaves. No research was done to try to understand the mechanisms involved in this virus movement. Guinchedi et al (1988) showed that BNYVV from natural infection occurred in the xylem tissue of the root. Typically, viruses move systemically in the phloem. It may be possible that whereas adaxial movement is in the phloem, abaxial movement is in the xyleme and different genetic factors are involved. These differences suggest that there may be several genetic components involved in systemic infection from local lesions in sugarbeet. As shown in Table 5, with only one or two exceptions, when a plant was visually scored as systemically infected, it always had BNYVV in the root and leaf tissue.

The results obtained from this research suggested that the difference in systemic infection from local lesions was under fairly simple genetic control. It is likely that a dominant allele in sugarbeet prevents typical sugarbeet genotypes from having the virus reaction of Bmac. The data from the F_2 generation usually fit the tests for one gene in which the dominant allele prevented systemic infection from local lesions and the homozygous recessive allele allowed systemic infection from local lesions. Beyond this F_2 generation, the data fit the expectations for one gene poorly. This lack of fit may be due to incomplete chromosomal pairing and lack of completely homologous genomes (Abe et al., 1987). From the literature reviewed by Bartsch & Ellstrand (1999), they concluded that *B.vulgaris* and *B.macrocarpa* are likely two separate but closely related species. Abe & Tsuda (1988) also showed similar problems with distorted genetic ratios and plant abnormalities.

They also used the Imperial Valley accession of *B.macrocarpa*. Their research showed: that the genetic ratios within backcross generations depended upon the direction of the cross; that when the F_1 was used as the male, there were greater distortions, that is, there were reciprocal cross differences; that parental selection also contributed to distortion; that aberrant ratios appeared to arise from linkage of markers with factors that affect gametogenesis, pollen function and abortion, and embryo development; and that distortions might have complex multi-chromosomal genetic origins.

Table 5. Examples of reactions of selected individual parental and F_3 plants for BNYVV by ELISA following mechanical inoculation with BNYVV. Plants with ELISA values greater than 3x the mean healthy check (=1.0) are considered positive. RR,R_ = red hypocotyl; rr = green. For visual leaf symptoms: + = systemic symptoms; - = none.

Parents	Hypoc Color	Leaf Symptoms	ELISA Value Root	ELISA Value Leaf
Bmac (mean)	RR	+	8.6 (+)	7.4 (+)
C37 (mean)	rr	-	0.9 (-)	1.0 (-)
F_3 from bulked F_2 plants				
1201 - 1	R_	+	10.4 (+)	12.8 (+)
- 2	R_	-	6.5 (+)	1.0 (-)
- 3	R_	-	4.9 (+)	1.0 (-)
- 5	rr	+	7.3 (+)	10.0 (+)
- 6	rr	-	2.4 (-)	0.9 (-)
- 7	R_	-	0.9 (-)	0.9 (-)
1202 - 1	rr	-	5.2 (+)	0.8 (-)
- 2	rr	+	5.9 (+)	13.0 (+)
- 3	R_	+	11.6 (+)	11.6 (+)
- 5	R_	-	5.6 (+)	1.2 (-)
F_3 lines from randomly selected F_2 plants				
1201 -101 (mean)	rr	-	1.1 (-)	1.2 (-)
-105 (mean)	rr	-	0.9 (-)	1.0 (-)
1201 -104 - 1	rr	+	7.1 (+)	12.6 (+)
- 2	rr	-	3.9 (+)	0.9 (-)
- 3	rr	-	4.6 (+)	1.0 (-)
- 4	rr	+	7.6 (+)	11.1 (+)
1209 -109 - 1	RR	+	8.8 (+)	15.2 (+)
- 2	RR	-	7.2 (+)	1.3 (-)
- 3	RR	+	9.1 (+)	15.1 (+)
- 6	RR	-	6.6 (+)	1.4 (-)
1201 -106 - 1	RR	-	0.8 (-)	0.8 (-)
- 3	RR	+	6.7 (+)	10.0 (+)
- 5	RR	+	9.5 (+)	17.4 (+)
- 6	RR	-	9.0 (+)	1.4 (-)
1201 107 - 1	rr	+	9.2 (+)	12.1 (+)
- 3	R_	+	9.3 (+)	17.4 (+)
- 4	R_	-	0.9 (-)	1.4 (-)
- 6	R_	-	5.3 (+)	1.3 (-)
- 8	rr	-	5.0 (+)	1.3 (-)

In the present study, for the inheritance of systemic infection, these are likely the same causes for the change from adequate fits in the F_2 generation to poorer fits in each succeeding generation. It is possible that the allele(s) from Bmac that condition BNYVV systemic reaction are differentially lost. In 2003, 24 randomly generated F_4 families from C37 x Bmac were evaluated for systemic infection from mechanical inoculation. Only one of these F_4 lines appeared to be homozygous for systemic infection. All of the plants within this F_4 line had red hypocotyls and leaf and plant types were unusual; about 25% of the seedlings had severe chlorosis and did not survive. Under normal Mendelian inheritance, about 10 or 11 (44%) of these F_4 lines should have been homozygous systemic if only one factor pair of alleles is involved.

Breeding, genetic, and virology research will be continued on the inheritance of BNYVV systemic infection from local lesions. A primary objective will still be to develop a true breeding biennial beet with the virus traits of *B.macrocarpa*.

CONCLUSION

From mechanical inoculation and the development of local lesions, BNYVV almost always goes systemic in *B.macrocarpa* developing typical BNYV symptoms in developing leaves. In C37 sugarbeet, BNYVV from mechanical inoculation almost always is nonsystemic. In *B.macrocarpa*, mechanically inoculated plants are positive for BNYVV in their roots and leaves. In C37, inoculated plants are negative for BNYVV in their roots and leaves.

In segregating lines from crosses between C37 x *B.macrocarpa*, individual F_3 plants show the phenotype of their parents or are different from their parental types. Some F_3 plants with local lesions develop BNYVV infection in their roots, but the virus does not go systemic to their leaves (a nonparental reaction). Plants that are negative for BNYVV for ELISA tests in their roots are always negative for systemic infection as judged visually or from ELISA tests. That is, F_3 plants that were scored as systemically infected, always had virus in their roots and those without leaf symptoms may or may not have virus in their roots.

In crosses between C37 x *B.macrocarpa*, the F_2 generation fits a segregation pattern of 3 nonsystemic infection: 1 systemic infection. This suggests that the differences in systemic infection are conditioned by one factor pair where resistance (nonsystemic) is the dominant allele over susceptibility (systemic infection), the recessive allele. If it is assumed that the parental types are C37 = *SS* and *B.macrocarpa* = *ss*, then *SS* and *Ss* are nonsystemic and *ss* is systemic. *s* may be linked to *B* (annualism) in *B.macrocarpa* and selection pressure to produce a biennial (*bb*) with *B.macrocarpa*'s virus reaction, results in loss of systemic infection.

In the F_3 and backcross generations, there are very poor fits to a one gene model. This distortion of genetic ratios may be due to chromosomal irregularities between sugarbeet and *B.macrocarpa* as suggested by Abe & Tsuda (1988). It also appears possible that more than one gene is involved in the expression of systemic infection and virus movement. That is, one genetic factor that controls movement from local lesions to the root and a second genetic factor that controls movement from the root to the shoot that results in systemic infection. These complexities are still being investigated.

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