ABERRANT RATIO: AN ANOMALY IN MAIZE ASSOCIATED WITH VIRUS INFECTION

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BARLEY stripe mosaic virus (BSMV) is an RNA virus (ATEBEKOV and NOVIKOV 1966; R. I. HAMILTON and D. J. GUMPF, personal communication). Since viruses replicate and occasionally undergo mutation, RNA is presumably the carrier of the genetic information in this group. The question naturally arises: Is there any interaction between the genetic information of the virus (RNA) and the genetic information of the host (DNA)? Virus-infected plants often exhibit symptoms which simulate known chlorophyll mutant types. Unless the virus is seed-transmitted, however, these symptoms do not reappear in subsequent generations. What appear to be true genetic effects have been reported by SPRAGUE *et al.* (1963). One such effect occurs rather frequently and has been designated aberrant ratio (AR).

Several types of locus related distortions have previously been reported in maize. One of these includes the gametophyte factors (EMERSON 1934). Typically these exhibit normal female transmission and distorted male transmission, the degree of distortion being related to the amount of crossing over between the gametophyte and marker loci. Chromosomal deficiencies may also lead to aberrant ratios for marker genes (STADLER 1933). Male transmission of the deficient chromosome is usually low. Female transmission may be nearly normal or deficient, depending upon the extent of chromatin loss. An extra segment of chromatin attached to chromosome 10, designated as abnormal 10, may lead to distorted ratios for genes close to the centromere on chromosome heterozygous for heterochromatic knobs (RHOADES and DEMPSEY 1966) through preferential recovery of the knobbed chromatids in the basal megaspores. In such cases female transmission may exhibit varying degrees of distortion dependent on the percent crossing over between the marker and the centromere.

None of these types of aberrations parallels the results obtained with AR. AR is characterized by essentially equivalent distortion of both male and female transmission. Neither male nor female sterility have been observed and this suggests that deficiencies are not an important causal factor in AR. Similarity of male and female transmission indicates the absence of gametophyte factor or abnormal-10 types of distortion. This paper presents some of the pertinent data and perplexing problems relating to AR.

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MATERIALS AND METHODS

The Argentina mild strain of the barley stripe mosaic virus (BSMV) (McKINNEY and GREELEY 1965) and strain "B" of the sugarcane mosaic virus (SUMMERS *et al.* 1948) were used. Since no genetic effects have been observed with sugarcane mosaic virus, no further mention will be made of the studies with it.

A multiple marked dominant stock A_1 *Su Pr Wx* (A_1-a_1, presence or absence of aleurone color; *Su-su*, starchy or sugary seeds; *Pr-pr*, purple or red aleurone color; *Wx-wx*, starch staining blue or red with iodine) was found to be susceptible to BSMV and has been used as the male parent in treatment and control crosses. In the male stock, the degree of systemic invasion by the virus differs among plants when inoculated in the seedling stage. With a high degree of invasion, the plant produces little or no pollen. Inoculation assays have failed to reveal active virus in the pollen or the seed from the infected plants, or in the healthy plants from this seed. Serological tests conducted by HOWARD A. SCOTT also failed to reveal virus in pollen from the infected plants. Latent strains occur in some cultures of the BSMV (McKINNEY and GREELEY 1965), but a phase comparable to the temperate phase found in certain bacteriophages, has not been recognized in any host-virus system in the seed plants.

A multiple recessive (a_1 *su pr wx*) stock was used in the female parent. This strain is highly resistant to BSMV; in rare instances, from one to three small yellow markings have appeared in from one to three leaves of an inoculated plant. Such leaves emerged from 9 to 23 days after inoculation; virus was transferred from the yellow markings, but could not be detected in transfers made from normal leaf tissue to Atlas barley which is a very sensitive test plant.

The frequency of AR in treated cultures is approximately 1:200. This estimate may involve some degree of heterogeneity. Virus-infected plants exhibit clear symptoms on the lower leaves. As the plant develops, however, symptom expression on the younger leaves may be slight or absent. Virus transmission from healthy appearing leaves usually has been negative. Infected plants exhibiting typical symptoms on the top leaf were used exclusively in the studies reported here. Virus assays were not routinely made from tassel branches, and therefore some of the plants used as males may have produced pollen which had not been exposed to the virus.

It should be emphasized that virus infection was limited to the male parent of the original cross. Virus symptoms have not been observed in F_1 or subsequent generations of the material reported here.

Aberrant ratio has been found for the marker genes *A*, *Su* and *Pr*. No cases have been demonstrated for the waxy locus. This may be an artifact as *wx* can be detected visually only in the *a Su* seed class.

RESULTS

Several thousand F_2 ears (control and treated) from the cross A_1 *Su Pr Wx* × a_1 *su pr wx* have been examined for deviations from expectation for each of the marker genes. When deviations were judged to be significant, the ear was shelled and separations were made. If deviations approximated twice the standard error, progeny tests were made. The dominant phenotype was planted in one row or series of rows and the recessive phenotype in adjacent rows. Paired plant reciprocals were made and the resulting kernels classified. Approximately 100 testcross progenies have been examined to verify the significance of these observed deviations. Roughly one third of these were obtained from control populations.

A typical set of control data involving an untreated A_1 allele (never exposed to virus) is presented in Table 1. The four aberrant F_2 ears, for which testcross data are presented, exhibited percentages of the a_1 phenotype ranging from 29.6 to 39.9. The testcross segregations are possibly somewhat more erratic than normal, but there is no indication of a consistent departure from expectation

TABLE 1

Individual ear records of male and female transmission of the A *and* a *alleles derived from a control stock*

Culture and plant number	%*a* in parent F_2 ear	Female transmission			Male transmission		
		A	*a*	%*a*	*A*	*a*	%*a*
64: 865–2 × 866–4	29.6	41	54	56.8	155	171	52.4
865–4 × 866–5		250	307	55.1	181	172	48.7
865–7 × 866–6		227	242	51.6	221	194	46.7
64: 869–1 × 870–2	39.9	193	184	48.8	196	219	52.8
869–5 × 870–4		133	185	58.2	229	267	53.8
869–8 × 870–1		192	192	50.0	119	180	60.2
64: 890–1 × 891–7	32.6	193	234	54.8	201	209	51.0
890–2 × 891–1		49	48	49.5	83	79	48.8
890–3 × 891–2		163	172	51.3	153	185	54.7
64: 924–2 × 925–2	30.2	243	246	50.3	181	255	58.5
924–3 × 925–5		194	203	51.1	222	178	44.5
924–6 × 925–6		244	197	44.7	231	178	43.5

affecting both male and female transmission. In over 2000 control F_2 ears examined and some 30 deviants subjected to test no cases of AR have been established.

Over 50 cases of presumed AR have been found in the F_2 cultures arising from virus infected P_1 males. Over 25 of these have been verified by plant reciprocal tests and a number of additional potential cases are still under test. The lower limit of AR merges into normal transmission. Population size determines whether significance can be established in such cases. Little work has been done on cases where distortion is less than 60:40. The greatest distortion observed has been approximately 95:5.

The data to be presented from treated cultures were chosen to illustrate the

TABLE 2

Individual ear records of male and female transmission of the A *and* a *alleles derived from an AR stock*

Culture and plant number	Female transmission			Male transmission		
	A	*a*	%*a*	*A*	*a*	%*a*
64: 748–1 × 749–5	117	138	54.1	113	180	61.4
748–2 × 749–18	230	218	48.7	...	...	...
748–3 × 749–12	214	193	47.4	210	198	48.5
748–4 × 749–15	212	284	57.3	260	236	47.6
748–5 × 749–13	54	99	64.7	119	237	66.6
748–11 × 749–2	156	209	57.3	160	164	50.6
748–14 × 749–7	232	229	49.7	159	190	54.4
748–15 × 749–6	58	112	65.9	94	238	71.7
748–16 × 749–11	155	257	62.4	102	106	51.0
748–17 × 749–17	184	215	53.9	103	183	64.0
748–19 × 749–16	121	339	73.7	86	328	79.2

TABLE 3

Individual ear records of male and female transmission of the Su *and* su *alleles, derived from an AR stock*

Culture and plant number	Female transmission			Male transmission		
	Su	*su*	%*su*	*Su*	*su*	%*su*
64: 748–1 × 749–5	135	120	47.1	154	139	47.4
748–3 × 749–12	222	185	45.5	196	212	52.0
748–4 × 749–15	231	265	53.4	234	262	52.8
748–5 × 749–13	79	74	48.4	166	190	53.4
748–11 × 749–2	156	180	53.6	164	160	49.4
748–14 × 749–7	219	132	51.4	165	184	52.7
748–15 × 749–6	84	86	50.6	178	154	46.4
748–16 × 749–11	212	200	48.5	104	104	50.0
748–17 × 749–17	219	180	45.1	145	141	49.3

different types of breeding behavior which have been encountered. As a result of this selection, these data do not provide critical evidence for either the constancy of degree of aberration among plant reciprocals within a given line of descent, or of the frequency with which one type of AR changes to an alternative form or to normal.

Aberrant ratio is locus-related rather than nonspecific. This is illustrated by the data in Tables 2 and 3. The parent culture from which 64:748 and 749 were derived exhibited 61.5% of *a* seeds where 50% were expected. The data in Table 2 represent an additional generation of test of this distortion. Of the 11 pairs of ears, three (748–2, 3, and 14) exhibited normal segregation; four (748–4, 11, 16 and 17) exhibited significant distortion in one direction only; four exhibited a degree of distortion characteristic of the parent; and one (748–19) exhibited a more extreme form of distortion. The behavior of pairs, 748–4, 11, 16 and 17, is not typical. These results suggest that AR is not completely stable and may undergo a change in degree as well as a reversion to normal. The data in Table 3 represent the same group of ears classified for starchy (*Su*) and sugary (*su*). In each case segregation is normal. The results presented in Table 3 are typical of control tests and for the nonaffected loci within AR cases. No instance has yet been found in which there is an AR effect for two loci within a single reciprocal pair. One case has been found, however, in which AR effects have been observed for two loci within the advanced generation progeny derived from a single F_2 ear.

One important feature of AR is the similarity of male and female transmission rates. In only rare exceptions do male and female transmission rates differ significantly from a mean rate calculated from the combined sample.

The results from a second case of AR, characterized by a deficiency of recessive *pr*, are presented in Table 4. In the previous generation this case exhibited 30.9% of *pr* kernels where 50% would normally be expected. In the data presented in Table 4, three (793–4, 8 and 9) of the 15 sets produced normal or near normal ratios. The remaining sets exhibit varying degrees of distortion, ranging from a mean of 37.5 to 22.6. Progeny tests of the two pairs marked by footnotes were conducted.

TABLE 4

Individual ear records of male and female transmission of Pr *and* pr *alleles derived from an AR stock*

Culture and plant number	Female transmission			Male transmission		
	Pr	*pr*	%*pr*	*Pr*	*pr*	%*pr*
63: 793–1 × 794–9	93	35	27.3	145	94	39.3
–3 × 794–5	158	62	28.2	195	100	33.9
–4 × 794–10	89	71	44.4	99	106	51.7
–5 × 794–15	233	118	33.6	111	88	44.2
–6 × 794–8	32	15	31.9	128	56	30.4
–7 × 794–2†	322	110	25.5	268	62	18.8
–8 × 794–12*	115	129	52.9	146	149	50.5
–9 × 794–13	164	127	43.6	177	154	46.5
–12 × 794–19	172	40	18.9	. . .	. . .	. . .
–13 × 794–3	193	47	19.6	176	51	22.5
–15 × 794–5	162	52	24.3	. . .	. . .	. . .
–16 × 794–16	40	59	59.6	138	82	37.3
–17 × 794–14	150	33	18.0	. . .	. . .	. . .
–18 × 794–18	204	113	35.8	210	128	37.9
–19 × 794–11	157	48	23.4	. . .	. . .	. . .

* Progeny test presented in Table 5.
† Progeny test presented in Table 6.

Data from a progeny test of 63:793–8 × 794–12 (* in Table 4) are recorded in Table 5. With one exception, all progeny pairs exhibited normal or near normal ratios. The exception, 792–11 × 793–9, exhibited a significant excess of *pr*. Thus, the progeny of a pair exhibiting normal segregation may give rise to AR segregations.

TABLE 5

Individual ear records for male and female transmission of the Pr *and* pr *alleles derived, in previous generations, from an AR stock. (Progeny test of culture 63:793–8 × 794–12 tested in Table 4)*

Culture and plant number	Female transmission			Male transmission		
	Pr	*pr*	%*pr*	*Pr*	*pr*	%*pr*
64: 792–1 × 793–4	195	197	50.3	177	158	47.2
–3 × 793–7	154	135	46.7	120	132	52.4
–4 × 793–8	141	120	46.0	121	128	51.4
–5 × 793–12	147	145	49.7	151	157	51.0
–6 × 793–13	228	125	35.4	53	69	56.6
–7 × 793–1	159	163	50.6	213	207	49.3
–8 × 793–2	186	174	48.3	102	101	49.8
–11 × 793–9	91	217	70.5	68	194	74.0
–12 × 793–3	140	152	52.1	160	128	44.4
–13 × 793–17	107	127	54.3	177	155	46.7
–14 × 793–5	192	178	48.1	163	189	52.7
–16 × 793–19	93	103	52.6	148	157	51.4
–17 × 793–11	186	132	41.5	165	131	44.3

TABLE 6

Individual ear records for male and female transmission of the Pr *and* pr *alleles derived, in previous generations, from an AR stock. (Progeny test of culture 63:793–7 × 794–2 listed in Table 4)*

Culture and plant number	Female transmission			Male transmission		
	Pr	*pr*	%*pr*	*Pr*	*pr*	%*pr*
64: 788–1 × 789–15	199	115	36.6	128	87	40.5
–2 × 789–5	279	123	30.6	314	119	27.5
–3 × 789–1	170	86	33.6	222	117	34.5
–4 × 789–2	250	96	27.7	234	76	24.5
–5 × 789–13	178	62	25.8	258	116	31.0
–6 × 789–11	179	114	38.9	215	87	28.8
–7 × 789–6	103	211	67.2	163	193	54.2
–8 × 789–3	214	139	39.4	208	129	38.3
–11 × 789–4	266	134	33.5	213	108	33.6
–12 × 789–5	301	104	25.7	178	96	35.0
–13 × 789–16	206	128	38.3	303	139	31.4
–15 × 789–12	78	239	75.4	65	160	71.1
–16 × 789–14	145	77	34.7	271	160	37.1
–18 × 789–20	174	114	39.6	263	135	33.9
–19 × 789–18	263	102	27.9	237	72	23.3

63:793–7 × 794–2 († in Table 4) produced a marked excess of *Pr* kernels. Data from the progeny test of this pair are presented in Table 6. Thirteen of the 15 pairs exhibit a continued deficiency of *pr*. In the two remaining pairs (788–7

TABLE 7

Individual ear records for male and female transmission of the A *and* a *alleles, derived from an AR stock*

Culture and plant number	Female transmission			Male transmission		
	A	*a*	%*a*	*A*	*a*	%*a*
63: 779–1 × 780–1	207	357	63.3	160	270	62.8
–2 × 780–3	193	179	48.1	144	138	48.9
–3 × 780–6	55	78	58.6	133	149	52.8
–4 × 780–2	219	228	51.0	190	182	48.9
–5 × 780–4*	187	194	50.9	202	213	51.3
–6 × 780–9	117	285	70.9	26	81	75.7
–7 × 780–10	236	250	51.4	183	190	50.9
–12 × 780–12†	105	254	70.8	70	196	73.7
–13 × 780–17	220	170	43.6	263	216	45.2
–14 × 780–16	107	204	65.6	189	293	60.8
–15 × 780–15	175	276	61.2	132	229	63.4
–16 × 780–13	70	105	60.0	90	148	62.2
–17 × 780–14	164	160	49.4	128	119	48.2
–18 × 780–18	176	300	63.0	106	188	63.9
–19 × 780–20	189	295	61.0	88	164	65.1

* Progeny tests presented in Table 8.
† Progeny tests presented in Table 9.

TABLE 8

Individual ear records for male and female transmission of the A *and* a *alleles, derived from an AR stock. (Progeny test of culture 63:779–5 × 780–4 listed in Table 7)*

Culture and plant number	Female transmission			Male transmission		
	A	*a*	%*a*	*A*	*a*	%*a*
64: 762–2 × 763–4	129	146	53.1	164	182	52.6
–5 × 763–1	204	211	50.8	202	263	56.6
–6 × 763–13	37	32	46.4	180	169	48.4
–8 × 763–2	248	241	49.3	11	11	50.0
–13 × 763–3	144	158	52.3	232	246	51.5
–16 × 763–7	284	258	47.6	280	289	50.8
–18 × 763–15	282	242	46.2	201	195	49.2
–19 × 763–11	196	231	54.1	208	228	52.3
–20 × 763–17	142	138	49.3	193	182	48.5
64: 764–1 × 765–3	53	61	53.5	166	173	51.0
–2 × 765–5	94	86	47.8	69	65	48.5
–4 × 765–4	116	115	49.8	119	159	57.2

and 15) distortion is also apparent but in a reverse direction producing an excess of *pr*.

An additional case of AR involved the *A* locus. Progeny test data are presented in Table 7. The parent culture exhibited 62.9% of *a* seeds where 50% were expected. Nine of 15 progeny pairs exhibited an excess of the *a* phenotype. The remaining six pairs (799–2, 4, 5, 7, 13 and 17) were characterized by near normal segregations. Additional progeny tests were made of cultures exhibiting normal or extreme distortion.

Progeny tests of 63:779–5 × 780–4 (* in Table 7) are presented in Table 8. Each of the progeny pairs in this test exhibited normal or near normal segregation. Progeny tests of 63:779–12 × 780–12 († in Table 7) are presented in Table 9.

TABLE 9

Individual ear records for male and female transmission of the A *and* a *alleles, derived from an AR stock. (Progeny test of cultures 63:779–12 × 780–12 listed in Table 7)*

Culture and plant number	Female transmission			Male transmission		
	A	*a*	%*a*	*A*	*a*	%*a*
64: 766–1 × 767–4	115	322	73.7	112	312	73.6
–2 × 767–2	127	312	71.1	114	388	77.3
–3 × 767–3	144	197	57.8	163	275	62.8
–5 × 767–11	103	374	78.4	64	237	78.7
–6 × 767–14	98	333	77.3	75	274	78.5
–7 × 767–15	163	250	60.5	195	244	55.6
–11 × 767–1	174	284	62.0	150	271	64.4
–15 × 767–13	143	151	51.4	209	218	51.1
–16 × 767–12	77	218	73.9	79	225	74.0
–20 × 767–16	68	210	75.5	72	201	73.6
–21 × 767–19	60	183	75.3	76	238	75.8

TABLE 10

Individual ear records for male and female transmission of the A *and* a *alleles, in a cross of* AA × *AR* aa

Culture and plant number	Female transmission			Male transmission		
	A	*a*	%*a*	*A*	*a*	%*a*
65: 937–1 × 938–1	109	113	50.9	47	57	54.8
–2 × 938–5	110	108	49.5	...	...	...
–4 × 938–4	221	225	50.4	91	96	51.3
–5 × 938–6	105	165	61.1	121	226	65.1
–6 × 938–8	135	175	56.4	104	106	50.5
–7 × 938–7	187	314	62.7	...	...	...
–8 × 938–2	80	151	65.4	...	...	...
–9 × 938–3	219	221	50.2	141	122	46.4
–10 × 938–9	129	251	66.0	121	266	68.7
–3 × 938–5	122	228	65.1	...	...	...
65: 935–2 × 936–2	197	170	46.3	...	...	...
–3 × 936–5	125	196	61.0	95	208	68.6
–4 × 936–1	108	282	72.3	91	189	67.5
–5 × 936–3	159	144	47.5	...	...	...
–7 × 936–8	69	110	61.5	...	...	...

Ten of the 11 pairs produced results roughly comparable to the parental type. The one exception, 64:766–15 × 767–12, exhibited a normal segregation.

The AR effects illustrated in the preceding tables have involved dominant alleles which trace back directly to a virus infected plant. Transmission of the AR effect however, can be obtained from derived recessives. Plants of *a* phenotype derived from a high *A* distortion stock were crossed to a healthy (never infected) *A A* stock and backcross segregation observed. The data are presented in Table 10. Some reciprocal combinations are lacking, but it is apparent that the progenies may be divided into two distinct groups. In the first group segregation is normal whether measured by either male or female transmission. The second group exhibits AR with an excess of the a phenotype. Thus, after AR becomes established it may, under proper conditions, be transmitted by either a treated allele (direct lineal descendent) or by an untreated allele (never directly exposed to the virus).

DISCUSSION

McCLINTOCK (1961) has reviewed some of the gene control mechanisms in maize, particularly *Spm*, and shown that the observed effects are consistent with the assumption of "operator" and "regulator" genes which influence the action of structural genes. JACOB *et al.* (1960) have suggested that the mechanism of control in maize may be comparable to episomes in bacteria.

The analysis of AR has not progressed sufficiently to establish the existence of an operator-regulator mechanism, or of an episome-like situation. The data currently available, however, are consistent with the assumption of an episome-like particle. This hypothetical particle is in some way related to virus infection, as no case of AR has been verified in the progeny of control crosses. It may be

assumed either that the particle is a normal extrachromosomal constituent of the maize cell, or that it represents some portion of the viral material. In either case the particle is without detectable effect until it comes under genetic control and is associated in some manner with a marker gene.

As mentioned earlier, AR may be exhibited in three phases (relative to the frequency of the dominant allele): high, low or normal. Each of these phases may change to either of the other two phases. The degree of AR for any locus may also vary. The extremes observed range from approximately 95:5 to 60:40. A possible explanation is that this variation is related to spatial distance between AR and the marker structural gene.

The data in Tables 2 through 9 represent cases in which the dominant allele was derived, three to six generations previously from a virus-infected male. The distortion observed, therefore, could possibly be attributed to some direct effect of the virus on the dominant allele. In Table 10, however, neither the A_1 nor the a_1 allele had been directly exposed to any viral effect. The transmission of AR in this case appears to require the assumption of some chromosomal element which can be transferred to an untreated chromosome by crossing over.

The change from high (excess of the dominant phenotype) to low can also be interpreted as arising from crossing over. Under this interpretation AR *A/–a* could give rise to *–A/AR a*. The data (e.g., Table 6, 788–15 × 789–12) are consistent with such an interpretation. When changes occur, from high to low, or low to high, the degree of distortion tends to remain similar but with the direction reversed. Exceptions to this correspondence have been noted, however.

A crossover mechanism can also account for the loss of AR. Loss could arise from either one of two conditions: loss of, or homozygosity for, the particle. Crossing over does not provide an equally apparent explanation for the origin of high or low aberration in the progeny tests of "normal" stocks (e.g., Table 6, 792–11 × 793–9) without additional assumptions. If normality of transmission is assumed to be due to homozygosity for the particle, then loss of one of the two particles would give rise to either high or low distortion, depending upon which allele is associated with the remaining particle. If normality of transmission is assumed to be due to the absence of the particle from the proximity of the structural marker site, then the reappearance of distortion would require the restoration of the lost particle. Data currently available will not distinguish between these and other possibilities. It is known, however, that distortion may be exhibited at more than one locus within a given family. This suggests either the presence of more than one particle or the ability to move to various sites within the chromosome complement.

The data in Table 10 indicate that the particle-bearing chromosome is the advantaged member of the pair. The high *A* AR parent stock would have been AR *A/–a* in genotype. The *a a* derivative could have carried AR through crossing over and would have been *–a*/AR *a* in constitution. Test crosses of hybrids with an untreated *A A* stock gave rise to "high a" distortion.

Indirect evidence suggests that the initiation of an AR effect occurs at or near meiosis. In many of the virus infected male plants, infection is systemic for the

lower leaves but with the upper leaves free of mosaic symptoms. It has been impossible to transmit virus with any degree of regularity from such normal-appearing leaves. No AR or other abnormal genetic effects have been observed when such plants are used in crosses.

The assumption of an episome-like particle provides a mechanistic bases which is consistent with data now available. This assumption, however, does not provide any direct clues as to how regulation is achieved.

SUMMARY

Aberrant ratio (AR) has been observed in F_2 and successive backcross generations in which the P_1 males were infected with barley stripe mosaic virus. The aberration is essentially equal in male and female transmission. Stocks exhibiting an excess of the dominant phenotype, "high A," may change to low or normal. Either of these types in turn may change to either of the two alternative conditions. AR has been observed for three of the four marker genes studied.—AR is tentatively interpreted as a result of an extrachromosomal particle which becomes attached to a chromosome at or near meiosis. Shifts from one type of transmission to another may be accounted for by a crossover mechanism.

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