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## Partial Production of A Normal Morphological Phenocopy in the *Zea Mays* L. Mutant, *Ramosa-1*

ALAN R. ORR<sup>1</sup>

**Abstract.** *ZEA MAYS* L. Mutant plants carrying the *ramosa-1* (*ra-1*) gene which effects inflorescence development, were treated with aqueous solutions of several growth substances. Some plants treated with TIBA exhibited tassels with a reduced *ra-1* phenotype. Several plants treated with GA produced ears showing little branching. Discussion is centered on evidence which supports the idea that hormone metabolism is part of a genetic control system governing maize inflorescence development at various stages in the pathway.

Although the development of numerous flowers and inflorescences have been described in considerable morphological detail, very little information is currently available concerning the biochemical and physiological aspects of the growth and differentiation of floral organs. To fully understand the morphogenesis of floral development we must be concerned with the relationships between form, metabolism and hereditary information.

Apical meristems are generally in a sustained embryonic condition. Morphogenesis continues in an active shoot apex thus providing an excellent experimental system for the study of plant development. A special problem which has attracted a number of investigators is the transformation of a vegetative shoot apex into a reproductive shoot apex. Through various experimental techniques, many of which disturb the normal metabolic activities of apical meristems—for example, by surgical operations (Snow and Snow, 1947; Wardlaw, 1959, 1963; Ball, 1960, 1963) by x-ray treatments (Stein and Steffensen, 1959) by tissue culture (Petru and Retovsky, 1957); and by application of natural and synthetic growth substances (Phinney, 1956, 1961; Nickerson, 1960; Anderson, 1963)—results indicate that many cells in an embryonic condition can be deflected from their normal destiny.

Another useful technique has been to substitute a mutant allele for its normal counterpart. Suitability of mutant genes for experimental studies of development was aptly explained by Phinney and West (1960). Experimental alteration, by gene substitution, of events leading to normal development of *Zea mays* L. inflorescence has led to the idea that an indispensable relay system of gene action must occur at specific stages in maize inflorescence development (See Postlethwait and Nelson's Fig. 1, 1964). A similar hypothesis was also proposed by Heslop-Harrison (1961) on the basis of studies involving inflorescence development of *Zea mays*.

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A previous portion of this study (Orr, 1968) was concerned with development of the inflorescence of the maize mutant, *Fasciated Ear (Fa)* following treatment with certain growth substances. Mutant (*Fa*) plants treated with a -naphthaleneacetic acid (NAA) and indoleacetic acid (IAA) responded in such a manner that normal appearing ears and tassels developed in place of the expected mutant form. A normal hormone balance may have been altered in the floral shoot apex of maize by the presence of the *Fa* gene. In other words, in the normal morphological development of a maize inflorescence it appears that an additional input of genetic information is required in the shoot apex to make the transition from vegetative growth to floral development.

The present study represent an extension of the investigation into the normal development of maize inflorescence to include the mutant gene *ramosa-1 (ra-1)*.

#### MATERIALS AND METHODS

Mutant *ramosa-1 (ra-1)* *Zea mays* L. seeds were planted and grown under a 16 hour photoperiod. Treatments of various growth substances (Table 1) were initiated to 13 day-old seedlings and maintained throughout the early development of both the tassel and the ear. Growth substances were applied every three days above the growing point by placing a 0.2 ml molar solution into the central leaf whorl. Control seedlings were simultaneously treated with 0.2 ml of an aqueous solution lacking the growth substances. Fifteen tassels per treatment were collected and the data summarized by the Duncan analysis of variance (Steel and Torrie, 1960). Representative samples of ears were collected from each treatment plot and analyzed for growth effects on phenotypic expression of the *ra-1* gene.

Table 1. Molar (M) Concentrations of Growth Substances Administered to Individual Plants

NAA	IAA	GA	MH	TIBA
.5 x 10 <sup>-4</sup> M	10 <sup>-4</sup> M	10 <sup>-4</sup> M	10 <sup>-4</sup> M	10 <sup>-4</sup> M
.5 x 10 <sup>-3</sup> M	10 <sup>-3</sup> M	10 <sup>-3</sup> M	10 <sup>-3</sup> M	10 <sup>-3</sup> M
.5 x 10 <sup>-2</sup> M	10 <sup>-2</sup> M	10 <sup>-2</sup> M	10 <sup>-2</sup> M	10 <sup>-2</sup> M

#### RESULTS

*Ramosa-1 (ra-1)*, a branched-ear mutant of maize was first reported by Gernert (1912). The gross morphology of the ear and tassel was described by Kempton (1921). Both ear and tassel are profusely branched (Fig. 1). The branches apparently arise

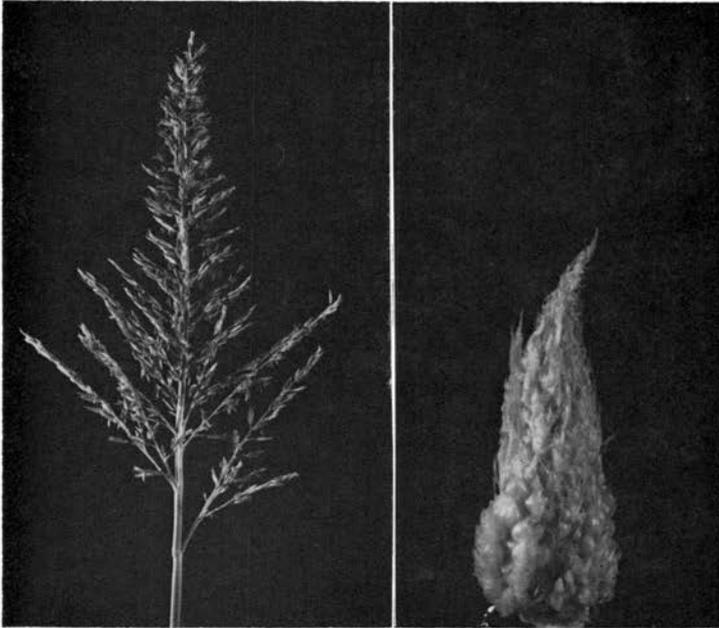


Figure 1. Mutant Forms of the ramosa-1 inflorescence. A. tassel. B. Ear.

as simple alternations in the developmental pathway. The shoot apex undergoes a normal switch from vegetative to inflorescence producing form and proceeds to the next morphological step, the production of branch primordia (Postlethwait and Nelson, 1964). The following morphological step, the formation of two spikelet primordia from the branch primordia, is blocked by the presence of the ra-1 gene. This results in the branch primordia continuing its activity as a single axis; the morphological result is a tapered branched inflorescence appearing much like a christmas tree.

*Tassel response.* Several morphological characteristics of ra-1 tassels were observed for their possible modifications from treatments with the growth substances (Table 2). Means and analysis of variance are given. All treatments proved to be ineffective in reducing the number of primary branches which characterizes the appearance of the ra-1 tassel. Every treatment slightly increased branching. NAA ( $0.5 \times 10^{-4}$  M) and GA ( $10^{-4}$  M) significantly increased the number of branches as compared to the control.

Table 2. Some Effects on  $\alpha$ -Naphthaleneacetic Acid (NAA), Maleic Hydrazide (MH), and Gibberellic Acid (GA) on the Tassel of *ramosa* ( $r_{a1}$ )<sup>a</sup>.

<u>Primary branch number</u>						
Cont.	GA $10^{-3}$	MH $10^{-4}$	NAA $.5 \times 10^{-3}$	MH $10^{-3}$	NAA $.5 \times 10^{-4}$	GA $10^{-4}$
24.93	25.71	30.86	32.36	34.33	44.94	51.59
a	a	a	a	a	b	b
<u>Peduncle (centimeter)</u>						
Cont.	MH $10^{-4}$	MH $10^{-3}$	NAA $.5 \times 10^{-3}$	NAA $.5 \times 10^{-4}$	GA $10^{-3}$	GA $10^{-4}$
7.64	9.10	10.57	12.23	12.71	23.32	25.50
a	ab	ab	b	b	c	c
<u>Tassel height (centimeter)</u>						
Cont.	MH $10^{-4}$	MH $10^{-3}$	NAA $.5 \times 10^{-3}$	NAA $.5 \times 10^{-4}$	GA $10^{-3}$	GA $10^{-4}$
10.28	12.73	13.13	15.50	16.39	16.62	19.15
a	ab	ab	bc	bc	bc	c
<u>Tassel branch area (centimeter)</u>						
Cont.	MH $10^{-4}$	MH $10^{-3}$	NAA $.5 \times 10^{-3}$	NAA $.5 \times 10^{-4}$	GA $10^{-3}$	GA $10^{-4}$
8.07	9.70	10.70	11.86	14.25	15.75	17.85
a	a	ab	bc	bcd	cd	d
<u>Male fertile spikelet number</u>						
GA $10^{-3}$	NAA $.5 \times 10^{-3}$	Cont.	MH $10^{-4}$	MH $10^{-3}$	GA $10^{-4}$	NAA $.5 \times 10^{-4}$
0.11	0.76	4.54	14.80	21.53	50.64	51.87
a	a	a	ab	abc	bc	c

<sup>a</sup>Within a row, means followed by different letters are significantly different at the 5% level by the Duncan (1955) analysis of variance test. Means followed by the same letter are not significantly different.

increase in the longitudinal growth of the central axis was observed with auxin treatments (Orr, 1968). Since the *ra-1* effect appears to be an abnormal stimulation towards elongation of the branch primordia axis, it was postulated that an auxin inhibitor might reduce the number of primary branches in the *ra-1* tassel. This idea was confirmed when *ra-1* plants were treated with 2, 3, 5—triiodobenzoic acid (TIBA). TIBA ( $10^{-3}$  and  $10^{-4}$  M) significantly (5% level) reduced the number of primary branches in the *ra-1* tassel.

*Ear response.* The typical phenotype of the *ra-1* ear was partially altered on plants treated with GA ( $10^{-3}$  and  $10^{-2}$  M). The number and length of initiated branches was greatly reduced (Fig. 2). Any branching that did occur was restricted to the basal portion of the ear (Fig. 2). All other treatments appeared to have little or no effect on the expression of the *ra-1* phenotype. In fact, it is interesting that TIBA which reduced branching in the tassel did not do so in the ear.

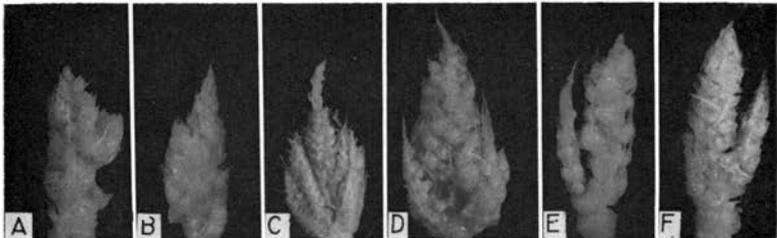


Figure 2. Effects of gibberellic acid on the *ramosa-1* ear. A-D. G. A. ( $10^{-3}$ M). E-F. G. A. ( $10^{-2}$ M).

## DISCUSSION

Inflorescence development in maize is produced by a series of predictable morphological events (Bonnett, 1940; Kiesselbach, 1949). Nevertheless, it is readily modified by a variety of environmental influences. Heslop-Harrison (1961) showed that photoperiod, temperature or auxin will affect a "switch" in the developmental pathway of the inflorescence at the change from vegetative to floral initiation, spikelet initiation, stamen and carpel development. Postlethwait and Nelson's (1964) studies with ear and tassel mutants of maize showed that additional "switch points" appeared to be involved in the normal sequence of morphological events. The later investigators concluded that morphological changes involved in inflorescence ontogeny are genetically programmed at specific points leading to a normal phenotype. They proposed a

developmental model which emphasized that morphogenesis of a maize inflorescence may be reflected through a series of sequential gene activations.

Heslop-Harrison (1963) earlier had proposed a more general model for a differentiating floral apex based upon control through a relay system of gene activation-deactivation. Heslop-Harrison argued that such a control mechanism might be augmented by a native hormone metabolism. Nickerson (1960 a and 1960 b) has shown that maize plants carrying inflorescence modifying mutant genes can become essentially normal in phenotype following hormone treatment. The Teopod (Tp) gene apparently causes the almost non-existent bract subtending each pair of spikelets to grow and form a leaf sheath homologue. Treatment with (GA) causes Tp carrying plants to produce inflorescence essentially normal in phenotype. Morphological effects of mutant genes *Vestigial glume* (Vg), *Tunicate* (Tu) and *ramosa-1* (ra-1) on inflorescence development could be partially modified by treatment with GA. Nickerson reports that plants carrying the ra-1 gene produced phenotypically normal tassels. In the present study GA failed to suppress branching in the tassel which typifies the ra-1 gene. Furthermore, Nickerson reports that ears of ra-1 plants treated with GA remained "characteristically multibranched structures". It is interesting that in the present study the phenotypic expression of some ra-1 ears was partially reduced with GA treatments. Postlethwait and Nelson (1964) reported that occasionally ra-1 plants may produce characteristic ra-1 tassels while ears of the same plant have little or no branching. During the course of this study all control plants produced ears and tassels exhibiting the ra-1 phenotype.

Anderson (1963) treated maize plants carrying the mutant gene *branched-silkless* (bd) with GA. The bd gene normally blocks the pathway at a point where the spikelet meristem gives rise to two floret meristems. However, in the presence of GA treatments a partial reduction in the typical bd phenotype occurs. The spikelet meristem of a maize inflorescence which typically divides into two floret meristems has been reported to undergo a typical elongation resulting from GA treatments (Orr, 1968).

It is possible that the morphogenetic effects of GA on inflorescence development depends in part on an auxin supply. The maize gene *lazy* (la), known to produce quantities of auxin (Van Overbeek, 1938) responds strongly to GA treatment (Nickerson, 1960). Several authors (Brian and Hemming, 1958; Galston and Warburg, 1959; Galston and McCune, 1961) have postulated a positive GA-auxin relationship in a variety of plants and plant parts. The possibility that some quantity of auxin plays a role in the switch from a branch primordia to two spikelet primordia in normal inflores-

cence development is suggested by the response of the ra-1 gene to auxin and antiauxin treatment. Whereas NAA and IAA treatments failed to reduce the ra-1 phenotype, TIBA treatments resulted in a partial reduction of the typical ra-1 tassel as reported in this study. The response obtained from TIBA treatments suggests the presence of auxin in the shoot apex. Perhaps in some manner GA treatment also helped to partially restore a proper auxin concentration in the ra-1 inflorescence.

Evidence presented above and elsewhere (Orr, 1968) suggests support for the hypothesis of Postlethwait and Nelson (1964) and Heslop-Harrison (1969) that the whole process of inflorescence ontogeny may be viewed in terms of a series of morphological changes each mediated by additional genetic information necessary to normal development. Obviously, it is still the task to demonstrate this experimentally. Nevertheless, it has been shown in this study and others that hormone metabolism may be a part of the mechanism governing maize inflorescence development.

## LITERATURE CITED

- ANDERSON, C. E. 1963. Ph.D. Thesis, Purdue University.  
 BALL, E. 1960. *Phytomorph.* 14:377-396.  
 ————. 1963. Scholar's Library, N. Y.  
 BONNETT, O. T. 1940. *Jour. Agric. Res.* 60:25-37.  
 BRIAN, P. W., & H. G. HEMMING. 1958. *Ann. Bot. N. S.* 22:1-17.  
 GALSTON, A. W. & H. WARBURG. 1959. *Plant Physiol.* 34:16-32.  
 ———— & D. C. McCUNE. 1961. In R. M. Klein (ed.) *Plant Growth Regulation*. The Iowa State Univ. Press, Ames, Iowa.  
 GERNERT, B. 1912. 46:616-622.  
 HESLOP-HARRISON, J. 1961. *Proc. Linn. Soc. Lond.* 172:108-123.  
 HESSLER, R. H. 1963. M. S. Thesis. Purdue University.  
 KEMPTON, J. H. 1921. U.S.D.A. Bull. 971.  
 KIESSELBACH, T. A. 1949. *Univ. Nebraska Col. Agric. Bull.* 161.  
 NICKERSON, N. H. 1960a. *Ann. Missouri Bot. Gard.* 47:243-261.  
 ————. 1960b. *Amer. Jour. Bot.* 47:809-815.  
 ————. 1960c. *Amer. Midl. Nat.*  
 ———— & T. N. EMBLER. 1960. *Ann. Missouri Bot. Gard.* 47:227-242.  
 ORR, A. R. 1968. *Iowa Acad. Sc.* 75:69-77.  
 PETRO, E. & R. RETOVSKY. 1957. *Folia Biol.* 3:319-320.  
 PHINNEY, B. O. 1956. *Proc. Natl. Acad. Sci.* 42:185-189.  
 ———— & C. A. WEST. 1960. *Ann. Rev. Plant Physiol.* 11:411-436.  
 ————. 1961. In R. M. Klein (ed) *Plant Growth Regulation*. Iowa State Univ. Press.  
 POSTLETHWAIT, S. N. & O. E. NELSON. 1964. *Amer. Jour. Bot.* 51:238-243.  
 SNOW, M. & R. SNOW. 1947. *New Phytol.* 46:5-19.

- STEBBINS, L. G. 1964. *Amer. Jour. Bot.* 51:220-230.  
\_\_\_\_\_. 1965. *Amer. Sci.* 53:104-126.
- STEEL, R. G. D. & J. H. TORRIE. 1960. McGraw-Hill Book Co., Inc., New York. 481 p.
- STEIN, O. L. & D. M. STEFFENSEN. 1959. *Zeitsch Vererbungslehre* 90:483-502.
- VAN OVERBEEK, J. 1938. *Jour. Heredity.* 29:339-341.
- WARDLAW, C. W. 1959. *Jour. Linn. Soc. Lond.* 56:154-159.  
\_\_\_\_\_. 1963. *Nature.* 198:560-561.
- WEATHERWAX, P. 1917. *Bull. Torrey Bot. Club.* 44:483-496.