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Suppressor of sessile spikelets1 (*Sos1*): A DOMINANT MUTANT AFFECTING INFLORESCENCE DEVELOPMENT IN MAIZE[1]

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Suppressor of sessile spikelets1 (*Sos1*) is a dominant mutant of maize that blocks branching of the spikelet-pair primordium to form the sessile spikelet during ear development. As a result, *Sos1* mutant ears and tassels possess single spikelets as opposed to the normal condition of paired spikelets, one sessile and the other pedicellate. *Sos1* also causes a reduction in the number of tassel branches and the number of orthostichies (or cupule ranks) in the ear. The *sos1* genetic locus maps to the short arm of maize chromosome 4. The *Sos1* single spikelet phenotype appears similar to the single spikelet phenotype found in teosinte, the probable progenitor of maize. This similarity invites the hypothesis that *sos1* had a role in the evolution of maize from teosinte. However, genetic mapping data and a comparison of the developmental basis of the single spikelet condition in the *Sos1* mutant and teosinte demonstrate that their similar phenotypes result from distinct genetic-developmental mechanisms. These results indicate that *sos1* was not involved in the evolution of maize and caution against drawing conclusions of homology based solely on similar adult phenotypes.

Genetic and development analyses of mutations that alter the normal pattern of inflorescence development in maize (*Zea mays* L. ssp. *mays*) offer powerful tools for dissecting the complex processes involved in inflorescence morphogenesis, determining the time of gene action during morphogenesis, and understanding the interactions among the genes involved in morphogenesis (Postlethwaite and Nelson, 1964; Veit et al., 1991; Irish, Langdale, and Nelson, 1994). One requisite for achieving the full power of this form of genetic analysis of developmental processes is a collection of mutants for the genes involved in all steps of the developmental pathway. In this paper, we describe a genetic locus for maize, *suppressor of sessile spikelets1* (*sos1*), that controls an early step in maize inflorescence development, namely, the branching of the spikelet-pair primordium to form a sessile-pedicellate spikelet pair. Plants carrying the mutant allele (*Sos1-Ref*) exhibit single (sessile) spikelets as opposed to paired (sessile-pedicellate) spikelets that typify normal maize. *sos1* provides a new tool for investigating inflorescence development in maize and other grasses.

Our interest in *sos1* arose in part because of its potential role in the evolution of maize and teosinte (*Zea* spp.), both members of the grass tribe Andropogoneae. Spikelets of the members of this tribe are typically borne in pairs, one sessile and one pedicellate. This paired arrangement is found in both the ears and tassels of maize, although the highly compact nature of the maize ear makes it difficult, but not impossible, to distinguish the sessile and pedicellate spikelets since both are essentially sessile (Cutler, 1946). In teosinte (*Zea* spp.), the probable progenitor of maize, sessile-pedicellate spikelet pairs are found in the tassel; however, only single spikelets are produced in the teosinte ear. Thus, it has been inferred that, during the evolution of teosinte from other Andropogoneae, one of the spikelets in the ear was aborted to yield single spikelets, and that during the evolution of maize from teosinte, the aborted spikelet was restored, giving paired spikelets in the maize ear (Galinat, 1983, 1985). A locus like *sos1* seems an excellent candidate for a gene involved in these evolutionary steps, motivating us to ask whether *sos1* was one of the genes involved in the evolution of maize from teosinte.

MATERIALS AND METHODS

Seeds of the *Sos1-Ref* mutant were taken from an ear showing the mutant phenotype in the University of Wisconsin Herbarium. According to Dr. Hugh Iltis (former Director of the Herbarium), he obtained the ear from Dr. John Lonnquist (formerly of the Department of Agronomy, University of Wisconsin) who reported to him that the mutant arose spontaneously in a maize population. Seeds originally obtained from the herbarium specimen were selfed for several generations until a line (91-31) was obtained that was true-breeding for the single spikelet trait. This line also had deformed tassels with fewer and shorter branches than normal. Subsequently, line 91-31 was crossed to several maize lines to observe the inheritance of the single spikelet phenotype and to determine its chromosomal location and linkage to several genetic markers. Line 91-31 was crossed to: 1) the maize inbred W22; 2) W22-TGA (a W22 derivative carrying a segment of teosinte chromosome 4; Dorweiler et al., 1993); 3) a *lazy1-sugary1* tester line obtained from the Maize Genetics Cooperative Stock Center (Urbana, IL); and 4) a teosinte (*Z. mays* ssp. *parviglumis* Iltis and Doebley, collection *Iltis and Cochrane 81*). The F_1s of each of these crosses were grown and either selfed, sib-mated, or backcrossed to the parent other than line 91-31. The progeny of these crosses were then analyzed for both the single spikelet trait and genetic linkage for marker loci segregating in the families.

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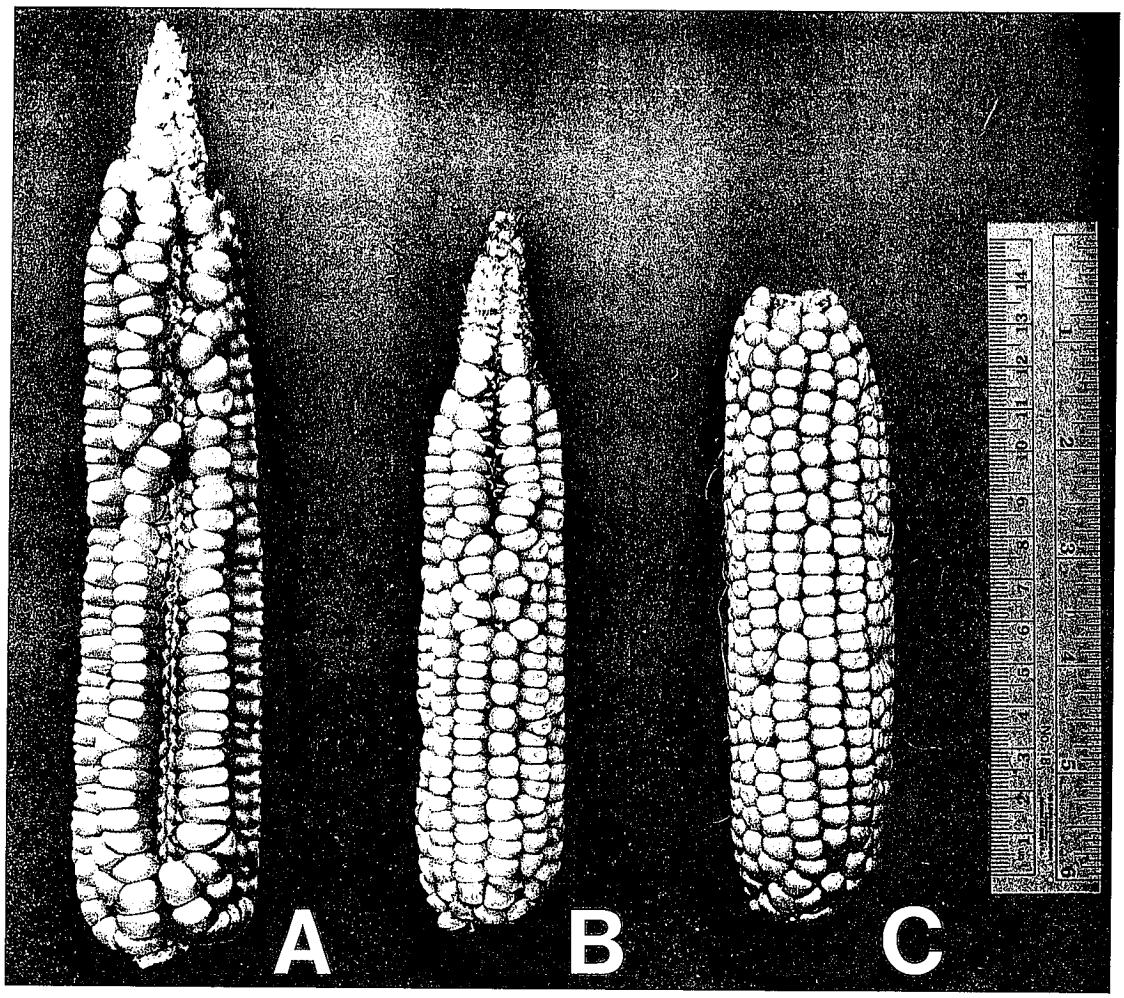


Fig. 1. Ears of *Sos1-Ref* maize with a strong phenotype (A), *Sos1-Ref* maize with a weak phenotype (B), and *sos1+W22* maize (C).

To determine the chromosomal location of the genetic locus controlling the single spikelet phenotype, we employed restriction fragment length polymorphisms (RFLPs) using DNA clones of known genomic locations as probes (Burr et al., 1988; Beavis and Grant, 1991; Gardiner et al., 1993). Molecular methods including DNA isolations, agarose gel electrophoresis, Southern blotting, radio-labeling of probes, and filter hybridizations have been previously described (Doebley and Stec, 1993). Genetic linkage analyses were performed using MAPMAKER MACINTOSH version 2.0 (Lander et al., 1987).

Finally, to determine the developmental basis of the single spikelet phenotype, we examined ear primordia of the *Sos1-Ref* mutant, normal maize, and teosinte using scanning electron microscopy (SEM). Ear primordia were removed from the plants ≈40 d after planting and preserved in FAA [50 ml EtOH, 10 ml formalin (37%), 5 ml glacial acetic acid, and 35 ml distilled water]. The primordia were subsequently dehydrated in an ethanol series, subjected to critical-point drying, gold-coated with a sputter-coater, and examined with a Hitachi S-450 scanning electron microscope (Williams and Sylvester, 1994).

RESULTS

Genetics—The F_1 hybrids of W22, W22-TGA, and the *lazy1-sugary1* tester with line 91-31 possessing the single spikelet phenotype all exhibited the single spikelet phenotype, demonstrating that this trait was dominant to the normal maize condition of paired spikelets (Fig. 1). The line 91-31 × W22 F_1 hybrid was backcrossed to W22, and this backcross population was analyzed during the summer of 1992. This was one of the coldest summers on record in Minnesota, and plant growth was poor. Among the 58 progeny analyzed, 31 exhibited paired spikelets and normal tassels. The remaining 27 plants were barren (without ears) and had tassels that consisted only of a central spike without any branches. The 31:27 ratio fit a 1:1 ratio ($\chi^2 = 0.28$, df = 1, $P > 0.75$) expected if a single dominant locus controlled the differences in tassel structure and barrenness. The high proportion of barren plants prevented us from scoring the single spikelet trait in the ear. One possibility we considered was that the cold weather had induced barrenness in plants carrying the factor that causes the single spikelet trait and that this factor

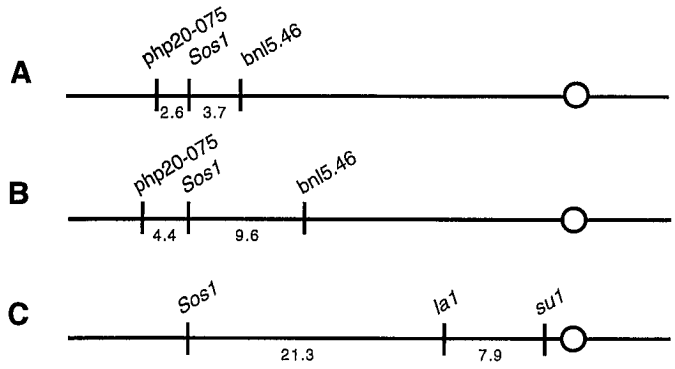
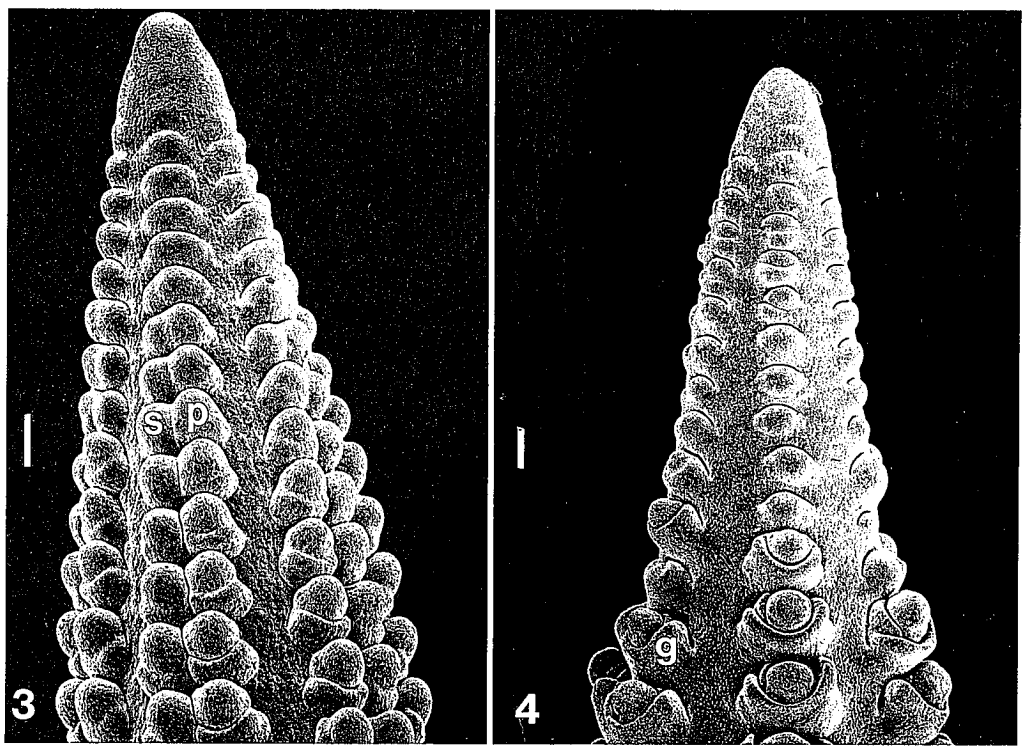


Fig. 2. Genetic maps for the short arm of maize chromosome 4 showing the position of *sos1* relative to other known loci. A. Map based on the (*Sos1-Ref* line 91-31 × W22) × W22 backcross population and scoring barrenness/unbranched tassels as the *Sos1* effect. B. Map based on the line *Sos1-Ref* 91-31 × W22-TGA F_2 population. C. Map based on the (*Sos1-Ref* line 91-31 × *lazy1-sugary1*) × *lazy1-sugary1* backcross population.

also affects the production of tassel branches. Working on this assumption, we analyzed the 58 plants for RFLP markers, using one marker per chromosome arm. We detected linkage between an RFLP marker (bnl5.46) on chromosome arm 4S and barrenness/unbranched tassels. After mapping additional RFLPs on this chromosome arm, we mapped these traits between php20-0725 and bnl5.46 (Fig. 2A).

In the summer of 1993, we again attempted mapping the single spikelet trait using an F_2 population obtained from selfing the line 91-31 × W22-TGA cross. Plant growth this year was better, and in a population of 58 F_2 plants, we observed 46 with single spikelets and 12 with paired spikelets in the ear. These numbers do not differ from the expected 3:1 ratio if the single spikelet trait is controlled by a single dominant locus ($\chi^2 = 0.32$, df = 1, $P > 0.5$). Thus, we designate this locus as *suppressor of sessile spikelets1* (*sos1*) and the mutant allele as *Sos1-Ref.* RFLP analysis of this F_2 population demonstrated that *sos1* maps between php20-0725 and bnl5.46 (Fig. 2B). We should also note that the introgressed teosinte chromosome segment in W22-TGA does not extend between php20-0725 and bnl5.46, and thus, *Sos1-Ref* was segregating with *sos1*+*W22* in this F_2 population.

Finally, to map *sos1* relative to morphological markers on the short arm of chromosome 4, the F_1 hybrid of line 91-31 × *lazy1-sugary1* tester was backcrossed to the *lazy1-sugary1* tester. The progeny of this backcross were grown in a winter (1993–1994) nursery on Molokai, Hawaii. Among 89 progeny, we observed 42 with single spikelets and 47 with paired spikelets, which fits the expected 1:1 ratio for a single dominant locus ($\chi^2 = 0.28$, df = 1, $P > 0.75$). This population enabled us to place *sos1* at 21.3 recombination units distal to *lazy1* (Fig. 2C). The results of all three mapping populations are consistent with placing *sos1* on the short arm of chromosome 4. Because the single spikelet trait mapped to the same location as barrenness/unbranched tassels in the 1992 backcross pop-



Figs. 3, 4. Scanning electron micrographs of maize ear primordia. **3.** Normal (*sos1/sos1*) maize. **4.** *Sos1-Ref* maize. g, lower glume primordium; s, sessile spikelet primordium; p, pedicellate spikelet primordium. Bars = 100 μm. On the ear primordium in Fig. 4, differentiation of the lower glume is exhibited in the third to the fifth spikelet primordia from the top though it is lacking from the seventh to the eleventh spikelet primordia. The absence of lower glume differentiation in these latter primordia is an anomaly that we cannot explain.

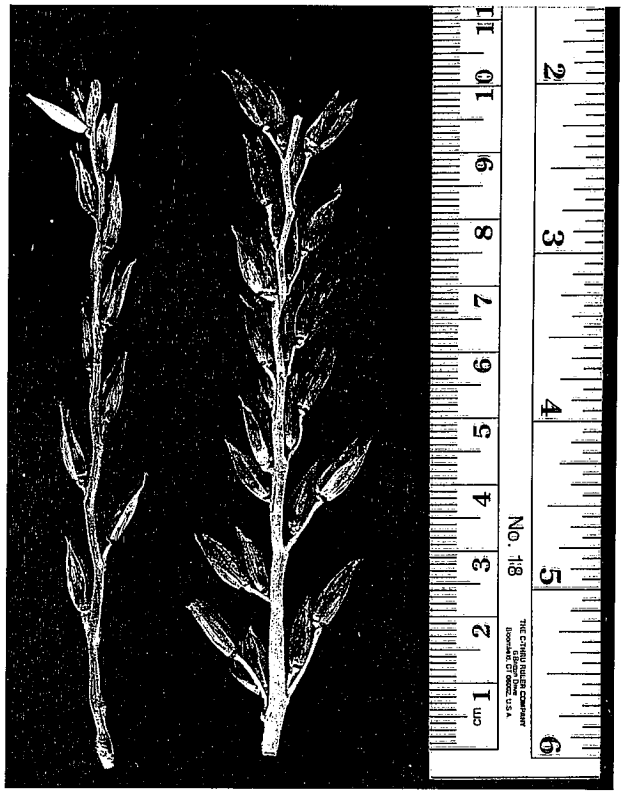


Fig. 5. Sections of lateral tassel branches of *Sos1-Ref* maize (left) showing single spikelets and normal (*sos1/sos1*) maize (right) showing paired spikelets.

ulation and because all these traits behaved as dominants, we infer that *sos1* alone controls these traits and that *Sos1-Ref* plants have a tendency toward barrenness under poor growth conditions.

Development—Figure 1A shows an ear carrying *Sos1-Ref*, causing the absence of the sessile spikelets and leaving a gap between the rows of the remaining pedicellate spikelets. This phenotype is readily distinguished from an ear of normal maize with paired (sessile-pedicellate) spikelets (Fig. 1C). In Fig. 1A, the absence of the sessile spikelet extends over the entire length of the ear. Figure 1B shows an ear carrying *Sos1-Ref* in which the absence of the sessile spikelet is restricted to the distal half of the ear. Ears of heterozygous (*Sos1-Ref/sos1*) plants in all three mapping populations occasionally exhibited paired spikelets on the basal 10–50% of their lengths. The occurrence of these ears suggests that *Sos1-Ref* is not fully penetrant.

To determine the developmental basis of the single spikelet phenotype, we examined ear primordia of both mutant (*Sos1-Ref*) and normal (*sos1/sos1*) maize. In normal maize, the ear primordia produce primary lateral initials (branches) or spikelet-pair primordia (Fig. 3). Subsequently, each spikelet-pair primordium branches again to form the sessile and pedicellate spikelet primordia. The sessile spikelet represents a secondary branch and the pedicellate spikelet is the continuation of the primary branch (Cheng, Greyson, and Walden, 1983; Sundberg and Orr, 1986). For developmentally young spikelet-pairs

TABLE 1. Comparison of *Sos1-Ref* mutant and normal (*sos1/sos1*) maize plants from the *Sos1-Ref* maize × W22-TGA F_2 population for the number of tassel branches and the number of orthostichies in the ear (SE = standard error of the mean, N = sample size and P = probability from a two-tailed t-test under the null hypothesis of the mean for normal being equal to that for mutant).

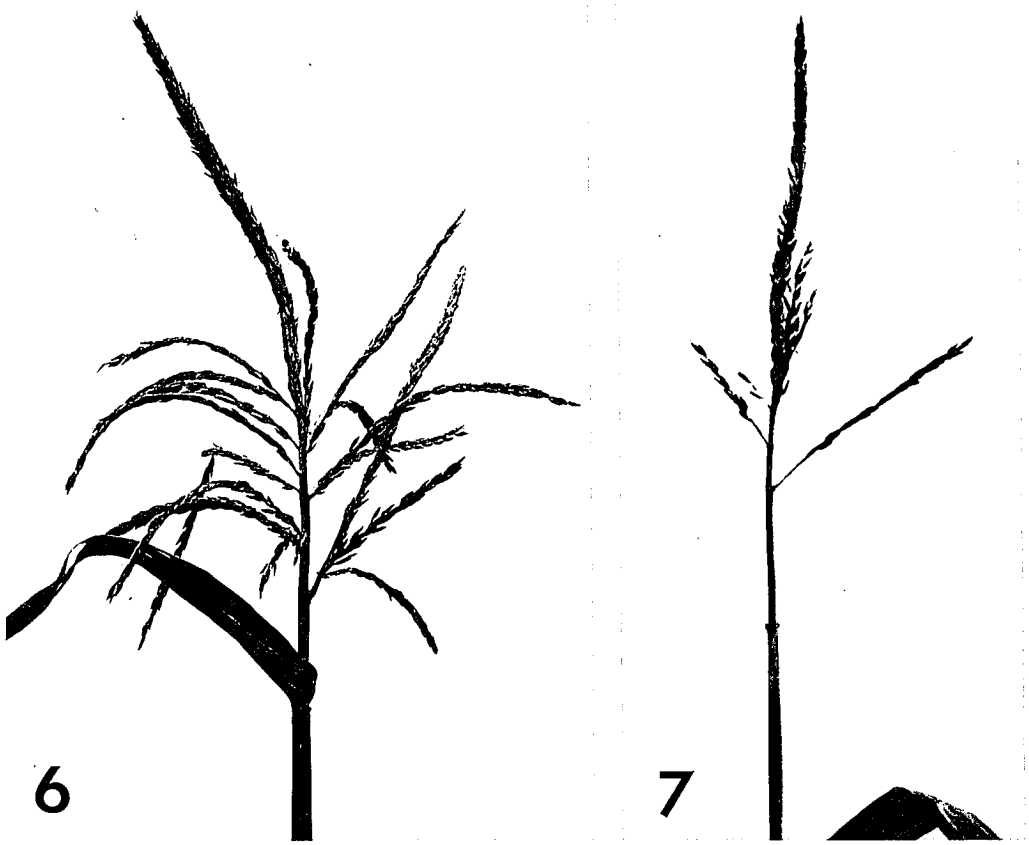
	Normal (*sos1/sos1*)	Mutant (*Sos1/sos1* and *Sos1/Sos1*)	P
Tassel branch number (SE; N)	11.1 (0.73; 28)	7.3 (0.48; 78)	0.0001
Ear orthostichies (SE; N)	7.1 (0.69; 28)	5.5 (0.82; 81)	0.0001

(those nearer the apex of the ear primordium), the spikelet primordia are unequal in size with the sessile spikelet primordium being smaller. These two branches subsequently begin to form a series of lateral organs, beginning with the lower glume (Fig. 3).

In the *Sos1* mutant, the ear primordium produces spikelet-pair primordia or primary branches as in normal maize; however, these primary branches fail to initiate the secondary branches that form the sessile spikelets (Fig. 4). It is this step in normal ear development that *Sos1-Ref* blocks. The primary branch subsequently begins to form lateral organs beginning with the lower glume, ultimately producing a normal spikelet. Because this spikelet represents the continuation of the primary branch, it is positionally homologous to the pedicellate spikelet of normal maize, and in this sense, *Sos1-Ref* suppresses the formation of the sessile spikelet that develops from the secondary branches of the ear.

Sos1 affects other aspects of inflorescence development. *Sos1-Ref* tends to have single rather than paired spikelets on the tassel branches (Fig. 5). However, this tassel phenotype is more variable than the single spikelet ear phenotype, and *Sos1-Ref* maize may have tassels with normal-paired spikelets. We also noticed that tassels of the *Sos1-Ref* mutant appeared to have fewer branches (Figs. 6, 7). To verify this observation, we counted the number of tassel branches on plants of the line 91-31 × W22-TGA F_2 population. We detected a statistically significant quantitative effect (Table 1). Similarly, we measured the number of orthostichies (or rows of cupules) in the ear, midway along its length. For normal maize, this equals one-half the number of rows of kernels, while for *Sos1-Ref* maize it equals the number of rows of kernels. Again, we observed a statistically significant quantitative effect (Table 1).

Evolution—While maize, like most members of the tribe Andropogoneae, has paired spikelets in its inflorescences, teosinte, the probable progenitor of maize, possesses single spikelets in its ears, and thus resembles the *Sos1-Ref* mutant in this regard. To investigate whether *sos1* is the gene controlling the single spikelet condition in teosinte, we performed a genetic segregation test. This involved crossing *Sos1-Ref* maize to teosinte and selfing or sibmating the F_1 hybrids to produce an F_2 population. Since both *Sos1-Ref* maize and teosinte have single spikelets in their ears, one should not observe any plants with paired spikelets if *sos1* controls the single spikelet trait in teosinte.



Figs. 6, 7. Maize tassels from the *Sos1-Ref* line 91-31 × W22-TGA F_2 population. **6.** Normal (*sos1/sos1*) maize with a well-branched tassel. **7.** *Sos1-Ref* maize with few tassel branches.

However, if a gene(s) other than *sos1* controls the single spikelet trait in teosinte, then among the F_2 segregants, one should observe some plants with paired spikelets. These plants would possess the teosinte allele of *sos1* and the maize allele at the gene(s) controlling paired spikelets in teosinte. This argument assumes that the maize and teosinte genomes are collinear, which is known to be true based on the identical linkage relationships for RFLP loci in maize and teosinte (Doebley and Stec, 1993).

Among the 67 *Sos1-Ref* maize-teosinte F_2 plants, we observed 40 with ears that possessed all single spikelets, 17 with ears that possessed a mixture of single and paired spikelets, and ten ears that consisted entirely of paired spikelets. The ten ears with all paired spikelets confirm that *sos1* is not the locus responsible for the difference of paired vs. single spikelets between maize and teosinte.

As an additional test of whether *sos1* was involved in the evolution of maize from teosinte, we examined ear primordia of teosinte to compare their development to that of *Sos1-Ref* maize. Previous reports indicated that, in teosinte, a pedicellate spikelet primordium is formed and then subsequently aborted to yield single sessile spikelets (Sundberg and Orr, 1990). This represents a very different mechanism for the production of the single spikelet phenotype than that of *Sos1-Ref* maize in which the branching of the spikelet pair primordia is blocked so that the sessile spikelet is never formed, yielding single pedicellate spikelets. Figure 8 shows a teosinte ear primordium. Just below its apex, spikelet-pair primordia are being initiated. Below this, one can see the spikelet-pair primordia forming the branch that will develop into the sessile spikelet. At this position, the sessile spikelet primordium is smaller than the pedicellate spikelet primordium. Near the base of the ear primordium, one can see the opposite situation, i.e., the sessile spikelet primordium is larger since the pedicellate spikelet primordium has been arrested in its development and will ultimately deteriorate, leaving a single sessile spikelet at each node. These observations confirm the previous report of Sundberg and Orr (1990) and demonstrate that the developmental basis of single spikelets in the *Sos1-Ref* maize is different from that in teosinte.

DISCUSSION

Sos1-Ref is a dominant mutant of maize that acts to suppress the branching of the spikelet-pair primordia in the ear and tassel. In the tassel, *Sos1-Ref* also reduces the number of tassel branches, and it reduces the number of orthostichies in the ear in a quantitative fashion. The observation that *Sos1-Ref* reduces the number of orthostichies in the ear indicates that it acts early in inflorescence development before the spikelet pair primordia are formed. The *sos1* gene product is probably expressed

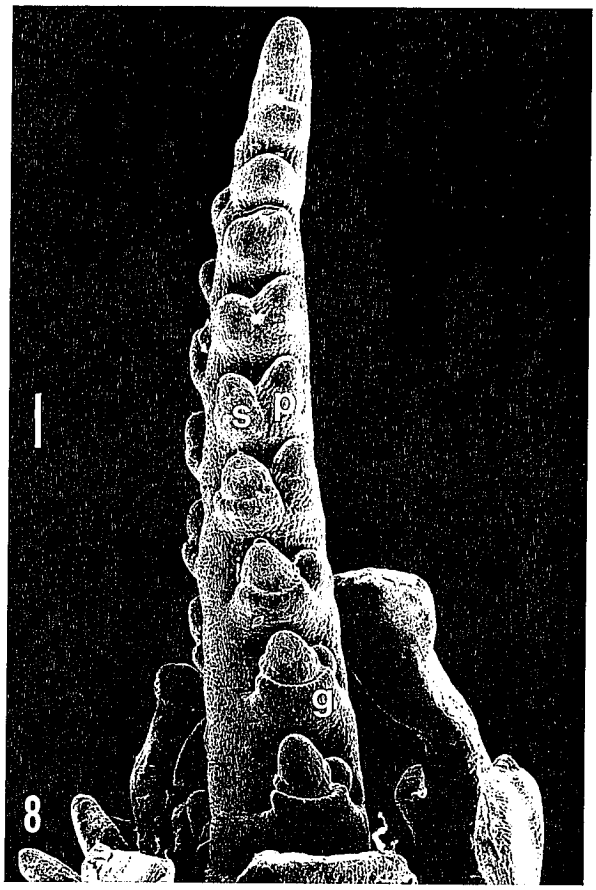


Fig. 8. Scanning electron micrograph of a teosinte ear primordium. Bar = 100 μm. g, lower glume primordium; s, sessile spikelet primordium; p, pedicellate spikelet primordium.

in the axillary and apical meristems during inflorescence development.

There have been several previous reports in the literature of *Sos1*-like mutants in maize. Langham (1940) reported that a single locus, which he designated *paired spikelet* (*pd*), controls the difference in single vs. paired spikelets between teosinte and maize. He also mentioned a mutation that arose spontaneously in a maize line and suggested that it was allelic to *pd*. Because this mutant has not survived and he did not perform formal genetic analyses, the nature of this putative mutant and its relationship to *sos1* cannot be investigated. Hepperly (1949) reported an *sos1*-like mutant. Although seeds of this mutant were deposited with the Maize Genetics Cooperative Stock Center under the name "unpaired spikelets," it was never subjected to formal genetic analysis and thus has not been given a gene symbol.

Hepperly's mutant was subjected to thorough developmental and morphological analyses by Wilcox (1950), although his results were never published. Wilcox found that the primary branch primordia in the ear generally fail to divide to form the sessile spikelet primordia. He concluded that it was the sessile spikelet that was lacking. He reported that at the base of the ear a few primary branch primordia would divide to form a sessile-pedicellate spikelet pair. He reported that the tassels had fewer branches and frequent single spikelets. Finally, he noted the presence of "rudimentary" spikelets in the tassel that have glumes but lack florets. We have seen these same rudimentary spikelets in the tassels of *Sos1-Ref* maize. In fact, in every regard, Wilcox's description of Hepperly's mutant is identical to *Sos1*. For this reason, it seems possible if not likely that *Sos1* is allelic to Hepperly's mutant.

Some confusion exists in the literature in regard to Hepperly's mutant ("unpaired spikelet") and Langham's *pd*. First, several authors have been unable to confirm Langham's report that the trait single vs. paired spikelets between maize and teosinte is controlled by a single locus, *pd* (Mangelsdorf, 1947; Rogers, 1950; Doebley and Stec, 1991, 1993). Rather, based on these reports, it seems likely that this trait is controlled by several loci and that Langham's result of monofactorial inheritance was anomalous. For this reason and because it is not possible to determine to which of the several loci governing the differences between maize and teosinte *pd* should refer, we recommend that Langham's *pd* be considered undocumented and dropped from use. Second, Galinat (1985) treated Hepperly's mutant and Langham's *pd* as allelic, although there are no data to support this view. Data from Wilcox (1950), Sundberg and Orr (1990), and our work show that the developmental basis of the single spikelet condition produced by Hepperly's mutant is different from that of teosinte, indicating that Hepperly's mutant is not allelic to the gene(s) differentiating maize and teosinte. For this reason, Wilcox (1950) also concluded that Hepperly's mutant did not represent a gene involved in the evolution of maize from teosinte.

Our interest in *Sos1-Ref* arose because the single spikelet trait of this mutant seemed to resemble the single spikelets of teosinte ears, the probable ancestor of maize. Thus, *sos1* was a candidate for a gene involved in the evolution of maize from teosinte. We can now reject this hypothesis for several reasons. First, as described above, the developmental basis of single spikelets in *Sos1-Ref* maize and teosinte is different. In teosinte ears, both the pedicellate and sessile spikelet primordia are formed, but then the pedicellate spikelet is aborted. With *Sos1-Ref*, only a single spikelet primordium is formed. Second, in teosinte, it is the pedicellate spikelet that is lacking, while in *Sos1-Ref* maize the sessile spikelet is lacking. Third, in our QTL (quantitative trait locus) mapping studies (Doebley and Stec, 1993), the QTLs controlling the difference of paired vs. single spikelets between maize and teosinte do not map to the region of chromosome 4 where *sos1* is located. Fourth, in the *Sos1-Ref* × teosinte segregation analysis reported above, we recovered plants with paired spikelets, demonstrating that *Sos1-Ref* is not allelic to the gene(s) controlling the difference in single vs. paired spikelets between maize and teosinte. Fifth, *Sos1-Ref* produces single spikelets in both the ear and tassel, while the gene controlling single spikelets in teosinte affects only the ear (see Wilcox, 1950).

Finally, the observation that the similar phenotypes of *Sos1-Ref* maize and teosinte have different genetic and developmental bases cautions against drawing conclusions of homology based on similar adult phenotypes alone. Only through detailed genetic and developmental

analysis can it be determined whether similar phenotypes are truly homologous or the result of convergence.

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