teosinte branched1 and the Origin of Maize: Evidence for Epistasis and the Evolution of Dominance

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Manuscript received November 29, 1994
Accepted for publication May 18, 1995

ABSTRACT

Two quantitative trait loci (QTL) controlling differences in plant and inflorescence architecture between maize and its progenitor (teosinte) were analyzed. Complementation tests indicate that one of these, which is on chromosome arm 1L, is the locus for the maize mutant *teosinte branched1* (*tb1*). This QTL has effects on inflorescence sex and the number and length of internodes in the lateral branches and inflorescences. This QTL has strong phenotypic effects in teosinte background but reduced effects in maize background. The second QTL, which is on chromosome arm 3L, affects the same traits as the QTL on 1L. We identify two candidate loci for this QTL. The effects of this QTL on several traits are reduced in both maize and teosinte background as compared to a maize-teosinte F_2 population. Genetic background appears to affect gene action for both QTL. Analysis of a population in which both QTL were segregating revealed that they interact epistatically. Together, these two QTL substantially transform both plant and inflorescence architecture. We propose that *tb1* is involved in the teosinte plant's response to local environment to produce either long or short branches and that maize evolution involved a change at this locus to produce short branches under all environments.

THE evolution of cultivated maize (*Zea mays* L. ssp. *mays*) from its probable wild progenitor teosinte (*Z. mays* ssp. *parviglumis* Iltis and Doebley) provides one of the most striking and complex examples of morphological evolution in plants. These taxa differ extensively in both plant and inflorescence architecture. The differences are so extreme that when teosinte was first discovered, taxonomists failed to recognize its close relationship to maize, placing it in a separate genus and tribe (WILKES 1967). After subsequent research demonstrated that teosinte and maize were fully interfertile and in essence members of the same biological species, BEADLE (1939) proposed that maize is simply a domesticated form of teosinte and that as few as five major genes largely controlled its morphological evolution from teosinte.

Our group has been investigating the inheritance of the morphological differences between maize and teosinte using quantitative trait locus (QTL) mapping with molecular markers (TANKSLEY 1993). Initially, we demonstrated that there are five regions of the genome that largely control the key morphological difference between maize and teosinte, a result consistent with BEADLE's 1939 hypothesis (DOEBLEY *et al.* 1990; DOEBLEY and STEC 1993). Subsequent investigation of one of these five regions demonstrated that it encompassed a single Mendelian locus [*teosinte glume architecture1* (*tga1*)] that controls the development of the cupulate fruitcase, a protective covering on teosinte seeds that is lacking in maize (DORWEILER *et al.* 1993). This result provided further support for BEADLE's hypothesis, although the question of whether the other four regions also possess single loci of large effect remains unanswered.

In this paper, we report the analysis of two QTL with large effects on the aspects of plant and inflorescence architecture that distinguish maize and teosinte. We use complementation tests to demonstrate that one of these QTL is the locus for the known maize mutant *teosinte branched1* (*tb1*). Based on map location and their morphological effects, we identify the maize loci *terminal ear1* (*te1*) and *tassel replaces upper-ear1* (*tru1*) as candidates for the second QTL. We also characterize the effects of the maize alleles of both QTL in teosinte background and the effects of the teosinte alleles in maize background. These analyses indicate that the maize alleles behave in a more dominant fashion in maize background relative to teosinte background, and that these QTL interact epistatically. Finally, a model for how *tb1* could alter morphogenesis and thereby contribute to the morphological evolution of maize is presented.

MATERIALS AND METHODS

Plant materials: The source of teosinte used in all experiments was *Z. mays* ssp. *parviglumis* collected near Teloloapan, Guerrero, Mexico by HUGH ILTIS and TED COCHRANE (Collection No. 81) of the University of Wisconsin-Madison. The primitive maize race Reventador (Collection Nay 15) was obtained from MAJOR GOODMAN, North Carolina State University. Maize

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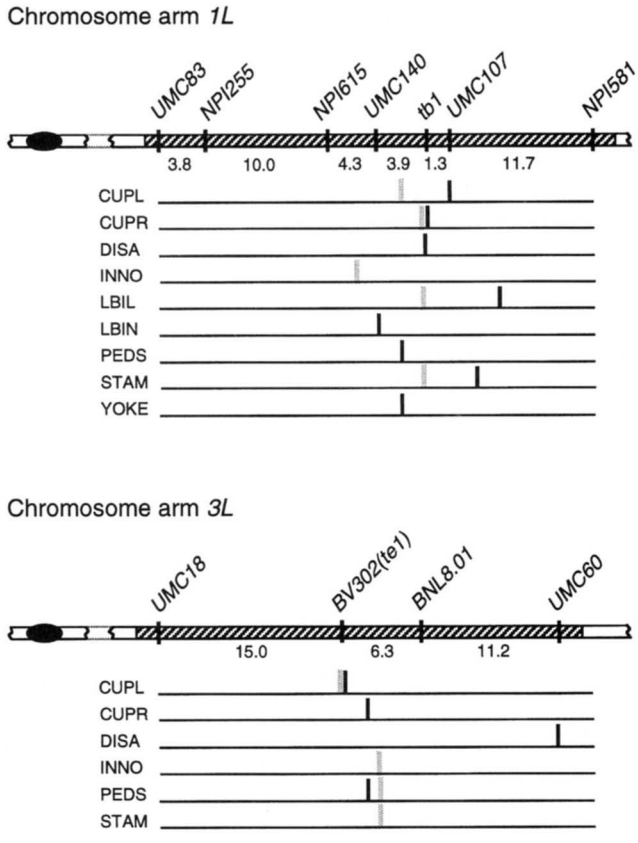


FIGURE 1.—Target regions on chromosome arms *1*L and *3*L. ▨, target regions moved into different genetic backgrounds. The molecular markers analyzed in each target region are shown. Beneath each target region, the positions of the QTL for each trait as estimated by MAPMAKER-QTL are shown (solid lines for the T-M1 and T-M3 populations, and stippled lines for the M-T1 and M-T3 populations). Distances (Haldane cM) between the molecular markers are from the T-M1 and T-M3 populations for target regions on *1*L and *3*L, respectively. The location of *tb1* is taken from DOEBLEY *et al.* (1995b). For the M-T1 population, the target region extended only from *NPI255* to *NPI581*. ●, centromeres. For a key to trait acronyms see Table 2.

inbred line W22 was obtained from JERRY KERMICLE, University of Wisconsin-Madison, and a line carrying *tb1-ref* was obtained from CHARLES BURNHAM of the University of Minnesota who originally described this mutant (BURNHAM 1959).

Line construction: We used backcross breeding with molecular marker-assisted selection to transfer chromosomal regions on chromosome arms *1*L and *3*L (hereafter target regions) encompassing previously identified QTL into two different genetic backgrounds (Figure 1). The starting material for this backcross breeding program was the R×P F_2 population based on a cross of Reventador maize (R) and *parviglumis* teosinte (P) that was previously analyzed by DOEBLEY and STEC (1993). Individual F_2 plants were selfed, providing a series of F_3 families. For each target region, a single F_3 plant homozygous for the maize allele at all marker loci in a target region but which carried teosinte alleles at most marker loci outside the target region was selected. The selected F_3 plants were backcrossed to teosinte for three generations before selfing to produce two BC_3F_2 populations. Molecular markers were employed each generation to insure we were retaining the maize alleles in the target regions as well as eliminating maize alleles outside the target regions. Marker loci outside the target regions were specifically chosen to eliminate regions of the genome that possess QTL contributing to the morphological differences between maize and teosinte. One BC_3F_2 plant for each target region was selfed to produce lines, Teosinte-M1L and Teosinte-M3L, homozygous for the maize alleles in the target regions on *1*L and *3*L, respectively. Lines Teosinte-M1L and Teosinte-M3L were each crossed again to teosinte, and the hybrids were selfed to produce two BC_4F_2 populations in which the individual target regions were segregating in teosinte background (Table 1). Additionally, Teosinte-M1L and Teosinte-M3L were crossed to each other, and the hybrid was selfed to produce a population in which the two target regions were segregating simultaneously in teosinte background.

Transfer of the teosinte target regions on chromosome arms *1*L and *3*L into maize genetic background was accomplished in a similar manner. For each target region, a single F_3 plant from the R×P F_2 population that was homozygous for the teosinte alleles at all marker loci in a target region but that carried maize alleles at most loci outside this target region was selected (Figure 1). The selected F_3 plant was then crossed to maize inbred W22. The hybrids of these crosses were backcrossed to W22 for four generations before selfing to produce two BC_4F_2 populations: one segregating for the target region on *1*L and the other for that on *3*L (Table 1). During this backcrossing program, selection was placed only on marker loci within the target region.

Quantitative trait analysis: Teosinte and maize have strikingly different plant architectures. In teosinte, the main culm produces many lateral branches, each terminated by a branched male inflorescence or tassel (Figure 2). The ears or female inflorescences of teosinte are borne in clusters at the nodes along the primary lateral branches. Maize produces relatively few lateral branches, each terminated by a normally unbranched and female inflorescence, *i.e.*, an ear (Figure 2). We refer to the inflorescence terminating the primary lateral branch as the primary lateral inflorescence whether it is a tassel as in teosinte or an ear as in maize. To analyze these differences, we measured four traits (Table 2): the number of internodes in the primary lateral branch (INNO), the average length of these internodes (LBIL), the proportion of male or staminate spikelets in the primary lateral inflorescence (STAM), and the number of branches in the primary lateral inflorescence (LIBN).

Teosinte and maize also show extreme differences in female inflorescence (ear) architecture. The teosinte ear is composed of a series of roughly 6–12 cupulate fruitcases (Figure 3A). These fruitcases are situated one on top of the other in the ear. The cupule of the cupulate fruitcase is formed from an invaginated rachis internode (Figure 3C). The cupule contains a single sessile spikelet that produces a single kernel. The cupulate fruitcases are separated from one another by abscission layers, enabling the ear to disarticulate at maturity for seed dispersal. The cob (rachis) of the maize ear, like that of its teosinte counterpart, is composed of invaginated internodes or cupules (Figure 3D). Maize cupules are arranged in several ranks around the ear so that cupules are both side-by-side and one on top of the other. There are usually 100 or more cupules in a single ear (Figure 3B). In contrast to teosinte, there are two spikelets associated with each cupule, one pedicellate and the other sessile. Finally, the maize ear lacks abscission layers as found in teosinte, so the ear remains intact at maturity. To analyze these differences, we measured five traits (Table 2): ear disarticulation (DISA), using a scale from 1 (100% disarticulating) to 5 (0% disarticulating); the number of cupules along a single row in the ear (CUPR); the average length of the cupules (internodes) in the ear (CUPL); the degree to which the cupules

TABLE 1

Populations for QTL analysis

Population (abbreviation)	Genetic background	Generation	Population size
Teosinte-M1L × Teosinte (T-M1)	Teosinte	BC_4F_2	111
Teosinte-M3L × Teosinte (T-M3)	Teosinte	BC_4F_2	79
Teosinte-M1L × Teosinte-M3L (T-M1+3)	Teosinte	BC_3F_2	183
W22-T1L × W22 (M-T1)	Maize	BC_4F_2	87
W22-T3L × W22 (M-T3)	Maize	BC_4F_2	87
Reventador × parviglumis (R×P)[a]	Maize-teosinte	F_2	290

[a] This population previously analyzed by DOEBLEY and STEC (1993).

were strictly one on top of the other like teosinte (a score of 1) or side-by-side to form yoked (YOKE) pairs as in maize (a score of 5, this trait measured only in populations with teosinte genetic background); and the proportion of cupules possessing single (sessile) spikelets as in teosinte *vs.* paired (sessile-pedicellate) spikelets as in maize (PEDS).

Plants for morphometric analyses were grown in a winter (1993–1994) nursery on the island of Molokai, Hawaii (T-M1, T-M3, and T-M1+3 populations) or a summer (1994) nursery in St. Paul, Minnesota (M-T1 and M-T3 populations). In both nurseries, plants were grown in 15-ft-long rows with plants spaced 1 ft apart. Plants for the two complementation tests with *tb1* were grown in a nursery in St. Paul during the summers of 1993 and 1994.

Molecular marker loci: For molecular markers, we employed restriction fragment length polymorphisms following procedures previously described by DOEBLEY and STEC (1993). Plasmid clones of low-copy-number nuclear DNA sequences of maize for use as probes were available from Brookhaven National Laboratory (BURR *et al.* 1988), Pioneer Hi-Bred International (BEAVIS and GRANT 1991), Native Plants Incorporated (HELENTJARIS *et al.* 1988), and University of Missouri-Columbia (GARDINER *et al.* 1993). We also used a clone of *te1* (*BV302*) generously provided by BRUCE VEIT and SARAH HAKE, US Department of Agriculture-Plant Gene Expression Center (Albany, CA). Figure 1 shows the target regions on chromosome arms 1L and 3L that were followed in the backcrossing program and the molecular marker loci in each region that were assayed.

Statistical analysis: Linkage maps for the marker loci in each target region were assembled using MAPMAKER version 3.0 (LANDER *et al.* 1987). Interval mapping of QTL was performed using the computer program MAPMAKER-QTL version 1.1 (LANDER and BOTSTEIN 1989). Interval mapping was used primarily for the purpose of estimating the chromosomal locations of the QTL. Single factor analysis of variance was used to test for significant associations between the molecular markers (*UMC107* and *BV302*) and morphological traits and to estimate the R^2 values (the proportion of the phenotypic variance explained by a QTL) for each significant association (EDWARDS *et al.* 1987). We chose *UMC107* because it lies 1.3 cM from *tb1* (Figure 1), the candidate for one of our QTL, and *BV302* (*te1*) because it is a candidate for our other QTL. The dominance/additivity ratio for each QTL was calculated as

$$d/a = \frac{MT - (MM + TT)/2}{(MM - TT)/2},$$

where *MM*, *TT*, and *MT* designate the mean trait values for plants having homozygous maize, homozygous teosinte or heterozygous genotypes at either *UMC107* or *BV302* (EDWARDS *et al.* 1987). To test for digenic epistatic interactions, trait performance for the nine possible two-locus genotypic classes at *UMC107* and *BV302* (*te1*) was subjected to two-factor analysis of variance. A significant interaction term was interpreted

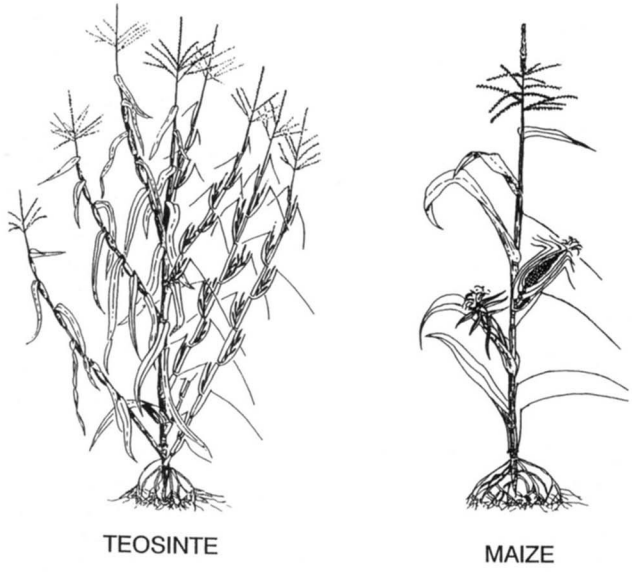


FIGURE 2.—Mexican annual teosinte and maize plant architectures. Adapted from ILTIS (1983) and DOEBLEY *et al.* (1990).

TABLE 2

List of morphological traits analyzed

Trait	Description
CUPL	Average length of cupules (internodes) in the inflorescence
CUPR	Number of cupules in a single rank of the ear
DISA	Tendency of ear to shatter (1–5 scale) 1 = 100% disarticulating, 5 = 0% disarticulating
INNO	Number of vegetative internodes in the lateral branch
LBIL	Average length of vegetative internodes in the primary lateral branch
LIBN	Number of branches in primary lateral inflorescence
PEDS	Percentage of cupules lacking the pedicellate spikelet
STAM	Percentage of staminate spikelets in primary lateral inflorescence
YOKE	Degree to which the fruitcases are in yoked pairs (1–5 scale) 1 = 0% yoked fruitcases, 5 = 100% yoked fruitcases

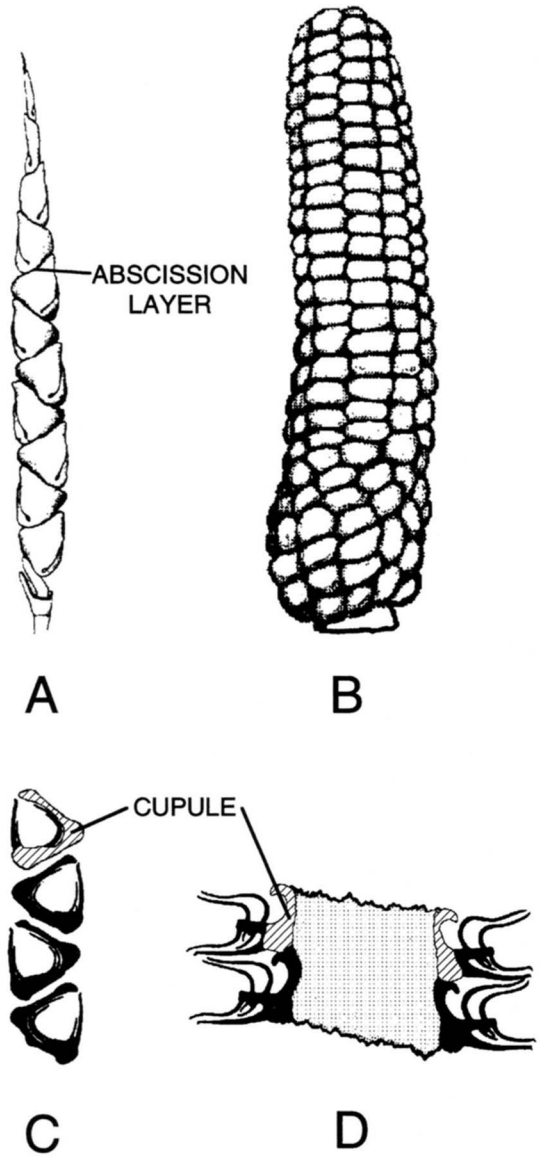


FIGURE 3.—Architecture of Mexican annual teosinte and maize ears (female inflorescences) adapted from ILTIS (1983), GALINAT (1969) and DOEBLEY (1993). (A) Teosinte ear. (B) Maize ear. (C–D) Longitudinal cross-sections of teosinte (C) and maize (D), both showing cupules (hatched).

as evidence of epistasis. Each interaction was further partitioned into four contrasts, additive by additive, additive by dominant, dominant by additive and dominant by dominant (COCKERHAM 1954).

RESULTS

QTL on chromosome arm *1*L: Chromosome arm *1*L has significant effects on each of the nine morphological traits in at least two of the four populations analyzed (Tables 3 and 4). We recognize that the effects on different traits could represent multiple linked QTL; however, for simplicity of discussion, we will consider all effects to represent the action of a single QTL, namely QTL-1L with alleles QTL-1L^M and QTL-1L^T for maize and teosinte, respectively. QTL-1L has significant effects on all traits except INNO in teosinte background (T-M1 population), suggesting that QTL-1L^M has strong phenotypic expression in teosinte background. However, there were significant effects on only five of the nine traits in maize background (M-T1 population). While the lack of significant effects in maize background could be a statistical artifact of the smaller population size, it is striking that some traits were invariant in maize background. For example, all ears were completely nondisarticulating (no variance for DISA) and unbranched (no variance for LIBN). The sex of the inflorescence (STAM) was affected in a similar way. Although there was a significant effect on STAM in all four populations, this effect is much larger in teosinte background (populations T-M1 and T-M1+3) than in maize background (Table 4). These observations indicate that QTL-1L^T has a reduced phenotypic effect on these traits in maize background relative to teosinte background.

Two other traits show noteworthy patterns of expression in the different backgrounds. An effect on ear DISA was detected in teosinte background but not in the original F_2 population despite a much larger sample size. Its effect in the F_2 population may have been obscured by other segregating QTL affecting this trait. The effect on PEDS is much larger in the F_2 population than in either maize or teosinte background (Table 4). This could occur if the effect of QTL-1L is dependent on epistatic interactions with other QTL that would be segregating in the F_2 population but not in either maize or teosinte background (see below).

QTL-1L^M reduces the length of the internodes in both the primary lateral branch (smaller LBIL) and inflorescence (smaller CUPL) (Table 4). This maize allele also acts to increase the number of internodes in both the branch (higher INNO) and inflorescence (higher CUPR). Thus, QTL-1L^M acts to produce a larger number of shorter internodes, and it is expressed in both the primary lateral branch and inflorescence. The degree to which the cupules are side-by-side as opposed to one on top of the other (YOKE) is another manifestation of this effect on internode elongation. If the internodes in the ear do not fully elongate, then the cupules will develop side-by-side.

Plants of the T-M1 population show how QTL-1L may have altered the morphology of the plant during the early evolution of maize. Plants homozygous for the teosinte allele tend to have long lateral branches tipped by tassels, while those homozygous for the maize allele have short branches tipped by ears (Figure 4). Ear structure is also altered with QTL-1L^M producing ears that show some yoking of the cupules, have a larger than normal number of cupules and fail to fully disarticulate (Figure 5).

Finally, the map positions of the effects on the individual traits associated with QTL-1L in the T-M1 and M-T1 populations are distributed within a narrow region surrounding *tb1* (Figure 1).

TABLE 3

R^2 values for the QTL

	Chromosome arm (marker locus)							
	1L (UMC107)				*3L (BV302)*			
	Population							
Traits	T-M1	R×P F_2	M-T1	T-M1+3	T-M3	R×P F_2	M-T3	T-M1+3
CUPL	35.8	3.4	52.2	35.2	37.6	30.7	39.2	37.8
CUPR	63.1	15.0	42.0	34.3	42.8	18.1	—	23.3
DISA	28.6	—	NV	26.9	8.2*	31.3	NV	19.5
INNO	—	4.6	11.7	7.4	—	14.8	26.9	10.3
LBIL	16.3	12.5	35.8	7.2	—	13.5	—	13.1
LIBN	21.1	7.7	NV	24.8	—	—	NV	—
PEDS	13.8	5.9	—	8.7	13.9	24.2	21.3	13.3
STAM	30.1	21.8	20.4	40.0	—	8.6	16.0	—
YOKE	19.8	4.0	NV	29.8	—	37.2	NV	27.9

R^2 values are expressed as percentages; *, statistical significance at $P = 0.05$, otherwise significant at $P = 0.01$; —, no significant effect; NV, no variance for the trait.

Complementation testing of *tb1*: *teosinte branched1* (*tb1*) is a recessive mutant of maize that affects plant architecture and maps to chromosome arm *1*L (BURNHAM 1959; BURNHAM and YAGYU 1961). Plants homozygous for the reference allele (*tb1-ref*) have long lateral branches tipped by tassels at upper nodes of the main culm, thus resembling teosinte in plant architecture. *tb1-ref* arose as a spontaneous mutant in a maize population and was not derived from teosinte (C. BURNHAM, personal communication). The effects of *tb1-ref* are similar to those of QTL-1L^T, and *tb1* and QTL-1L map to the same region of chromosome arm *1*L (DOEBLEY and STEC 1991) (Figure 1). Because of the coincident map positions and similar phenotypes, we proposed that QTL-1L^T and *tb1-ref* are allelic (DOEBLEY and STEC 1993).

To test this hypothesis, we performed two complementation tests. Plants heterozygous for QTL-1L in maize (W22) background were crossed to a maize plant carrying *Tb1+Maize/tb1-ref* and a second plant homozygous for *tb1-ref*. We considered two possible outcomes: complementation in which case all plants should have normal maize plant architecture and noncomplementation, if QTL-1L^T is allelic to *tb1-ref*, in which case $^1/_4$ or $^1/_2$ of the plants should have teosinte branched plant architecture. These tests assume that QTL-1L^T (= *tb1+teosinte*) is recessive to the dominant maize allele (*Tb1+Maize* = QTL-1L^M), an assumption supported by the fact that heterozygous (QTL-1L$^{M/T}$) plants exhibit normal maize plant architecture.

In both tests, we observed two classes of progeny: plants with normal maize plant and ear architecture and plants with a *tb1*-like phenotype, having primary lateral

TABLE 4

Phenotypic effects for the QTL

	Chromosome arm (marker locus)							
	1L (UMC107)				*3L (BV302)*			
	Population							
Traits (units)	T-M1	R×P F_2	M-T1	T-M1+3	T-M3	R×P F_2	M-T3	T-M1+3
CUPL (mm)	−1.74	−0.79	−1.05	−2.95	−2.86	−2.71	−0.79	−3.17
CUPR (number)	2.49	3.93	5.13	3.69	1.48	4.60	—	3.01
DISA (1–5 scale)	1.13	—	—	2.10	0.25	2.06	—	1.80
INNO (number)	—	0.49	0.88	0.52	—	0.93	1.34	0.60
LBIL (cm)	−5.68	−2.13	−2.51	−3.05	—	−2.17	—	−4.08
LIBN (number)	−2.29	−1.61	—	−4.30	—	—	—	—
PEDS (%)	1.9	14.3	—	2.4	2.0	28.0	0.1	2.4
STAM (%)	−40.6	−34.8	−2.1	−65.8	—	−23.9	−0.5	—
YOKE (1–5 scale)	0.75	0.74	—	2.07	—	2.35	—	1.92

Effects are reported as the change in trait units observed by substituting two maize alleles for two teosinte alleles at the marker locus. —, signifies no significant effect observed. Mean trait values for teosinte and maize (Table 8) provide a context for the interpretation of the magnitude of these phenotypic effects.

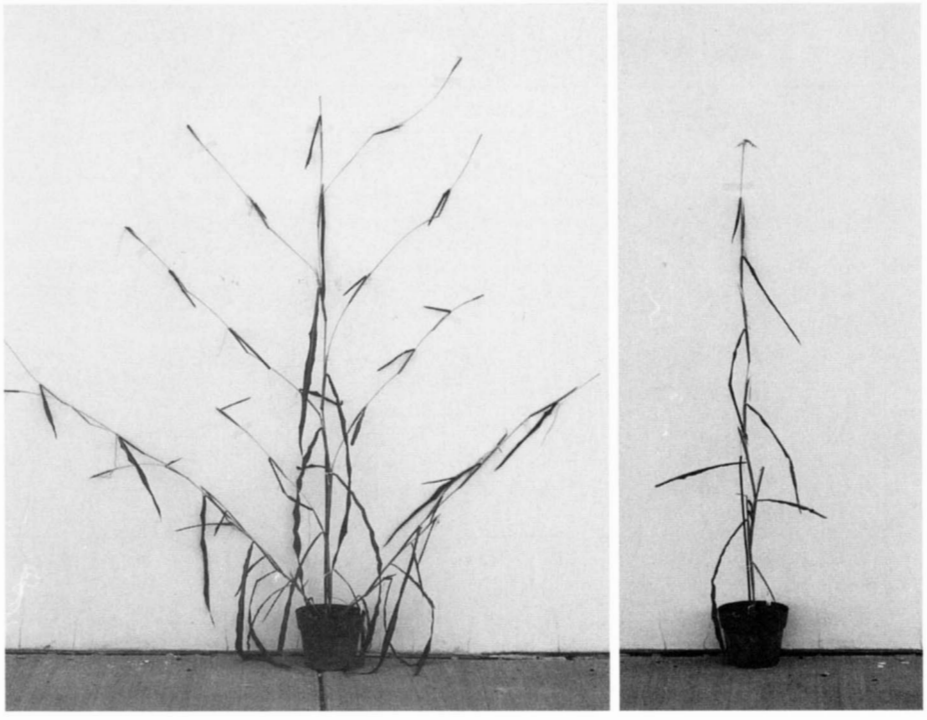


FIGURE 4.—Plants from the T-M1 population. Plants homozygous for the teosinte (left) and maize (right) alleles at the marker loci in the target region on chromosome arm *1*L are shown. These plants demonstrate how QTL-1L^{M} severely reduces lateral branch length. These plants were grown in a growth chamber separately from the main population that was grown in a nursery in Hawaii.

inflorescences that were part tassel and part ear (Figure 6). The proportions of these two phenotypic classes fit the 3:1 and 1:1 segregation ratios expected for our two complementation tests if QTL-1L^{T} failed to complement *tb1-ref* (Table 5). Although none of the progeny had a strong teosinte-branched phenotype, since the appropriate proportions of the progeny in both tests showed a weak teosinte branched phenotype, we conclude that QTL-1L^{T} is allelic to *tb1-ref*, but that QTL-1L^{T} represents a weak, semiquantitative allele (*tb1+teosinte*) relative to the qualitative maize mutant (*tb1-ref*).

QTL on chromosome arm *3*L: Chromosome arm *3*L has an effect on all traits except LIBN in at least two of the four populations analyzed (Tables 3 and 4). As with chromosome arm *1*L, we will consider the effects on all traits to represent the action of a single QTL, namely QTL-3L with alleles QTL-3L^{M} and QTL-3L^{T} for maize and teosinte, respectively. Again, differences in the population sizes complicate interpretations of the absence of a significant phenotypic effect in one population relative to another; however, the absence and reduction of some effects do not appear to be statistical artifacts. For example, DISA was invariant in the M-T3 population (all ears were fully non-disarticulating), indicating that QTL-3L^{T} has no effect on this trait in maize background. PEDS shows a much reduced effect in both maize and teosinte backgrounds relative to the F_2 population. Below, we will give evidence that the effect on PEDS is influenced by epistasis (a primary source of genetic background effects), and thus this reduction in its expression is not simply an artifact of differential sampling. Similarly, there is a large effect on cupule yoking (YOKE) in the F_2 and T-M1+3 populations but no significant effect in either maize or teosinte backgrounds (Table 4). Other evidence for the importance of background for QTL-3L is that it has a large effect on inflorescence sex (STAM) in the F_2 population, but only a negligible or no effect in both teosinte and maize backgrounds (Table 4). Overall, this QTL has its greatest effects in the F_2 population and shows reduced phenotypic expression when moved into either maize or teosinte background. These observations suggest that QTL-3L is influenced by epistatic interactions with other loci (see below).

QTL-3L^{M} alters the morphology of the plant in a manner similar to that for QTL-1L^{M}, namely it produces a larger number of shorter internodes in both the primary lateral branch and inflorescence (Table 4). Thus, both QTL affect a common developmental process. Plants of the T-M3 population reveal how QTL-3L^{M} may have altered ear morphology during the early evolution of maize (Figure 7). The effects are slight and seem almost trivial. QTL-3L^{M} renders the cupulate fruitcases

TABLE 5

Complementation tests with *teosinte branched1*

Cross	Expected ratio	Progeny phenotype[a]	
		Normal maize	*tb1*-like
W22: QTL-1L$^{T/M}$ × Tb1/tb1	3:1	57	15
W22: QTL-1L$^{T/M}$ × tb1/tb1	1:1	56	48

[a] The number of progeny in each phenotypic class is indicated. The observed ratios do not differ significantly from those expected [χ^2 = 0.67 (3:1) and 0.62 (1:1), $P > 0.25$].

FIGURE 5.—Immature ears from plants of the T-M1 population. Ears from plants homozygous for the teosinte (left) and maize (right) alleles at the marker loci in the target region on chromosome arm *1*L are shown. These ears demonstrate how QTL-1L^{M} alters ear morphology by producing some yoking of the cupulate fruitcases and a larger number of fruitcases. These plants were grown in a growth chamber separately from the main population that was grown in a nursery in Hawaii. Bar, 1 cm.

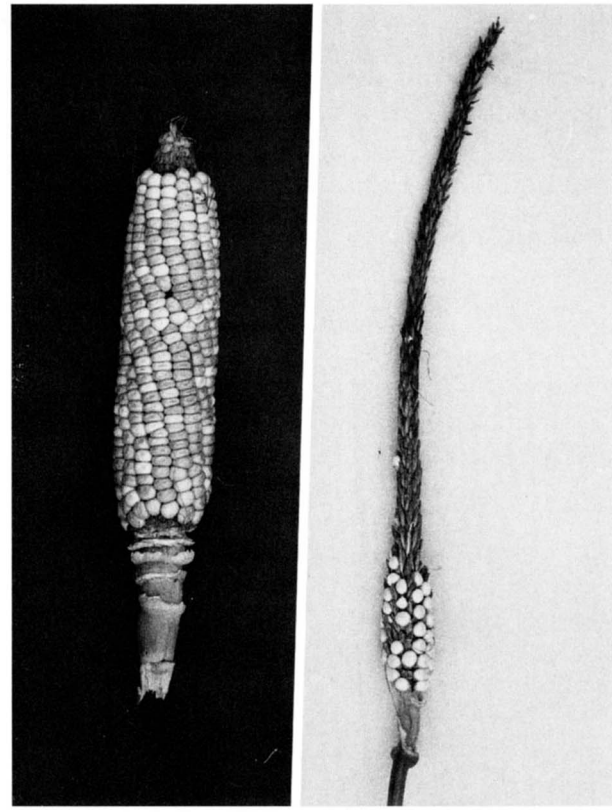


FIGURE 6.—Inflorescences terminating the primary lateral branch from the complementation test discussed in the text. A female inflorescence (ear) showing the phenotype of the *Tb1+Maize* allele (left) and a mixed male-female inflorescence showing the phenotype of a *tb1-ref/tb1+teosinte* plant (right).

shorter or plumper and increases the number of fruitcases per ear. There is very little effect on ear disarticulation, cupule yoking, or the overall appearance of the ear (Figure 7; Table 4).

The effects associated with QTL-3L on all of the traits except one in the T-M3 and M-T3 populations map to the middle of the target region near *te1* (Figure 1). The one exception is DISA in the T-M3 population that maps near *UMC60*. The effect on DISA was rather small (Table 4), and thus the estimation of its position is subject to greater variance.

Genetic background and gene action: The fact that we studied the QTL in teosinte, F_2 and maize backgrounds enabled us to assess the effects of genetic background on gene action. This was done by calculating the dominance/additivity ratio for each trait as affected by each QTL. This ratio will equal −1.0 when the maize allele is fully recessive, 0.0 when additive and +1.0 when fully dominant. In the six cases where a significant effect on a trait was seen in both maize and teosinte backgrounds, the *d/a* ratio is always larger in maize background (Table 6). For QTL-1L, the mean *d/a* ratio is smallest in teosinte background, intermediate in the F_2, and largest in maize background. For QTL-3L, the maize allele shows its least dominance in teosinte background, its greatest dominance in the F_2, and intermediate dominance in maize background. As a rough guide to the significance of these numbers, we regressed the *d/a* ratios onto the percentage of maize germplasm in the population (0% for T-M1 and T-M3, 50% for the F_2, and 100% for M-T1 and M-T3). This test suggests a significant association ($R^2 = 0.26$, $P < 0.01$).

The effect on the presence of the PEDS shows the greatest change in gene action among the populations (Table 6). QTL-3L^{M} is almost fully recessive for PEDS in teosinte background and almost fully dominant in maize background. Similarly, QTL-1L^{M} is almost fully recessive for PEDS in teosinte background but partially dominant in the F_2. This might reflect the fact that epistasis plays a significant role in the expression of

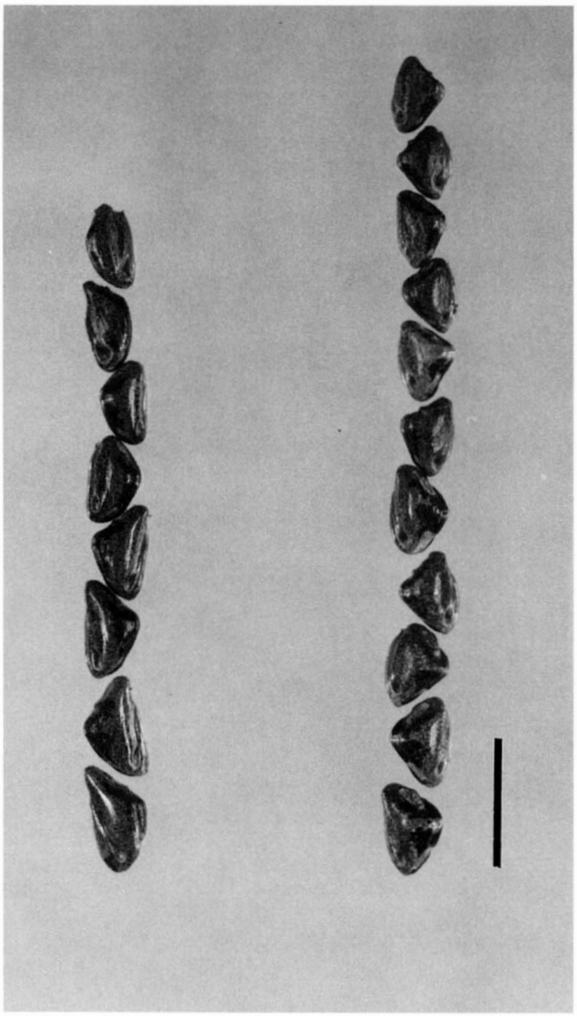


FIGURE 7.—Mature ears from the T-M3 population. Ears from plants homozygous for the teosinte (left) and maize (right) alleles at the marker loci in the target region on chromosome arm 3L are shown. These ears demonstrate how QTL-3L^M alters ear morphology by producing somewhat shorter (or plumper) cupulate fruitcases and a larger number of fruitcases in the ear. Bar, 1 cm.

PEDS. The situation for inflorescence sex (STAM) is similar, with the maize allele at both QTL showing full dominance in maize background.

The T-M1+3 population in which both target regions were segregating enabled us to ask if each of these regions affected the type of gene action exhibited by the other. We calculated the mean d/a ratio of the traits for QTL-1L when QTL-3L was homozygous for the teosinte ($d/a = -0.40$) and maize ($d/a = -0.08$) alleles. Similarly, we calculated the mean d/a ratio for QTL-3L when QTL-1L was homozygous for the teosinte ($d/a = -0.44$) and maize ($d/a = -0.15$) alleles. In both cases, the maize allele at one QTL shows greater dominance when the plants are fixed for the maize allele at the second QTL.

Epistasis: In our first QTL study of a maize-teosinte F_2 population, we detected a significant epistatic interaction between QTL-1L and QTL-3L for the presence of the pedicellate spikelet (DOEBLEY and STEC 1991). In our second study involving different types of maize and teosinte, we did not detect significant epistasis between these QTL, although the results were just below the level of statistical significance, $P = 0.08$ (DOEBLEY and STEC 1993, unpublished). The combined results of these two F_2 populations were suggestive but did not provide compelling evidence that these two QTL interact epistatically.

The T-M1+3 population was constructed to test whether QTL-1L and QTL-3L interact epistatically. Because this population has a uniform teosinte genetic background, other segregating QTL should not interfere with the detection of epistasis as they could in a maize-teosinte F_2 population. Analysis of this population confirms that these two QTL interact epistatically to control PEDS and YOKE (Table 7). For PEDS, the R^2 for epistasis is nearly as large as the R^2 for the main effect associated with *BV302* and larger than the R^2 for the main effect associated with *UMC107*. For YOKE, the epistatic variance is smaller. The interaction term from

TABLE 6

Dominance/additivity ratios for QTL

	Chromosome arm (marker locus)					
	1L (*UMC107*)			3L (*BV302*)		
	Population					
Traits	T-M1	R×P F_2	M-T1	T-M3	R×P F_2	M-T3
CUPL	0.20	0.33	0.23	−0.23	0.35	−0.20
CUPR	0.03	0.39	0.53	−0.39	0.45	—
DISA	—	—	—	−0.62	0.36	—
INNO	—	−0.40	0.23	—	0.25	−0.35
LBIL	−0.44	0.60	0.24	—	0.43	—
LIBN	0.52	0.28	—	—	—	—
PEDS	−0.84	0.23	—	−0.80	0.64	0.71
STAM	0.44	0.52	1.00	—	0.36	0.99
YOKE	−0.28	0.08	—	—	—	—
Mean	−0.05	0.25	0.45	−0.51	0.41	0.29

TABLE 7

Epistatic interactions between QTL

	Main effects		Interaction	Contrasts[a]			
Trait	*UMC107*	*BV302*	*UMC107* × *BV302*	A × A	D × D	D × A	A × D
PEDS	8.6	13.3	12.3	4.0	(1.5)	1.8*	3.5*
YOKE	30.1	27.9	3.4	2.9	(0.0)	(0.9)	(0.0)

R^2 values expressed as percentages for the individual QTL, the interaction between them, and the four contrasts are shown. Values in parentheses are not statistically signficant. * signifies $P = 0.05$, otherwise $P = 0.01$. R^2 values for the main effects here and in Table 3 differ slightly because of missing data.

[a] A, additive; D, dominance.

the analyses of variance approached significance ($P < 0.20$) for four additional traits (INNO, LBIL, LBIN and STAM), suggesting that with a larger population size further evidence for epistasis may have been observed.

Another way of visualizing the contribution from epistasis is to look at the effects of substituting two maize for two teosinte alleles at each QTL when the other QTL is homozygous teosinte in the T-M1+3 population. One can then compare the sum of these values to the effect of substituting maize for teosinte alleles at both QTL simultaneously. For example, QTL-1L$^{M/M}$ plants have an average of 1.1% more pedicellate spikelets in their ears (PEDS) than do QTL-1L$^{T/T}$ plants among plants that are homozygous teosinte at QTL-3L (Table 8). Similarly, QTL-3L$^{M/M}$ have 0.8% more pedicellate spikelets than QTL-3L$^{T/T}$ plants among plants that are homozygous teosinte at QTL-1L. Thus, without epistasis, these two QTL should have a combined effect of 1.9%. The actual combined effect is a 7.3% increase, suggesting that there is a much greater contribution from the interaction than from the main effects of the individual QTL. Similarly for YOKE, the individual effects are 1.1 and 0.4 (on a 1–5 scale) for an expected total main effect of 1.5 without epistasis (Table 8). However, the actual combined effect of both QTL is 3.5, indicating the contribution from epistasis exceeds the total main effects of the two individual QTL.

The fact that these two QTL show epistasis is not surprising given that both affect a common developmental pathway that regulates the number and length of internodes. Plants from the T-M1+3 population demonstrate how these two QTL can radically transform the teosinte ear from one that is fully disarticulating and without any yoking to one that is fully nondisarticulating and with all yoked cupules (Fig. 8).

DISCUSSION

Teosinte branched1: *tb1-ref* is a recessive, null or loss of function mutant that produces plants with long lateral branches tipped by tassels at some upper nodes of the main culm and tillers at the basal nodes (BURNHAM 1959; VEIT *et al.* 1993; IRISH *et al.* 1994). This contrasts with normal maize plant architecture conferred by the dominant maize allele (*Tb1+Maize*): short lateral branches tipped by ears at some upper nodes and few or no tillers at the basal nodes. Since both tillers (basal lateral branches) and upper lateral branches arise from

TABLE 8

Mean trait values for the T-M1+3 population

		Genotypic classes								
	UMC107	*TT*	*TT*	*TT*	*MT*	*MT*	*MT*	*MM*	*MM*	*MM*	
	BV302	*TT*	*MT*	*MM*	*TT*	*MT*	*MM*	*TT*	*MT*	*MM*	
Trait	Teosinte										Maize
CUPL (mm)	11.9	11.5	10.7	8.7	10.7	9.1	7.4	8.9	7.8	6.5	3.9
CUPR (number)	4.6	6.1	7.1	8.3	7.7	9.2	10.5	9.2	10.6	12.4	44.5
DISA (1–5 scale)	1.0	1.2	1.8	2.4	1.7	2.9	3.6	2.9	3.9	4.8	5.0
INNO (number)	2.7	2.7	2.6	3.0	2.4	3.0	3.1	3.0	3.2	3.4	9.7
LBIL (mm)	173.4	101.7	66.5	61.1	83.6	47.6	40.9	47.8	54.6	18.0	6.9
LIBN (number)	5.2	4.9	4.4	5.9	0.6	2.4	2.5	0.6	0.6	0.0	0.0
PEDS (%)	0.0	0.0	0.0	0.8	0.3	0.2	1.3	1.1	0.4	7.3	100.0
STAM (%)	94	94	72	72	38	49	40	10	15	5	0
YOKE (1–5 scale)	1.0	1.0	1.1	1.4	1.1	2.0	3.2	2.1	3.1	4.5	5.0

The mean trait values for the race Reventador maize and *Z. mays* ssp. *parviglumis* teosinte parents are based on plants grown in a different year (DOEBLEY and STEC 1993). Thus, they provide only a rough guide for comparison with the mean values of the different genotypic classes from the T-M1+3 population.

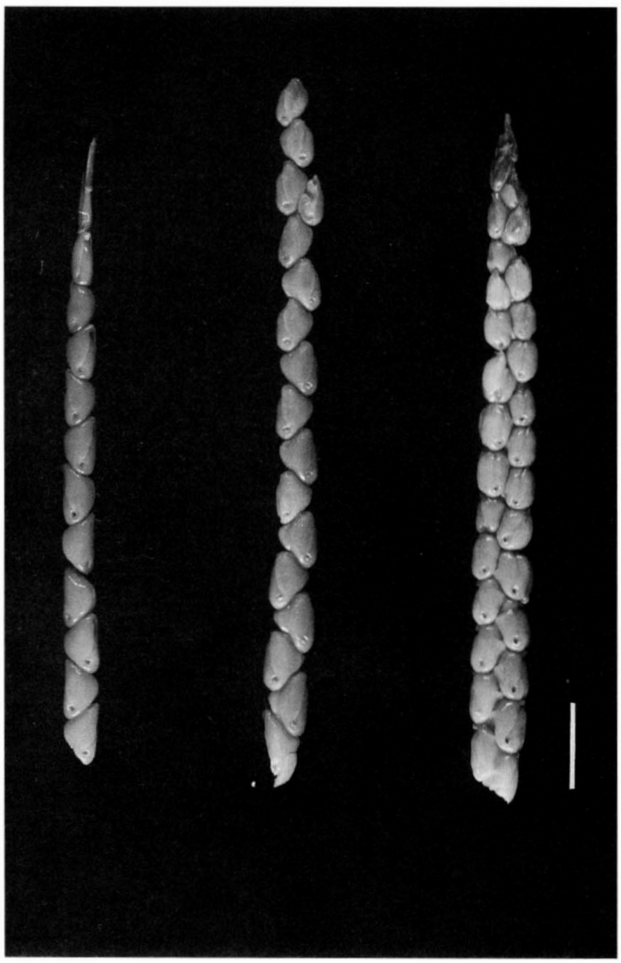


FIGURE 8.—Immature ears from the T-M1+3 population. Ears from plants homozygous for the teosinte (left) and maize (right) alleles at the marker loci in both target regions on chromosome arms *1*L and *3*L, and one ear from a plant heterozygous for the marker loci on *1*L and homozygous for teosinte alleles for the marker loci on *3*L (center) are shown. These ears demonstrate the dramatic effect that the combination of the maize alleles at QTL-1L and QTL-3L has on ear morphology by producing a non-disarticulating ear with fully yoked cupulate fruitcases and twice the number of fruitcases. Bar, 1 cm.

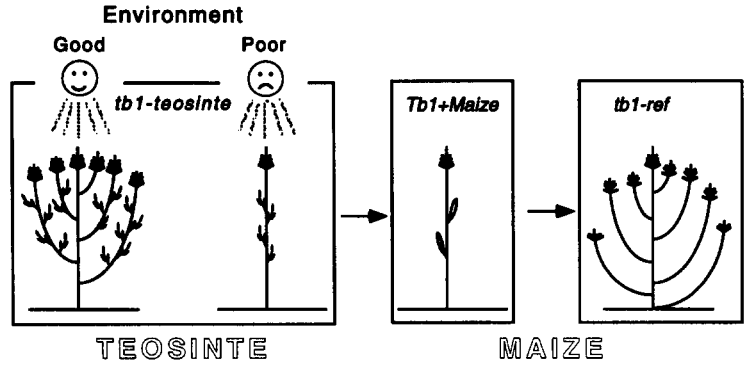
axillary meristems, in a general sense, *tb1* governs the fate of the axillary meristems. As discussed above, QTL-$1L^T$ transforms upper lateral branches in a manner similar to *tb1-ref.* QTL-$1L^T$ was also found to promote tillering in another study (DOEBLEY and STEC 1991), although we did not measure tillering in the populations analyzed for this paper.

The complementation tests indicate that *tb1-ref* and QTL-$1L^T$ (= *tb1+teosinte*) are allelic. Although complementation tests cannot provide absolute proof that two mutants are allelic, they offer powerful evidence for that conclusion. The morphology of the *tb1-ref/tb1+teosinte* plants showed only a weak or intermediate *tb1*-like phenotype (Figure 6), indicating that *tb1+teosinte* is a weak "mutant" allele. The intermediate nature of *tb1+teosinte* suggests that the fully dominant maize allele (*Tb1+Maize*) evolved from *tb1+teosinte* by enhancement of the ability to suppress axillary meristems, as might be accomplished by overexpression of the gene product. Thus, *tb1-ref* (loss of function) engenders a strong teosinte branched phenotype, *tb1+teosinte* (weak function) a partial teosinte branched phenotype, and *Tb1+Maize* (enhanced function) the normal maize phenotype.

A model for *tb1*: Plants of many species can respond to unfavorable environmental conditions (such as a high level of competition from surrounding vegetation, shading, and restricted moisture) by growing into slender unbranched plants (strong apical dominance), or correspondingly they can respond to favorable local environmental conditions by growing into robust highly branched plants (weak apical dominance). Based on the observations of one of us (J.D.), teosinte also appears capable of this same type of plastic response to local environment where it grows naturally in Mexico. Given the phenotype it produces, it is easy to imagine that *tb1* is involved in regulating this response by specifying the fate of axillary meristems. Thus, we propose the following model for the function of *tb1* in teosinte (Figure 9). Under favorable environmental conditions, *tb1+teosinte* is turned off, allowing axillary meristems to develop fully into tillers or long lateral branches tipped by tassels. Under unfavorable conditions, *tb1+teosinte* is turned on so that the plant produces few or no tillers and only short lateral branches tipped by ears. Thus, *tb1* is hypothesized to be a locus involved in the plastic response of the teosinte plant to its local environment by altering plant architecture.

This model can be extended to explain the evolution of maize plant architecture by hypothesizing that in maize the expression of *tb1* is no longer tied to an environmental signal (*e.g.*, degree of shading) but rather that *Tb1+Maize* is expressed during the development of the branches in all environments, keeping both tillering and full elongation of the upper lateral branches repressed (Figure 9). Under this model, both the *tb1+teosinte* and *Tb1+Maize* alleles would encode functional products, although ones that are differentially regulated. *tb1+teosinte* is more or less recessive to *Tb1+Maize* in maize background because the latter will be expressed whether or not the former allele is activated by an environmental signal. Finally, under this model, the maize mutant (*tb1-ref*) can be explained as a recessive loss of function allele. With complete loss of function, the axillary meristems of homozygous *tb1-ref* plants elongate to produce either basal tillers or elongate upper lateral branches tipped by tassels (Figure 9).

Candidates for QTL-3L: There are two known mutant loci that map near QTL-3L and that have phenotypes suggesting that one of them could be this QTL. First, *terminal ear1* is a recessive mutant on chromosome arm *3*L that affects internode elongation in the main vegetative culm and sex expression in the tassel. Mutant



tb1	off	on	on	lost
regulation	environmental		constitutive	---
repression	no	yes	yes	no

FIGURE 9.—Model for the function of *teosinte branched1* in teosinte and maize (see text).

te1-ref plants have a larger number of shorter internodes in the vegetative culm of the plant (MATTHEWS *et al.* 1974; VEIT *et al.* 1993; IRISH *et al.* 1994). *te1-ref* also typically converts the basal portion of the terminal tassel into an ear, the feature after which the locus was named. *te1-ref* has no reported effects on the internodes of the ear or primary lateral branch (ear shank). A model considered by B. VEIT (personal communication) is that *Te1* functions to restrict the initiation of lateral organs (leaves) such that when this function is lost, the plant produces a larger number of leaves and the nodes to which they are attached.

Like *te1-ref,* QTL-3L^{M} affects internode number and length and sex expression but in a spatially different pattern. Specifically, this QTL effects the numbers and lengths of internodes in the ear and the primary lateral branch (not the main stalk), as well as the sex of the primary lateral inflorescence (not the terminal tassel on the main culm). Based on these phenotypic effects and the map positions of *te1* and QTL-3L (Figure 1), *te1* can be considered a candidate for QTL-3L. If *te1* is QTL-3L, then *te1* should have some effect on the numbers and lengths of internodes in the ear and the primary lateral branch of maize and/or teosinte. These effects may be difficult to detect in maize because its internodes in both the ear and ear shank are already very short.

A second candidate is *tassel replaces upper-ear1* (*tru1*), which has a phenotype somewhat similar to *tb1* (SHERIDAN 1988). In *tru1* plants, the ears and ear shanks at the upper nodes of the plant are replaced by long lateral branches tipped by tassels, although the lower nodes produce ears. *tru1* maps in or near our target region on chromosome arm 3L (W. F. SHERIDAN, personal communication). The phenotype of *tru1* is similar to that of QTL-3L^{T}, making it an attractive candidate locus for our QTL.

Pleiotropy *vs.* linkage: The two target regions we have analyzed each affect a number of different aspects of plant morphology (Table 4). These multiple effects indicate either pleiotropy or linkage of several QTL each affecting different traits. We prefer the former interpretation because the traits involved are often simultaneously affected by single pleiotropic mutants in maize and other plants. The most relevant examples are *tb1-ref,* which affects internode lengths (CUPL and LBIL) and inflorescence sex and structure (STAM and LIBN), and *te1-ref,* which affects internode lengths (CUPL and LBIL) and numbers (CUPR and INNO) and inflorescence sex (STAM). Ethylene response mutants of Arabidopsis affect both internode elongation (CUPL and LBIL) and abscission layer formation (DISA) (KIEBER and ECKER 1993). Finally, *suppressor of sessile spikelets1* of maize affects both branching in the inflorescence (LIBN) and the presence of single *vs.* paired spikelets in the ear (PEDS) (DOEBLEY *et al.* 1995a). Thus, there is ample precedent for extensive pleiotropy of the nature we envision.

Genetic background and dominance: The expression of our two QTL is affected by genetic background. QTL-1L^{M} shows strong expression for most traits in teosinte background, while the effects of QTL-1L^{T} on several traits are absent or much reduced in maize background. The strong expression in teosinte background indicates that selection for QTL-1L^{M} during the early evolution of maize should have been highly effective. The strong expression in teosinte background also confirms that this QTL represents a locus of large effect. Loci of large effect have not been considered an important force in evolution by many authors (see ORR and COYNE 1992). In teosinte background, QTL-1L^{M} increases the mean number of seeds per ear (2 × CUPR) from 12 to 18, a 50% increase (Table 8). Similarly, it reduces the mean length of the lateral branch (INNO × LBIL) from 28 to 14 cm, a 50% decrease. Both of these qualify as large effects.

QTL-3L has a different dynamic in that it shows weaker expression in both maize and teosinte backgrounds as compared to the R×P F_2 population. For some traits, the effect of this QTL is strongly enhanced when in combination with QTL-1L as a result of epistasis. Its reduced effects on other traits in maize and teo-

sinte background could result from epistatic interactions with other loci that we have not analyzed. Nevertheless in teosinte background, QTL-3L^M increases the mean number of seeds in the ear from 12 to 17 (42%), which could provide enough of an additive effect to allow for effective selection during the early evolution of maize (Table 8).

Another indication of the importance of genetic background on QTL behavior was the observation that the maize alleles at the two QTL exhibit greater dominance in maize background than in teosinte background. This change in gene action could have resulted from selection during the domestication process for modifier loci that enhanced the expression of the trait in the heterozygote. This mechanism would only work if the QTL remained polymorphic within the population under selection so that there were heterozygotes upon which selection for the dominance modifiers could be applied, or if there were hybridization between populations with and without the maize allele at the QTL such that new heterozygotes were generated even after the maize allele was brought to fixation in one population. A second mechanism to explain the change in dominance would be as a byproduct of selection for alleles at other loci that enhance the expression of the traits controlled by the QTL. Experimental work in Drosophila has shown that selection of this nature on the homozygotes can modify the dominance properties of a gene (THOMPSON and THODAY 1972).

Epistasis: QTL mapping experiments conducted over the last several years have typically uncovered little evidence for epistasis, suggesting that for natural polygenes it is uncommon (TANKSLEY 1993). Nevertheless, the methodological limitations of QTL mapping, such as the small population sizes typically employed and recombination between the marker loci and the QTL, require one to exercise some caution in accepting this evidence. Another methodological problem is the limitation of the analysis of variance for detecting interaction effects (WADE 1992). In a simulation study where a factor **A** had a small negative main effect but a large positive effect due to interactions with two other factors, the analysis of variance incorrectly determined the main effect of **A** to be positive and failed to detect the large interaction in over 80% of the trials (WADE 1992). These results undermine conclusions one might draw about the frequency and strength of epistasis based on the analysis of variance in QTL mapping studies.

One could view the results of our current attempt to detect epistasis as either the glass half full or half empty. We have detected significant epistasis for two of nine traits. The loss or reduction in expression for some other traits when QTL are transferred into maize or teosinte background also suggests a role for epistasis. Nevertheless, both QTL retain large main effects on some traits regardless of genetic background, suggesting that interactions with other genes are not always important. Even for the presence of the pedicellate spikelet that showed a large epistatic effect, we detected significant (although reduced) main effects when the individual QTL were isolated in maize or teosinte background. Still overall, as compared to our failure to clearly detect epistasis between these two QTL in the R×P F_2 population, we conclude that epistasis is more important than one would be led to believe from the F_2 study alone. As discussed by TANKSLEY (1993), it will be necessary to employ nearly isogenic lines to allow more precise measurement of epistasis.

Our results indicate an important role for epistasis in the inheritance of paired *vs.* single spikelets. For PEDS in the R×P F_2 population, QTL-1L and QTL-3L had a combined effect (including their interaction) of 60%, *i.e.,* converting an ear with 0% paired spikelets to one with 60% paired spikelets. When transferred to teosinte background, these two QTL have a combined effect of only 7.3% including the interaction. This nearly 10-fold reduction in the effects of these two QTL in teosinte background suggests that higher order epistatic interactions are involved in the genetic control of this trait and that the estimates of the effects of QTL are strongly dependent on the context in which they are analyzed.

Evolution of maize: There have been several attempts to integrate genetic and morphological data to form a general explanation for the origin of the maize ear. One view proposes that about five major genes were involved and that each controlled one key trait. For example, one gene would control disarticulating *vs.* non-disarticulating ears, one single *vs.* paired spikelets, one distichous *vs.* polystichous phyllotaxy, and one soft *vs.* hard glumes (BEADLE 1939, 1980; LANGHAM 1940; GALINAT 1971, 1978). There have been various versions of this general view over the years, but its essence has remained the same. This view focuses mostly on ear architecture, largely ignoring the differences in overall plant architecture between maize and teosinte.

A second view holds that the major genes of BEADLE (1939) and LANGHAM (1940) differentiating maize and teosinte "do not exist" and that the differences are controlled by polygenes "unlikely now to be identified individually" (ILTIS 1983). Morphologically, this view proposes that the ear of maize arose by the "catastrophic" feminization of the tassel terminating the primary lateral branch. Under this view, soft glumes and paired spikelets were the automatic byproducts of feminization and thus not under genetic control separate from feminization. In a sense, this view focuses mostly on plant architecture, treating some key changes in ear structure as byproducts of a change to short lateral branches tipped by feminized tassels.

The results of our QTL mapping analyses are consistent with the view that a few major identifiable genes largely control the differences between maize and teosinte. In fact, we demonstrated that one of the QTL

represents a single major locus (*tga1*) that in essence controls hard *vs.* soft glumes (DORWEILER *et al.* 1993). A second of our QTL seems to represent a single locus that largely controls distichous *vs.* polystichous phyllotaxy (DOEBLEY and STEC 1993). Both of these QTL conform well to the model under which a single gene controls a single trait. The two QTL analyzed in this paper also appear to represent the type of major loci invoked by BEADLE and LANGHAM, but they do not as easily fit the "one gene-one trait" model (BEADLE 1972, 1980).

Results of the present study indicate that the difference in plant architecture between teosinte and maize is largely controlled by *tb1*. In teosinte background, this locus controls a switch from long primary lateral branches tipped by tassels to short ones tipped by essentially normal teosinte ears with hard cupulate fruitcases and mostly single spikelets (Figures 4 and 5). The resulting ears are altered to be less than fully disarticulating and the length of the internodes in the ears is reduced. Nevertheless, the structure is clearly a teosinte ear and not a feminized tassel. It is partly on this basis that our results fail to support the view that the maize ear arose as a feminized tassel (ILTIS 1983). QTL-3L enhances the effects of *tb1*, and together these two loci substantially transform both ear and plant architecture. In the T-M1+3 population, substitution of maize for teosinte alleles at both QTL radically transforms the plant (Table 8). On average, the primary lateral inflorescence changes from 94% male to 95% female, the number of cupules in the ear (2 × CUPR) from 12 to 25, the degree of cupule yoking from 0% to 88%, the extent of non-disarticulation from 0% to 95%, the percentage paired spikelets from 0% to 7.3%, and the length of the lateral branch (INNO × LBIL) from 28 to 6 cm. Thus, with the exception of paired spikelets, these two QTL produce a nearly complete transformation from teosinte to maize plant architecture and dramatically alter the ear.

The two QTL analyzed in this paper transform plant architecture and produce an ear as shown in Figure 8. However, additional steps are needed to change this modified teosinte ear into a maize ear. First, one would need to incorporate the maize allele for *tga1* to block the formation of the cupulate fruitcase and provide softer glumes (DORWEILER *et al.* 1993). Second, the maize allele for the QTL on chromosome arm 2S would provide a polystichous arrangement of the cupules (DOEBLEY and STEC 1993). Thus, a total of four loci could be sufficient to go most of the way from teosinte to a plant that has most of the key features of cultivated maize. The only additional changes required would be those providing fully paired spikelets.

Implications for plant evolution: The processes involved in evolution under domestication are not fundamentally different from those operating during evolution under natural selection. For this reason, studies of crop evolution can reveal processes operative in plant evolution in general. Several results of the present study may apply more broadly. As in our previous work, the analyses presented here indicate that genes of large effect can be an important force in morphological evolution (HILU 1983; GOTTLIEB 1984; ORR and COYNE 1992). This is especially true for *tb1*, which the present study indicates was largely responsible for the changes in plant architecture. Similarly, the combined effects of alleles at only two QTL transform the ear extensively (Figure 8). The differences in ear structure among the wild teosintes (see WILKES 1967) are minute in comparison to the change conferred by the maize alleles of these two QTL. Had such a difference occurred in nature, it would be judged sufficient by taxonomists to name a new genus. This provides further evidence that a few genes can induce a major morphological shift. Other recent studies of natural species provide similar evidence that genes of large effect can be involved in species differentiation (VLOT *et al.* 1992; COYNE *et al.* 1994; VAN HOUTEN *et al.* 1994).

Another issue concerns the role of epistasis and the influence of genetic background on QTL expression. QTL-3L^{M} has rather modest effects in teosinte background even when homozygous (Figure 7). This maize allele could probably exist as a natural variant in a teosinte population. Some forms of teosinte may provide backgrounds in which its effects are almost entirely suppressed. If this is true, then hybridization among teosinte populations would produce new combinations of such cryptic alleles and rapidly generate novel phenotypes, even where one sees little phenotypic differentiation among populations. Such a mechanism would be consistent with the shifting balance theory of WRIGHT (1969) with its emphasis on adaptive gene complexes and interdemic migration. In Arabidopsis, such a cryptic locus (*cauliflower*) has recently been discovered that in combination with a standard major mutant (*apetala1*) radically transforms the inflorescence into a cauliflower-like mass of undifferentiated flowers, despite the fact that the *cauliflower* locus has no discernible effects of its own (BOWMAN *et al.* 1993).

Finally, although the two QTL analyzed in this paper and *tga1* studied previously (DORWEILER *et al.* 1993) have large effects, they are not the types of amorphic or complete loss of function alleles commonly studied in genetics. We draw this conclusion from the general intermediacy of the heterozygotes for these QTL. Rather, these QTL represent modification of function mutants. Under our model, *Tb1+Maize* can be classified as a hypermorphic allele with increased expression of the normal *tb1+teosinte* function. In contrast, if loss of function at *te1* causes more frequent initiation of internodes, then QTL-3L^{M}, as a putative allele of *te1*, could be classified as a reduced function or hypomorphic allele. Finally, *tga1*, which disrupts the normal development of the cupulate fruitcase, could be classified as an antimorphic allele. Although these classifications

require some vigorous arm waving, they can serve to guide further research. They also reaffirm the intuitive conclusion that evolution proceeds more often by the modification of rather than the elimination of existing functions.

We thank RUTH SHAW, TOM OSBORN, JANE DORWEILER, RON PHILLIPS, and two anonymous reviewers for their comments on this paper; FRANK ENFIELD, ZHAO-BANG ZENG and CHEN-HUNG KAO for help with the analysis of epistasis; and BRUCE VEIT and SARAH HAKE for providing the molecular clone of *te1* (BV302). This research was supported by the National Science Foundation grant BSR-9107175, USDA-NRI Competitive grant 93-37300-8773, and the Minnesota Agricultural Experiment Station. This paper is publication number 21,788 of the Minnesota Agricultural Experiment Station.

LITERATURE CITED

BEADLE, G. W., 1939 Teosinte and the origin of maize. J. Hered. **30:** 245–247.

BEADLE, G. W., 1972 The mystery of maize. Field Mus. of Nat. Hist. Bull. **43:** 2–11.

BEADLE, G. W., 1980 The ancestry of corn. Sci. Amer. **242:** 12–119, 162.

BEAVIS, W., and D. GRANT, 1991 A linkage map based on information from four F_2 populations of maize (*Zea mays* L.). Theor. Appl. Genet. **82:** 636–644.

BOWMAN, J. L., J. ALVAREZ, D. WEIGEL, E. M. MEYEROWITZ and D. R. SMYTH, 1993 Control of flower development in *Arabidopsis thaliana* by *APETALA1* and interacting genes. Development **119:** 721–743.

BURNHAM, C. R., 1959 *teosinte branched.* Maize Genet. Coop. Newsl. **33:** 74.

BURNHAM, C. R., and P. YAGYU, 1961 Linkage relations of teosinte branched. Maize Genet. Coop. Newsl. **35:** 87.

BURR, B., F. A. BURR, K. H. THOMPSON, M. C. ALBERTSON and C. W. STUBER, 1988 Gene mapping with recombinant inbreds in maize. Genetics **118:** 519–526.

COCKERHAM, C. C., 1954 A extension of the concept of partitioning hereditary variance for analysis of covariance among relatives when epistasis is present. Genetics **39:** 859–882.

COYNE, J. A., A. P. CRITTENDEN and K. MAH, 1994 Genetics of a pheromonal difference contributing to reproductive isolation in *Drosophila.* Science **265:** 1461–1464.

DOEBLEY, J., 1993 Genetics and the morphological evolution of maize, pp. 66–78 in *The Maize Handbook,* edited by M. FREELING and V. WALBOT. Springer-Verlag, New York.

DOEBLEY, J., and A. STEC, 1991 Genetic analysis of the morphological differences between maize and teosinte. Genetics **129:** 285–295.

DOEBLEY, J., and A. STEC, 1993 Inheritance of the morphological differences between maize and teosinte: comparison of results for two F_2 populations. Genetics **134:** 559–570.

DOEBLEY, J., A. STEC, J. WENDEL and M. EDWARDS, 1990 Genetic and morphological analysis of a maize-teosinte F_2 population: implications for the origin of maize. Proc. Natl. Acad. Sci. USA **87:** 9888–9892.

DOEBLEY, J., A. STEC and B. KENT, 1995a *Suppressor of sessile spikelets1* (*sos1*): a dominant mutant affecting inflorescence development in maize. Am. J. Bot. **82:** 571–577.

DOEBLEY, J., P. VANDER WEIL and A. STEC, 1995b Map position of *teosinte branched1.* Maize Genet. Coop. Newsl. **69:** (in press).

DORWEILER, J., A. STEC, J. KERMICLE and J. DOEBLEY, 1993 *Teosinte glume architecture1:* a genetic locus controlling a key step in maize evolution. Science **262:** 233–235.

EDWARDS, M. D., C. W. STUBER and J. F. WENDEL, 1987 Molecular-marker-facilitated investigations of quantitative-trait loci in maize. I. Numbers, genomic distribution and types of gene action. Genetics **116:** 113–125.

GALINAT, W. C., 1969 The evolution under domestication of the maize ear: string cob maize. Mass. Agric. Exp. Stn. Bull. **577:** 1–19.

GALINAT, W. C., 1971 The origin of maize. Annu. Rev. Genet. **5:** 447–478.

GALINAT, W. C., 1978 The inheritance of some traits essential to maize and teosinte, pp. 93–111 in *Maize Breeding and Genetics,* edited by D. B. WALDEN. John Wiley and Sons, New York.

GARDINER, J. M., E. H. COE, S. MELIA-HANCOCK, D. A. HOISINGTON and S. CHAO, 1993 Development of a core RFLP map in maize using an immortal F_2 population. Genetics **134:** 917–930.

GOTTLIEB, L. D., 1984 Genetics and morphological evolution in plants. Am. Nat. **123:** 681–709.

HELENTJARIS, T., D. WEBER and S. WRIGHT, 1988 Identification of the genomic locations of duplicate nucleotide sequences in maize by analysis of restriction fragment length polymorphisms. Genetics **118:** 353–363.

HILU, K. W., 1983 The role of single-gene mutations in the evolution of flowering plants. Evol. Biol. **26:** 97–128.

ILTIS, H. H., 1983 From teosinte to maize: the catastrophic sexual transmutation. Science **222:** 886–894.

IRISH, E. E., J. A. LANGDALE and T. M. NELSON, 1994 Interactions between *tassel seed* genes and other sex determining genes in maize. Dev. Genet. **15:** 155–171.

KIEBER, J. J. and J. R. ECKER, 1993 Ethylene gas: it's not just for ripening any more! Trends Genet. **9:** 356–362.

LANGHAM, D. G., 1940 The inheritance of intergeneric differences in *Zea-Euchlaena* hybrids. Genetics **25:** 88–107.

LANDER, E. S. and D. BOTSTEIN, 1989 Mapping Mendelian factors underlying quantitative traits using RFLP linkage maps. Genetics **121:** 185–199.

LANDER, E. S., P. GREEN, J. ABRAHAMSON, A. BARLOW, M. DALY *et al.,* 1987 MAPMAKER: an interactive computer package for constructing primary genetic linkage maps of experimental and natural populations. Genomics **1:** 174–181.

MATTHEWS, D. L., C. O. GROGAN and C. E. MANCHESTER, 1974 Terminal ear mutant of maize (*Zea mays* L.). J. Agric. Sci. **82:** 433–435.

ORR, H. A. and J. A. COYNE, 1992 The genetics of adaptation: a reassessment. Am. Nat. **140:** 725–742.

SHERIDAN, W. F., 1988 Maize developmental genetics: genes of morphogenesis. Annu. Rev. Genet. **22:** 353–385.

TANKSLEY, S. D., 1993 Mapping polygenes. Annu. Rev. Genet. **27:** 205–233.

THOMPSON, J. N., and J. M. THODAY, 1972 Modification of dominance by selection in the homozygote. Heredity **29:** 285–292.

VAN HOUTEN, W., L. VAN RAAMSDONK and K. BACHMANN, 1994 Intraspecific evolution of *Microseris pygmaea* (Asteraceae, Lactuceae) analyzed by cosegregation of phenotypic characters (QTL) and molecular markers (RAPDs). Plant Syst. Evol. **190:** 49–67.

VEIT, B., R. J. SCHMIDT, S. HAKE and M. F. YANOFSKY, 1993 Maize floral development: new genes and old mutants. Plant Cell **5:** 1205–1215.

VLOT, E. C., W. H. VAN HOUTEN, S. MAUTHE and K. BACHMANN, 1992 Genetic and nongenetic factors influencing deviations from five pappus parts in a hybrid between *Microseris douglasii* and *M. bigelovii* (Asteraceae, Lactuceae). Int. J. Plant Sci. **153:** 89–97.

WADE, M. J., 1992 Sewall Wright: gene interaction and the shifting balance theory. Oxf. Surv. Evol. Biol. **8:** 35–62.

WILKES, G., 1967 *Teosinte: The Closest Relative of Maize.* Bussey Institute, Harvard University, Cambridge, MA.

WRIGHT, S., 1969 *Evolution and the Genetics of Populations,* Vol. 2. University of Chicago Press, Chicago.

Communicating editor: B. S. WEIR