

**Supplementary Table 1** Patterns of nucleotide diversity reveal selection on *ramosa1* in the maize lineage. Statistics were calculated on DNA sequence obtained as described in Methods. The “3 selected” row contains aggregate data for the three starch biosynthetic pathway loci (*bt2*, *sh2* and *su1*) that show strongest evidence for selection during domestication and/or improvement and “11 neutral” contains aggregate data for the 11 average (neutral) loci, all previously analyzed<sup>1</sup> in the same panel, which includes the inbred lines a272, a6, b103, b14a, b37, b73, b97, ci187, cml254, cml258, cml333, d940y, ep1, f2, i205, ids28, il101, kui2007, kui21, ky21, m162w, mo17, n28ht, nc260, nc348, oh43, pa91, t232, tx601 and w153r. “Random” is a collection of 20 other, non-selected loci evaluated on a separate diverse germplasm set<sup>2</sup>.

| loci       | Total nucleotide sites |           |            | HKA tests, silent nucleotide sites |        |                    |                            |                       |                    |
|------------|------------------------|-----------|------------|------------------------------------|--------|--------------------|----------------------------|-----------------------|--------------------|
|            | N <sup>a</sup>         | pi Silent | pi Nonsyn. | N                                  | pi     | Ratio <sup>b</sup> | f(P<0.05) <sup>c</sup> , % | Outgroup <sup>d</sup> | P all <sup>e</sup> |
| <i>ral</i> | 1517                   | 0.0010    | 0.0027     | 625                                | 0.0016 | 0.04               | 64                         | <i>Td</i>             | <0.0001            |
| 3 selected | 22230                  | 0.0033    | 0.0009     | 4829                               | 0.0022 | 0.04               | 70                         | <i>Td</i>             |                    |
| 11 neutral | 6649                   | 0.0107    | 0.0053     | 4568                               | 0.0152 | 0.23               | 2                          | <i>Td</i>             |                    |
| Random     | 10908                  | 0.0122    | 0.0038     |                                    |        |                    |                            |                       |                    |

<sup>a</sup>total number of nucleotide sites; <sup>b</sup>ratio of diversity within maize to average maize-*Tripsacum* divergence; <sup>c</sup>fraction of significant HKA tests with 11 neutral loci; <sup>d</sup>*Td* = *Tripsacum dactyloides*; <sup>e</sup>combined P value for the collection of pairwise HKA tests<sup>3</sup>.

## References:

1. Whitt, S. R., Wilson, L. M., Tenailon, M. I., Gaut, B. S. & Buckler, E. S. Genetic diversity and selection in the maize starch pathway. *Proc Natl Acad Sci U S A* **99**, 12959-62 (2002).
2. Tenailon, M. I. et al. Patterns of DNA sequence polymorphism along chromosome 1 of maize (*Zea mays* ssp. *mays* L.). *Proc Natl Acad Sci U S A* **98**, 9161-6 (2001).
3. Sokal, R. & Rohlf, F. *Biometry* (W. H. Freeman and Company, New York, 1995).

## Supplementary Methods - Cosegregation analysis

Mutable *ral* alleles were derived in high-*Spm* copy number lines. In deriving single-*Spm* cultures from these isolates we never observed mutability in the absence of *Spm* activity, which indicated apparent genetic linkage between mutable *ral* alleles and *Spm* transposon activity. To test the linkage between *Spm* and the mutable phenotype associated with the alleles *ral-m2* and *ral-m3*, we outcrossed pollen from the mutant portion(s) of single-*Spm*, mutable tassels onto a marked tester for *ral* and *Spm*, genotype *ral-R gll; c1-mr*, where *c1-mr* (a gift from Dr. P. A. Peterson, Iowa State University) is an *Inhibitor* (*I*, or defective *Spm*) insertion allele of *colored aleurone1* (*c1*) that is spotted in the presence of *Spm*, while *gll* is *glossy1*, a seedling marker within a few cM of *ral*. *Spm*-containing kernels were sown, scored for normal, mutable or mutant tassel phenotype, and testcrossed onto the same stock (*e.g.*, *ral-R gll; c1-mr* X + *ral-m2 +/o2 ral-R gll; c1-mr*). 24 such testcrosses were evaluated by sowing 50 spotted (+*Spm*) and 50 colorless (–*Spm*) kernels from each, scoring the resulting plants for *glossy1* and *ramosa1* phenotypes, and confirming *Spm* genotypes where relevant by another testcross with the aleurone marker. In this way we identified 12 transposition events that broke the tight linkage between *Spm* and *glossy1* and/or *ral*. In all 12 cases, *Spm* transposition was accompanied by conversion of the mutable allele to a genetically stable normal or mutant allele, suggesting that the *Spm* elements linked to *ral-m2* and *ral-m3* directly or indirectly caused the mutant phenotypes. We reasoned that if the following three criteria were satisfied, then *Spm* insertion was likely the direct cause: first, the single active (hypomethylated<sup>1</sup>) *Spm* element in the *ral-m2* cultures should be in a different location from the active *Spm* associated with *ral-m3* (the events should be different); second, neither *Spm* should have been present in the parent (they should be new events); finally, both elements should nevertheless reside near or within the same gene. A few DNA gel blots confirmed the first two criteria, so we cloned the *Spm* element from the *ral-m2* chromosome and used genomic DNA that flanked the element to confirm the third condition (*e.g.*, Fig. 2a, see main text).

## References

1. Banks, J. A., Masson, P. & Fedoroff, N. Molecular mechanisms in the developmental regulation of the maize Suppressor-mutator transposable element. *Genes And Development* **2**, 1364-80 (1988).