The Genomic Basis for Short-Term Evolution of Environmental Adaptation in Maize

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ABSTRACT Understanding the evolutionary capacity of populations to adapt to novel environments is one of the major pursuits in genetics. Moreover, for plant breeding, maladaptation is the foremost barrier to capitalizing on intraspecific variation in order to develop new breeds for future climate scenarios in agriculture. Using a unique study design, we simultaneously dissected the population and quantitative genomic basis of short-term evolution in a tropical landrace of maize that was translocated to a temperate environment and phenotypically selected for adaptation in flowering time phenology. Underlying 10 generations of directional selection, which resulted in a 26-day mean decrease in female-flowering time, 60% of the heritable variation mapped to 14% of the genome, where, overall, alleles shifted in frequency beyond the boundaries of genetic drift in the expected direction given their flowering time effects. However, clustering these non-neutral alleles based on their profiles of frequency change revealed transient shifts underpinning a transition in genotype–phenotype relationships across generations. This was distinguished by initial reductions in the frequencies of relatively large positive effect alleles and subsequent enrichment of many rare negative effect alleles, some of which appear to represent allelic series. With these genomic shifts, the population reached an adapted state while retaining 99% of the standing molecular marker variation in the founding population. Robust selection and association mapping tests highlighted several key genes driving the phenotypic response to selection. Our results reveal the evolutionary dynamics of a finite polygenic architecture conditioning a capacity for rapid environmental adaptation in maize.

KEYWORDS recurrent selection; flowering time; genetic diversity; plant breeding; agriculture; climate change

AFTER ~150 years of progress toward understanding evolution—since The Origin of Species (Darwin 1859)—burgeoning experimental results fueled by advances in genomic technology are shedding light on still unresolved questions about the nature of phenotypic change, including: the impact of mutation (e.g., Levy et al. 2015) and standing variation (e.g., Burke et al. 2010; Jones et al. 2012); the role of epistasis (e.g., Tenaillon et al. 2012); and the relationship between natural and artificial selection (e.g., Chan et al. 2012). A key question, especially in the face of biological invasions and climate change, is how genomes confer and constrain the capacity for organisms to adapt to new environments (Orr 2005). Genetic dissection of experimentally evolved populations is a tractable framework for elucidating adaptive evolution.
since the experimenter can control selection and mating in particular environmental settings (Barrick and Lenski 2013; Schlötterer et al. 2015). As a new extension to this framework, we implemented an efficient study design for dual inference about the population and quantitative genomic basis of phenotypic evolution (Wisser et al. 2011). This was used to investigate the response to a decade of directional phenotypic selection for tropical-to-temperate adaptation in maize—a model species for plant genetics and a crop of global importance.

**Genomic Basis of Response to Phenotypic Selection**

The rate and history of mutations, the numbers and positions of functional variants, the distribution of allele effects, and the modes of gene action are among the genetic factors that shape the response to selection and influence the maintenance of phenotypic and genetic variability (Barton and Keightley 2002).

Considering theoretical population and quantitative genetic expectations for the response to directional selection, alleles at one or few loci with large effects on a selected trait should rapidly change in frequency, resulting in a corresponding phenotypic response (Falconer and Mackay 1996). As these alleles approach fixation, genetic variance is reduced and the response diminishes. Sustained responses may be attributed to standing polygenic variation, new mutations, epistatic interactions, or heritable epigenetic effects. For polygenic traits controlled by numerous loci of small effects, modeled at the extreme of infinite loci (Barton et al. 2017; Fisher 1918), responses to selection can arise from subtle changes in allele frequencies across many loci. Consequently, allelic variation is retained and the causal-genic variance is expected to undergo negligible change. However, directional selection also creates negative disequilibrium covariance between allele effects across loci, resulting in temporary reductions in genetic variance for the trait under selection, a phenomenon referred to as the Balmer effect (Bulmer 1971; Walsh and Lynch 2018). Qualitatively similar expectations arise under a so-called finite polygenic architecture where tens or more loci with allele effects of varying magnitudes are at play (Chevalet 1994; Fernando et al. 1994; Turelli and Barton 1994).

An empirical understanding about the genetics of adaptation has been advanced through experimental population and quantitative genetic approaches (Savolainen et al. 2013). The relative importance of genes with major and minor effects varies among traits, populations and species. At one extreme, relatively rapid or dramatic phenotypic changes have resulted from a few alleles at loci with large effects on traits such as flowering behavior (Lowry and Willis 2010) and toxin resistance (Baxter et al. 2011). In contrast, other dramatic shifts in adaptive phenotypes have been ascribed to a polygenic architecture (Burke et al. 2010; Berg and Coop 2014). Drawing a clear line of distinction between the two is not straightforward and is partially confounded by differences in experimental systems and their statistical power, but adaptation from a mixture of genes with major and minor effects have been reported (Levy et al. 2015). Moreover, “evolve-and-reshape” studies have exposed unforeseen outcomes in the genomic changes underlying phenotypic evolution, including unique patterns of allele frequency change and the maintenance of molecular genetic diversity (Burke and Long 2012), the direct causes of which are unresolved.

**Phenological Adaptation in Maize**

Pivotal to adaptation and productivity in crop species is synchrony between the growing season and flowering time (Jung and Müller 2009). Numerous studies have investigated the genetic architecture of natural variation in flowering time for maize using a variety of methods, including genic analysis, linkage and association mapping, ecogeographical genetics and historical genetic analysis. Emerging from this body of literature is a consensus that allele effects dispersed across a finite polygenic architecture capture the major proportion of genotypic variation in flowering time (e.g., Chardon et al. 2004; Buckler et al. 2009; Li et al. 2016), and that certain flowering time genes—Vg1 (Salvi et al. 2007; Ducrocq et al. 2008), ZmCCT10 (Hung et al. 2012; Yang et al. 2013), ZmCCT9 (Huang et al. 2018), and ZCN8 (Guo et al. 2018)—appear to have been instrumental to the postdomestication spread of maize from its tropical origin to many different environments. However, with one exception (Durand et al. 2015), to our knowledge, the genomic basis of this adaptive trait has not been investigated in experimentally evolved populations. This could fill gaps in knowledge about the evolution of adaptation and lay a foundation to innovate breeding methods for rapidly adapting populations to new environments.

In this study, we investigated the genomic basis of adaptation from a distinct vantage point, where the entire period of evolution to an adapted state was captured in a single, multi-generational population—”Hallauer’s Tusón” (Teixeira et al. 2015). Selection was initiated within an admixed founder population formed by intermatting separate seed bank populations of Tusón, a landrace historically adapted to lowland tropical environments (Goodman and Brown 1988). Remarkably, 10 generations of directional phenotypic selection for early female-flowering time, with secondary selection for other traits in a temperate U.S. environment (Ames, IA; 42.03° N latitude), recapitulated the temperate-adapted state of maize achieved by early farmers; this is thought to have occurred over the course of thousands of years (Swarts et al. 2017), albeit by less deliberate breeding methodology. A study design, in which families derived from genotyped individuals sampled across generations were phenotypically evaluated, allowed us to employ methods for dissecting both the population and quantitative genomic basis of phenotypic evolution (Figure 1). Because tropical maize is a rich resource of potentially useful genetic diversity that has largely been under used by maize breeders in temperate environments.
(Goodman 1998), findings from this study can guide future maize breeding for climate change and address fundamental questions about the genomic basis of environmental adaptation in plants.

Materials and Methods

Front matter

Unless otherwise noted, data analysis was performed using R (R Core Team 2016); R packages are cited accordingly. The following abbreviations are used: AFPC (allele frequency profile cluster); BLUEs (best linear unbiased estimates); FDR (false discovery rate); FITR (frequency increment test with reference loci); GWA (genome-wide association); LD (linkage disequilibrium); SIM (simulation test statistic).

Plant material

The subject of this study was Hallauer’s Tusón, a multigenerational population of maize derived from a landrace of tropical origins that was subjected to 10 generations of phenotypic truncation selection for early female-flowering time in a temperate environment (Ames, IA; 42.03° N latitude) (Teixeira et al. 2015; Hallauer and Carena 2016). Figure 1 depicts the breeding scheme for Hallauer’s Tusón and our study design.

As described by Hallauer and Carena (2016), the base population (g0) used to initiate selection was produced in Iowa by isolated, open pollination (no intentional selection) among multiple seed-bank accessions of the Tusón landrace sampled from different countries; however, the origins of some of these accessions remain unclear. It should be noted that maize is a monoecious species with female and male organs on separate parts of the same plant, such that open pollination includes the possibility of self-fertilization. Using the base population, selection ensued where ≈ 8000 – 10,000 plants were grown in isolation and allowed to intermate at random, which again included the possibility of selfing. During flowering, 300 – 500 of the earliest female-flowering individuals (based on their silk-emergence phenotype), secondarily selected for other traits, were tagged and later harvested. An equal number of seeds per ear were mixed to form the subsequent generation of ≈ 8000 – 10,000 individuals for selection. This recurrent selection scheme was applied for 10 generations (from 1995 to 2004) until the population was deemed phenologically adapted by comparison with other temperate adapted maize.

Phenotype data

Previously, Teixeira et al. (2015) phenotypically evaluated 297 self-pollinated (S0) families derived from the even numbered generations of Hallauer’s Tusón (g0 : n = 18; g2 : n = 56; g4 : n = 56; g6 : n = 56; g8 : n = 56 and g10 : n = 55) for two years at multiple locations in North America, including the Iowa location where Hallauer’s Tusón was originally selected. Under the design depicted in Figure 1, the present study combined the available phenotypic data for female-flowering time measured in the selection environment (Ames, IA) and a highly correlated environment (Newark, DE) with new genotype data.

Genotype data

DNA was isolated from the 297 parents (noninbred) of the S0 population that were evaluated phenotypically, plus an additional 90 random individuals from g0 that were not phenotyped (this was done to provide a larger sample size of g0 for more reliable genomic inference). Lyophilized leaf tissue was pulverized using a Geno/Grinder 2000 and extracted with the DNeasy 96 Plant Kit (Qiagen). Genotyping with Illumina’s MaizeSNP50 Beadchip (Ganal et al. 2011) was performed by DNA LandMarks (Québec, Canada), producing genotype data at 56,110 SNP sites with an average of 2.8% missing data per sample (min: 0.2%; max: 20.9%). Additional genotype data were produced for variant sites upstream of ZmCCT10 (Zm00001d024909) (Supplemental Material, Supplemental Methods), including a presence-absence causal variant for photoperiod sensitivity (Hung et al. 2012; Yang et al. 2013). See Supplemental Methods for genotypic quality control (Table S1) and projection of markers onto the consensus linkage map for a maize nested association mapping population (McMullen et al. 2009).

Analysis of genetic diversity

Specific subsets of markers were used in examining different aspects of genetic diversity (File S1 and Table S2). Some subsets use marker names prefixed with “PZA” or “PZE,”
which have the lowest ascertainment bias among the MaizeSNP50 SNPs (Frascaroli et al. 2013).

Summary statistics of genetic diversity were computed with hierfstat v. 0.04–14 (Goudet 2005) to calculate $H_o$ (average observed heterozygosity within generations), $H_s$ (average expected heterozygosity within populations) and $H_T$ (average expected heterozygosity for the total population), as well as $F_{IS}$ (average inbreeding coefficient within generations), $F_{ST}$ (average differentiation between generations), and $F_{IT}$ (average inbreeding coefficient for the total population) according to Nei (1986)—this corresponds to the sample-level scope of inference. HardyWeinberg v. 1.5.5 (Graffelman 2015) was used to perform exact tests for Hardy–Weinberg equilibrium.

The relationship of Hallauer’s Tusón with maize more broadly was assessed using 934 samples representative of global maize germplasm for which genotype data for the same markers was available (Ganal et al. 2011). A two-dimensional projection of relationships among samples was computed using PHATE (Potential of Heat-diffusion for Affinity-based Trajectory Embedding) (Moon et al. 2017) implemented in phateR v. 0.2.7, using default settings with a precomputed simple matching distance matrix as input for the knn.dist.method.

Results from PHATE using only Hallauer’s Tusón suggested some structure was present among samples from $g_0$ but not other generations. Therefore, STRUCTURE (Pritchard et al. 2000) was used to examine subpopulations among $g_0$ samples assuming admixture and independent allele frequencies (Table S3; see Supplemental Methods for details). The $\Delta K$ method informed our selection of $K$ (Evanno et al. 2005). Although the exact accessions of Tusón used to form $g_0$ remain unclear, several were used (Hallauer and Carena 2016). Therefore, we ignored the stronger signal of $\Delta K$ suggesting $K = 2$ and chose the next highest peak at $K = 6$ (Figure S1). To examine whether any one subpopulation was favored in the initial generations of selection, STRUCTURE was also used to ascertain the $g_0$ ancestry present in $g_2$ samples, assuming the aforementioned subpopulation classification for individuals in $g_0$. A paired t-test was used to test the difference between the proportion of individuals per subpopulation in $g_0$ (Table S3) and the average per individual admixture proportion estimated by STRUCTURE for $g_2$ (Table S4).

LD (Hill and Robertson 1968), measured as $r^2$, was computed with genetics v. 1.3.8.1 (Warnes et al. 2013). The structure of LD between chromosomes was characterized per generation using low-ascertainment biased markers with no missing data and a minimum allele count of 12 in the given generation (Table S2). The structure of LD within chromosomes was similarly characterized, but with a larger number of markers (Table S2) and examined at different intervals of genetic distance: (0,1],(1,5],[5,10],[10,50],>50. In addition, $r^2$ was computed among generations (all samples) between sequential pairs of markers per chromosome. Using these latter estimates of LD, lokern 1.1.8 (Herrmann 2016) was used to perform kernel regression of the $r^2$ values as a function of the midpoint basepair coordinate between each pair of markers. A plot of pairwise $r^2$ between markers flanking the ZmCCT10 associated causal site for photoperiodism was made using LDheatmap v. 0.99.2 (Shin et al. 2006).

**Population genetic analysis**

**Allele frequency mapping:** Three statistical tests were used to detect markers with nonrandom patterns in allele frequency change across generations: (i) a customized whole genome SIM test for departures from genetic drift; (ii) the Bayenv test for robust correlations between allele frequencies and generations (Coop et al. 2010); and (iii) the FITR for robust departures from genetic drift (Nishino 2013).

A detailed description of the SIM test can be found in Supplemental Methods. Briefly, by customizing simuPOP (Peng and Kimmel 2005), a simulator was constructed to generate in silico genomes (genome-wide genotypes) for individuals constituting a population that undergoes breeding according to the design for Hallauer’s Tusón (with random selection to model genetic drift) and sampling according to our study design (to account for sampling variance). The simulator used the fixed $g_0$ genotype matrix, fixed recombination rates estimated from projection onto the genetic map (Supplemental Methods) and the fixed STRUCTURE matrix (Table S3) to initially generate 10,000 random in silico genomes (derived based on the structured $g_0$ sample data) from which simulated breeding ensued. At each marker across the genome, the probability of the observed sample allele frequency change was computed relative to the expected distribution created from 10,000 replicates of simulation. Marker $p$-values were adjusted for multiple testing (Benjamini and Hochberg 1995), and those with a 1% FDR are referred to as SIM$^+$ markers, while the remaining markers are referred to as SIM$^-$. Bayenv 2.0 was used to identify robust correlations between allele frequencies (response variable) and generations (explanatory variable). Bayenv was originally designed to test for correlations with an environmental variable; here, generation numbers (standardized) were used instead. The covariance matrix used to model the background expectation of allele frequency change was estimated from the low-ascertainment biased SNPs (Table S2). Markers with the top 1% Bayes factor values were considered robust outliers, which we refer to as Bayen$^+$ markers.

The FITR statistic is conditioned on the variance in allele frequency change estimated from neutral markers in the sample (Nishino 2013). For this, we used SIM$^-$ markers declared at a 10% FDR. To minimize bias from using a single set of reference markers, we modified the test by bootstrap resampling SIM$^-$ markers and computing the proportion of times in which each marker was significant (at a 1% FDR) among 10,000 bootstrap samples. In each bootstrap sample, the reference data comprised 1% of the markers ($n = 444$) sampled at random and without replacement. The test is not
definable for fixed sites, yet the alleles at markers used for testing were not always observed in each generation. Therefore, alleles with an observed frequency of zero in a particular generation were set to 1/2N_s, where N_s is the number of individuals in the g^th generation. Markers with a 1% FDR in at least 75% of the bootstrap sample tests are henceforth referred to as FITR^+.

**Localizing footprints of selection:** Chromosomal regions with a local footprint of selection (i.e., deviation from genetic drift across a segment of the genome) were delimited using chromosome-specific kernel regression functions of the −log10(q) values from the SIM test on the physical coordinates of markers. To obtain a definable −log10(q) input value for markers where the SIM test p-value equaled 0 (where the observed data fell outside the limits of the null distribution of simulated drift), p = 0 was set to 0.00057, which was half the minimum p-value among all SIM tests. Regions along each chromosome where the kernel regression line surpassed a threshold of −log10(q = 0.05) were considered local footprints of selection, and are henceforth referred to as SIM^+ regions.

**Characterizing features of allele frequency change:** Divisive analysis of hierarchical clustering (Kaufman and Rousseeuw 1990) was performed with cluster v. 2.0.7 (Maechler et al. 2018) in order to group SIM^+ markers with similar profiles of allele frequency change for the minor allele in g_o. The number of clusters was determined using clusGap based on the Tibshirani et al. (2001).

Additional data summaries, using the minor allele in g_o as the reference allele, were used to compare features of allele frequency change between SIM^- and SIM^+ markers, including: (a) the slope and intercept from regression of allele frequency change on generations; (b) the mean absolute change in allele frequency among the highest to lowest ranking changes in frequency per marker across sequential pairs of generations (the largest amount of change between a given pair of generations was assigned a ranking of 1; the least amount of change was assigned a ranking of 5); (c) the distribution of the longest run (i.e., number of generations) of positive and negative monotonic change in allele frequency across sequential generations; and (d) the rank distribution for the amount of allele frequency change across generation pairs.

**Quantitative genetic analyses**

**Genetic differentiation:** QST, a measure of the proportion of genetic variance distributed among populations for quantitative traits (Spitze 1993), was used to estimate genetic differentiation in female-flowering time between g_o and each subsequent generation of Hallauer’s Tusön, as well as to examine QST relative to the distributions of FST for SIM^- and SIM^+ markers. Knowing that flowering time was under selection, the QST − FST comparison was used to characterize the relationship between population genetic and quantitative trait divergence (Le Corre and Kremer 2012). Following from Spitze (1993):

\[
\hat{Q}_{ST} = \frac{Q^2_{GB}}{(Q^2_{GB} + 2Q^2_{GB})}
\]

where \(\hat{Q}_{ST}\) and \(Q_{GB}\) are estimates of the among-generation and average within-generation additive genetic variances, respectively. See the next section and Supplemental Methods for details on the estimation of variance components. For QST − FST comparison, we used the Hudson estimator, \(F_{ST}^{HI}\) (Bhatia et al. 2013), which is compatible with restricted maximum likelihood estimation of genetic variances used for QST, since both estimates correspond to the population-scope of inference in the broad sense.

**Partitioning of the genotypic variance:** Using ASReml v. 3 (Gilmour et al. 2009), the following mixed linear model was used to partition the phenotypic variance and decompose the genotypic variance into additive, dominance, and residual genetic variance components:

\[
y = X_n\beta + Z_e\varepsilon + Z_{[R,E]}i + Z_{F(G)}a + Z_{F(G)}d + Z_{F(G)}r + Z_{F(G)}X^* + Z_{F(G)}e + \varepsilon
\]

where y corresponds to the vector of observations (female-flowering time), \(\beta\) is the fixed overall mean effect, and \(e, i, a, d, r, f^*, e^*\) and \(\varepsilon\) correspond to the vectors of random environment effects, incomplete block nested in replication within environment effects, additive genetic family effects, dominance genetic family effects, residual genetic family effects, family × environment interaction effects, and residuals, respectively. The effect of replication nested in environments was excluded as it was not significant according to a likelihood ratio test. The respective design matrices \([X_n, Z_e, Z_{[R,E]}, Z_{F(G)}]\) (this has same structure for \(a, d, r, i\), \(Z_{F(G)}\)) relate observations to their corresponding vectors of effects. The additive, dominance, and residual genetic family effects were assumed to be distributed independently of one another, where: \(a \sim (0, G\sigma^2_a), d \sim (0, D\sigma^2_d), i \sim (0, I\sigma^2_i).\) The G matrix was computed according to VanRaden (2008) and the D matrix according to Su et al. (2012), while I is an identity matrix.

Variance component estimates from Equation 2 were used to compute heritability in the broad (\(H^2\)) and narrow (\(h^2\)) sense on an entry mean-basis according to Holland et al. (2003). In addition, extensions of Equation 2 were used to examine the amount of genetic variance in female-flowering time explained by each chromosome and for SIM^- vs. SIM^+ markers (Supplemental Methods).

**Genome-wide association mapping:** Kinship-controlled GWA was performed using a standard two-step procedure (Yu et al. 2006). First, BLUESs of female-flowering time for the S_0:1 families were estimated using Equation 2, but replacing additive, dominance, and independent random genetic effects with a single term for families fit as a fixed
effect. Second, using BLUEs as the response variable, markers were tested for trait association using the mixed linear model in TASSEL (Bradbury et al. 2007) standalone v. 5.2.12, while controlling for the random polygenic background with the aforementioned G matrix.

To reduce false-positive associations due to rare genotypes co-occurring with outlier phenotypes, if the sample size of phenotyped lines for a given genotypic class at a marker was less than five, individuals with the corresponding genotypic state (typically the homozygous minor allele class) were set to missing for that marker. The QQ plot of GWA p-values is shown in Figure S2. A 10% FDR was used to declare significant trait-marker associations; henceforth, these are referred to as GWA+ markers.

When estimating additive allele effects, some markers had only two genotypic classes (heterozygous and one homozygous class). The effects of minor variants at these loci were estimated as the difference between the heterozygous class and the homozygous class. For markers with three genotypic classes, the additive effect was uniformly reported as half the difference between the heterozygous class and the homozygous class. For markers with three genotypic classes, the estimated additive allele effect. Phenotype and genotype data are available at Dryad: phenotype - https://doi.org/10.5061/dryad.8f64f; genotype - https://doi.org/10.5061/dryad.q573n5dt. Python code used for genome simulation is available via GitHub: https://github.com/maizeatlas/saegus.

**Synthesis map**

A graphical map of the maize genome integrating multiple results was created. The map included local linkage disequilibrium estimated by kernel regression, the difference in heterozygosity between generations 0 and 10 (H_{0g0} – H_{10g10}), SIM test results (-log10-transformed q-values), Bayenv test results (log10-transformed Bayes factor values), FITR test results (bootstrap values >75%), GWA test results (-log10-transformed q-values), previously mapped QTL associated with flowering time {flowering time per se [Table S3 in Buckler et al. (2009)] and photoperiod sensitivity [Dataset S3 in Hung et al. (2012)]}, and candidate genes for flowering time [Dataset S8 in Hung et al. (2012)].

**Data availability**

The authors state that all data necessary for confirming the conclusions presented in the article are represented fully within the article. Supplemental methods, tables, and figures are available at figshare: https://doi.org/10.25386/genetics.9936284. Supplemental Tables: Tables S1 and S2 details results from quality control filtering and lists subsets of the genotype data used for analysis; Tables S3 and S4 contain results from STRUCTURE analysis; Table S5 summarizes SIM^+_region identified by kernel regression; Table S6 shows chromosome-specific genetic variance component estimates; Table S7 lists the markers detected by GWA; and Table S8 shows the candidate gene for flowering time nearest to each GWA+ marker. Supplemental Figures: Figure S1 shows results from STRUCTURE used to select K; Figure S2 is a QQ plot of observed vs. expected p-values for GWA tests; Figure S3 is a Venn diagram of SIM^+ markers detected when the SIM test was applied to sequential pairs of generations; Figure S4 summarizes various features of allele frequency change for SIM^- and SIM^+ markers; Figure S5 shows the allele frequency profile clusters and corresponding distributions of additive allele effects for SIM^- markers; Figure S6 shows the structure of LD within and between chromosomes for pairwise combinations of SIM^- and SIM^+ markers; Figure S7 shows the synthesis map of multiple analysis results. Supplemental Files: File S1.txt contains a list of the quality control markers, their map locations on B73 AGPv2 and AGPv4 reference assemblies, and the analysis-specific, Table S2 subset to which they belong. File S2.txt contains summary statistics and test results for each marker, including: allele frequencies per generation for the corresponding minor allele in g_0; observed heterozygosity (H_0) per generation; P^HST between g_0 and g_10; p-values and q-values for the SIM test; Bayes factor values and correlation statistics for Bayenv, bootstrap values for FITR; p-values and q-values for GWA; and the estimated additive allele effect. Phenotype and genotype data are available at Dryad: phenotype - https://doi.org/10.5061/dryad.8f64f; genotype - https://doi.org/10.5061/dryad.q573n5dt. Python code used for genome simulation is available via GitHub: https://github.com/maizeatlas/saegus.

**Results**

**Artificial selection generated a tropical genome with a temperate-adapted phenotype**

Hallauer’s Tusón population was founded by intermating multiple seed bank accessions of a maize landrace historically adapted to tropical environments (Figure 1). Teixeira et al. (2015) demonstrated the capacity of this population to become phenologically adapted to a temperate environment within 10 generations of artificial selection, based primarily on selection for early female-flowering time. Here, we found that the population was highly diverse; nearly the entire set (96%) of ≈ 50,000 SNPs on the MaizeSNP50 chip (Ganal et al. 2011) segregated within or among generations (Table 1).

When compared to a global sample of maize, all of the individuals across generations of Hallauer’s Tusón clustered with tropical germplasm (Figure 2a), notwithstanding the temperate-adapted phenotype of individuals belonging to the later generations (Teixeira et al. 2015). Thus, to tackle challenges associated with crop vulnerability through plant breeding, Hallauer’s Tusón highlights the adaptive potential of maize landrace populations, and provides a unique source of germplasm likely to contain novel alleles for temperate maize breeding programs.

**Retrospective analysis reveals admixture during selection on a structured founder population**

Genomic inference combined with knowledge about the population development scheme was used to illuminate the
breeding history of Hallauer’s Tusón. Although generations showed some SNP-based differentiation (mean $F_{ST} = 0.014$), as expected for progeny generated by random mating among selected individuals, overall inbreeding was minimal ($F_{IT} = 0.021$). However, on a per generation basis, $F_{IS}$ was noticeably higher in $g_0$, where 26% of the markers significantly deviated from Hardy–Weinberg equilibrium, with 78% of those deviations being due to an excess of homozygotes (Table 1). This suggested a Wahlund effect (Hartl and Clark 2007) from sampling genetically separated accesses that remained after the intermatting step to form $g_0$, but subsequent generations showed evidence of random mating during selection (Table 1).

STRUCTURE analysis also indicated that individuals in $g_0$ had formed largely by intermatting within separate founder accessions and hybridization between specific pairs of accessions (Figure 2b and Table S3). These findings are congruent with the way in which $g_0$ was bred, whereby the original Tusón accessions were planted in adjoining blocks and allowed to open-pollinate, which would have favored mating within and between pairs of subpopulations. Although selection could potentially favor specific subpopulations under these conditions, the genomic ancestral composition for $g_2$ individuals showed admixture profiles that were proportional to that of the subpopulation sizes in $g_0$ (Table S4). Thus, randomized bulking and planting of seed between each generation of artificial selection minimized subsequent inbreeding and population structure during selection.

**Differentiation across a fraction of the genome potentiated strong phenotypic change**

A decade of directional phenotypic selection, resulting in an overall mean decrease of 26 days to female-flowering time, caused generations to become strongly differentiated phenotypically, as measured by $Q_{ST}$ (Figure 3). Simulation of neutral allele frequency changes that would occur under the breeding scheme used for phenotypic selection allowed us to identify 6115 of 43,628 (14%) markers with non-neutral allele frequency changes (referred to as SIM+ markers). These markers were widely dispersed across the genome (but with some clusters of linked SIM+ markers, as described later), and were distinguished from SIM− markers by their increasing levels of $F_{ST}$ across generations relative to $g_0$ (Figure 3). The identification of a sizeable fraction of genomewide markers as SIM+ suggested a finite polygenic architecture (i.e., possibly tens to hundreds of loci affecting flowering time) could underlie the phenotypic response to selection. Similarly, based on $Q_{ST} – F_{ST}$ comparisons, the very large increases in $Q_{ST}$ could be explained by much smaller levels of $F_{ST}$ amplified across a large number of loci.

**Population genetic analysis pinpoints shifts in the genetic architecture underlying response to selection**

Although $F_{ST}$ increased overall across generations at SIM+ markers, changes in the frequencies of alleles at a locus varied mostly among generations. Using the simulator to test for non-neutral allele frequency changes between sequential pairs of generations showed that a majority of marker-specific departures were exclusive to one consecutive pair (Figure S3). This was coincident with the common observation of “bursts” in allele frequency change within a few generations, rather than monotonic changes across all generations (Figure S4, A–C).

Because transient changes in allele frequency were a prominent feature of the genomic response to selection, we used clustering to examine the temporal structure of allele frequencies among SIM+ markers. A total of 15 allele frequency profile clusters (AFPCs) were resolved with a high degree of overall clustering structure (divisive coefficient = 0.98; Figure S5). Minor alleles in $g_0$ with negative frequency trajectories were captured in AFPCs 1–3 comprising $\approx 10\%$ of the SIM+ markers. The remaining $g_0$ minor alleles, however, were enriched from starting frequencies that spanned the minor allele frequency spectrum. Across clusters, some notable transitions in allele frequency responses were observed: (i) alleles in AFPC1, which included the photoperiod sensitive allele ($ZmCCT10-s$), substantially reduced in frequency to become rare or removed within the first four generations; (ii) several AFPCs (6, 10, 11, 13, 14, and 15) showed clear increases in allele frequency within the first few generations that were limited thereafter; and (iii) for AFPC4, a dominant cluster comprising 43% of all SIM+ markers, initially rare alleles appreciably increased after $g_4$.

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**Table 1 Summary statistics for molecular genetic diversity in Hallauer’s Tusón**

| Generation | Sample size | Proportion polymorphica | $H_0^b$ | $H_5^c$ | $H_{1t}^d$ | $F_{ST}^e$ | HWD−f | HWD− : $H_0$/$H_5$g |
|------------|-------------|-------------------------|---------|---------|-----------|-----------|-------|---------------------|
| 0          | 105         | 0.897                   | 0.285   | 0.320   | NA        | 0.111     | 0.256 | 0.784               |
| 2          | 56          | 0.893                   | 0.323   | 0.315   | NA        | −0.026    | 0.071 | 0.400               |
| 4          | 55          | 0.902                   | 0.325   | 0.314   | NA        | −0.037    | 0.067 | 0.373               |
| 6          | 54          | 0.914                   | 0.315   | 0.320   | NA        | 0.016     | 0.072 | 0.475               |
| 8          | 56          | 0.923                   | 0.328   | 0.325   | NA        | −0.006    | 0.072 | 0.440               |
| 10         | 55          | 0.929                   | 0.333   | 0.327   | NA        | −0.017    | 0.069 | 0.411               |
| 0–10       | 381         | 0.963                   | 0.318   | 0.320   | 0.305     | 0.021     | 0.262 | 0.704               |

a Results in the column are based on 49,477 markers (Table S1), while all remaining results are based on 44,445 markers with a minor variant count $\geq 12$ among all samples.
b Average observed heterozygosity within generations. For all samples, this corresponds to the average among generations.
c Average expected heterozygosity within generations. For all samples, this corresponds to the average among generations.
d Average expected heterozygosity for the total population.
e Average inbreeding coefficient within generations. For all samples, this is $F_{IS}$, the average inbreeding coefficient for the total population.
f The proportion of HWD+ markers where $H_0$ was less than $H_5$.
g The proportion of HWD− markers where $H_0$ was less than $H_5$.
There were few instances, like ZmCCT10-s, where alleles were purged from the population. The loss of SNP variants in Hallauer’s Tusón was actually rare—1.00% (n = 439 markers) of all SNPs in g0 were purged during selection (10 of these were SIM+), and this was 3.4 times less than the average proportion of SNPs purged across replicate simulations of neutral frequency change (range: 2.8%–4.0%). Taken together, population genetic analysis suggested that selected genotype–phenotype relationships temporally shifted across a finite polygenic architecture with a predominant enrichment of initially minor alleles, resulting in the maintenance of genetic variation.

**Quantitative genetic analysis contextualizes genome-wide population genetic dynamics**

Our experimental design included phenotypic data on self-pollinated families of genotyped individuals sampled across generations evaluated in common environments (Figure 1), permitting estimation of quantitative genetic parameters and interpretation in the context of population genetic results. Underlying a 50-day range for female-flowering time in Hallauer’s Tusón, high broad and narrow sense heritabilities ($H^2 = 0.96 ± 0.01 \cdot h^2 = 0.81 ± 0.08$) indicated a large fraction of the phenotypic variance could be explained by genotypic effects, and the genotypic variance partitioned into 85% additive and 15% dominance variance with no residual genetic variance remaining. Including an additive-by-additive epistatic relationship matrix did not improve the model fit nor did it explain any of the genotypic variance.

The genetic variance explained by individual chromosomes varied widely (Table S6). For instance, chromosome 10, in which the ZmCCT10-associated causal variant is located, was an outlier that accounted for a large proportion (28%) of the additive variance, while chromosome 2 included no additive or dominance variance. This supports and extends the population genetic inference of a finite polygenic architecture, showing variability in the genetic effects across the genome available for selection.

**Figure 2** Genetic diversity and population structure in Hallauer’s Tusón. (A) 2D PHATE plot showing relationships for Hallauer’s Tusón and a broad range of maize germplasm. Aside from distinguishing samples from Hallauer’s Tusón and teosinte inbreds, population structure groups were assigned to samples based on Table S1 in Romay et al. (2013) (several samples were unclassified but retained in the analysis). (B) Population structure in g0. Admixture profiles for six subpopulations are shown. Values at the top of the STRUCTURE plot correspond to days to female flowering time for individuals with phenotype data. Subpopulation frequencies of ZmCCT10-s, the deletion (“lack-of-insertion”) variant associated with photoperiod sensitivity is shown.

Genome-wide additive allele effects (estimated among families across generations) correlated with average changes in allele frequency per generation ($r = −0.39, p < 2.2e−16$), whereby, as expected, alleles with negative effects on female-flowering time (contributing to early flowering) tended to have positive slopes in allele frequency change and vice versa (Figure 4; the Bayenv and GWA hits highlighted in the Figure are discussed in the next section). This relationship was largely driven by SIM+ markers, which accounted for 60% of the additive variance (none of the dominance variance)—an excessive enrichment given these constituted $\approx 14\%$ of the SNPs.

Nonlinear changes in the phenotypic mean and additive variance for female-flowering time across generations reflected some of the observed dynamics in allele frequency change (Figure 5). Changes in the mean could be modeled as a cubic function with significant ($\alpha = 0.05$) coefficients $[f(x) = −0.07gen^3 + 1.39gen^2 − 9.64gen + 100.27]$, where female-flowering time decreased across all generations but to a larger degree between generations 0–4 and 8–10. These generations contained greater numbers of SIM+ markers with larger magnitudes of allele frequency change (Figure S4d). The corresponding change in additive variance could be modeled as a quadratic function of generations with significant coefficients $[f(x) = −14.11gen^2 + 1.06gen + 31.58]$, where initially the variance was greatly reduced but later increased across generations 6–10; by g10, the additive variance exceeded that of g4. These changes were coincident with initial reductions in the frequencies of few relatively large positive effect alleles (AFPC1) and subsequent enrichment of many rare negative effect alleles (AFPC4) (Figure 5 and Figure SS).

**Robust selection and association mapping identify associations with key flowering time genes**

The simulation test clearly enriched for markers that differentiated generations (Figure 3), but not all of these are necessarily linked to causal variants underlying female-flowering time selection. For instance, chromosome 2 captured none of the genetic variance in female-flowering time; however, it contained SIM+ markers (Table S6). The model used to simulate breeding events does not include components of potentially important sources of variation, such that...
departs from the null distribution may also be due to factors such as individual differences in gametic fitness, or the secondary selection that was exerted for other traits (Hallauer and Carena 2016). On the other hand, covariance between causal and neutral allele frequency changes may generate false positives (Coop et al. 2010). Although we could not control for deviations due the former, selection and association mapping tests that control for genomic background effects were used to identify markers exhibiting robust changes in allele frequency across generations and robust associations with trait variation among generations, respectively.

Prior to applying these tests, we assessed the transgenerational structure of LD across the genome using subsets of low-ascertainment biased markers with a standardized minimum allele count per generation (Table S2). Linkage disequilibrium was examined for each combination of SIM− and SIM+ markers. Based on all pairwise estimates of LD, median $r^2$ showed little variation across generations, and did not exceed 0.04 within chromosomes and 0.01 between chromosomes (data not shown). At increasingly higher percentiles of the $r^2$ distribution, LD between pairs of SIM+ markers within chromosomes (but not between chromosomes) was elevated relative to other combinations of SIM− and SIM+ markers (Figure S6). We found that the heightened LD between SIM+ markers was restricted to local linkage blocks, and tended to increase across generations (Figure S6)—a hallmark footprint of selection.

Given that the structure of LD gave rise to nonindependent sets of linked SIM hits, kernel regression was used to delimit 29 SIM+ regions encompassing 1008 (16%) of the SIM+ markers (Table S5). The Bayenv test, which controls for genome-wide covariance in sample allele frequencies, produced Bayes factor values that were correlated with the SIM test (Spearman’s $r = 0.62$). The top 1% of Bayenv hits were located on all chromosomes and were present in most SIM+ regions, with regions on chromosome 9 being heavily populated with Bayenv+ markers (Figure 6 and Figure S7). Similarly, the FITR test, which is conditioned by variance in allele frequency change estimated from the sample, primarily implicated SIM+ regions on chromosome 9 as robust outliers.

GWA mapping performed on mean female-flowering time resulted in few genome-wide significant associations, notwithstanding the SNP-based polygenic model that explained essentially all of the genotypic variance. Between 2 and 12 GWA hits were detected across 1–10% FDR thresholds (Table S7). All but one of these showed the expected relationship between the sign for the additive allele effect and slope in frequency change (we note this one marker was on chromosome 2, which explained none of the genetic variance when the whole chromosome was modeled under a polygenic architecture). However, no GWA hits were detected on chromosome 9 nor within any of the SIM+ regions. The top GWA hit was the presence–absence causal variant for ZmCCT10-regulated photoperiodism, which was also detected as hits by the SIM and Bayenv tests but not the FITR test. Otherwise, the strength of signals (slopes vs. effects) for top hits by selection and association mapping tests tended to differ (Figure 4).

Taken together, robust tests to dissect the genetic basis of the response to selection implicated a number of genes previously associated (causally or as a candidate gene) with variation in flowering time and photoperiodism in maize, several of which are highlighted in Figure 6.

Evidence for multiple local haplotypes underlying the phenotypic response to selection

The transition from selection on common to rare alleles occurred at some of the same regions of the genome. For instance, SIM+ regions on different chromosomes included SIM+ markers in both AFPC1 and AFPC4, in which allele frequencies showed strong shifts during different periods of selection (Figure 5). Similarly, at the ZmCCT10 locus, robust associations were detected for SNPs that responded to selection even after the elimination of ZmCCT10-s. Pairwise LD between significant markers at the ZmCCT10 locus indicated two separate haplotypes were responsive to selection (Figure 7). These results reinforce the conclusion of a finite polygenic architecture underlying the response to selection, and extend...
that to suggest selection on multiple local haplotypes was an important aspect of short-term evolution.

Discussion

Genetic analysis of adaptation in crop species provides a lens into evolution and generates relevant information for plant breeding. Although flowering time phenology has been widely studied in plants (Jung et al. 2017), we are aware of no study (in plants) that has dissected the transgenerational genomic basis of adaptive evolution (here, for flowering phenology) in a population translocated to a new environment. We help close this knowledge gap by investigating a tropical landrace of maize that was adapted to a temperate environment across a decade of artificial selection (Teixeira et al. 2015; Hallauer and Carena 2016). Using an efficient study design (Figure 1; Wisser et al. 2011), we simultaneously elucidated population and quantitative genetic components underlying the 10 generations of selection required for the population to reach a state of phenological adaptation similar to modern temperate maize lines.

The evolutionary capacity of the tropical Tusón landrace to become rapidly adapted to a temperate environment was attributed to a finite polygenic architecture, yet two genomic phases underlying the phenotypic response to selection could be discerned. The first phase, from generations 0 – 4, was distinguished by an oligogenic-like architecture, where marked reductions of a relatively small number of moderate-frequency minor variants in $g_0$ (AFPCs 1 and 2), with relatively large positive effects on flowering time, contributed to an initial strong response to phenotypic selection and a large reduction in genetic variance (Figure 5). Afterward, the genomic basis of the response transitioned to become dominated by the enrichment of a large number of rare-minor variants in $g_0$ with smaller-sized negative effects on flowering time, leading to a genome-wide increase in heterozygosity (Table 1) and consequent increase in additive variance (Figure 5).

The observed changes in phenotypic mean and variance are similar to expected outcomes theorized for a finite polygenic architecture with additive allele effects (Chevalet 1994). Consistent with an additive genetic model, several AFPCs showed linear trends across all generations reflective of unconditionally (un)favorable alleles in Hallauer’s Tusón (Figure S5). However, AFPCs with transient shifts in allele frequency were also detected, such as mid-to-late generational responses and plateaus in allele frequency change, highlighting a context-dependent component of the genetic architecture underlying the response to phenotypic selection. The same pattern of plateauing allele frequencies after an initially strong shift was found by temporal analysis of natural populations of Drosophila melanogaster adapted to a novel laboratory environment, which Orozco-terWengel et al. (2012) reasoned was due to overdominant or antagonistic pleiotropic effects. It has been demonstrated (mathematically) that the selection coefficient for an additive allele can vary across generations also as a result of changes in background polygenic variance (Chevin and Hospital 2008). The genotypic variance in Hallauer’s Tusón partitioned into additive (primarily) and dominance genetic variance with no apparent epistatic-genetic variance, but epistatic genetic effects will contribute to the additive genetic variance component in many cases, such that inferences about gene action should not be drawn from variance components estimates (Hill et al. 2008; Huang and Mackay 2016). Thus far, genetic studies on flowering time in maize have described an architecture with predominantly additive genetic variance (e.g., Buckler et al. 2009; Coles et al. 2010; note that these studies use inbred lines, which precludes estimation of dominance variance), but reports of dominant, overdominant (Coles et al. 2011) and epistatic (Blanc et al. 2006; Durand et al. 2012) allele effects on variation in flowering characteristics also exist. With a limited sample size for quantitative genetic dissection per generation, our study is unable to clarify the causes or relative contribution of context-dependent effects on the response to phenotypic selection.

Maize is highly diverse (Buckler et al. 2006), and landraces of maize are locally adapted to a wide range of environments (Committee on The Preservation of Indigenous Strains of Maize 1952–1963). Still, it was surprising that Hallauer’s Tusón captured nearly all of the SNPs on the MaizeSNP50 chip (Ganal et al. 2011). This high level of molecular genetic
variation, as well as the detection of an increasing proportion of polymorphic platform SNPs across generations (Table 1), led us to question whether migrant pollen had entered the population, particularly since it was open-pollinated during selection; although the population was bred in spatial and temporal isolation of other maize populations. Separate lines of evidence indicate the population could have very high diversity while remaining a closed system with no migration or pollen flow. First, similar to our finding, another study has found that individual populations of maize landraces can capture >90% of the SNPs on the same MaizeSNP50 platform (Arteaga et al. 2016). Because the base population of Hallauer’s Tusón was admixed from multiple, geographically dispersed populations of the landrace Tusón, there is a greater likelihood for the level of diversity to be high. Second, the binomial sampling probabilities for our study limited detection of rare variants within generations despite their putative presence in the population. For instance, based on our sample sizes (which was larger for g₀), SNPs at a frequency of 1% have an 11% chance of being undetected in g₀ and a 33% chance of being undetected in the other generations, but subtle increases above 1% result in large increases in the probability of their detection. Therefore, variants that were not detected in one generation but detected in another may exist at low frequencies, and the transgenerational increase in polymorphic platform SNPs can be explained by selection of initially rare alleles. Finally, considering the most frequent migrant sources in Iowa where the population was selected would be of temperate origin, all of the genotyped individuals in Hallauer’s Tusón clustered with other maize samples of tropical rather than temperate origin (Figure 2).

A fundamental question in genetics is how populations acquire and maintain variation that conditions them with the capacity to adapt to a novel environment. At the locations where the source populations were already adapted and grown, we presume stabilizing selection occurred on flowering time, as flowering time affects fitness in an environmentally dependent manner (Hall and Willis 2006; Mercer and Perales 2019). Stabilizing selection is expected to deplete genetic variation (Barton and Keightley 2002), such that the extensive functional variation for flowering time in Hallauer’s Tusón suggests evolutionary forces beyond mutation and selection for a single optimal flowering time affected the founding populations. Teixeira et al. (2015) showed that flowering time variation in Hallauer’s Tusón is under strong genetic and environmental control, with relatively little genotype-by-environment interaction (however, GxE effects were present across latitude and greater in the initial generations). Therefore, seasonal fluctuations that affect the relationship between flowering time and fitness (Giuffrèt et al. 2000) and multivariate constraints to evolution (Walsh and Blows 2009) likely contributed to the maintenance of substantial standing variation for this trait, and therefore its capacity for adaptation to a novel environment.

As the population was subjected to directional selection in a temperate environment, alleles contributing to earlier flowering tended to be enriched (Figure 4). Although SIM⁺ alleles with negative effects on flowering time spanned the full allele frequency spectrum in g₀, including initially

Figure 5 Quantitative and population genetic components of the response to selection. (A) Zero-centered (not standardized) “Z” values corresponding to the mean (BLUE) and additive variance (Va) for female-flowering time per generation. (B) Box plots of allele frequencies per generation for SIM⁺ markers in AFPCs 1 and 4. (C) Histograms of additive allele effects for SIM⁺ markers in AFPCs 1 and 4. (D) SIM hits (vertical lines colored by chromosome) within SIM⁺ regions (x-axis) that belonged to AFPCs 1 and 4. The silhouette width (y-axis) is a measure of a markers fit to the cluster (values > 0.5 can be considered a good fit). Facet labels indicate the AFPC identifier and the proportion of SIM⁺ markers per cluster (in (B) and (C) this corresponds to the proportion among all SIM hits; in (D) this corresponds to the proportion among regional SIM hits).
frequent variants such as the photoperiod insensitive allele ZmCCT10-i (present at 75% in g0), most of these (∼ 75%) existed in the minor frequency domain. This was in contrast to minor alleles in g0 at SIM+ markers, for which ∼ 50% had negative effects on flowering time. Hence, in the base generation of Hallauer’s Tusón, favorable alleles for temperate adaptation primarily exist in the minor frequency spectrum.

Due to the admixture of multiple Tusón populations to form g0, however, the allele frequencies reflect those among (not within) the founder populations, such that native population allele frequencies of temperate-adaptive variants are confounded. To address this, subpopulation assignments for samples from g0 (assumed to correspond to the founding populations) were used to compute subpopulation-specific allele frequencies for the corresponding minor allele in the whole g0 sample (data not shown). Across all SIM+ markers, most alleles were shared among multiple subpopulations; only 8% of these markers included private alleles. This suggests that geographically separated populations of the landrace Tusón have retained shared alleles that enable latitudinal adaptation, a finding that is congruent with geographical association results from a diverse sample of maize landraces (Romero Navarro et al. 2017).

Our inference from allele frequency data, however, should be considered with caution. The inferred number of loci (finite polygenic architecture) and effect of selection on variants across the allele frequency spectrum is based on markers that are unlikely to be causal variants themselves, but are expected to tag local haplotype blocks containing causal variants (Nuzhdin and Turner 2013; Kelly and Hughes 2019). Moreover, any initially rare variants with late flowering time effects would likely have been purged or kept at low frequencies, leading to low power of detection for both selection and association mapping, despite the importance of such potential variants for environmental adaptation. This represents a bias to evolutionary inference in experimentally evolved populations and highlights the need for deeper sampling within generations and other approaches to elucidate the structure of natural variation for adaptation.

Our study design allows for the mapping and characterization of specific genomic loci underlying phenotypic evolution. Although the relatively small sample (∼ 300 families) and low marker density (tens of thousands of SNPs) limited...
the power of our study, we nevertheless detected robust associations with known genes involved in flowering time adaptation, raising confidence in our discovery of other unique loci (Figure S7). For instance, selection against photoperiod sensitivity was a major component of adaptation in Hallauer's Tusón (Teixeira et al. 2015). Regions encompassing ZCN8, CON1, COL9, CRY2, and ZmCCT9 on chromosomes 8 and 9, and a causal regulatory variant of ZmCCT10 on chromosome 10, were detected by selection or association mapping (Figure 6). These genes are regulators of photoperiodism in plants (Guo et al. 1989; Cheng and Wang 2005; Miller et al. 2008; Meng et al. 2011) that have contributed to latitudinal adaptation (Yang et al. 2013; Guo et al. 2018; Huang et al. 2018), all of which were identified in a large-scale mapping study on photoperiod sensitivity in maize (cf. Figure 6 and Figure 2 in Hung et al. 2012). Moreover, we found that different local haplotypes were responsive to selection at several of these same loci where Hung et al. (2012) detected allelic series [e.g., SIMregion21–23 and 25 (Figure 5) and the ZmCCT10 locus (Figure 7)]. On chromosome 8, no robust association was detected for the Vegetative to Generative Transition 1 (VGT1) gene (Salvi et al. 2007) involved in earliness per se, but a maize homolog of Arabidopsis thaliana de-etiolated 1 (DET1) involved in photomorphogenesis (Pepper et al. 1994), not previously highlighted for maize adaptation, mapped to a conspicuous SIMregion across the centromere of the chromosome.

Development of next-generation crop varieties is crucial to ensuring ample production and quality of plant-based products for society. Reliance on limited pools of diversity for breeding and the creation of monocultures can lead to constrained and vulnerable production systems, but genetic diversity is ecologically structured whereby alleles that could be useful in a target production environment reside in germplasm that suffers from maladaptive syndromes (Teixeira et al. 2015). Our study shows the potential for maize landraces to be adapted to temperate environments by simple recurrent selection for early flowering time while resulting in minimal loss of molecular genetic variation to reach the adapted state. The lack of a linked footprint of selection (SIMregion) encompassing the ZmCCT10 causal site, which had the strongest association with flowering time and underwent a rapid complete-sweep, suggests that some critical adaptive mutations in maize are embedded in regions of low LD, which would permit the maintenance of linked variation during directional selection. Moreover, the multidimensional nature of the genetic architecture underlying response to phenotypic selection, involving multiple loci and alleles with context-dependent effects, appears to enable a rapid shift toward an adapted state with limited loss in diversity. We anticipate that further characterization of these additional layers of the genetic architecture and dynamics of the genomic response to selection will lead to new advances in genomic prediction across generations. By bridging population and quantitative genetic inference, this study advances our understanding of short-term evolution, providing unique insights that aid in developing approaches to adapt crops to climate change.

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