Persistence of sub-genomes in paleopolyploid cotton after 60 million years of evolution

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keywords: whole genome duplication, gene fractionation, biased fractionation, transposable element, gene expression

Abstract

The role of gene duplication as an evolutionary force has long been recognized. Indeed, the process of gene duplication, via whole genome multiplication (WGM), is cyclical and ubiquitous in flowering plants (Jiao, Wickett et al. 2011, Jiao, Leebens-Mack et al. 2012), with many lineages having experienced multiple rounds of WGM, each followed by substantial duplicate gene loss (fractionation). The process of fractionation may be biased with respect to homoeologs, often with over-expression of genes in the less fractionated (LF) relative to more fractionated (MF) regions (Woodhouse, Schnable et al. 2010, Schnable, Springer et al. 2011). The correlation between fractionation and expression has been hypothesized to arise via positional-effect down-regulation of genes via silencing of local transposable elements (TEs)(Woodhouse, Cheng et al. 2014). We assess this hypothesis using comparative genomics and analysis of synteny, and report that the genomic signatures of biased fractionation remain evident in the paleopolyploid genome of modern diploid cotton (*Gossypium*), which underwent a 5- to 6-fold ploidy increase around 60 million years ago (Paterson, Wendel et al. 2012). Our data stand in contrast to other reports by extending the evolutionary time over which signatures of biased gene loss can be detected, suggesting that, hitherto, the long term impact of this process has not been fully appreciated. As in other species, biased fractionation is associated with over-expression of genes in LF regions. Furthermore, we report that LF and MF fractions can be clearly differentiated via several genomic signatures despite the antiquity of the WGM event. For example, MF regions have elevated GC content, higher transposon (TE) density and elicit preferential mapping of siRNAs. However, contrary to previous observations (Hollister, Smith et al. 2011), we show that global gene expression levels are not influenced by TE proximity, nor local siRNA targeting, both of which are therefore unlikely to be the primary drivers of biased fractionation. We propose an alternative scenario in which repeat content *per se* and differential recombination rates may be responsible for biased fractionation of sub-genomes following WGM.

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