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doi:10.1016/j.tig.2005.08.002

Novel patterns of gene expression in polyploid plants

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Genome doubling, or polyploidy, is a major factor accounting for duplicate genes found in most eukaryotic genomes. Polyploidy has considerable effects on duplicate gene expression, including silencing and up- or downregulation of one of the duplicated genes. These changes can arise with the onset of polyploidization or within several generations after polyploid formation and they can have epigenetic causal factors. Many expression alterations are organ-specific. Specific genes can be independently and repeatedly silenced during polyploidization, whereas patterns for other genes appear to be more stochastic. Three recent reports have provided intriguing new insights into the patterns, timing and mechanisms of gene expression changes that accompany polyploidy in plants.

Introduction

Most eukaryotic genomes have numerous duplicated genes, many of which appear to have arisen from one or more cycles of polyploidy (genome doubling), either by allopolyploidy or autopolyploidy (see Glossary). Well-documented examples of polyploidy exist in various groups of vertebrates, insects, yeasts and plants [1,2]. Ancient polyploidy events (paleopolyploidy) have been inferred to have occurred during the evolutionary history of vertebrates, yeast and flowering plants [3]. Following paleopolyploidy there has been extensive loss of duplicated genes. Polyploidy has been especially common in flowering plants, where most species are inferred to have experienced at least one polyploidy event in their evolutionary history [4]. For example, at least two and probably three paleopolyploidy events are thought to have occurred during the evolutionary history of *Arabidopsis thaliana* [5]. Approximately 27% of the gene pairs that were formed by polyploidy have been retained in *A. thaliana* [6] and more than half of these gene pairs show evidence of functional divergence [7].

The merging and doubling of two genomes sets in motion extensive modifications of the genome and/or transcriptome, creating cascades of novel expression patterns, regulatory interactions and new phenotypic variation for evaluation by natural selection [5,8,9]. Recent studies

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Available online 10 August 2005

Glossary

AFLP-cDNA: amplified fragment length polymorphisms, using cDNA as template. This is an mRNA fingerprinting method that can be used to detect putative cases of gene silencing. By comparing parental diploid transcriptomes with those of their derived allopolyploid, this approach permits the detection of 'missing' parental AFLP fragments in the polyploid. Double-stranded cDNA is digested with two restriction enzymes, adapters are ligated to the ends and two round of PCR are performed. Gel electrophoresis generates a set of banding patterns; comparison of polymorphic bands between a polyploid and its diploid parents can reveal bands that are 'missing' in the polyploid, potentially reflecting gene silencing.

Allopolyploidy: polyploids arising from hybridization between genetically distinct parents, typically different species. Meiotic segregation in strict allopolyploids is disomic, with chromosome-pairing being restricted to homologous chromosomes within each progenitor genome.

Autopolyploidy: polyploids arising by chromosome doubling within an individual or from merger of two similar genomes from different individuals within a species. In these polyploids, meiotic segregation often is tetrasomic, and chromosome pairing can include both homologous and homoeologous associations.

Homoeologs: duplicated, orthologous genes that originated by polyploidy and are present in each of the two parental genomes in an allopolyploid (sometimes called homoeologues).

Paleopolyploidy: an ancient polyploidy event. At least two and probably three rounds of polyploidy have occurred during the evolution of the *Arabidopsis thaliana* lineage. Paleopolyploidy is usually followed by considerable loss of duplicated genes.

Subfunctionalization: partitioning of ancestral gene function or expression patterns among a gene pair originating from a duplication event (e.g. Ref. [33]). Such partitioning of aggregate function is envisioned to evolutionarily preserve both genes.

have demonstrated that many of these effects arise following the onset of polyploid formation, whereas others play out over a longer evolutionary timescale. Key to these recent insights is the use of newly created, synthetic plant polyploids that mimic natural systems. These have proven to be excellent systems for studying the immediate consequences of polyploidy and they provide insights into processes that occur following polyploidy in many eukaryotes. Extensive genomic rearrangements, including exchanges between genomes and gene loss, have been documented in certain systems [10–12] but not others [13]. Studies of synthetic polyploids have shown that genes duplicated by polyploidy, termed 'homoeologs', can become silenced immediately or soon after polyploidy [14–18], similar to tandemly repeated rRNA genes [19,20]. Here we highlight recent studies of synthetic plant polyploids that have provided intriguing new insights into the patterns, timing and mechanisms of gene expression changes in plant polyploids [21–23].

Patterns of duplicate gene expression and silencing following polyploidy

To explore patterns of duplicate gene silencing immediately following polyploidization, newly created polyploids of *Arabidopsis* and cotton were examined using amplified fragment length polymorphisms (AFLP)-cDNA screens to identify silenced genes. By comparing parental diploid transcriptomes with those of their derived allopolyploid, this approach permits the detection of 'missing' parental AFLP fragments in the polyploid, which, following verification by other techniques such as RT–PCR, leads to inferences of polyploidy-induced gene silencing. The *Arabidopsis* polyploids included an autotetraploid *A. thaliana*, formed by spontaneous doubling of the chromosomes of diploid *A. thaliana* in tissue culture,

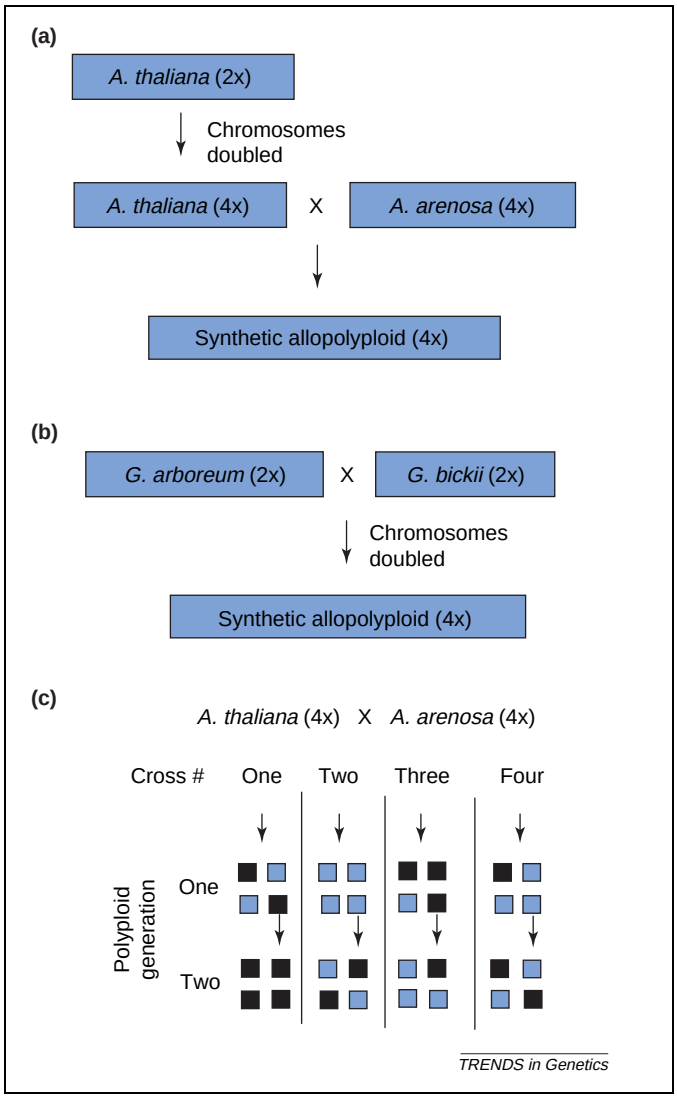

Figure 1. The creation of synthetic plant polyploid lines and examination of their gene expression patterns. **(a)** Synthesis of *Arabidopsis* allopolyploids (from *Arabidopsis thaliana* and *Arabidopsis arenosa*).**(b)** Synthesis of cotton allopolyploids (from *Gossypium arboreum* and *Gossypium bickii*) **(c)** Illustration of stochastic gene silencing in four independent lines of the synthetic *Arabidopsis* allotetraploid derived from crosses of the same tetraploid parents; also showing four siblings in a generation. Black boxes indicate silencing of the gene; blue boxes indicate expression. See the main text and Wang *et al.* [21] for specific examples.

and an allotetraploid, formed by hybridization between the newly created autotetraploid *A. thaliana* and the natural tetraploid *A. arenosa* [14] (Figure 1). The cotton polyploids were formed by hybridization between two diploid cotton species followed by chromosome doubling [24] (Figure 1). Several putatively silenced genes were identified in *Arabidopsis* and cotton by the AFLP-cDNA screens. Wang *et al.* [21] and Adams *et al.* [22] performed RT–PCR experiments for seven and 13 putatively silenced genes, respectively, to verify gene silencing. In both of the allopolyploids, formed from *Arabidopsis* and cotton, gene silencing occurred during the first or second generation after synthesis. Particularly noteworthy in the *Arabidopsis* polyploids were two genes that were expressed in diploid *A. thaliana*, silenced in the autotetraploid *A. thaliana* and then reactivated following hybridization with *A. arenosa* in the allopolyploid. Moreover, one gene (*RAD54*) whose expression was not detected in diploid *A. thaliana* leaves

was activated in leaves following autopolyploid formation. These results show that both chromosome doubling and hybridization can affect gene expression. To examine the timing of onset of duplicate gene silencing, Wang *et al.* [21] examined gene expression and silencing patterns in generations two through four following initial polyploid formation. Three genes showed variation in expression levels in different generations. For example, a gene encoding a serine threonine kinase was expressed initially only from one homoeolog then only by the other homoeolog in generation four, suggesting that there is period of a few generations following polyploidy when partitioning of duplicate gene expression occurs.

Is duplicate gene silencing stochastic or repeatable?

Because the transcriptome appears to be so radically altered by genome doubling, the question arises as to whether the gene expression changes detected are in some fashion 'directed' by the dictates of genomic structure, regulatory control, dosage, or other factors (and therefore are repeatable) or if the changes are more *ad hoc* or stochastic in nature. This issue can be explored by examining the expression of the same duplicate gene in multiple polyploid genotypes or lines.

Wang *et al.* [21] compared expression patterns in four independently synthesized *Arabidopsis* allotetraploid lines with the same parents (Figure 1c), first by AFLP-cDNA screens and then for several genes using more precise RT–PCR experiments. Some genes showed expression in all lines, whereas others were silenced or downregulated only in one or two lines, suggesting a process of stochastic silencing. Further evidence for stochastic gene silencing in the *Arabidopsis* allopolyploids was obtained by showing variation in expression, including silencing of certain genes, among siblings within the same line and generation. For example, the expression of gene encoding a serine threonine kinase was mostly or entirely from one copy in seven of eight siblings examined, whereas only the other copy was expressed in the final individual. Adams *et al.* [22] compared expression patterns in three synthetic cotton allopolyploids formed from the same maternal species (albeit different individuals) but having different paternal genome donors. One gene (encoding a monooxygenase) showed striking similarities in organ-specific, gene-silencing patterns in all three genotypes, suggesting repeatable silencing of this gene, whereas silencing patterns were variable among genotypes for two other genes, consistent with stochastic silencing. These results suggest that gene expression in newly formed polyploids can be both plastic and stochastic; such variation might help explain the unstable phenotypes seen within allopolyploid *Arabidopsis* genotypes [14]. By contrast, the newly created cotton allopolyploids have relatively stable phenotypes, so it will be interesting to determine if this will be correlated with lower variability in gene expression among generations and individuals.

In contrast to the experiments with synthetic plant polyploids, Soltis *et al.* [25] examined expression patterns in natural allopolyploid *Tragopogon* by AFLP-cDNA to determine if gene-silencing patterns are reproducible in natural polyploid populations that have similar but reciprocal parentage. Expression variation was observed between reciprocal allopolyploid populations, potentially reflecting parental effects but also possibly arising from parental polymorphism or a more stochastic process.

Organ-specific changes in duplicate gene expression and subfunctionalization

An intriguing twist on gene expression in polyploids was recently revealed: silencing and relative expression levels of genes duplicated by polyploidy can be variable in different parts of the plant, indicating differential regulation of the two homoeologs during plant development [18,21,22]. In cotton, there is considerable variation in the relative expression levels and silencing patterns of duplicated gene pairs among organ types, especially in different floral whorls, beginning at the first generation after polyploidy. For example, expression of a calmodulin-binding protein and an ubiquitin-protein ligase ranged from silencing of one homoeolog in some organs to equal expression of both homoeologs in other organs [22]. Complete partitioning of expression between homoeologs can occur in different organs. For example, only one homoeolog of the alcohol dehydrogenase A (*adhA*) gene is expressed in cotton petals and only the other homoeolog in styles [18], indicative of subfunctionalization. The developmentally regulated patterns of reciprocal *adhA* homoeolog silencing are strikingly similar in natural cotton polyploids and in two synthetic allopolyploids. These observations suggest the possibility of instantaneous, epigenetic regulatory alteration that might be evolutionarily stable for more than one million years. Subfunctionalization of homoeologs has important consequences for our understanding of the long-term preservation of duplicated genes. Specifically, because organ or tissue-specific expression partitioning can arise quickly after the onset of polyploid formation, some duplicated genes are inferred to have a 'time to subfunctionalization' near zero, thereby escaping the otherwise inexorable fate of mutational obliteration. Future studies will provide insights into the types of genes, gene networks, or perhaps genomic contexts that are more susceptible to organ-specific expression modulation.

Transposon activation in newly synthesized polyploids

An additional and potentially evolutionarily significant dimension to our understanding of polyploidy has emerged from studies demonstrating polyploidy-induced activation of dormant transposable elements. Madlung *et al.* [23] used an *Arabidopsis* genomic microarray that surveys a heterochromatic region of chromosome 4 containing multiple transposons. They found that in *A. thaliana*, certain enhancer suppressor mutator (*En-Spm*)-like transposons belonging to the sunfish family displayed activation in an allopolyploid hybrid compared with its autotetraploid parental lines. They further showed loss of methylation associated with allopolyploidy concomitant with transcriptional activation of the element. Interestingly, transposition of the sunfish transposon appears to have occurred in autopolyploid *A. thaliana* and possibly in some allopolyploid lines too.

Transposon activation also occurs in newly synthesized wheat allopolyploids. Remarkably, activation of certain transposable elements can result in silencing of the adjacent downstream genes [16]. For example, activation of the Wis transposon caused silencing of the downstream *purB* gene (Figure 2). These studies show that allopolyploidy can induce transposable element activity, thereby creating the potential for insertional mutagenesis and potentially relevant changes in phenotype, while altering local patterns of gene expression by readout transcription. At present these phenomena have not been directly connected to adaptive variation, but it seems likely that evolutionarily significant examples will be forthcoming in the future.

Mechanisms of duplicate gene silencing in polyploids

What is the spectrum of mechanisms that can cause silencing of duplicated genes in polyploids? Initial insights into this question were provided by experiments where treatment of the natural allopolyploid *Arabidopsis suecica* with the methyltransferase inhibitor 5′-aza 2′-deoxycytosine (aza-dC) was shown to cause reactivation of two silenced genes, *RFP* and *TCP3*, suggesting epigenetic gene silencing caused by hypermethylation [26]. Wang *et al.* [21] used a different approach to determine if hypermethylation of DNA cytosines was responsible for silencing of some of the genes whose expression patterns had been characterized in *A. suecica*. RNA interference (RNAi) methodology (Figure 2) was used to silence two genes involved in DNA methylation, *DDM1* (encoding DNA methylation 1) and *MET1* (encoding methyltransferase 1), in *A. suecica* and to create DNA-hypomethylation lines. Two out of five genes tested, *RAD54* and *PP1* (encoding a putative protein) showed reactivation in the RNAi lines. The reactivation was attributed to inhibition of methylation-induced silencing of the genes. *RAD54* derepression in the RNAi lines was correlated with demethylation of the region containing the promoter sequence. Three other silenced genes were not reactivated in the RNAi lines, suggesting that mechanisms other than DNA methylation were responsible for gene silencing in those cases.

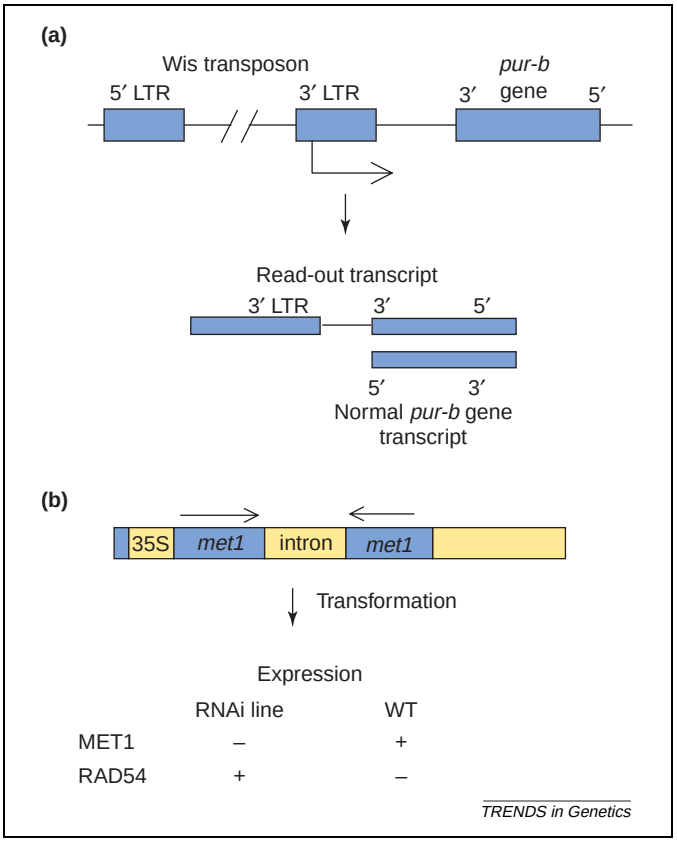

Figure 2. Mechanisms of duplicate gene silencing in plant polyploids. **(a)** Read-out transcription from a transposon (Wis) in wheat, causing silencing of the next gene downstream (*pur-b*), which is in the opposite orientation [13]. **(b)** RNAi methodology was used by Wang *et al.* [21] for silencing methyltransferase gene (*met1*) to inhibit methylation of hypermethylated and silenced genes, causing reactivation of RAD54 and indicating that hypermethylation acts as a silencing mechanism. Abbreviations: LTR, long terminal repeat; WT, wild type.

What other mechanisms cause silencing of duplicated genes in polyploids? Possibilities include deacetylation, methylation, other modifications of histones and positional effects from higher-order changes in chromatin structure (reviewed in Refs [5,8,27,28]). Transposon activation is another mechanism, as discussed earlier. Small RNAs and RNAi might have a role in homoeologous-gene silencing. An additional possibility is that of altered gene expression arising from regulatory mismatch in allopolyploid nuclei [29,30]. This mismatch might interact with altered cell volumes and stoichiometries of transcription factors and regulatory proteins in complex ways to generate widespread changes in gene expression. The foregoing factors are not mutually exclusive and mechanisms will undoubtedly vary depending on the gene.

Concluding remarks and perspective

Significant advances have been achieved in the past year in our understanding of the patterns, timing and mechanisms of duplicate gene expression in polyploids, building on the many advances realized over the preceding five years. Future studies will provide a greater understanding of the scale and scope of up- and down-regulation of genes in polyploids and the interactions of these genes in expression networks. These observational improvements will lead to a deeper understanding of underlying mechanisms, and also help us understand why some expression alterations in polyploids are repeatable among different lines and genotypes, whereas others appear to be more stochastic. Fine-scale studies of specific gene classes will be insightful and so will large-scale microarray surveys, such as those currently being performed in *Arabidopsis* [31] allotetraploids. Further characterization of mechanisms is likely to reveal a diversity of epigenetic responses including those related to histone modifications and other chromatin structural changes, small RNA- and RNAi-based mechanisms [32] and regulatory mismatches between parental genomes and their stoichiometric requirements for particular promoter-binding and effector proteins [29]. The scale of the phenomenon of expression alteration suggests that this process will prove to be a significant facet of polyploid evolution.

Update

While this paper was in preparation a study of gene expression in allopolyploid Senecio, using microarrays,

was published. Hegarty *et al.* [34] found considerable variation in the expression levels of many genes among diploid, allohexaploid and triploid Senecio species.

Acknowledgements

We thank Jeff Chen for comments on the article. Research in the authors' laboratories is supported by the National Science Foundation (USA), the United States Department of Agriculture and the National Science and Engineering Research Council of Canada.

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0168-9525/$ - see front matter
doi:10.1016/j.tig.2005.07.009