glucosylphosphatidylinositol (GPI)-anchored protein expressed in the synergids [16]. It was thus hypothesized that both LORELEI and FERONIA participate in the same signaling pathway. Interestingly, the paralogs of *LORELEI*, *SETH1* and *SETH2*, encode GPI-anchored proteins in pollen vegetative cells and their loss of function prevents pollen-tube growth [17]. It is thus likely that the arrest of pollen-tube tip growth requires the function of different family members for each component of the signaling cascades acting in parallel in the synergids and pollen tube.

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Small RNAs: How Seeds Remember To Obey Their Mother

The endosperm is one of two products from the double fertilization event that occurs during sexual reproduction in flowering plants. A series of recent reports highlights the unusual genetic regulatory mechanisms that occur in endosperm and suggests a role for transposon regulation in imprinting.

Nathan M. Springer

Sexual reproduction in plants involves a double fertilization event that produces the embryo (2n) and the endosperm (3n) (reviewed in [1]). The triploid endosperm is created when the central cell of the female gametophyte (2n) is fertilized by a 1n sperm cell. The balanced development of the endosperm and embryo is critical for the production of viable off-spring. The majority of cereal grain is endosperm tissue (Figure 1) and a large proportion of our food supply is derived directly or indirectly from the endosperm. The endosperm is important in determining the viability of crosses [2] and has unusual chromatin organization [3] and gene expression patterns [4], including imprinting [1].

Genomic imprinting, which occurs in mammals and flowering plants [5], refers to the differential expression of the maternal and paternal alleles of a gene. The first example of genomic imprinting was identified in plant endosperm tissue [6]. Subsequent research has identified a handful of genes that are imprinted in plant endosperm tissue and many of these genes play important roles in regulating endosperm development [1,5]. The parental conflict theory suggests that imprinting arose due to competition between the maternal and paternal genomes and predicts maternally expressed growth inhibitors and paternally expressed growth promoters [7].

Detailed studies of the mechanism of genomic imprinting in plants have identified roles for DNA methylation and histone modifications [1,5]. Expression of the DEMETER (DME) protein, a DNA glycosylase that can remove DNA methylation [8], in the central cell leads to activation of the maternal alleles for some imprinted genes [1,5]. The paternal allele is kept silent by DNA methylation for some genes and/or through histone modifications by Polycomb group proteins [1,5]. Interestingly, there is evidence that the maternally expressed allele of some imprinted genes is required for maintaining the silence of the paternal allele [1,5].

Recent work by Gehring *et al.* [9] and Hsieh *et al.* [10] provides evidence for genome-wide demethylation of the maternal genome in *Arabidopsis* endosperm tissue. The genome-wide demethylation of the maternal genome is consistent with previous observations of maize endosperm [11]. Gehring *et al.* [9] combined endosperm methylation profiling with expression

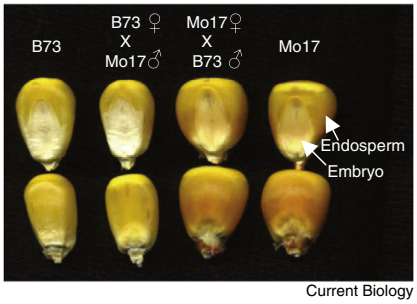

Figure 1. Maternal effects in maize.

Maize seeds from the inbred genotypes B73 and Mo17 are shown along with both of the reciprocal F1 hybrids (the two rows show different sides of the seed). Note the strong similarities between the F1 hybrid seed and the maternal parent. The positions of the embryo and endosperm are also indicated.

analyses to identify a set of genes that might exhibit imprinting and were able to confirm complete or partial imprinting for six of the twelve genes tested. The authors estimate that ~50 genes might exhibit imprinted expression in *Arabidopsis* endosperm tissue [9].

The majority of loci that exhibit demethylation in endosperm tissue are transposable elements or fragments derived from transposons [9,10]. Interestingly, while the majority of sequences are hypomethylated, there is also evidence for a number of sequences that are hypermethylated in CHG or CHH contexts [10]. CHG and CHH methylation is generally produced by a DNA methylation pathway that is distinct from the CG pathway, specifically, a pathway in which small interfering RNAs (siRNA) target DNA methylation [12]. A careful examination of the sequences with increased levels of CHG and CHH methylation in endosperm reveals that these are often associated with siRNA and suggests that genome-wide demethylation may stimulate production of siRNA from some loci.

The increase of siRNA-mediated DNA methylation at some loci in endosperm fits very nicely with the recent findings of Mosher *et al.* [13]. There are thousands of siRNAs, corresponding to over 1% of the *Arabidopsis* genome, that are produced by RNA polymerase IV (PolIV) [14]. However, PolIV mutant lines have no obvious morphological defects [15,16]. To further understand the role of PolIV-dependent (p4)-siRNAs in *Arabidopsis*, Mosher *et al.* [13] studied developmental expression patterns. A large class of p4-siRNAs is only expressed in the central cell and developing endosperm. These p4-siRNAs are predominantly expressed from the maternal allele in the endosperm tissue, and the expression of this class of p4-siRNAs requires that the p4-siRNA biosynthetic genes (including *NRPD1A*, *DCL3* and *RDR2*) are functional in the female gametophyte.

Together these findings suggest an endosperm-specific regulatory network of demethylation and p4-siRNA production. Expression of DME in the central cell results in genome-wide demethylation of the maternal genome. The hypomethylated genome then provides a substrate for the production of p4-siRNAs. Following fertilization by a sperm cell, there is continued expression of the maternal p4-siRNAs but the paternal genome does not provide a template for production of p4-siRNAs. The maternal p4-siRNAs can regulate maternal and paternal transposons and genes. This model would have implications for the evolution of imprinting and suggests several potential roles for p4-siRNAs.

The model suggests that the process of imprinting in plants may be based on a genome-wide phenomenon of maternal demethylation and p4-siRNA production [10]. This view of imprinting would suggest that the imprinting of specific genes is controlled by adjacent transposons. Gene-specific imprinting would occur when these adjacent transposon-related sequences are subject to DME-mediated demethylation and act as allele-specific enhancers. In contrast to the parental conflict theory [7], this would suggest that imprinting originally arose as a mechanism for repression of transposable elements in female gametes. Indeed, there is evidence that imprinting of *FWA* is controlled by fragments of transposable elements [17,18]. This view of imprinting would suggest that the imprinting of specific genes is controlled by adjacent transposons. In some cases this imprinting might be stabilized by selection if there is a fitness advantage to maternal- or paternal-specific expression of a given gene in agreement with the parental conflict theory.

One possible role for the p4-siRNAs may be to reinforce transposable element silencing. In another recent study, Slotkin *et al.* [19] found a similar phenomenon in pollen. Pollen has a vegetative nucleus in addition to the two sperm cell nuclei that will fertilize the egg and central cell. There is an increase in expression of transposable elements in the vegetative nucleus, which is accompanied by increased production of 21-nucleotide siRNAs. Slotkin *et al.* [19] propose that the release of transposon silencing in the vegetative nucleus allows for production of *trans*-acting siRNAs that can reinforce silencing of transposable elements in the sperm cell nuclei and that this may be a conserved feature of germline companion cells. A similar system may be at work in the central cell of the female gametophyte. The demethylation of transposons in the central cell, a companion cell for the egg cell, provides the potential for increased expression of p4-siRNAs that may be able to act in *trans* to reinforce silencing of repetitive elements in the egg cell or developing embryo. The identification of systems to reinforce silencing of transposable elements in both the male and female gametes may suggest the importance of silencing transposons in this haploid phase of the reproductive cycle and provides a mechanism to reinforce silencing of transposable elements in each generation.

An additional role for the p4-siRNAs may be to condition maternal effects. The genotype and environment of the maternal plant can condition a number of seed phenotypes (Figure 1) [20]. The p4-siRNAs that are produced from the maternal allele could act in *trans* to regulate the production or stability of both maternal and paternal transcripts in the developing endosperm. Indeed, gene expression studies of developing maize endosperm tissue identified a large number of genes with expression patterns that are conditioned by the maternal parent but are not imprinted [3]. These maternal-like expression levels may be conditioned by a subset of p4-siRNAs that target genes instead of transposons.

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Gaze Perception: Is Seeing Influenced by Believing?

Gaze perception has been thought to be stimulus-driven. This view is challenged by a new demonstration that a gaze direction aftereffect can be influenced by beliefs about the gazer's ability to see.

Stephen R.H. Langton

Stare for a while at a photograph of a face of someone whose eyes are gazing over your left shoulder. If, after having done this, you look at a photograph of someone whose eyes are actually directed towards your left ear, you are likely to mistakenly perceive this person to be looking straight at you. In other words, prolonged exposure to a face gazing in one direction will bias subsequent perception of gaze direction in the opposite direction — a complex example of a perceptual aftereffect [1–4]. In a paper in this issue of *Current Biology*, Teufel *et al.* [5] report that judgements of eye-gaze direction can be similarly influenced after repeated exposure to a person wearing mirrored goggles whose head was angled in a particular direction, *but only when participants believed that the gazer could see through the goggles*. The implication is that the perceptual coding of gaze direction can be influenced by the attribution of a mental state to the gazer.

The process by which perceptual aftereffects arise is known as adaptation and is thought to reflect changes in the responses of neural mechanisms that encode the visual property in question [6]. The classic example occurs when staring for a minute or two at a waterfall — unchanging downward motion — results in the perceptual distortion of a subsequently viewed stationary object, which appears to be moving upwards. Similar effects occur with other relatively low-level perceptual properties such as colour, size and tilt [6]. More recently, however, researchers have observed that adaptation can occur with more complex stimuli such as faces [7–9].

Aftereffects are important because they tell us something about the mechanisms underlying perceptual experience. For example, the work on gaze adaptation [4] has suggested that gaze direction is likely to be signalled by the pooled output of separate cell populations each broadly tuned to a different gaze direction (for example, left, right and direct). Aftereffects that have been observed following adaptation to heads rotated at different angles have led to similar conclusions about the coding of head orientation [10].

The gaze and head adaptation studies marry reasonably well with earlier work by Perrett and colleagues [11,12], whose recordings of single cells in macaque brains identified separate populations of cells that were maximally responsive to different eye-gaze directions, different views of the head, and also for bodies adopting upright or bent-over postures. Their influential suggestion was that a neural mechanism functions to signal the direction of another individual's social attention by combining information from eye-gaze, head orientation and body posture. Teufel *et al.*'s [5] finding that adaptation transfers from head direction to the perception of eye-gaze direction seems to implicate this neural mechanism.

According to one view, this neural circuitry is hard-wired and functions to compute attention direction when provided with the appropriate input [13,14]. Indeed, given how readily a pair of white circles containing smaller black circles is perceived as a pair of