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GENE EXPRESSION

Small but dominant RNA

> the epigenetic silencing of recessive alleles by dominant ones through small RNAs could represent a general mechanism

As Mendel observed over a century ago, the manifestation of a phenotype can crucially depend on the dominant–recessive relationship between alleles at a given locus. A study of plant self-incompatibility suggests a new mechanism by which such dominant–recessive relationships arise: in this system, a small RNA encoded by the dominant allele acts in *trans* to epigenetically silence the expression of the recessive allele.

A plant uses a 'self-incompatibility' mechanism to reject fertilization by pollen that is genetically too similar to itself. In *Brassica* species, this trait is controlled by three tightly linked, multiallelic loci and particularly by the genes encoding the male-specific protein *S*-locus protein 11 (SP11) and its female-specific receptor *S*-receptor kinase (SRK). Rejection occurs if the parent plants carry the same combination of *SP11* and *SRK* alleles (known as the '*S* haplotype'). But the rejection response also depends on the interaction between dominant and recessive *S* haplotypes within the pollen-generating plant. In these diploid plants, recessive alleles are 'masked' in incompatibility reactions. For example, pollen from a heterozygous plant that carries a dominant haplotype (S_9) and a recessive haplotype (S_{60}) will still be accepted by a homozygous S_{60} plant, as the expression of the donor S_{60} allele will be suppressed (see the figure).

The authors had previously found that in a heterozygote, the silencing of expression of the recessive haplotype correlated with cytosine methylation at the silenced *SP11* promoter. But by what mechanism? *In silico* searches for sequences that were complementary to the methylated promoter regions led the authors to identify a new, fourth gene, which they named *SP11* methylation inducer (*SMI*). *SMI* flanks the *SP11* gene and is expressed in anthers before methylation occurs.

SMI encodes a small 24-nucleotide RNA (*Smi*) that when transcribed from the dominant haplotype directs the DNA methylation and consequent silencing of the pollen *SP11* gene on the recessive haplotype. The specificity of this interaction is determined by the degree of sequence complementarity of *Smi* to its target: the *Smi* on the dominant haplotype is highly complementary only to the promoter of the recessive *SP11* allele — *Smi* cannot, therefore, silence the neighbouring, dominant *SP11* itself.

Surprisingly, an *SMI* locus is also present on the recessive haplotype; however, a base substitution makes this recessive *Smi* non-functional. Indeed, when this recessive *Smi* sequence was inserted on the dominant S_9 haplotype in transgenic plants, it abolished the dominant properties of this locus.

This rather intricate system probably exists for a good reason, as theory suggests that a dominant–recessive architecture would increase the number of compatible mates. Aside from the particular biological phenomenon explored in this study, the epigenetic silencing of recessive alleles by dominant ones through small RNAs could represent a general mechanism by which heterozygote organisms achieve widespread monoallelic expression.

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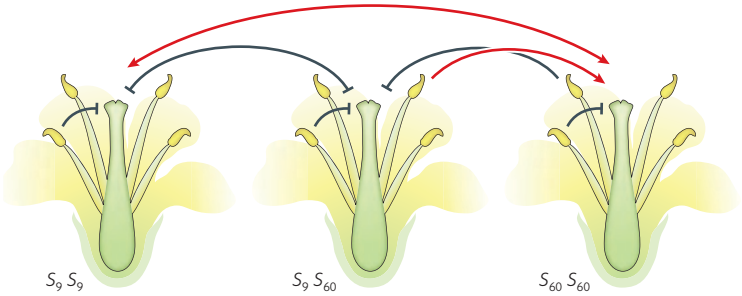

ORIGINAL RESEARCH PAPER Tarutani, Y. *et al.* *Trans*-acting small RNA determines dominance relationships in *Brassica* self-incompatibility. *Nature* **466**, 983–986 (2010)
FURTHER READING Pastinen, T. Genome-wide allele-specific analysis: insights into regulatory variation. *Nature Rev. Genet.* **11**, 533–538 (2010)