S011 Flavonoid biosynthesis and endosperm cellularization – controlling seed size in *Arabidopsis*

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Barriers to hybridisation between species and within species are frequently post zygotic in nature and act by perturbing normal seed development. A classic example of such a breeding barrier is the 'triploid block' where crosses between diploid (2x) and tetraploid (4x) parents leads to an over- or under-proliferation of seed endosperm tissue resulting in death of the triploid embryo. It has been established that genomic imprinting, the parent-of-origin-specific expression of genes in the endosperm, plays a major role in this phenomenon. In *Arabidopsis thaliana* many ecotypes (e.g. L*er*) tolerate 2x X 4x interploidy crosses whereas the Columbia-0 ecotype (Col) exhibits triploid block when the paternal parent is tetraploid, with over-proliferation of endosperm being the cause of seed abortion. Previous work identified the *TRASPARENT TESTA GLABRA 2* locus (*TTG2*) as a potent modifier of the triploid block in *A. thaliana*, indeed *ttg2* mutants (maternal) are effective in rescuing Col4x-induced seed lethality in crosses to both Col-0 and L*er* 2x mothers. *TTG2* encodes a WRKY transcription factor that plays a regulatory role in the accumulation of flavonoid pigments in the endothelial layer of the seed coat. Here we describe recent work on mutants of the flavonoid biosynthesis pathway that reveal a key role for flavonoids in the regulation of seed size through their impact on the timing of endosperm cellularisation. The potential mechanism for their influence on this critical step of seed development will be discussed.