

# A WRINKLE in the time of seed maturation: chromatin-level control of fatty acid biosynthesis

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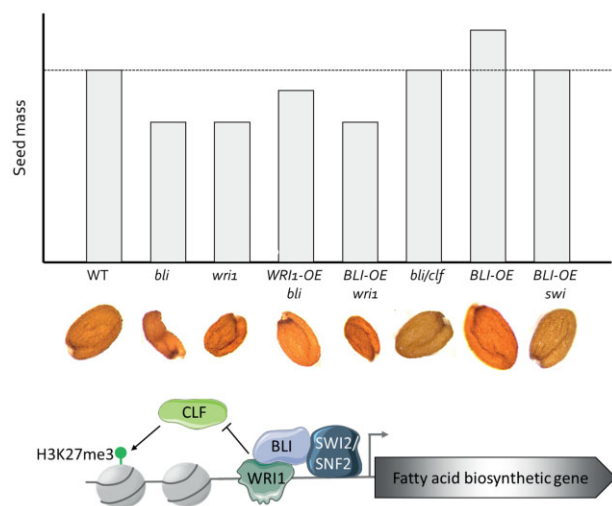
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Fatty acid biosynthesis during seed development is essential for storing carbon and energy to support seedling growth after germination. Aside from their significance to plant growth, plant fatty acids also play important roles in animal nutrition and as biofuel feedstocks. The transcription of fatty acid biosynthetic genes requires several transcription factors whose detailed mechanisms of action remain obscure.

The WRINKLED 1 (WRI1) transcription factor is essential for seed maturation by regulating fatty acid biosynthetic genes through elusive mechanisms (Kong et al., 2020). Now in *The Plant Cell*, Ruihua Huang, Mengling Liu and colleagues (Huang et al., 2022) describe how the WRI1-interacting protein BLISTER integrates multiple protein–protein interactions with chromatin and histone remodeling factors to activate WRI1 target genes for seed maturation (see Figure).

To begin their investigation, the authors screened a cDNA library from maturing *Arabidopsis* seeds using the WRI1 protein as a bait and uncovered BLISTER as a WRI1-interacting protein. The WRI1–BLI interaction was validated by in vitro pull-down and bimolecular fluorescence complementation (BiFC). A yeast two hybrid (Y2H) assay revealed that the structural maintenance of chromosome (SMC)-like domain of BLI was required to interact with WRI1. The *bli* and *wri1* mutant seeds contained fewer fatty acids compared with wild-type seeds, which was correlated to decreased biosynthetic gene expression in both mutants. Trans-complementation analysis showed that WRI1 overexpression in the *bli* mutant restored normal seed maturation, whereas overexpression of BLI in the *wri1* mutant did not (see Figure). By combining chromatin immunoprecipitation and reporter gene trans-activation assays, the authors revealed that BLI and WRI1 function together for full transcriptional



**Figure** BLISTER regulates WRI1-dependent seed maturation through chromatin regulation. Adapted from Huang et al. (2022), Figures 1, 5, and 9.

activation of fatty acid biosynthetic genes, while BLI depends on WRI1 to stimulate transcription.

Using MNase to generate nucleosome-protected footprints, the authors showed that BLI prevents excessive nucleosome loading at WRI1-binding motifs. Consistent with that, BLI interacted with a subunit of the SWI2/SNF2 nucleosome remodeler complex in Y2H and BiFC experiments. In addition, BLI interacted with the CURLY LEAF (CLF) histone methyltransferase required for H3K27me<sub>3</sub>, facultative heterochromatin, and developmental gene silencing (Schatlowski et al., 2010). Interestingly, H3K27me<sub>3</sub> increased at WRI1 targets in *bli* mutant seeds in comparison to wild-type, while a distinct histone modification that correlates

with active transcription was decreased. In line with that, introducing a *clf* mutation in the *bli* mutant background restored a normal seed phenotype (see [Figure](#)). The authors then performed an elegant series of in vivo co-immunoprecipitation assays to investigate how BLI integrates multiple interactions with WRI, CLF, and the SWI2/SNF2 complex. This revealed that WRI1 and CLF compete to interact with the SMC-like domain of BLI ([Schatlowski et al., 2010](#)), whereas WRI1 requires BLI as a bridge to interact with SWI2/SNF2 (see [Figure](#)). Chromatin immunoprecipitation experiments revealed increased CLF binding and concurrent decreased SWI2/SNF2 binding to WRI1 target genes in the absence of BLI, revealing the regulatory role of BLI in the orchestration of WRI1-dependent fatty acids gene activation through the regulation of chromatin.

This study thus presents a novel regulatory function for BLI in chromatin-based fatty acid gene transcriptional activation, which expands the previously assumed role of BLI in H3K27me3-related transcriptional repression ([Schatlowski](#)

[et al., 2010](#)). Indeed, the study of Huang, Liu and colleagues suggests that BLI repels CLF away from WRI1 target genes and allows their activation by increasing chromatin accessibility in cis-regulatory regions. In future studies, genome-wide analysis of BLI-interacting proteins and genomic targets may clarify this dual role in transcriptional regulation.

## References

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