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**IN BRIEF**

# A Functional Link between Mitochondria and the Cell Wall in Stress Responses OPEN

Both mitochondria and the cell wall are targeted by and respond to stresses. The cell wall is physically affected by abiotic stresses and is the site of initial attack by pathogens. During responses to such stresses, the cell wall is remodeled to maintain integrity and allow flexibility (reviewed in Hamann, 2015; Tenhaken, 2015). Stresses can also alter mitochondrial function, especially in terms of the rate of respiratory electron transport, and mitochondria are important hubs for signaling to other parts of the cell in response to stress conditions (reviewed in Ng et al., 2014; Huang et al., 2016). Now, **Hu et al. (2016)** establish that not only are both compartments involved in stress responses, there is a functional connection between mitochondrial function under stress and cell wall integrity.

A chemical screen for inhibitors of cell division identified C17 (see figure), which inhibited cytokinesis, inhibited hypocotyl elongation and decreased cellulose content in plants. To learn more about what mediates these effects, the authors searched for suppressor mutations. Several mutants that showed C17 tolerance had alterations in invariant residues of transmembrane regions of CESA1 and CESA3, catalytic subunits of the cellulose synthase complex. Consistent with these findings, cells treated with C17 resembled those of *cesa* mutants, with fewer cellulose synthase complexes in the plasma membrane and correspondingly weakened cell walls. Thus, Hu et al. have discovered a novel inhibitor of cellulose synthase—in itself a notable accomplishment.

Intriguingly, a screen for further suppressors led the authors to two pentatricopeptide repeat proteins that are targeted to the mitochondria. Despite being altered in mitochondrial proteins with no known link to the cell wall, *cell wall maintainer1* (*cwm1*) and *cwm2* were tolerant of C17 as well as other inhibitors of cellulose synthase. In addition, both mutants could rescue the growth of the *je5* mutant (disrupted in *CESA3*) under osmotic stress. Thus, the lack of mitochondrial proteins CWM1 and CWM2 seems to allow plants to maintain cell wall integrity in the face of suboptimal cellulose synthase activity.

Hu et al. found that, like other pentatricopeptide repeat proteins, CWM1 and CWM2 are involved in editing mitochondrial transcripts, in this case those encoding subunits of various respiratory complexes. The *cwm1* and *cwm2* mutants had correspondingly reduced levels of those complexes, including complex III. Importantly, antimycin A, another inhibitor of complex III, also conferred tolerance to C17. This finding supports the conclusion that modulation of mitochondrial respiratory activity, in particular that related to complex III, is linked to the maintenance of cell wall integrity under stress.

Finally, Hu et al. provide evidence that this signaling from the mitochondria to affect the cell wall involves ANAC017, a transcription factor important in retrograde signaling to the nucleus upon mitochondrial dysfunction. This work thus provides exciting opportunities to explore mitochondrial transcript editing, mitochondrial dysfunction, cellulose biosynthesis, and the signaling that connects them all.

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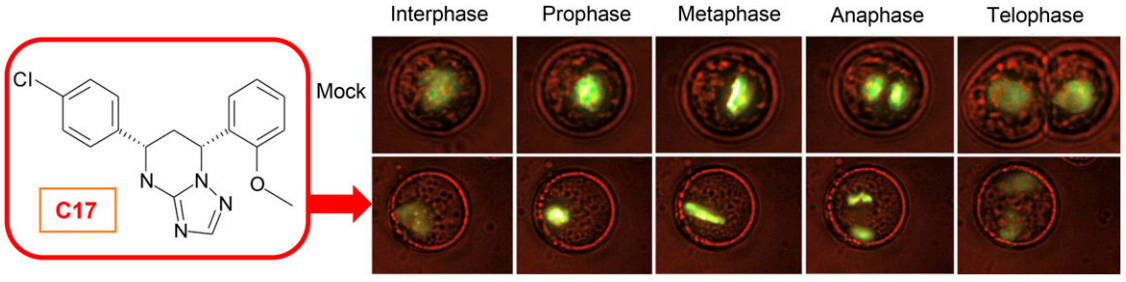


Novel cellulose synthase inhibitor C17 (left) inhibits cytokinesis in Arabidopsis suspension cells (right). Histone 2B in the nuclei is labeled with yellow fluorescent protein. (*Adapted from Hu et al. [2016], Figure 1.*)

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