



LETTER TO THE EDITOR

Phytomelatonin and post-drought stomatal recovery: psychotropic-like signaling in guard-cell dynamics

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Waseem and colleagues (2025) provide a timely synthesis of why rapid post-drought stomatal reopening is physiologically important, and they discuss how phytomelatonin, 5-aminolevulinic acid, and brassinosteroids may counter abscisic acid (ABA)-induced closure (Waseem *et al.* 2025). Their central proposal, that these regulators promote reopening largely by limiting ABA accumulation and attenuating downstream H_2O_2/Ca^{2+} signaling in guard cells, is valuable because it shifts attention from drought-phase closure to the recovery bottlenecks that constrain carbon gain after rewatering.

One aspect that would benefit from clearer framing is the conditional nature of phytomelatonin effects on guard-cell behavior. In the review, phytomelatonin is discussed as an ABA-opposing signal, acting through reduced ABA biosynthesis or enhanced ABA catabolism and thereby lowering H_2O_2 -triggered Ca^{2+} elevations that suppress K^+ influx (Waseem *et al.* 2025). Yet broader literature describes both antagonistic and cooperative melatonin–ABA interactions, with outcomes that depend on stress context, dose, and timing. For example, a recent review in “Frontiers in Plant Science” emphasizes that melatonin can downregulate ABA synthesis in some settings but may also support ABA-related responses in others, including stomatal behavior, depending on experimental conditions and the physiological goal, such as water conservation during stress *vs.* rapid photosynthetic recovery after rewatering (Ali *et al.* 2023).

Importantly, there is also direct experimental evidence that exogenous melatonin can promote stomatal closure and reactive oxygen species production as part of defense signaling, at least in *Nicotiana benthamiana*, where melatonin and its homologs induced closure and reactive oxygen species *via* identified melatonin receptors (Kong *et al.* 2021). This type of evidence makes it more challenging to treat phytomelatonin as a uniformly pro-opening agent and suggests that the same molecule can participate in either closing or reopening programs, depending on the phase of the stress trajectory and the signaling network engaged.

A practical way to prevent overgeneralization would be to add a short synthesis that separates recovery-phase reopening from drought-phase closure and distinguishes

evidence based on guard-cell measurements from evidence inferred from transcript changes in bulk tissues. Even a brief boxed paragraph could help readers track which studies directly quantify ABA levels, guard-cell reactive oxygen species, and Ca^{2+} dynamics and stomatal conductance under matched designs, and which ones extrapolate mechanistic links from gene expression or antioxidant markers.

Finally, terminology matters more than it seems. Because melatonin is strongly associated with animal neuroendocrinology outside plant science, the term phytomelatonin is useful not only taxonomically but also to limit cross-domain semantic carryover that can bias how non-specialists interpret exogenous applications. A cautious wording around dose, delivery, and physiological phase would help keep the focus on plant recovery physiology while reducing the risk of misplaced pharmacological analogies.

Overall, Waseem *et al.* (2025) provide a helpful framework for thinking about stomatal reopening as an active, regulated process. Clarifying when phytomelatonin promotes reopening *vs.* when it participates in closure-associated signaling would further strengthen the review and make it easier to translate the proposed mechanisms into experimental designs and agronomic hypotheses.

References

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