Ethylene induced macromolecule catabolism - the switch required for bud meristem growth resumption?

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Chemical and physical stress agents induce bud dormancy release

hydrogen cyanamide (HC) and Heat Shock (HS)

Stress-related signals may have central role in the execution of the dormancy release cascade

We may identify core functions by comparative analysis of responses to such stimuli

Azid (AZ) Hypoxia
Our initial model for the molecular cascade that activates dormancy release (based on years of comparative analyses of response to dormancy release stimuli...)

Here we bring on the tip of the fork support for the model and suggest that Ethylene induced catabolism may be a central switch of dormancy release.

Pang et al., 2007, JExBot
Halali et al., 2008, Planta
Ophir et al., 2009, PMB
Azid, HC and HS temporarily induce anaerobic respiration, to face energy shortage caused by impaired aerobic respiration.

Anaerobiosis induce bud dormancy release

Temporary induction of fermentation also occur under vineyard conditions during deep dormancy, indicative of an energy crisis.

Ophir et al., 2009, PMB
Or, unpublished
- Sucrose degradation is activated during deep dormancy.
- It is probably induced in response to enhanced Glycolysis needed to supply pyruvate for anaerobic respiration.
- Sucrose degradation decrease during dormancy release in parallel with increased sucrose synthesis capacity and sucrose level.
- Similar regulation appears in response to HC and additional stimuli (not shown).
Ethylene biosynthesis

- HC and AZ upregulate Ethylene synthesis by temporary induction of ethylene synthesis genes (ACS, ACO)

- Ethylene induce dormancy release

- Temporary increase in ethylene biosynthesis capacity is also regulated at the transcription level during the natural dormancy cycle

- Inhibition of ethylene signaling inhibit bud break and the effect is timing dependent

Shi et al, 2018, submitted
**Ethylene signaling**

We formerly identified ERF genes, which are known sensors of energy crisis and activate hypoxic response.

- As expected, they accumulates in response to hypoxia.
- Less expected, they directly respond to HC induced signal.
- They are positively regulated during deep dormancy in transcript or protein level.

Ophir et al., 2009, PMB

Shi et al., in preparation.
We identified all the ERFs, as well as other genes that are regulated by HC, Azid, hypoxia **AND** ethylene...assuming that they are primary regulators of the cascade.
ABA delay bud break and reduce the enhancing effect of HC, HS, Azid and hypoxia on dormancy release.

Recovery from the inhibition was demonstrated in the combined ABA-HC treatment whereas no recovery was evident in the ABA-treated, compared to the control.

Zheng et al., 2015, JExBot
HC lead to reduction of ABA levels and increase of level of ABA degradation products in the buds.

Down-regulation of VvNCED1 and up-regulation of VvA8H-CYP707A4 levels by HC may be responsible together for decreased ABA level and increased ABA catabolites level in response to HC.

Zheng et al., 2015, JExBot
Zheng et al., 2018, PCE
The OE VvA8H-CYP707A4 grapevine lines presented significantly improved rate and level of dormancy release.
Profiling the expression of GA metabolism throughout the natural dormancy cycle suggests during endodormancy release:

- levels of active GA biosynthetic enzymes increased
- levels of active GA degradation enzyme decreased

These results are in agreement with the initial model

However... In reality, things appears to be more complicated...

Zheng et al., 2018, JExBot
During initial steps of meristem activation, GA has a strong inhibiting effect. Once meristem is activated, GA has an enhancing effect, probably on primordia growth.
Regulation of grape bud dormancy release

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Thank you and thanks to

The Bud Dormancy team

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