ABA is a modulator of endodormancy release in grapevine buds

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In warm-winter regions, bud dormancy release poses a major obstacle to commercial viticulture. Artificial substitutes for chilling are thus mandatory. Induction of respiratory stress by artificial stimuli s(uch as hydrogen cyanamide, HC) leads to dormancy release of endodormant buds via an uncharacterized biochemical cascade of events, which we try to dissect in order to design proper alternatives. We formerly proposed that (1) ABA inhibits bud dormancy release via repression of meristem activity; (2) the enhancing effect of dormancy release stimuli involves reduction in ABA level and/or changes in response to ABA. Our data show that ABA indeed inhibits dormancy release in grapevine buds and attenuates the advancing effect of HC. However, HC-dependent recovery was detected, and was affected by dormancy status. Regulation of central players in ABA metabolism (VvNCED and VvABA8'OH) correlated with decreased ABA and increased ABA-catabolite levels in HC-treated buds. The activity of the major bud VvNCED and VvABA8'OH was verified in vivo, and as predicted, transgenic vines over expressing VvABA8'OH presents enhanced bud break and had an interesting effect on apical dominance. Expression profiling during the natural dormancy cycle revealed that at maximal dormancy, VvNCED1 expression is down regulated while that of starts to drop while levels of VvABA8'OH transcript and ABA catabolites increase sharply. This may provide initial support for the involvement of ABA metabolism in the execution of natural dormancy as well. We will additionally report on 1) behavior of transgenes with modified ABA response; 2) potential interaction between ABA and other growth regulators (GA, JA, Ethylene); 3)effect of ABA and other stimuli on the bud transcriptom.