Chilling induces sugar and ABA accumulation that antagonistically signals for symplastic connection of the dormant bud

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Abstract

Bud endodormancy (ED) release requires accumulating a certain amount of cold exposure, measured as chilling units. We used the potato tuber as a model system to investigate the mechanism underlying ED duration following chilling. Sweetening is a well-known response of potato tuber to chilling. We showed that ED release correlated better with sugar units' (SU) accumulation than chilling units. In turn, sweetening was associated with shorter ED. A logistic function was developed to predict ED duration based on SU measurements. Modifying SU level by knockout of the vacuolar invertase gene (*StVInv*) extended ED, while *StVInv* overexpression shortened it. We induce higher SU and shorter ED by silencing the energy sensor SNF1-related protein kinase 1 (SnRK1). We found that the time gap between sugar accumulation and bud burst is associated with abscisic acid (ABA) level that serve as a growth inhibitor. SU accumulation induced by chilling or transgenic lines reduces plasmodesmata (PD) closure in the dormant bud meristem. Chilling or knockout of ABA 8'-hydroxylase induces ABA accumulation, parallel to sweetening, and antagonistically promotes PD closure. Our results suggest that chilling induces SU and ABA accumulation that antagonistically signals for symplastic connection of the dormant bud.

Ref: Danieli et al. PC&E 2023.