

**Title** Postharvest sprouting suppression of potato tubers as related to the role of cell lipids and patatin phospholipase

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### **Abstract**

Potato is the world's most widely grown tuber crop and the fourth largest food crop in terms of fresh produce after rice, wheat and corn. A major problem in tubers supply to the fresh, industrial and seed markets is sprouting during storage. Sprouting due to tuber dormancy release causes weight loss and tuber softening, and is associated with the conversion of starch to sugar, which is undesirable for the processing industry. The use of sprout-preventing chemicals, such as chlorpropham (CIPC), is problematic due to possible negative impact to the environment and human health. Moreover, commonly used sprout-preventing chemicals delay germination of seed tubers in the field. We developed a scalable method to inhibit potato tuber sprouting during storage by fogging with monoterpene L-carvone which is extracted from peppermint/spearmint species and is commercially available. Preliminary results showed that sprouting suppression by L-carvone is associated with cell sap leakage. A similar induction of membrane leakage occurs when potato parenchymal cells are treated with lipophilic organic acid molecules. This event facilitates migration and binding of patatin 03 to the cell wall. Patatins are a family of protoplasmic proteins, some of which have been demonstrated to be acyl hydrolases. Using anti-patatin polyclonal antibodies we showed that patatins concentrate around the base of the apical buds during sprouting. To investigate the relationship between patatin and sprouting inhibitor action, we determined the effect of L-carvone on potato tuber buds and also on Arabidopsis leaves. Our preliminary study shows that level of lysolipids, including lysophosphatidylcholines (lysoPC) and lysophosphatidylethanolamines (lysoPE), potential products of patatin acyl hydrolase activity, are altered in both Arabidopsis leaves and in sprouting buds of potato tubers treated with L-carvone. Our working hypothesis is that the lipophilic organic molecules which are sprouting inhibitors, cause cell membrane distortion, resulting in leakage, alterations in lipids with signaling properties, differential expression of patatin and migration of patatin 03.