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Ethylene, phospholipid signalling and programmed cell death in plants

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Treatment of plant cells with a variety of biotic (e.g. pathogenic bacteria, virus, fungal toxins) and abiotic (UV radiation, ozone, heavy metals) elicitors induces cell death exhibiting typical features associated with apoptosis in animal cells such as cytoplasmic shrinkage, chromatin condensation and nuclear and DNA fragmentation. During developmental events, cell death is often accompanied by symptoms of autophagy in which the vacuole size increases and organelles disappear until the cytoplasm is virtually devoid of structure. In addition, also apoptotic-like features are observed in developmental cell death. Common to all these cell death events is the prominent involvement of ethylene. Ethylene's mode of action in programmed cell death has not yet been elucidated. Suspension-cultured tomato cells were used to study the signal transduction events and the role of ethylene in elicitor-induced plant cell death. Treatment of cells with either the anti cancer drug camptothecin, cadmium sulphate or fungal elicitors induced an increased production of H_2O_2. Both H_2O_2 production and cell death were effectively suppressed by simultaneous addition of antioxidants, indicating that ROS production is instrumental in cell death. Cell death was associated with increased activity of caspase-like proteases and treatment of the cells with inhibitors specific to human caspases blocked both H_2O_2 production and cell death. Apart from the involvement of caspase-like proteases and H_2O_2 we showed the involvement of phospholipase-C and -D signaling in cell death. Cell death in elicitor-treated tomato cells was dependent on ethylene. Blocking ethylene production or perception alleviated the effect of the chemicals on both H_2O_2 production and cell death. The interaction of different signaling pathways and the role of ethylene in cell death are discussed.