

## Capacity to control oxidative stress-induced caspase-like activity determines the level of tolerance to salt stress in two contrasting maize genotypes

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### Abstract

The response of two maize (*Zea mays* L.) genotypes, named GR (salt-tolerant) and SK (salt-sensitive), to salt stress (150 mM NaCl) was investigated under controlled environmental growth conditions. Genotype SK experienced more oxidative damage than the GR genotype when subjected to salt stress, which corresponded to higher  $O_2^-$  production rate and  $H_2O_2$  content in the SK genotype than the GR genotype. Induction of caspase-like activity in response to salt stress was stronger in the SK genotype than in the GR genotype. On the other hand, induction of antioxidant enzyme activity to scavenge  $O_2^-$  and  $H_2O_2$  in response to salt stress was weaker in the SK genotype than in the GR genotype. Consequently, the higher level of oxidative damage in the SK genotype in response to salt stress was manifested as more extensive cell death and biomass reduction in the SK genotype than it was in the GR genotype. Our results suggest that a direct relationship exists between salt stress-induced oxidative damage and cell death-inducing caspase-like activity, with tolerance to the salt stress being controlled by the efficiency of the plant antioxidant enzymes in limiting salt stress-induced oxidative damage and thus limiting cell death-inducing caspase-like activity.

### Introduction

Salinity stress adversely affects plant growth and can lead to plant cell death and severe reduction of crop yield because of its negative effects on diverse plant biochemical and physiological processes (Parida and Das 2005). The effects of salinity on these processes are partly due to generation of reactive oxygen species (ROS) such as the superoxide anion ( $O_2^-$ ) and hydrogen peroxide ( $H_2O_2$ ), which trigger augmented antioxidant enzyme activities as a defence mechanism against ROS-induced oxidative damage (Gémes et al. 2011; Mallik et al. 2011; Noreen et al. 2010; Sairam et al. 2005). One of the consequences of ROS overproduction in response to salt stress is lipid peroxidation, manifested as oxidative damage to lipids that constitute cell and organelle membranes that can be estimated on the basis of malondialdehyde (MDA) content (Ellouzi et al. 2011). Plants with enhanced ability to scavenge ROS (which we refer to as enhanced antioxidant capacity) and improved ability to prevent cell death under salinity stress may thus have





























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