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Meeting abstract

Alteration in ascorbate and ascorbate peroxidase in programmed cell death and oxidative stress

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Different stress conditions, of biotic and abiotic nature, enhance the cellular production of reactive oxygen species (ROS) [1]. Due to their reactive nature, ROS are potentially harmful to all cellular components. Apart their destructive nature, ROS behave as metabolic regulators, being considered as secondary messengers. Indeed, ROS can trigger pathways aimed at saving cells from demise; however, under certain conditions, they can also impair the cellular red/ox balance as to activate a programmed cell death (PCD) process [2,3]. These differences in the ROS-dependent responses seem to be due to different localization, timing and level of ROS production under different stimuli [4,5]. Moreover, the possibility that the co-production of other reactive species is a critical point for the activation of different defence responses has been recently underlined. Increasing attention has been paid to nitric oxide (NO) as a signal molecule synergically acting with ROS in the activation of PCD [6,7]. The level of ROS and the cellular redox homeostasis are regulated by different antioxidant systems; among these, ascorbate (ASC) plays a pivotal role, it being both a direct scavenger of ROS and the electron donor of ascorbate peroxidase (APX), a key enzyme for scavenging hydrogen peroxide in plant cells [8,9].

Here we report data showing that in tobacco cultured cells a moderate oxidative stress did not lead to apoptotic or necrotic events whereas, when H_2O_2 production was increased over a certain range of concentration, an induction of cell death with the features of necrosis was achieved. On the other hand, when the tobacco cells were simultaneous treated with NO and H_2O_2 generators a PCD program was triggered. The scavengers ASC and APX change differently under the various stress conditions. During the moderate oxidative stress a transient increase in APX activity occurred whereas, in the induction of cell necrosis, the activity of APX decreased proportionally to cell death. Under such conditions, no alteration in the APX gene expression was evident and 24 hours after the generation of the oxidative stress, APX activity was significantly increased in the surviving cells, in order to overcome the oxidative stress and to avoid further cell death. When the PCD program was triggered in these cells by the contemporary increase of NO and H_2O_2 the suppression of APX occurred both at the translation or post-translation level. The decrease in APX seems to be one of the first alteration in the redox regulating systems induced in the plant cells in route to PCD.

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