Prevention of Copper-Induced Calcium Influx and Cell Death by Prion-Derived Peptide in Suspension-Cultured Tobacco Cells

Tomoko Kagenishi[a], Ken Yokawa[a], Masaki Kuse[b], Minoru Isobe[c], François Bouteau[a,d], and Tomonori Kawano[a,d,*]

[a] Faculty and Graduate School of Environmental Engineering, The University of Kitakyushu, Kitakyushu 808–0135, Japan. E-mail: kawanotom@env.kitakyu-u.ac.jp
[b] Nagoya University Chemical Instrument Center, Nagoya University, Chikusa, Nagoya 464–8601, Japan
[c] Graduate School of Bioagricultural Sciences, Nagoya University, Chikusa, Nagoya 464–8601, Japan
[d] Laboratoire d'Electrophysiologie des Membranes (LEM) EA 3514, Université Paris Diderot, Case 7069, 2 Place Jussieu, F-75251 Paris Cedex 05, France

* Author for correspondence and reprint requests

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Impact of copper on the oxidative and calcium signal transductions leading to cell death in plant cells and the effects of the copper-binding peptide derived from the human prion protein (PrP) as a novel plant-protecting agent were assessed using a cell suspension culture of transgenic tobacco (*Nicotiana tabacum* L., cell line BY-2) expressing the aequorin gene. Copper induces a series of biological and chemical reactions in plant cells including the oxidative burst reflecting the production of reactive oxygen species (ROS), such as hydroxyl radicals, and stimulation of calcium channel opening, allowing a transient increase in cytosolic calcium concentrations. The former was proven by the action of specific ROS scavengers blocking the calcium responses and the latter was proven by an increase in aequorin luminescence and its inhibition by specific channel blockers. Following these early events completed within 10 min, the development of copper-induced cell death was observed during additional 1 h in a dose-dependent manner. Addition of a synthetic peptide (KTNMKHMA) corresponding to the neurotoxic sequence in human PrP, prior to the addition of copper, effectively blocked both calcium influx and cell death induced by copper. Lastly, a possible mechanism of peptide action and future applications of this peptide in the protection of plant roots from metal toxicity or in favour of phytoremediation processes are discussed.

Key words: Aequorin, Copper Phytotoxicity, Prion Protein