

# *the plant journal*



Volume 18, Number 11, November 2000  
ISSN 1097-3809

WILEY  
Blackwell Science

REVIEW PAPER

# Stress-induced electrolyte leakage: the role of K<sup>+</sup>-permeable channels and involvement in programmed cell death and metabolic adjustment

Vadim Demidchik<sup>1,\*</sup>, Darya Straltsova<sup>1</sup>, Sergey S. Medvedev<sup>2</sup>, Grigoriy A. Pozhvanov<sup>2</sup>, Anatoliy Sokolik<sup>1</sup> and Vladimir Yurin<sup>1</sup>

<sup>1</sup> Department of Plant Cell Biology and Bioengineering, Biological Faculty, Belarusian State University, Independence Avenue 4, 220030, Minsk, Belarus

<sup>2</sup> Department of Physiology and Biochemistry of Plants, Biological Faculty, Saint Petersburg State University, Universitetskaya em. 7–9, 199034, Saint Petersburg, Russia

\* To whom correspondence should be addressed. E-mail: [dzemidchyk@bsu.by](mailto:dzemidchyk@bsu.by)

Received 2 September 2013; Revised 18 December 2013; Accepted 20 December 2013

## Abstract

Electrolyte leakage accompanies plant response to stresses, such as salinity, pathogen attack, drought, heavy metals, hyperthermia, and hypothermia; however, the mechanism and physiological role of this phenomenon have only recently been clarified. Accumulating evidence shows that electrolyte leakage is mainly related to K<sup>+</sup> efflux from plant cells, which is mediated by plasma membrane cation conductances. Recent studies have demonstrated that these conductances include components with different kinetics of activation and cation selectivity. Most probably they are encoded by GORK, SKOR, and annexin genes. Hypothetically, cyclic nucleotide-gated channels and ionotropic glutamate receptors can also be involved. The stress-induced electrolyte leakage is usually accompanied by accumulation of reactive oxygen species (ROS) and often results in programmed cell death (PCD). Recent data strongly suggest that these reactions are linked to each other. ROS have been shown to activate GORK, SKOR, and annexins. ROS-activated K<sup>+</sup> efflux through GORK channels results in dramatic K<sup>+</sup> loss from plant cells, which stimulates proteases and endonucleases, and promotes PCD. This mechanism is likely to trigger plant PCD under severe stress. However, in moderate stress conditions, K<sup>+</sup> efflux could play an essential role as a ‘metabolic switch’ in anabolic reactions, stimulating catabolic processes and saving ‘metabolic’ energy for adaptation and repair needs.

**Key words:** Electrolyte leakage, ion channels, metabolic adjustment, potassium efflux, programmed cell death, reactive oxygen species, stress response.

## Introduction

Electrolyte leakage is a hallmark of stress response in intact plant cells. This phenomenon is widely used as a test for the stress-induced injury of plant tissues and ‘a measure’ of plant stress tolerance (Levitt, 1972; Blum and Ebercon, 1981; Bajji *et al.*, 2002; Lee and Zhu, 2010). The electrolyte leakage is ubiquitous among different species, tissues, and cell types, and can be triggered by all major stress factors, including

pathogen attack (Atkinson *et al.*, 1985, 1990, 1996; Ebel and Mithofer, 1998; Blatt *et al.*, 1999; Maffei *et al.*, 2007), salinity (Nassery, 1975; Maathuis and Amtmann, 1999; Shabala *et al.*, 2006; Demidchik *et al.*, 2010), heavy metals (De Vos *et al.*, 1991; Murphy and Taiz, 1997; Demidchik *et al.*, 2003), oxidative stress (Demidchik *et al.*, 2003, 2010), high soil acidity (pH <4) (Marschner *et al.*, 1966), wounding (Nassery,

Abbreviations: GLR, glutamate receptor; GORK, guard cell ‘outward-rectifying’ K<sup>+</sup> channel; I–V curve, current–voltage curve; MIFE™, microelectrode ion flux estimation; MSL, mechanosensitive-like channel; NSCC, non-selective cation channel; ROS, reactive oxygen species; SKOR, stelar K<sup>+</sup> ‘outward-rectifying’ channel.

© The Author 2014. Published by Oxford University Press on behalf of the Society for Experimental Biology. All rights reserved.  
For permissions, please email: [journals.permissions@oup.com](mailto:journals.permissions@oup.com)