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RESEARCH ARTICLE

Protective roles of trehalose in *Pleurotus pulmonarius* during heat stress response

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Abstract

High temperature is one of the major abiotic stresses that limit edible mushroom growth and development. The understanding of physiological alterations in response to heat stress and the corresponding mechanisms involved is vital for the breeding of heat-resistant edible mushroom strains. Although trehalose functions as a protectant against abiotic stresses in fungi, the putative role of trehalose in thermotolerance remains to be elucidated. In this study, we found heat stress inhibited the growth of two *Pleurotus pulmonarius* strains, heat-sensitive and less-sensitive, and the inhibition was more significant for the sensitive strain. Heat stress leads to the increase of lipid peroxidation and intracellular trehalose accumulation, with a higher level in the heat-sensitive strain, and this effect is independent of exogenous trehalose application. In addition, a lower concentration of exogenous trehalose application in sensitive strain than in less-sensitive strain was found to alleviate the inhibition of mycelium growth and further increase the intracellular trehalose concentration by heat stress. Thus, the protective effects of trehalose were more remarkable in the sensitive strain. The activities of intracellular trehalose metabolic enzymes, i.e., trehalose-6-phosphate synthase, trehalose phosphorylase and neutral trehalase, were determined, and our data indicated that the changes of these enzymes activities in the sensitive strain were more beneficial to accumulate trehalose than that in the less-sensitive strain.

Keywords: edible mushroom, heat stress, *Pleurotus pulmonarius*, thermotolerance, trehalose

1. Introduction

Temperature is an important environmental factor that influences edible mushrooms mycelial growth, fruiting formation, spore and spawn production (Chang and Miles 2004; Kashangura 2008; Hoa and Wang 2015; Zhang R Y *et al*. 2016). Many physiological and biochemical injury cases could be involved in heat stress. For example, heat stress increases anaerobic respiration (Zhang R Y *et al*. 2016), activates apoptosis-like cell death (Song *et al*. 2014) and damages proteins and cell membranes (Ancín-Azpilicueta *et al*. 2012). Accumulation or exogenous application of osmoprotectants, such as proline, glycine

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