Title:	Glutathione depletion activates the yeast vacuolar transient receptor potential channel, Yvc1p, by reversible glutathionylation of specific cysteines
Authors:	Chandel, Avinash (/jspui/browse?type=author&value=Chandel%2C+Avinash) Das, Krishna K. (/jspui/browse?type=author&value=Das%2C+Krishna+K.) Bachhawat, A.K. (/jspui/browse?type=author&value=Bachhawat%2C+A.K.)
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Abstract:	Glutathione depletion and calcium influx into the cytoplasm are two hallmarks of apoptosis. We have been investigating how glutathione depletion leads to apoptosis in yeast. We show here that glutathione depletion in yeast leads to the activation of two cytoplasmically inward-facing channels: the plasma membrane, Cch1p, and the vacuolar calcium channel, Yvc1p. Deletion of these channels partially rescues cells from glutathione depletion–induced cell death. Subsequent investigations on the Yvc1p channel, a homologue of the mammalian TRP channels, revealed that the channel is activated by glutathionylation. Yvc1p has nine cysteine residues, of which eight are located in the cytoplasmic regions and one on the transmembrane domain. We show that three of these cysteines, Cys-17, Cys-79, and Cys-191, are specifically glutathionylated. Mutation of these cysteines to alanine leads to a loss in glutathionylation and a concomitant loss in calcium channel activity. We further investigated the mechanism of glutathionylation and demonstrate a role for the yeast glutathione S-transferase Gtt1p in glutathionylation. Yvc1p is also deglutathionylated, and this was found to be mediated by the yeast thioredoxin, Trx2p. A model for redox activation and deactivation of the yeast Yvc1p channel is presented.
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