Title:	Heart failure and the glutathione cycle: an integrated view
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Abstract:	Heart failure results from the heart's inability to carryout ventricular contraction and relaxation, and has now become a worldwide problem. During the onset of heart failure, several signatures are observed in cardiomyocytes that includes fetal reprogramming of gene expression where adult genes are repressed and fetal genes turned on, endoplasmic reticulum stress and oxidative stress. In this short review and analysis, we examine these different phenomenon from the viewpoint of the glutathione cycle and the role of the recently discovered Chac1 enzyme. Chac1, which belongs to the family of γ-glutamylcyclotransferases, is a recently discovered member of the glutathione cycle, being involved in the cytosolic degradation of glutathione. This enzyme is induced during the Endoplasmic Stress response, but also in the developing heart. Owing to its exclusive action on reduced glutathione, its induction leads to an increase in the oxidative redox potential of the cell that also serves as signaling mechanism for calcium ions channel activation. The end product of Chac1 action is 5-oxoproline, and studies with 5-oxoprolinase (OPLAH), an enzyme of the glutathione cycle has revealed that down-regulation of OPLAH can lead to the accumulation of 5-oxproline which is an important factor in heart failure. With these recent findings, we have re-examined the roles and regulation of the enzymes in the glutathione cycle which are central to these responses. We present an integrated view of the glutathione cycle in the cellular response to heart failure.
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