

Author's Accepted Manuscript

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PII: S0891-5849(18)31018-9
DOI: <https://doi.org/10.1016/j.freeradbiomed.2018.08.014>
Reference: FRB13878

To appear in: *Free Radical Biology and Medicine*

Received date: 8 June 2018
Revised date: 1 August 2018
Accepted date: 13 August 2018

Cite this article as: Aravind T. Reddy, Sowmya P. Lakshmi, Asoka Banno and Raju C. Reddy, Role of GPx3 in PPAR γ -induced protection against COPD-associated oxidative stress, *Free Radical Biology and Medicine*, <https://doi.org/10.1016/j.freeradbiomed.2018.08.014>

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Role of GPx3 in PPAR γ -induced protection against COPD-associated oxidative stress

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ABSTRACT

Cigarette smoke, a source of numerous oxidants, produces oxidative stress and exaggerated inflammatory responses that lead to irreversible lung tissue damage. It is the single, most significant risk factor for chronic obstructive pulmonary disease (COPD). Although an intrinsic defense system that includes both enzymatic and non-enzymatic players exists to protect lung tissues against oxidative stress, impairment of these protective mechanisms has been demonstrated in smokers and COPD patients. The antioxidant enzyme GSH peroxidase (GPx) is an important part of this intrinsic defense system. Although cigarette smoke has been shown to downregulate its expression and activity, the underlying mechanism is not known. Peroxisome proliferator-activated receptor γ (PPAR γ) is a nuclear hormone receptor with antioxidant effects. PPAR γ activation has demonstrated protective effects against cigarette smoke-induced oxidative stress and inflammation. Molecular mechanisms for PPAR γ 's antioxidant function likewise remain to be elucidated. This study explored the link between PPAR γ and GPx3 and found a positive association in cigarette smoke extract (CSE)-exposed human bronchial epithelial cells.

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