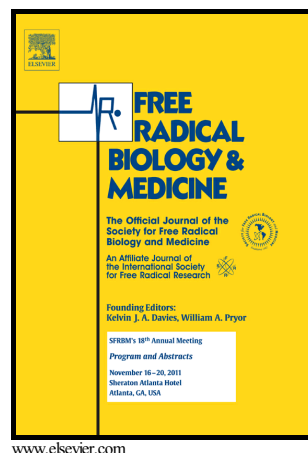


Author's Accepted Manuscript

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PII: S0891-5849(17)30758-X
DOI: <http://dx.doi.org/10.1016/j.freeradbiomed.2017.09.014>
Reference: FRB13452

To appear in: *Free Radical Biology and Medicine*

Received date: 20 July 2017
Revised date: 8 September 2017
Accepted date: 17 September 2017

Cite this article as: Xiaodan Han, Junling Zhang, Xiaolei Xue, Yu Zhao, Lu Lu, Ming Cui, Weimin Miao and Saijun Fan, Theaflavin ameliorates ionizing radiation-induced hematopoietic injury via the NRF2 pathway, *Free Radical Biology and Medicine*, <http://dx.doi.org/10.1016/j.freeradbiomed.2017.09.014>

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Theaflavin ameliorates ionizing radiation-induced hematopoietic injury via the NRF2 pathway

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Highlights

- TF scavenges ROS and inhibits DNA damage in irradiated HSCs
- TF inhibits IR-induced HSC senescence
- TF activates the NRF2 pathway in irradiated HSCs
- TF-mediated protection against IR is abrogated by NRF2 deficiency

Abstract

It has been well established that reactive oxygen species (ROS) play a critical role in ionizing radiation (IR)-induced hematopoietic injury. Theaflavin (TF), a polyphenolic compound from black tea, has been implicated in the regulation of endogenous cellular antioxidant systems. However, it remains unclear whether TF could ameliorate IR-induced hematopoietic injury, particularly the hematopoietic stem cell (HSC) injury. In this study, we explored the potential role of TF in IR-induced HSC injury and the underlying mechanism in a total body irradiation (TBI) mouse model.

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