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## Protective role of frataxin against myocardial ischemia reperfusion injury

**Abdullah Al Asmari**
Auburn University, USA

Oxygen free radicals associated with ischemia-reperfusion (IR) injury in cardiomyocytes is known to cause mitochondrial damage. However, the exact mechanism of how the oxygen free radicals develop in the mitochondria due to IR stress is currently unclear. In the current study, we focus upon understanding the role of frataxin (FXN) in regulating mitochondrial damage associated with IR injury. FXN, a nuclear encoded mitochondrial matrix protein, has been observed to regulate mitochondrial iron homeostasis and thus mitochondrial energy regulation. Loss of FXN, in Friedreich's ataxia, is associated with mitochondrial iron overload and increased ROS formation and cellular damage. In this study, we hypothesized that FXN protects cardiomyocytes against IR injury by preventing the dysregulation of myocardial bioenergetics. We identified that FXN expression is increased in response to IR injury and that increase is mediated by hypoxia inducible factor (HIF-1α) which results in regulation of mitochondrial iron homeostasis and the ensuing mitochondrial ROS formation. Most surprisingly, we observed that enhanced FXN expression displayed elevated levels of glutathione (GSH) and superoxide dismutase (SOD). Furthermore, these findings were supported in our FXN over-expressing and knock down cells under the same IR condition. Together, these results demonstrate that increased expression of FXN is cardio-protective against IR injury through its anti-oxidant effect and by improving mitochondrial energetics.

afa0006@tigermail.auburn.edu

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