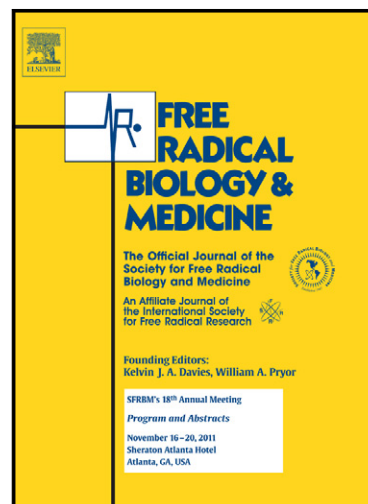


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Alpha B-crystallin induction in skeletal muscle cells under redox imbalance is mediated by a JNK-dependent regulatory mechanism

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Keywords: myoblasts; sodium arsenite; p38; c-Jun; Nrf2.

ABSTRACT

The small heat shock protein α B-crystallin (CRYAB) is critically involved in stress-related cellular processes such as differentiation, apoptosis and redox homeostasis. The up-regulation of CRYAB plays a key role in the cytoprotective and antioxidant response, but the molecular pathway driving its expression in muscle cells during oxidative stress still remains unknown.

Here we show that non-cytotoxic exposures of sodium meta-arsenite (NaAsO₂) inducing redox imbalance are able to increase CRYAB content of C2C12 myoblasts in a transcriptional-dependent manner. Our *in silico* analysis revealed a genomic region up-stream of the *Cryab* promoter containing two putative Antioxidant Responsive Elements (ARE) motifs and one AP-1-like binding site. The redox-sensitive transcription factors Nrf2 and the AP-1 component c-Jun were found to be up-regulated in NaAsO₂-treated cells and we demonstrated a specific NaAsO₂-mediated increase of c-Jun and Nrf2 binding activity to the genomic region identified, supporting their putative involvement in CRYAB regulation following a shift in redox balance.

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