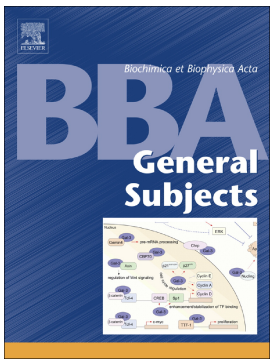

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Synergistic Cellular Responses to Heavy Metal Exposure: a minireview

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Abstract

Background: Metal-responsive transcription factor 1 (MTF-1) induces the expression of metallothioneins (MTs) which bind and sequester labile metal ions. While MTF-1 primarily responds to excess metal exposure, additional stress response mechanisms are activated. Evidence suggests potential crosstalk between responses mediated by MTF-1 and stress signaling enhances cellular tolerance to metal exposure.

Scope of Review: This review aims to summarize current understanding of the interaction between the stress response mediated by MTF-1 and other cellular mechanisms, notably the nuclear factor κB (NF-κB) and heat shock response (HSR).

Major Conclusions: Crosstalk between MTF-1 mediated metal response and NF-κB signaling or HSR can modulate expression of stress proteins in response to metal exposure via effects on precursor signals or direct interaction of transcriptional activators. The interaction between stress signaling pathways can enhance cell survival and tolerance through a unified response system.

General Significance: Elucidating the interactions between MTF-1 and cell stress response mechanisms is critical to a comprehensive understanding of metal-based cellular effects. Co-activation of HSR and NF-κB signaling allows the cell to detect metal contamination in the environment and improve survival outcomes.