

Homocysteine treatment alters redox capacity of both endothelial and tumor cells

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Homocysteine is a non-proteinogenic amino acid playing key roles in two interconnected metabolic pathways, namely, the activated methyl cycle and the linear trans-sulfuration pathway that allows the conversion of methionine to cysteine. A dysregulation of intracellular homocysteine metabolism could yield an increased export of this amino acid, leading to hyperhomocysteinemia, which has been associated with an increased risk of cardiovascular diseases. In spite of decades of experimental effort, there is no definitive consensus on what could be the molecular mechanisms whereby hyperhomocysteinemia could contribute to cardiovascular disease. The redox active nature of homocysteine has favored the idea of an induction of oxidative stress as the underlying mechanism of homocysteine toxicity. In contrast, homocysteine can also behave as an anti-oxidant. The present work is aimed to further analyze the capacity of homocysteine to modulate the redox capacity of both endothelial and tumor cells.

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