Influence of Bicarbonate Buffer on Ischemic Damage in Rat hearts, HL-1 Cells and C. elegans

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The role of carbonate radicals in pathological processes is still poorly understood. During ischemia, the concentration of bicarbonate/CO₂ may increase due to the absence of oxygen and blood circulation. Furthermore, ischemic damage is known to be mediated by reactive oxygen species (ROS). To understand the role of bicarbonate in ischemia damage we used 3 different models: isolated rat hearts, immortalized mouse cardiomyocytes (HL-1 cells) and C. elegans. Our results showed that when higher CO₂ concentrations were present (buffer gassed with 10% CO₂ and 90% O₂), the ischemic hearts presented delayed recovery, HL-1 cells had an increase in death percentage and C. elegans displayed increased axonal damage and a dysfunctional touch response. These conditions weren’t observed in non-ischemic exposure to buffer gassed with 10% CO₂ + 90% O₂. Also, hearts perfused with buffer gassed with 10% CO₂ + 90% O₂ had a significant increase in the amount of proteins containing nitrated tyrosine, oxidized methionine and carbonylated residues relative to hearts perfused without CO₂. We conclude that bicarbonate-derived oxidants are important in ischemic damage and may play a major role in post-infarct cardiac injury.

Word Keys: Ischemia, Heart Infarct, Bicarbonate Buffer, Reactive Oxygen Species, C. elegans

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