



Effect of High Oxygen Partial Pressure on 4-Hydroxy-2-Nonenal Induced Myoglobin Oxidation, Oxidation-Reduction Potential, and Myoglobin Unfolding

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Objectives

High oxygen modified atmosphere packaging (MAP) is often employed by the meat industry to enhance the appearance of fresh meat. 4-hydroxy-2-nonenal (HNE) is an α -, β -unsaturated aldehyde derived from the oxidation of ω 6-polyunsaturated fatty acids. Previous research has examined the effect of HNE on oxymyoglobin oxidation at atmospheric oxygen partial pressure (20%). However, limited knowledge is currently available on the effects of HNE on myoglobin redox stability under high oxygen conditions. Therefore, the objective of this study was to examine the effect of high partial pressure of oxygen on HNE induced myoglobin oxidation.

Materials and Methods

Oxymyoglobin (0.15 mM; pH 5.6) prepared via hydrosulfite-mediated reduction was mixed with HNE (1.05 mM; dissolved in water), whereas controls received an equal volume of water. Following mixing, both samples were assigned to either high oxygen partial pressure (78%) or atmospheric oxygen partial pressure packaging. The samples were incubated either in a coffin-style display case maintained at $2 \pm 1^\circ\text{C}$ (under continuous lighting) for 0, 2 or 6 d, or in an incubator for 0, 1, 2, or 3 h at 25°C . Metmyoglobin formation, oxidation-reduction potential, and myoglobin unfolding were evaluated on each of the storage time points.

The experiments were replicated 3 times ($n = 3$) and the data were analyzed using the Mixed Procedure of SAS (SAS Inst. Inc., Cary, NC).

Results

Myoglobin oxidation, oxidation-reduction potential, and protein unfolding properties were not different ($P > 0.05$) between treatments on time 0 for samples incubated at 4 and 25°C . On d 2 and 6, the high oxygen partial pressure treatment resulted in increased metmyoglobin formation and protein unfolding ($P < 0.05$) than the atmospheric oxygen partial pressure samples. Moreover, atmospheric oxygen partial pressure treatment had greater oxidation-reduction potential than high oxygen partial pressure treatment. At 25°C , high oxygen partial pressure treatments exhibited greater myoglobin oxidation from 1 to 3 h of incubation ($P < 0.05$) than atmospheric conditions.

Conclusion

The results suggest that high oxygen partial pressure accelerated HNE induced oxidation irrespective of temperature. Further research using molecular protein biomarkers may provide an understanding of the underlying mechanistic steps involved in the interaction of HNE with the myoglobin at higher oxygen partial pressure.