

Effect of high-pressure treatment on collagen fibrillogenesis in presence of decorin

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Objectives: High-pressure treatment has been the most adopted nonthermal processing technology in the food industry with current ever-growing implementation. High-pressure treatment affects actomyosin toughness and background toughness, leading to meat tenderization. The mechanism of weakening actomyosin toughness by high-pressure treatment has been already reported (Suzuki *et al.*, 1991). The effect of highpressure treatment on background toughness ascribes to connective tissue. Intramuscular connective tissue is mainly composed of collagen. Ichinoseki *et al.* (2006) reported that high pressure did not degrade collagen molecules but dissociated collagen fibrils. Decorin, a small proteoglycan, binds to collagen fibrils and stabilizes them. Hosono *et al.* (2014) reported high-pressure treatment up to 400 MPa causes reversible tertiary structural changes in decorin. It has been suggested that the weakening of intramuscular connective tissue may result from alteration of the decoding-collagen interaction due to structural changes of the decorin molecule. Decorin has been shown to influence the kinetics of collagen fibrillogenesis, the fibrils' diameter, and the distance between the fibrils. In this study, as the first step in investigating the effect of high-pressure treatment on the decorin-collagen interaction, we observed the effect of high-pressure treatment on collagen fibrillogenesis in the presence of decorin.

Materials and Methods: Decorin isolated and purified from bovine skeletal muscle and type I collagen derived from bovine dermis was used. On the decorin solution and collagen solution using PBS as a solvent, a high-pressure treatment was performed in each situation using a small high-pressure pump. Then, decorin-collagen interaction was investigated by observing collagen fibrillogenesis. For that, a spectrophotometer was used to measure the absorbance at 310 nm over time. Before starting the major study, it was confirmed how decorin affects collagen fibrillogenesis without high-pressure treatment. Next, the effect of high-pressure treatment on decorin and collagen before collagen fibrillogenesis was investigated. Decorin and collagen solution were treated respectively with high pressure up to 400 MPa. After that, the measurement of collagen fibrillogenesis was monitored. Finally, the effect of high-pressure treatment after collagen fibrillogenesis was investigated. High-pressure treatment up to 400 MPa was applied to collagen fibrils bound with decorin. Then, their interaction under and after high-pressure treatment was monitored.

Results and Discussion: Inhibition of collagen fibrillogenesis by decorin without high-pressure treatment was confirmed. Furthermore, it was shown that there was no effect of high-pressure treatment before collagen fibrillogenesis. This result can be explained by the reports that collagen molecules are not affected and the tertiary structure of decorin is reversible on high-pressure treatment up to 400 MPa. In the examination of the effect of high-pressure treatment after collagen fibrillogenesis, it was observed that the absorbance increased when the pressure was released. That is, the self-association of collagen molecules progressed. From this result, it is considered that decorin was dissociated from collagen due to changing of the tertiary structure of decorin under high pressure. In addition, since decorin prevents the association of collagen molecules more than necessary, it is possible that the dissociation of decorin caused the free collagen molecules to associate with the collagen fibrils. However, the results of this study showed that the highpressure treated decorin was able to bind to collagen molecules even after the treatment, therefore, it is considered that decorin can rebound to collagen fibrils.

Conclusions: In this study, optical measurements provided information on the decorin-collagen interaction. It was shown that decorin may dissociate from collagen fibrils under high pressure at 300 and 400 MPa. It has been reported that intramuscular connective tissue is also tenderized at high-pressure treatments at 300 and 400 MPa. Therefore, it is considered that the cause of the tenderizing may be the dissociation of decorin from collagen fibrils. To elucidate the effect of high-pressure treatment on the decorin-collagen interaction, observation of the decorin-collagen interaction by several other approaches is expected.

References:

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