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Combined Effects of High-pressure and Thermal Treatments on Lipid Oxidation and Enzymes in Pork

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Abstract The relationship between lipid oxidation and related enzymes in pork during combined pressure-heat treatments was investigated. Minced pork was treated under a pressure range of 0.1-750 MPa and a temperature range of 30-60°C for 20 min. Thiobarbituric acid-reactive substances (TBARS) values and activities of acid lipase (AL), neutral lipase (NL), phospholipase (PL), and lipoxygenase (LOX) in pork were evaluated. NL and LOX were completely inactivated at 600 MPa-50°C and 450 MPa-60°C, and AL at 600 MPa-55°C. PL had the greatest stability and was completely inactivated at 600 MPa-60°C and 750 MPa-50°C. PL activity was strongly related to lipid oxidation caused by high pressure, followed by AL and LOX. TBARS values at 600 and 750 MPa were strongly related to the inactivation rate and the ratio of PL during treatment. PL was the most important enzyme related to lipid oxidation induced by pressure.

Keywords: pork, high pressure, lipid oxidation, lipase, lipoxygenase

Introduction

High pressure (HP) technology is gaining increasing importance in the food industry; however, HP application for processing of meat products is limited because HP can enhance intramuscular lipid oxidation (1). The reported reasons for lipid oxidation due to HP are inconsistent. Tanaka *et al.* (2) found that lipid oxidation may be related to a synergistic effect of denatured proteins under high pressure. Angsupanich and Ledward (3) concluded that there was no relationship between them. Release of metal ions from inactive complexes present in meat under HP is believed to be a major cause of lipid oxidation (3,4). However, Defaye and Ledward (5) and Orlien *et al.* (6) did not observe a significant increase in amounts of free metal ions with an increase in lipid oxidation following HP treatment in studies of beef and chicken, respectively. Moreover, cell membrane damage caused by HP was thought to be another cause for lipid oxidation in meat (6,7).

In a previous study (8), lipid oxidation in pressurized pork was suggested to be related to changes in the activities of certain enzymes, such as lipase, phospholipase (PL), and lipoxygenase (LOX). An influence of HP on the activities of these enzymes in meat products has been reported. Homma *et al.* (9), Ohmori *et al.* (10), and Jung *et al.* (11) investigated the effects of HP on PL activity in rabbit meat, bovine liver, and beef, respectively. He *et al.* (12) studied the effect of HP on lipase activity in oysters. However, no studies have investigated the relationship between changes in enzyme activities and lipid oxidation due to HP treatment.

Ma (1) observed that levels of thiobarbituric acid-reactive substances (TBARS) decreased rather than increased in a report on the effect of HP at 800 MPa at 70°C on beef. In addition to induced decomposition of lipid oxidation products under these conditions, it may be related to lipase inactivation. Yagiz *et al.* (13) observed that treatment with a pressure of 150 MPa increased lipid oxidation in fish meat, while the oxidative stability increased after treatment with 300 MPa, perhaps related to some degree to changes in lipase activity under HP. Wada and Ogawa (14) and Clariana *et al.* (15) proposed that changes in lipase activity caused by HP could influence the oxidative stability of muscles. Thus, the relationship between changes in lipase activity and lipid oxidation in pressurized meat merits further investigation. Fresh muscle contains small amounts of free fatty acids that are more readily oxidized than bound fatty acids. If lipase is inactivated under HP, triacylglycerols and phospholipids will not be hydrolyzed, or will undergo a reduced level of hydrolyzation, and lipid oxidation will be inhibited.

The objective of this study was to investigate the combined effect of HP at 0.1-750 MPa and thermal treatments at 30-60°C on lipid oxidation and activities of acid lipase (AL), neutral lipase (NL), PL, and LOX, in minced pork. Particular attention was directed toward the

relationship between lipid oxidation and changes in enzyme activities.

Materials and Methods

Preparation of pork samples Fresh longissimus muscles from 3 Rongchang pigs each weighing approximately 100 kg were provided in October of 2012 by the Animal Sciences Academy of Chongqing, China for this study. After 24 h of chilling at 4°C, visible fat and connective tissue were removed and pork was vacuum-packed in polythene bags (20 cm×15 cm), operating at 99% vacuum (DZ400; Ruili Machinary Co., Ltd, Hangzhou, China) immediately, and frozen at −20°C (BC/BD-429H Freezer; Haier, Qingdao, China) until use.

HP treatment Pork was thawed at 4°C for 24 h and minced (JYL-G12; Joyoung Household Electrical Appliance Co., Ltd, Jinan, China), after mixing with artificial way, minced pork samples were divided into 180 ones of approximately 50 g each. After vacuum-packing (DZ400; Ruili) in polythene bags (15 cm×10 cm), 30 groups of 6 minced pork samples each were randomly treated with combinations of pressure levels at 0.1, 150, 300, 450, 600, and 750 MPa and temperatures at 30, 40, 50, 55, and 60°C for 20 min in an HPP.L2 HP apparatus (HTSM Bio-Tech Co., Ltd., Tianjin, China) using bis(2-ethylhexyl) sebacate (Aladdin Industrial Corporation, Shanghai, China) as a pressure-transmission medium. The capacity of the pressure vessel was 1 L with a maximum operating pressure and temperature of 800 MPa and 60°C, respectively. Following treatment, half of the pork samples (3 samples) from each group were immediately analyzed for TBARS values and activities of AL, NL, PL, and LOX. The remaining 3 pork samples in each group were stored in the dark at 4°C for 7 days in oxygen permeable bags. TBARS values of stored pork samples were subsequently assayed. Pork samples held at 30°C under ambient pressure of 0.1 MPa for 20 min were used as controls.

Lipase extraction and activity assays Extraction and activity assays for AL, NL, and PL were performed using previously described procedures (16). One unit (U) of activity was defined as the amount (nmol) of released 4-methylumbelliferone per h at 37°C. Activities were expressed as U/g of protein.

LOX activity assays The activity of LOX was determined using a previously described method (16). One unit (U) of LOX activity was defined as the amount of enzyme required for an absorption increment of 0.001 per min and per g of protein.

Determination of TBARS values TBARS values were assessed following the method of Siu and Draper (17). Results were expressed as mg of malondialdehyde (MDA) per kg of pork.

Effects of a pressure pulse on PL activity Twenty-four pork samples of approximately 50 g each were prepared and randomly divided into 4 equal groups with 6 samples in a group for investigation of the relationship between PL activity and lipid oxidation. The 4 sample groups were randomly assigned for treatment with pressure-temperature combinations of 600 MPa-60°C, 750 MPa-50°C, 750 MPa-55°C, and 750 MPa-60°C. For 3 pork samples in each group, the pressure was immediately released when the pressure-temperature reached set values and the samples were withdrawn. The remaining 3 pork samples were treated under the set conditions for 20 min. After treatment, PL activity was assayed immediately.

Statistical analysis The activities of AL, NL, PL, and LOX were expressed as percentages of activity in a control ($A/A_0 \times 100$), where A (U) is the enzyme activity in pork treated at different preset conditions, and A_0 (U) is the enzyme activity in controls. TBARS values and enzyme activity data obtained from different treatments were analyzed using a one-way analysis of variance (ANOVA) with SPSS statistical software (Version 13, SPSS Inc., Chicago, IL, USA). The level of significance was set at $p<0.05$.

Results and Discussion

Effect of pressure-heat treatments on lipid oxidation in pork Effects of pressure-heat treatments on TBARS values immediately after treatment and after 7 days of storage at 4°C are shown in Table 1. TBARS values of pork increased to different extents during cold-storage, in agreement with a previous report (18).

Under ambient pressure of 0.1 MPa, pork TBARS values significantly ($p<0.05$) increased in the pork samples treated at 55 and 60°C prior to cold-storage, compared with controls. Under 150 MPa, pork TBARS values under treatment at 60°C increased significantly ($p<0.05$), compared with pork treated at 30°C. During cold-storage, pork TBARS values significantly ($p<0.05$) increased with temperature, compared with pork treated at 30°C. Under 300 and 450 MPa, pork TBARS values increased with an increase in temperature. After cold-storage, TBARS values initially increased to a peak with temperature, then began to decrease. For pork samples treated with 600 and 750 MPa, the evolution of TBARS values during treatment and cold-storage was similar and did not significantly ($p>0.05$) change with an initial increase in temperature, compared with pork treated at 30°C, but markedly ($p<0.05$) decreased thereafter.

Pork TBARS values under treatment at different temperatures initially increased with an increase in pressure, independent of treatment or cold storage. In particular, pork TBARS values under treatment at 300 MPa were significantly ($p<0.05$) higher than for pork treated with 150 MPa, except for pork samples treated at 60°C prior to cold-storage, consistent with a previous report (18) where 350 MPa was the key pressure leading to a significant increase in lipid oxidation. After TBARS values reached a peak with an increase in pressure, values decreased and the critical pressure decreased with an increase in temperature. Pork samples treated at 30 and 40°C

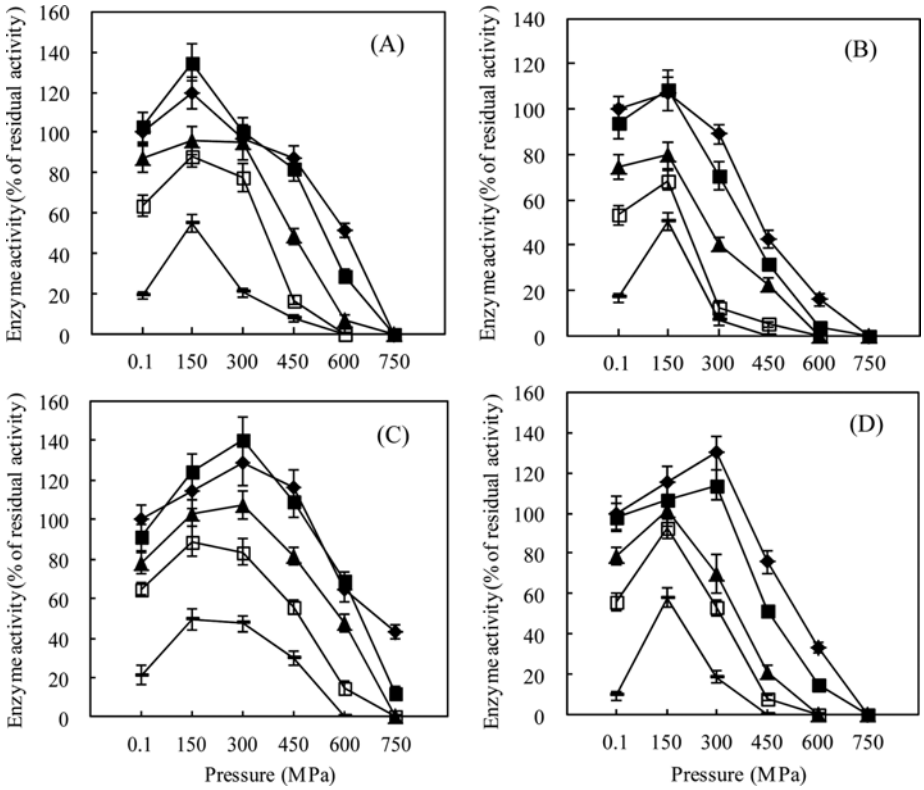

Fig. 1. Effects of combined pressure-heat treatments on acid lipase (A), neutral lipase (B), phospholipase (C), and lipoxygenase (D) activities in pork. ◆, ■, ▲, □, and ▬ represent pork samples treated at 30, 40, 50, 55, and 60°C, respectively.

were under a pressure of 600 MPa while the pressure was 450 MPa for pork treated at 50, 55 and 60°C. Following cold-storage, the critical pressure for pork treated at 30, 40, and 50°C was 450 MPa, and was 300 MPa for pork treated at 55 and 60°C. The decrease in TBARS value under combinations of more intense pressure-temperature may have been related to changes in activities of lipases, PL, and LOX.

Effects of pressure-heat treatments on AL in pork The effects of pressure-heat treatments on the activity of AL in pork are shown in Fig. 1A. Under ambient pressure, AL activity was not affected at 50°C, whereas AL activity significantly (p<0.05) decreased to approximately 64 and 20% of original activity values at 55 and 60°C, respectively. Under 150 MPa, AL activity in all pork samples increased to different degrees, compared to the activity 0.1 MPa. Enzymes were activated under 150 MPa, particularly for pork treated at 30 or 40°C for which the activity was over 100%, perhaps due to release of enzymes from lysosomes under HP (9,19) or due to pressure activation of enzymes via conformational changes that eliminated the extension polypeptide shielding the active site of the enzyme (20). Activation induced by pressure has been reported, but the critical pressure value that conferred activation varied with different media and different enzymes. For acid phosphatase in muscles (9), proteolytic enzymes in cold-smoked salmon (19), acid phosphatase in beef (11), and LOX in tomato (21), the activating pressures were 500, 300, 520, and 400 MPa, respectively. For treatment at 300 MPa, AL activities in all pork samples in this study decreased, compared with pork treated at 150 MPa. Under 450 and 600 MPa, enzyme activities continued to decline and there was no activity in pork treated at and above 55°C when the pressure was 600 MPa. No AL activity was detected in pork treated at 750 MPa.

Effect of pressure-heat treatments on NL in pork Evolution of NL was similar to AL, but NL stability was lower than for AL (Fig. 1B). Under ambient pressure, NL activity remained unchanged at 40°C, whereas only 74, 53, and 17% of the original activity was retained after treatments at 50, 55, and 60°C, respectively. For treatment at 150 MPa, pressure activation was still observed, but the activation extent was lower than for AL, perhaps due to lesser amounts of NL than AL present in lysosomes. At 300 MPa, pork NL activity was significantly (p<0.05) reduced, compared with pork samples under 150 MPa. Under 450 MPa, pork NL activity was continuously reduced, and no activity was observed in pork treated at 60°C. At a pressure of 600 MPa, 16% and 4% of the original activity was detected in pork treated at 30 and 40°C, respectively, with no activity was observed in other pork samples.

Treatment of 600 MPa-50°C or 450 MPa-60°C caused total inactivation of NL with no effect on TBARS values in pork samples. Thus, there was no association between NL activity and lipid oxidation induced by HP, indicating that NL was not the major

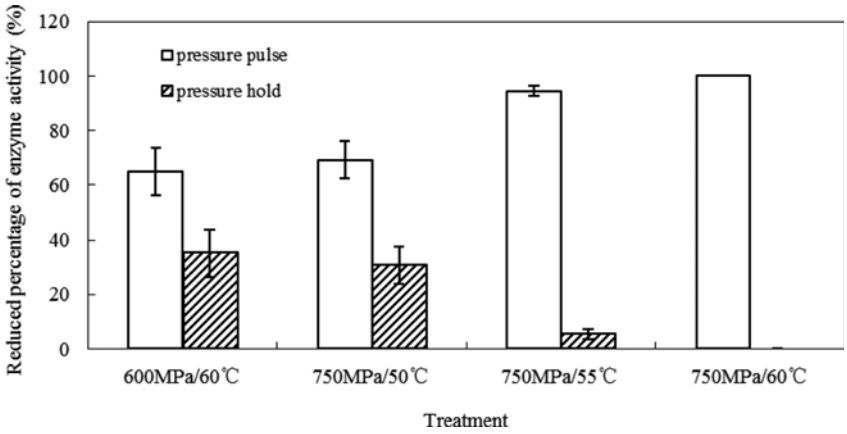

Fig. 2. Comparison of pressure hold and pressure pulse on phospholipase activity in pork.

enzyme responsible for triacylglycerol hydrolysis. Previous studies have reported (16,22) that AL was the main enzyme for hydrolyzation of triacylglycerols. Under 600 MPa-55°C, AL was completely inactivated, and pork TBARS value under this condition was lower than for treatment at 600 MPa-50°C, particularly after 7 days of storage at 4°C. Inactivation of AL was probably the cause as AL can catalyze hydrolysis of triacylglycerols into free fatty acids, which are more readily oxidized than bound fatty acids. A reduced amount of triacylglycerols was hydrolyzed when AL was inactivated, leading to reduced release of free fatty acids. Under both 750 MPa-30°C and 750 MPa-40°C, AL was also completely inactivated, whereas TBARS values in pork treated under these conditions were high after 7 days of storage at 4°C, indicating that AL inactivation was not sufficient to cause a reduction in lipid oxidation due to pressure-heat treatments. Independent studies evaluating the pressure stability of AL and NL have not been reported; however, lipase has been studied. Seyderhelm *et al.* (23) found that lipase was completely inactivated in a pH 7.0 buffer when treated with 700 MPa at 45°C for 5 min and the pressure stability was higher than for both AL and NL in this study. This difference may be related to higher enzyme concentrations in the buffer. Bang and Chung (24) observed that treatment of yeast and *E. coli* lipase with 448 MPa for 30 s at room temperature resulted in complete inactivation of enzyme activity, and the pressure stability of lipase was lower than values observed in this study. Noël *et al.* (25) observed that treatment of lipase in a pH 7.0 phosphate buffer at 480 MPa for 40 min at room temperature resulted in loss of half of the enzyme activity, comparable to observations herein.

Effect of pressure-heat treatments on PL in pork The pressure stability of PL was strongest among AL, NL, PL, and LOX (Fig. 1C). Under ambient pressure, the thermal resistance of PL was comparable to the resistance of AL. Under 150 MPa, activation due to pressure was still observed. The activating effects of pressure on both AL and NL were seen only under 150 MPa, whereas a stronger activating effect of pressure on PL was observed under 300 MPa than under 150 MPa when the treatment temperature was at or lower than 50°C, probably related to different distributions of individual enzymes in lysosomes (10). After treatment at 450 MPa, PL activity in all pork samples was significantly (p<0.05) reduced, compared with pork samples treated at 300 MPa, but the activity of samples treated at 30 and 40°C was still above 100%, consistent with reports by Homma *et al.* (9) and Jung *et al.* (11) in which PL activity increased when muscles were treated with 500 and 520 MPa, respectively. Under 600 MPa, PL activity continued to decrease significantly (p<0.05) compared with pork samples treated at 450 MPa, and there was a complete lack of activity in pork treated at 60°C. At 750 MPa, PL activity was detected only in pork treated at 30 and 40°C. Seyderhelm *et al.* (23) observed that treatment of PL in Tris buffer at 600 MPa at 55°C for 30 min inactivated the enzyme by 90%, whereas bovine milk had a protective effect on PL because only a 10% reduction of enzyme activity was achieved under identical conditions. The stability of PL in this study was comparable to the stability observed in Tris buffer and was lower than the stability observed in milk, as reported by Seyderhelm *et al.* (23).

Under conditions of 600 MPa-60°C, 750 MPa-50°C, 750 MPa-55°C, and 750 MPa-60°C, PL was completely inactivated. TBARS values under these 4 treatments were significantly lower (p<0.05) than those of other groups treated under 600 or 750 MPa after 7 days of storage at 4°C (Table 1), perhaps related to PL inactivation because after PL inactivation, phospholipids could not be continuously hydrolyzed for release of free fatty acids during storage. The percentage of polyunsaturated fatty acids in phospholipids was high and oxidized free fatty acids were primarily polyunsaturated fatty acids. In addition, pork TBARS values under treatment with combinations of 750 MPa-55°C and 750 MPa-60°C were significantly (p<0.05) lower than for treatment under 750 MPa-50°C and 600 MPa-60°C.

Effect of pressure-heat treatments on LOX in pork The pressure stability of LOX was lower than for AL and PL, but was comparable to the stability of NL (Fig. 1D), consistent with the report of Seyderhelm *et al.* (23) where PL was more stable under pressure, followed by lipase, and finally LOX, which was weakest in the buffer. Under 0.1

Table 1. Effects of combined pressure-heat treatments on TBARS values in pork

Items	0.1 MPa	150 MPa	300 MPa	450 MPa	600 MPa	750 MPa
0d						
30°C	0.35±0.05[1)]aA[2)]	0.38±0.05aA	0.93±0.08bA	1.48±0.16cA	1.74±0.19cB	1.35±0.18cB
40°C	0.32±0.07aA	0.40±0.09aA	1.05±0.11bAB	1.39±0.14bcA	1.81±0.22cB	1.52±0.23cB
50°C	0.37±0.04aA	0.44±0.12aA	1.28±0.10bBC	1.76±0.14cAB	1.71±0.20cB	1.07±0.18bB
55°C	0.47±0.05aA	0.58±0.10aAB	1.47±0.12bC	2.14±0.27cB	1.43±0.21bAB	0.58±0.10aA
60°C	0.54±0.11abA	0.81±0.13bB	2.05±0.19dD	2.25±0.22dB	1.18±0.10cA	0.49±0.09aA
7d						
30°C	0.46±0.08aA	0.65±0.10aA	3.48±0.20bA	4.96±0.37cBC	4.87±0.50cC	4.29±0.34cC
40°C	0.52±0.07aA	1.49±0.09bB	4.77±0.34dB	5.76±0.28eCD	4.56±0.29dC	3.81±0.27cC
50°C	0.55±0.12aA	2.24±0.23bC	4.45±0.39cB	6.33±0.49dD	5.34±0.51cdC	1.94±0.20bB
55°C	1.26±0.14bB	3.09±0.38cD	4.68±0.17dB	4.29±0.30dB	2.98±0.22cB	0.75±0.14aA
60°C	2.55±0.27cC	3.87±0.29dD	3.98±0.32dAB	3.28±0.42cdA	1.76±0.20bA	0.58±0.22aA

[1)]Results were expressed as mean values±standard error (SE), data were mean values of triplicate.
[2)]Values with different small letters in the same row were significantly different (p<0.05); Values with different capital letters in the same column and under the same storage day were significantly different (p<0.05).

MPa, the effect of temperature on LOX was similar to the effect on NL. Pressure activation of LOX was consistent with PL and was observed at 150 and 300 MPa. Under a pressure of 450 MPa, pork LOX activity was significantly (p<0.05) reduced, compared with pork samples treated at 300 MPa and was undetected in pork treated at 60°C. When the pressure increased to 600 MPa, pork enzyme activities continued to drop significantly (p<0.05), and no activity was detected at 50°C. Seyderhelm *et al.* (23) and Heinisch *et al.* (26) reported that treatment of LOX in a buffer at 600 MPa at 45°C for 10 min and with at 600 MPa at 40°C for 30 min, respectively, resulted in complete inactivation. Rodrigo *et al.* (21) and Tedjo *et al.* (27) found that the inactivation pressures for tomato LOX and soybean LOX were 550 and 570 MPa, respectively, comparable to results obtained in this study.

A previous study (18) identified 350 MPa as the key pressure causing an increase in lipid oxidation of pork at 20°C. This increase was probably related to an increase in LOX activity because the activity reached a high point with treatment 300 MPa and 30°C, exceeding 130% of the original activity (Fig. 1D). After treatment with 450 MPa-60°C and 600 MPa-50°C for 20 min, LOX was completely inactivated, but pork TBARS values under these conditions remained high (Table 1), indicating that lipid oxidation was not decreased with LOX inactivation under these conditions. Thus, under a pressure of 300 MPa, LOX stimulated lipid oxidation. However, under an increased pressure of 600 MPa, LOX activity was no longer related to lipid oxidation. In this case, lipid oxidation may primarily be a result of autoxidation.

Effect of a pressure pulse on PL in pork A majority of PL activity under the 4 treatment conditions was lost during the process of pressure buildup or release caused by a pressure pulse (Fig. 2). For pork treated under 750 MPa-55°C and 750 MPa-60°C, almost all enzyme activities were lost due to the pressure pulse. For pork treated under 750 MPa-50°C and 600 MPa-60°C, loss of PL activity was approximately 70 and 65% during pressure buildup and release, respectively. The remaining 30 and 35% of enzyme activity, respectively, was lost during the pressure holding period. The pressure pulse resulted in instantaneous disruption of enzyme protein structures, whereas the pressure hold probably caused mechanical stress due to the pressure-time combination, resulting in damage to the functional and structural components of cells (28). Pork TBARS values under treatment at both 750 MPa-55°C and 750 MPa-60°C were significantly (p<0.05) lower than values for treatment under both 750 MPa-50°C and 600 MPa-60°C. PL in the first 2 groups was probably almost completely inactivated during pressure buildup. Thus, PL did not play a role during the pressure hold. Free fatty acid generation was reduced and amounts of oxidized free fatty acids were correspondingly decreased. Pork TBARS values under treatment at both 750 MPa-55°C and 750 MPa-60°C changed only slightly during 7 days of cold-storage, indicating that, following pressure treatment, the contents of polyunsaturated free fatty acids were already low due to inactivation of PL and AL as direct oxidation of bound fatty acids was difficult. Riahi and Ramaswamy (28) investigated the effect of a pressure pulse on amylase from apple juice and found that at 20°C, pulse inactivation was approximately 30.5% at 100 MPa, which increased to 93.1% at 400 MPa. Indrawati *et al.* (29) reported that pulse inactivation of LOX in green beans was approximately 10% and 50% under 200 and 500 MPa, respectively, at room temperature. Results of this study were somewhat different, perhaps due to different materials and/or enzymes.

Pork TBARS values for a pressure pulse immediately after treatment were comparative with values for treatment using 0.1 MPa (data not shown), perhaps due to a short time under HP. After 7 days of cold-storage, pork TBARS values under treatment at 600 MPa-60°C and 750 MPa-50°C were significantly (p<0.05) increased to 2.42 and 2.73 respectively, compared with controls, and were higher than corresponding pressure hold values of 1.76 and 1.94, respectively (Table 1). On the contrary, pork TBARS values under treatment at

750 MPa-55°C and 750 MPa-60°C did not exhibit significant (p>0.05) changes after cold-storage, compared with controls, perhaps related to retained PL activity in pork after pressure pulse treatment.

In conclusion, among the 4 enzymes (AL, NL, PL, and LOX) related to lipid hydrolysis and oxidation in pork, PL showed the strongest pressure-heat stability, followed by AL, and that of LOX and NL was comparable. Lipid oxidation caused by HP was primarily related to PL, followed by AL and LOX, and the role of NL was weak. However, the oxidation stability of lipids in meat during HP was also related to many factors, such as antioxidants and metal ions present in meat. Relationships between these factors and lipid oxidation during pressure treatment merit further study.

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Disclosure The authors declare no conflict of interest.

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