2019 Reciprocal Meat Conference – Muscle and Lipid Biology and Biochemistry

Meat and Muscle Biology™

Modifications of Membrane Phospholipids in Response to Extended Aging from Pork Loins

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Keywords: aging, lipid oxidation, lipidomics, phospholipid, pork
Meat and Muscle Biology 3(2):160

Objectives

It is well established that fresh meat shelf-life deteriorates during aging process. We hypothesize that part of the shelf-life reduction is due to membrane phospholipid deterioration through phospholipase activity and/or phospholipid oxidation during aging. Therefore, the objective of this study was to characterize the modifications/deterioration of phospholipid classes/species in pork loins from 3 different aging periods.

Materials and Methods

Loins from 20 carcasses were collected at a commercial harvest facility in the Midwest 1 d postmortem from carcasses of Duroc sired crossbred pigs. Four chops from each carcass containing only the longissimus muscle were vacuum packaged and aged at 4°C for 1, 8, and 21 d. A sensitive approach based on electrospray ionization tandem mass spectrometry was used to comprehensively analyze phospholipid composition using the lipid extract from each sample at each aging period (n = 60). Unsaturation index (UI; measurement of the number of double bonds) for each phospholipid species class was also calculated to quantify fatty acyl chain unsaturation for each sample in each aging period.

Results

Total phospholipid quantity in pork loins was not different between 1d and 8d aged chops but decreased significantly from 8d to 21d of aging (806.6 vs. 297.5 nmol phospholipid/mg lipid; $P < 0.01$). On the other hand, the mol% data (distribution of each phospholipid species in relative % of total phospholipid) revealed that phosphatidylinositol (PI) and phosphatidylserine (PS) increased in mol% from 1d to 21d of aging in pork loins ($P < 0.01$). This increase was mainly due to the increase of PI 38:4 (primarily 18:0/20:4) and PS 36:2 (primarily 18:0/18:2) between 1d and 21d samples ($P < 0.01$). The results showed that phospholipid degradation products like lysophosphatidylcholine (LPC) mol% rose quickly after short term aging (8d) but remained constant through the rest of the 21d aging period ($P < 0.01$). Conversely, lysophosphatidylethanolamine (LPE) was unaltered between 1d and 8d of aging but decreased between 8d and 21d aged pork loins ($P < 0.01$). The mol% of phosphatidic acid (PA) also increased between 1d and 21d aged pork loins ($P < 0.05$). Extended aging did not alter the mol% of total phosphatidylcholine (PC), ether-linked PC (ePC), sphingomyelin (SM), phosphatidylethanolamine (PE) or ether-linked PE (ePE; $P > 0.05$). Surprisingly, UI revealed the exact opposite trend as the mol% data. The UI of PI and PS decreased ($P < 0.01$) from 1d to 21 d of aging in pork loins due to the disappearance of many minor PI and PS species with very long chain fatty acids and multiple double bonds such as PI 42:10 and PS 44:10. There was also a slight increase of PC UI after 8 d of aging in pork loins ($P < 0.01$). The UI for LPC, ePC, SM, LPE, PE, ePE and total phospholipid were not altered in any of the aging periods ($P > 0.05$).

Conclusion

These results confirmed our hypothesis that phospholipids undergo extensive degradation during aging. The data also indicated that the majority of phospholipids in pork loins may maintain integrity over short period aging (1–8d). Among the phospholipid classes, PI and PS were slightly more resistant to deterioration compared with the others due to their ability to modify fatty acyl chain saturation. Additional investigations are necessary to define the role of phospholipid modifications in fresh pork shelf-life and flavor.