P-09-06

# Carnosine reduction of AGEs (CML) formation during digestion of meat (#157)

Yi Yao Li[1, 5], Marie France Palin[2], Varoujan Yaylayan[1], Brian Sullivan[3], Frédéric Fortin[4], Simon Cliche[5], Claude Gariépy[5]

[1] Mcgill University, Food Science and agricultural chemistry, Ste Anne de Bellevue, Canada; [2] AAFC Sherbrooke R&D Centre, AAFC Sherbrooke R&D Centre, Quebec, Canada; [3] Canadian Centre for Swine Improvement, Canadian Centre for Swine Improvement, Ottawa, Canada; [4] Centre de Développement du Porc du Québec, Centre de Développement du Porc du Québec, Quebec, Canada; [5] AAFC St-Hyacinthe R&D Centre, AAFC St-Hyacinthe R&D Centre, Saint-Hyacinthe, Canada

## Introduction

Red meat consumption, in spite of offering a high density of important nutrients, has been associated with increased risk of developing a range of chronic diseases. The advanced glycation end products (AGEs) formed in vivo [1-2] and potentially also those derived from the meat [1,3] have been reported to contribute to the development of these diseases [1,3]. Although further validation is warranted for the dietary AGEs contribution to consumer's health [4], it was recently suggested that AGEs could be formed directly in the gastro intestinal tract (GIT) [5], but supporting data are warranted. Carnosine ( -alanyl-L-histidine), a meat dipeptide possessing multiple properties, including antiglycation, is recognized for its capacity to inhibit AGEs formation in biological tissues [2,6]. However, compared to the experimental use of carnosine supplement, very few studies have involved carnosine in its meat matrix. The pro-oxidative environment of the GIT can contribute to the oxidation of meat components during digestion leading in turn to potential health implications [7]. Therefore, assessing the assumptive AGEs formation in the GIT and their potential *in situ* inhibition by carnosine from meat could shed a new light on the health attributes of meat.

## Methods

Fresh ground pork *longissimus* muscle was prepared as per Table 1. *In vitro* digestion (salivary, gastric, and duodenal phases) was conducted on cooked meat based on Van Hecke et al. [8]. Digests from each phase were analyzed for N(epsilon)-(carboxymethyl)lysine (CML), a common AGEs maker [3], with the OxiSelect™ CML competitive ELISA kit (Cell Biolabs, Inc, San Diego, USA). Data were analyzed by the MIXED procedure of SAS version 9.4 (SAS, 2002-2012; SAS Institute Inc., Cary, USA).Table 1. Preparation of meat samples

| Low Carnosine (LCar=309.8mg/100g longissimus muscle)* | | High Carnosine (HCar=600.0mg/100g longissimus muscle)** | |
|---|---|---|---|
| Low Fat (LF=1.3%)* | High Fat (HF=10.0%)** | Low Fat (LF=1.3%)* | High Fat (HF=10.0%)** |

* intrinsic levels of carnosine and fat in *longissimus* muscle, **targeted levels of carnosine and fat (increased).

## Results

Table 2 shows a clear increase in CML during the digestion of meat irrespective of treatments suggesting an overall increase in AGEs formation. Statistical effects of either increased fat content or carnosine level were obtained in each of the salivary and duodenal phase in line with their respective enhancing or reducing effect on CML. However, it is the interaction between carnosine and fat content in the saliva ($P < 0.0001$) and duodenal phase ($P=0.0112$) that shows the inhibition of AGEs formation by meat carnosine in the GIT. Although high fat content enhanced CML formation, increasing carnosine level in the meat from HF groups was clearly effective in decreasing the CML level compared to that of the LCar-HF group. In the gastric phase, the trend for decreased CML in the HCar-LF group only ($P = 0.0505$), compared to the other treatments, also supports the carnosine reduction of AGEs (CML) formation in the GIT.Table 2. CML levels

| Phase | LCar | | HCar | | P Values | | |
|---|---|---|---|---|---|---|---|
| LF | HF | LF | HF | Car | Fat | Car*Fat | |
| Salivary | 18.06 | 46.98 | 15.75 | 13.89 | <.0001 | <.0001 | <.0001 |
| Gastric | 113.18 | 111.60 | 69.45 | 114.64 | .0857 | .0668 | .0505 |
| Duodenal | 733.90 | 1281.37 | 669.16 | 889.31 | .0009 | <.0001 | .0112 |

Unit: ng/g meat; Car: carnosine.

## Conclusion

This study shows that AGEs formation as CML occurred during the digestion of meat relative to its fat content. However, the significant reduction of CML by carnosine, particularly in the duodenum where CML formation was the most prevalent and potentially available for absorption, bring forward a potentially important role of carnosine for reducing AGEs formation in the GIT.

## References

1. McAfee, A. J., McSorley, E. M., Cuskelly, G. J. et al. (2010). Red meat consumption: an overview of the risks and benefits. Meat Sci. 84: 1-13.
2. Boldyrev, A. A., Aldini, G., & Derave, W. (2013). Physiology and pathophysiology of carnosine. Physiol. Rev. 93: 1803-1845.
3. Poulsen, M. W., Hedegaard, R. V., Andersen, H. M. et al (2013). Advanced glycation end products in food and their effects on health. Food Chem. Toxicol. 60: 10.37.
4. Nowotny, K., Schröter, D., Schreiner, & M. Grune, T. 2018. Dietary advanced glycation end products and their relevance for human health.

Notes

1. Ageing Res. Rev. 47: 55-66.
2. Hipkiss, A. R. (2018). Glycotoxins: Dietary and metabolic origins; possible amelioration of neurotoxicity by carnosine, with special reference to Parkinson's disease. Neurotox. Res.. 34: 164-172
3. Freund, M. A., Chen, B., & Decker, E. A. (2018). The inhibition of advanced glycation end products by carnosine and other natural dipeptides to reduce diabetic and age-related complications. Comp. Rev. Food Sci. Food Safety, 17: 1367-1378.
4. Van Hecke, T., Van Camp, J., & De Smet, S. (2017). Oxidation during digestion of meat: Interactions with the diet and Helicobacter pylori gastritis, and implications on human health. Comp. Rev. Food Sci. Food Safety. 16: 214-233.
5. Van, Hecke. T., Vossen, E., Vanden, B. J. et al. (2014). Fat content and nitrite-curing influence the formation of oxidation products and NOC-specific DNA adducts during In Vitro digestion of meat. Plos One. 9: e101122.

**Notes**