Lipid oxidation and its protection by plant polyphenols in an *in vitro* model of gastric digestion

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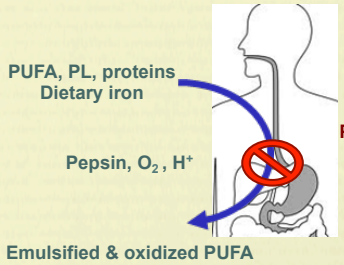
Introduction

The gastric tract may be the first site where food is exposed to postprandial oxidative stress and antioxidant activity by plant micronutrients. After food intake, dietary iron, dioxygen and emulsified polyunsaturated fatty acids (PUFA, mostly as triglycerides) come into close contact and substantial lipid oxidation may take place [1-2]. The primary lipid oxidation products, i.e. hydroperoxides, may not reach the intestine but rather decompose in the stomach into hydroxy fatty acids, aldehydes and epoxides [3], which once absorbed may participate in the atherogenicity of Low Density Lipoproteins [4].



Aim of the study

The *in vitro* investigation of lipid oxidation possibly taking place in the gastric tract and its inhibition by common dietary polyphenols (PP). In particular, oligomeric proanthocyanidins (OPAs), whose content in the diet is probably largely underestimated [5], deserve more detailed investigations.

Materials & Methods

In vitro model of gastric digestion

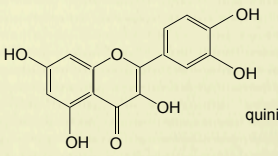
- 10% sunflower o/w emulsions were stabilized by bovine serum albumin (BSA) or egg yolk phospholipids (PL) in order to model the physical state of lipids after ingestion of a western diet
- The pH was set at 5 (initial stage of gastric digestion) and then at pH 3 (mid-phase of digestion) [6]
- Addition of pepsin at 0.06 mg/mL (gastric protease, 4220 U/mg)
- Initiation of lipid oxidation by 20 µM (metmyoglobin, $MbFe^{III}$) at 37 °C
- Lipid-derived conjugated dienes (mostly hydroperoxides) were monitored by UV spectroscopy at 234 nm

Tested polyphenols

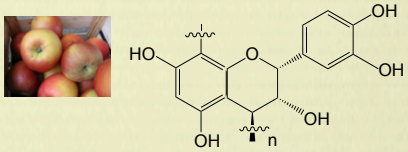
- Quercetin (Q) and (-)-epicatechin (EC) were added at 100 µM final concentration
- Oligomeric proanthocyanidins (OPAs) were extracted from the cider apple variety Kermerrien and were added at 100 µM expressed as concentration of EC monomer

I. Oligomeric mix with DP 1 to 12 (OPAs DPav8): OPA purity 68 % + chlorogenic acid (5-CQA) 6 % or 7 µM as final concentration

II. Trimers (OPAs DP3): purity 77 %



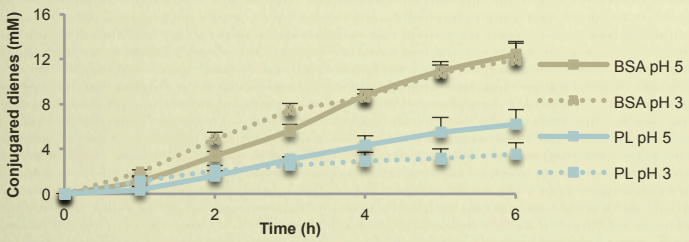
Quercetin (Q) **Chlorogenic acid (5-CQA)**



OPAs: n > 1
(-)-Epicatechin (EC): n = 1

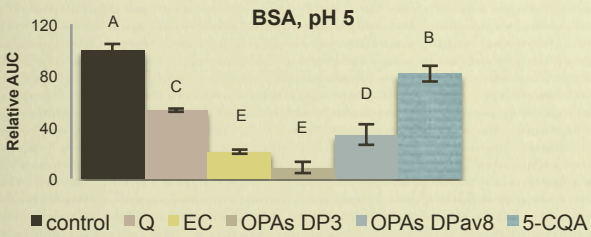
Results & Discussion

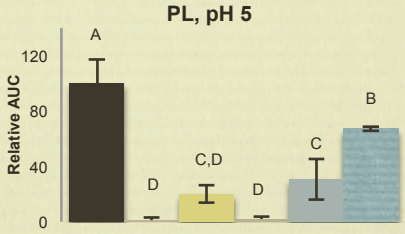
I. Metmyoglobin-initiated lipid oxidation

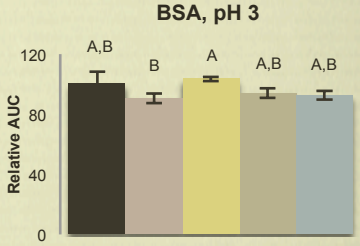


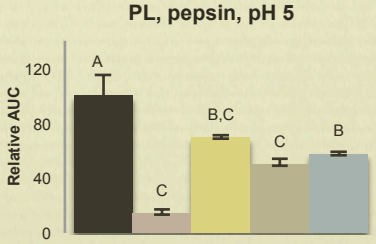
- Lipid oxidation is strongly modulated by the type of emulsifier: PL is a small surfactant that gives more homogeneous interfaces than BSA ⇒ PL-stabilized emulsions are less vulnerable to oxidation
- pH has no significant impact on the rate of lipid oxidation

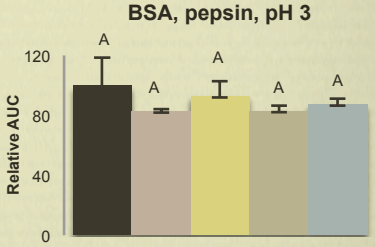
II. Inhibition by dietary polyphenols : Effect of the emulsifier type, pH and pepsin











- At dietary concentrations, Q, EC and OPAs proved to be highly inhibitory in the presence of BSA and PL at pH 5
- The OPA trimer is as efficient as the EC monomer, implying a high reactivity of all the covalently bound EC units with $MbFe^{III}$
- Chlorogenic acid (5-CQA) at 7 µM contributed little to the overall reactivity of the OPA fractions
- The antioxidant activity of polyphenols is lost at pH 3 due to the denaturation of $MbFe^{III}$ (release of hematin cofactor)
- Pepsin does not influence the antioxidant effect of polyphenols with both emulsifiers

Conclusion

This work demonstrated that a diet rich in fruit and vegetables can efficiently protect lipid nutrients from oxidation during the initial stage of gastric digestion (pH 5).

Furthermore, the rate of lipid oxidation depends on the emulsifier type whereas its inhibition by dietary polyphenols is more strongly modulated by pH than by pepsin or the emulsifier type.

References

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