Evaluation of different dietary lipid supplements on oxidatively generated biomarkers in periparturient dairy goats.

Bellagamba Federica[1], Busetto Maria Letizia[1], Caprino Fabio[1], Invernizzi Guido[1], Moretti Vittorio Maria[1], Savoini Giovanni[1].

[1] Dipartimento di Scienze e Tecnologie Veterinarie per la Sicurezza Alimentare, Università degli Studi di Milano, Via Trentacoste 2, 20134 Milano, Italy

The study of biomarker of oxidative stress in ruminant is a field of research recently explored [1]. Consequences of high-fat diet and lipid peroxidation on the health of ruminants and quality of their products have been considered and discussed [2].

This study aimed at analyzing the impact of different dietary lipid supplements on oxidative stress status in 26 periparturient Alpine dairy goats. At day 130 of gestation (about 20 days before kidding), goats, chosen homogeneous for parity and milk yield in previous lactation, were housed in single boxes, divided in three groups and fed with experimental diets just differing in lipid sources: (a) a dietary protected fish oil (FO) group, rich in n-3 long chain polyunsaturated fatty acids (n-3 LC-PUFAs); (b) a dietary calcium stearate (ST) group, rich in 16:0, 18:0 saturated fatty acids (SFAs) and (c) a dietary control group (C), without any supplement. Experimental fatty acid enriched diets (FO and ST) were both formulated to administer 30 g/day and 50 g/day of fatty acids before and after kidding, respectively. Blood samples (98 samples) were collected weekly starting from day 130 of gestation until 21 days of lactation; serum and plasma were obtained immediately after sampling and stored at -80° C until the analysis. Analytical determination of malondialdehyde (MDA) by high performance liquid chromatography (HPLC) and enzyme-linked immunosorbent assay (ELISA) assessment of 8-oxo-7,8-dihydro-2'-deoxyguanosine (8-oxodGuo) have been carried out in serum samples. Since the 8-oxodGuo derives after the ROS attack to DNA, this DNA-adduct is widely used as specific marker of oxidative damage. This DNA adduct can be measured in tissues, serum and urine and often used as prognostic factor in cancer lesions and degenerative diseases. MDA is the most abundant individual aldehyde resulting from lipid peroxidation and commonly employed in studies involving oxidative alteration of lipids. Gas chromatographic determination of plasma fatty acid was also performed. The same sampling from animals fed without any supplements (C group) has been considered as control and analysed. Data were processed by analysis of variance (ANOVA) and values presented as mean ± SEM. Data were subjected to a Student–Newman–Keuls post hoc test for homogeneous subsets, and a P value of ≤ 0.05 was considered as significant.

MDA and 8-oxodGuo resulted both higher in FO group when compared to ST and C group (822.02±50.45 μmol ml^{-1} and 1.83±0.24 ng ml^{-1}, respectively), being the 8-oxodGuo level from FO group statistically different ($P \leq 0.05$) from other groups. Dietary lipid supplements produced remarkable change in plasma fatty acid (expressed as g/100g total FAs), and particularly, n-3 LC-PUFAs, 20:5 n-3 (2.06 %), 22:5 n-3 (1.94 %) and 22:6 n-3 (1.61 %), in FO group resulted significantly different ($P \leq 0.05$) from ST and C groups. In ST group plasma, 18:0 level was the highest (19.20 %) and significant different from FO (17.47 %) diet group. The n-6 LC-PUFAs (20:3 n-6; 20:4 n-6) as well as linoleic acid were not different at statistical level among experimental groups. MDA and 8-oxodGuo did not show any significant difference amongst different times of sampling; otherwise the effect of sampling was evident in fatty acids of plasma with a significant ($P \leq 0.05$) increase of EPA (20:5 n-3, 3.9 %) and DHA (22:6 n-3, 2.25 %) in FO diet after 21 days of supplementation Concerning the metabolic variation around peripartum, the results did not produce any significant difference within the experimental groups, however the highest value of 8-oxodGuo was found in peripartum period of goats receiving FO diet. The present study confirms that modifications of dietary fatty acid composition derived from the utilisation of different dietary lipid sources in feed formulations can have appreciable impact on oxidative DNA damage also in ruminants and produce valuable variations of plasma fatty acids. No evidence of a significant increase of serum MDA concentrations has been verified.

References

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