IJAE Vol. 115, n. 1/2 (Supplement), 2010

Sialoglycoconjugate expression in the intestinal mucosa of obese Zucker rats

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Growing interest is focused on exogenous and endogenous factors which may modulate the critical equilibrium of the intestinal environment. In this context, we previously investigated the glycosylation pattern in the colonic mucosa of rats under different dietary conditions. In the gastrointestinal tract glycoconjugates, mainly represented by sialoglycoconjugates, contribute to the properties of the intestinal mucins. They also act in various events, such as inflammation, neoplastic transformation, and cancer metastasis. Sialic acids, as terminal sugars in the glycan chains, represent specific binding sites for microorganisms, thereby regulating intestinal flora and pathogenity. Recent results support the emerging view that gut microbiota contribute to metabolic disease and suggest their possible role in obesity and, consequently, in other aspects of metabolic syndrome.

Based on these suggestions, the present study has investigated and characterized *in situ* the sialoglycoconjugates expressed in the intestinal mucosa of obese Zucker rats (OZR). OZR represent a model of type 2 diabetes exhibiting a moderate degree of arterial hypertension and increased oxidative stress. The occurrence and distribution of sialic acids differently linked to D-Gal and/or to D-GalNAc were demonstrated with SNA and MAL II lectin binding. Moreover, to have additional and complementary information about sialic acid acetylation degree and sites, PNA and DBA lectin histochemistry was combined with sialidase predigestion, potassium hydroxide deacetylation, and differential periodate oxidation.

The binding patterns obtained, which represent the distribution sites of sialylated glycocomponents expressed within the epithelium of the intestinal tract and/or are secreted as components of the cellular glycocalix, showed an overall lower intensity of lectin reactivity in OZR compared with control animals. Both the sialic acid (α2,6)-D-Gal/D-GalNAc and sialic acid (α2,3)-D-Gal sequences were affected. The indirect evaluation of sialic acid residues along the intestinal tract confirms the above observations and further details the structural features of the sialic acid residues in OZR rats. These results are worthy to be further investigated to establish possible correlations between the modified sialylation expression and the characteristics of microbial modulation reported in metabolic syndrome.

Key words
Intestinal mucosa, Sialoglycoconjugate, metabolic syndrome, obese Zucker rats