

# Immunoexpression of type-1 adiponectin receptor in the human intestine

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## SUMMARY

Adiponectin is an important biomarker of metabolic syndrome that was recently identified in human breast milk. We demonstrate the presence of type-1 adiponectin receptor (adipoR1) by immunoperoxidase method in 21 biopsic specimens – duodenum ( $n = 6$ ), terminal ileum ( $n = 7$ ) and colon ( $n = 8$ ) from 14 human subjects (6 females and 8 males aged 9 months–47 years). In all the samples, adipoR1 was detected. The positivity was observed in enterocytes and colonocytes as well as in lymphocytes in the submucosa and in the smooth muscle of the intestinal wall. Thus, adiponectin may influence intestinal physiology through its type-1 receptor.

**Keywords:** adiponectin – adiponectin receptor – intestine – nutritional programming – breast milk

## Imunoexprese adiponektinového receptoru typu 1 v lidském střevě

### SOUHRN

Adiponektin je důležitý biomarker metabolického syndromu, který byl v nedávné době identifikován také v mateřském mléce člověka. V naší práci prokazujeme přítomnost adiponektinového receptoru typu 1 (adipoR1) imunoperoxidázovou metodou ve 21 biopatických vzorcích – duodenum ( $n = 6$ ), terminální ileum ( $n = 7$ ) a kolon ( $n = 8$ ) od 14 jedinců (6 žen a 8 mužů ve věku 9 měsíců – 47 let). Ve všech vzorcích jsme prokázali přítomnost adipoR1. Pozitivita byla pozorována v enterocytech, kolonocytech i v lymfocytech submukózy a v hladké svalovině střevní stěny. Předpokládáme, že adiponektin může ovlivňovat intestinální fyziologické děje prostřednictvím adiponektinového receptoru typu 1.

**Klíčová slova:** adiponektin – adiponektinový receptor – střevo – nutriční programování – mateřské mléko

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Adiponectin represents an important regulatory hormone produced by adipocytes. It influences the metabolism of fatty acids and increases insulin sensitivity. Low serum levels of adiponectin were described in patients with metabolic syndrome (1). Recently, adiponectin has been identified in human breast milk suggesting that it may play a role in nutritional programming of newborns and thus lead to lower risk of symptoms of metabolic syndrome later in adulthood (2,3,4). Previously, the presence of adiponectin was described also in human fetal tissues (5) and also epithelial cells of the salivary gland were identified as a source of adiponectin (6). Moreover, recent findings have pointed to the fact that expression of adiponectin in the mesenteric adipose tissue may be related to development of inflammatory bowel diseases (7). The mechanism of adiponectin action in the gut remains unknown as of yet.

Two main types of adiponectin receptors have been recognized - adipoR1 and adipoR2 (8). AdipoR1 is a dominant type of the

receptor expressed in humans, mainly in the skeletal muscle, but also in the liver, adipose tissue, kidney, bone, prostate, placenta, endometrium, adrenal cortex and other organs. AdipoR2 is predominantly expressed in the hepatocytes. Adiponectin receptors were previously identified in the intestine of mouse embryos (9). AdipoR1 in mice is generally highly expressed in tissues derived from primitive gut.

Up to date there has been no evidence of the presence of these receptors in human intestine. The aim of the present study was to demonstrate the presence of adipoR1 in the human intestinal wall by use of immunohistochemistry.

## MATERIALS AND METHODS

Twenty one intestinal biopsic samples from 14 subjects (6 females and 8 males aged 9 months – 47 years) undergoing upper or lower endoscopic investigation of the gastrointestinal tract with normal macroscopic and histological findings were enrolled into the study. All the participants or their parents subscribed an informed consent form and the study was approved by the local ethical committee. The samples were taken from the duodenum ( $n = 6$ ), terminal ileum ( $n = 7$ ) and colon ( $n = 8$ ). Immunohistochemistry using immunoperoxidase method with the antibody against adipoR1 (Anti-Adiponectin Receptor-1 (357-375), Phoenix Pharmaceuticals, cat. no. H-001-44, diluted 1:250, microwave pretreatment) was per-

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