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Metalloproteinases and their inhibitors gene expression profiles in leukocytes of primary hypertension (PH), non-alcoholic fatty liver disease (NAFLD), and obese children

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Primary hypertension (PH), non-alcoholic fatty liver disease (NAFLD) and obesity are all related to a systemic low-grade inflammation, vascular remodeling, visceral obesity, insulin resistance, and dyslipidemia. However, NAFLD children rarely develop PH. The aim of this study was to assess peripheral blood leukocyte (PBLs) gene expression profiles of mediators involved in extracellular matrix degradation, vascular remodeling and inflammation in the normal weight PH, obese PH, NAFLD and obese, normotensive, non-NAFLD, healthy children, aged 9-16 yrs, compared to healthy, age-matched, normal weight (only for obese PH - obese healthy) control group. This approach makes possible to find out which of tested factors is related to PH or NAFLD regardless of obesity. Total leukocyte mRNA expression levels were measured by quantitative RT-PCR (real-time reverse transcriptase-polymerase chain reaction). Relative target gene expression level, compared to control group was normalized by expression of the reference gene - G3DPH. We tested the expression of a) matrix Metalloproteinases (MMPs) and their inhibitors (TIMPs), including MMP-9, MMP-2, MMP12, MMP14, and TIMP-1, TIMP-2, respectively, and b) mediators: Insulin like growth factor -1 receptor (IGF-1R); transforming growth factor beta (TGF); and interleukin-6 (IL-6).

The normal weight PH patients (pts) expressed high levels of MMP-14, increased MMP-2, slightly elevated MMP-9, TIMP-1, 2 with only moderately increased IGF-1R, TGF-beta and IL-6 compared to healthy, normal weight control. The obese PH pts had 2-fold higher expression of MMP-9, MMP-2, and IL-6, slightly increased MMP-12, 14, and decline in TIMP-1/2, TGF-beta, and IGF-1R, as compared to normal weight PH pts. The NAFLD pts were characterized by increased of MMP-9 (equal to that of normal weight PH pts) and rather low expression of other mediators studied, except for high TGF-beta gene expression levels.The obese, normotensive, non-NAFLD pts had increased MMP-9, MMP-12, but very low MMP-2 expression as compared to normal weight controls, with no significant changes in the expression of other mediators studied.

Conclusions:

1. Increase in leukocyte MMP-2, MMP-14 and IL-6 gene expression seems to be more related to PH than to obesity. It possibly predisposes to increase in vascular remodeling and elevation of arterial stiffness due to vessel wall elastin degradation and immature collagen deposition
2. In contrast, MMP-9 and MMP-12 gene expression levels are related rather to obesity but less to PH
3. NAFLD was related to selective increase in leukocyte TGF-beta gene, more balanced expression of MMPs and TIMPs, with low IL-6 and IGF-1R gene expression levels. It may reflect less vigorous arterial wall remodeling and vascular activation

Biography

Joanna Trojanek has completed the PhD of Biochemistry from Institute of Biochemistry and Biophysics Polish Academy of Sciences in 1991. She was postdoctoral fellow at Center for Neurovirology and Cancer Biology, Department of Biology, Temple University, Philadelphia, USA for three years and afterwards at Department of Translational Pulmonology, University Children Hospital, and Heidelberg, Germany for next three years. From 2010 she is Research Associate in Microbiology and Clinical Immunology Department at The Children's Memorial Health Institute. She has published about 18 papers in peer-reviewed journals. The main research areas are reflected to gene and protein expression profiles involved in pathogenesis and development of primary hypertension and other metabolic syndromes in children's leukocytes.

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