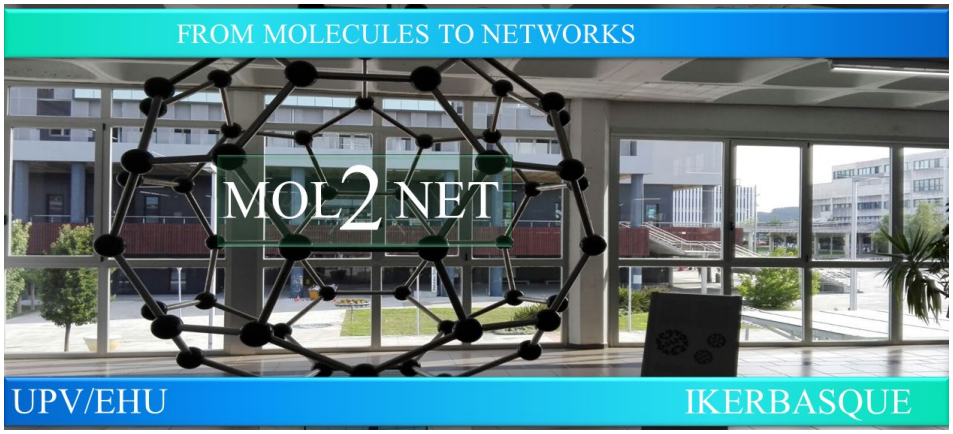

Changes in the expression pattern of genes encoding selected adipokines in endometrial cancer

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Introduction

Leptin (LEP) is primarily secreted by differentiated adipocytes, influencing neoplastic angiogenesis through the activation of the JAK/STAT pathway, which results in the increased proliferation of vascular endothelial cells as well as an increased expression of the vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF) and anti-apoptotic proteins, namely Bcl-2. In women whose BMI is higher than 25, the risk of endometrial cancer is doubled and in those whose BMI is above 30, this risk is tripled. A higher concentration of LEP in the serum of women with endometrial cancer compared to healthy volunteers was also confirmed.

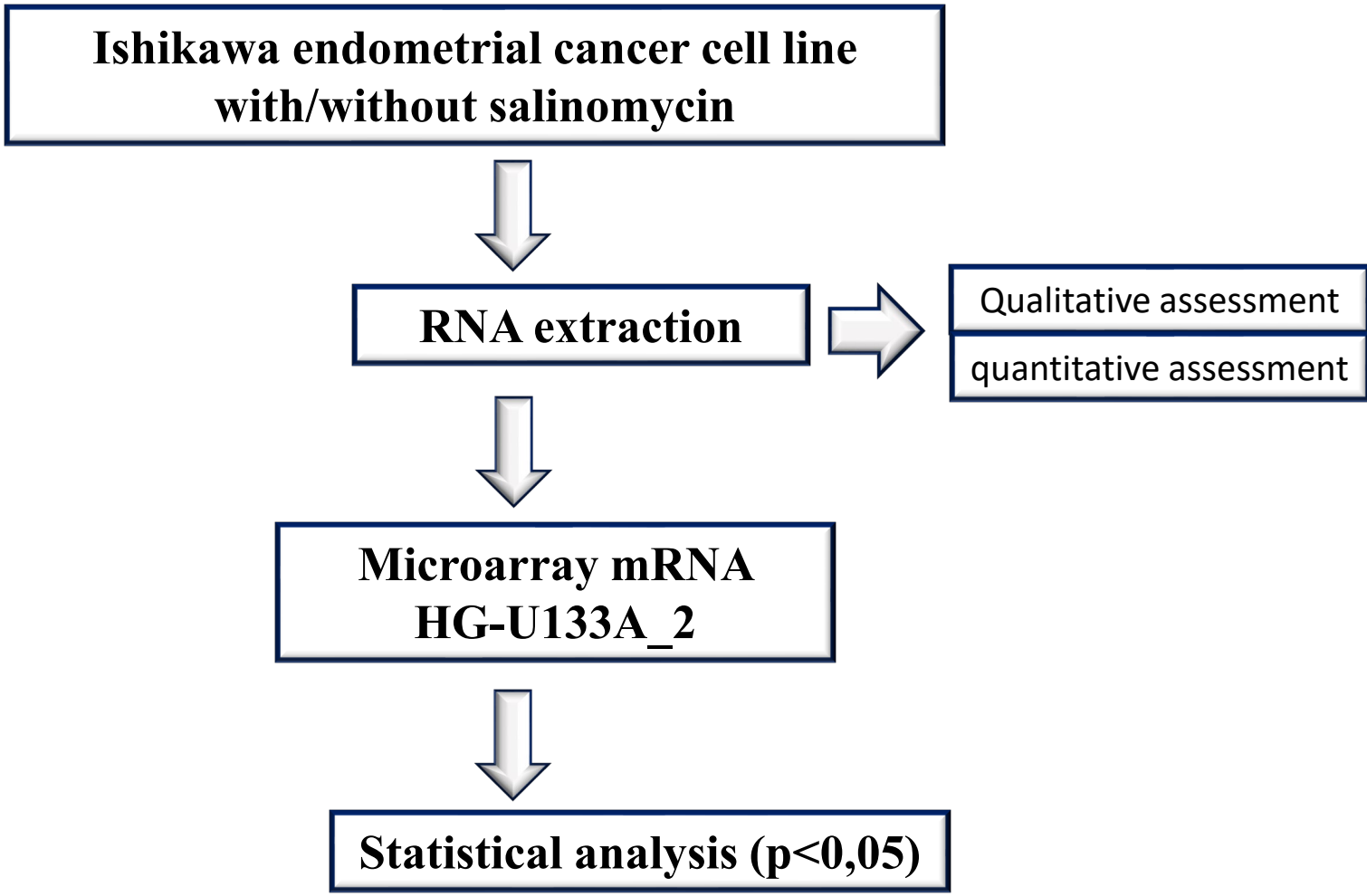
Introduction

Adiponectin (ADP) demonstrates an insulin-sensitizing effect, increasing the expression of nitric oxide synthase, furthermore, it also has anti-inflammatory activity through inhibiting the expression of tumor necrosis factor-alpha (TNF-α) and interleukin 6 (IL-6). A 6-times higher risk of cancer development is noted in obese people, in whom the concentration of ADP was lower than in the group of healthy volunteers. It is highlighted that it is this adipokine that is most strongly associated with the risk of endometrial cancer.

Aim

The overriding aim of this study was the assessment of changes in the expression pattern of genes coding chosen adipokines in an Ishikawa line endometrial cancer cell culture exposed to the effects of cisplatin, compared to a control culture.

Material and methods



Results

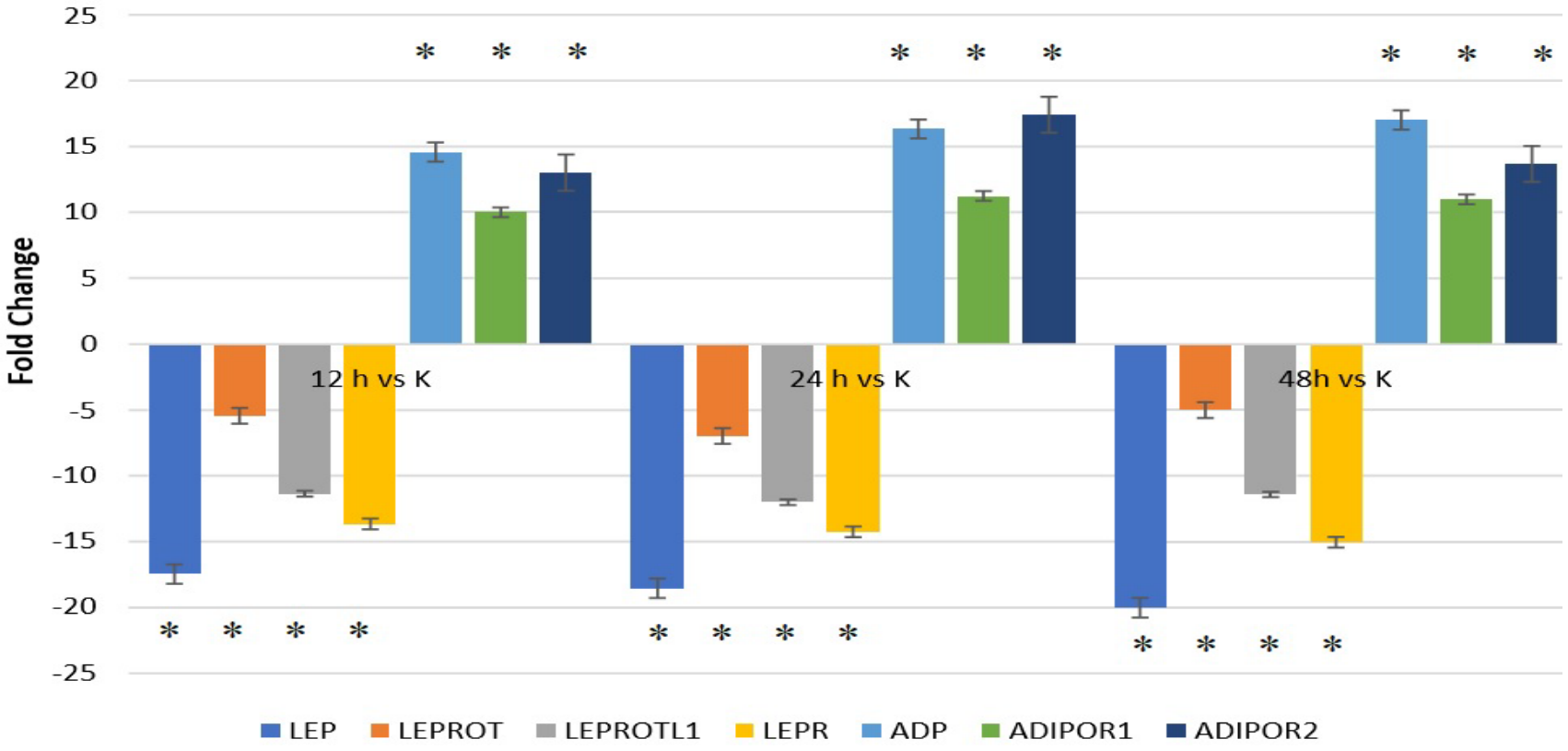


Figure 1. Expression of adiponectin, leptin and their receptors depending on the exposure time of the Ishikawa cell line to cisplatin.

Conclusion

Changes in the expression profiles of leptin, adiponectin, can be explored in the context of utilizing them as supplementary diagnostic markers of endometrial cancer, monitoring the effectiveness of cisplatin therapy, and in the creation of new therapeutic strategies aimed toward the JAK/STAT signaling pathway.

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