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Differential expression of various LncRNAs and microRNAs in abdominally obese and non-obese individuals

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Motivation and Aim: Obesity is a complex, multifactorial disease characterized by excessive fat accumulation that may impair health [1]. Despite significant advances in our understanding of obesity, many aspects of its underlying molecular mechanisms remain elusive. Long non-coding RNAs (lncRNAs) and microRNAs (miRNAs) are increasingly explored as critical regulators or biomarkers in various diseases [2], with their roles in adipogenesis, lipid metabolism, and insulin response currently being studied [3]. However, studies linking lncRNAs and miRNAs to obesity are relatively scarce and sometimes yield inconsistent results [4]. Our research investigates the differential expression of a wide range of lncRNAs (ASMER1, SNHG9, P5549, p19461, GAS5 and MALAT1) and miRNAs (MIR26A1, MIR222, MIR221 and MIR155) in the visceral adipose tissues of individuals with and without abdominal obesity.

Methods and Algorithms: Population and Sampling: The study included a case group with abdominal obesity and a control group without obesity, in total 100 participants. Abdominal obesity was defined accordingly Body Mass Index (BMI) ≥ 25.0 kg/m² for whites, and for Asians BMI ≥ 23.0 kg/m² with a waist circumference (WC) ≥ 80 cm for women and WC ≥ 94 cm for men [5]. White visceral adipose tissue samples were collected from individuals during elective surgery.

RNA Extraction: Tissue samples were preserved immediately in RNAlater (Thermo Fisher Scientific, USA) and stored at –20 °C until RNA extraction. RNA was extracted and cDNA was synthesized according to manufacturers protocols (Biolabmix, Russia). Quantitative PCR (qPCR) was performed using SYBR dye (Biolabmix, Russia) to determine the relative expression levels of targeted microRNAs and lncRNAs using the LightCycler96 system (Roche, Switzerland). The ΔΔCq method was utilized for expression level calculations.

Statistical Analysis: The normality of data distribution and homogeneity of variances were assessed using Levene's test. Correlations between RNA levels and various clinical indicators were determined using Student's t-tests and Mann–Whitney U tests. The Bonferroni correction was applied to adjust for multiple comparisons.

Results: Expression of ASMER1 and MALAT1 as well as miR222 and miR221 was undetectable in the homogenate of visceral fat tissue. RNA levels of P5549 and P19461 were at the borderline of detection levels and showed no reliable differences between the control and case groups. Expression levels of SNHG9, GAS5, and miR155 showed

no significant difference between participants with and without obesity, while the expression of miR26A1 was significantly lower ($P<0.01$) in the case group.

Conclusion: Our study is the first to show that miR26A is significantly downregulated in the visceral adipose tissue of obese individuals. This finding is consistent with previously described functions of miR26A, such as suppressing adipocyte progenitor differentiation and reducing visceral fat mass and blood lipid levels in mice [6].

In contrast, GAS5 levels remained nearly unchanged between groups and showed no correlation with miR26A levels. This result is inconsistent with multiple studies that suggest a regulatory 'sponge' interaction between GAS5 and miR26A [7]. This discrepancy suggests that an alternative mechanism may be responsible for the downregulation of miR26A in human visceral adipose tissue, necessitating further investigation.

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