

## ABSTRACT

**The Resistance Mechanism of Adipogenesis Caused By Electroacupuncture at The S 40 Point on Male White Rats (*Rattus Norvegicus* Wistar Strain) Exposed to A High Fat Diet**

**Methods and materials:** 42 white male rat within the age of 1-2 months were used and divided into KN Group (normal diet, without EA), K- Group (high-fat diet, without EA), K+ Group (high-fat diet with nonakupoin EA), FRIR Group (high-fat diet and with a low frequency and low intensity ST 40 EA), FRIT Group (high-fat diet with a low frequency and high intensity ST 40 EA), and FTIR Group (high-fat diet and with a high frequency and low intensity ST 40 EA). Electroacupuncture is performed once a day five times a week for four weeks. At the end of treatment, the test subjects were dissected and the PPARG expression in adipose tissue is examined to see the most optimal dose to inhibit of adipogenesis. After discovering the optimal dose, further investigation to find a mechanism by measuring the levels of BMP4 and TGF beta blood levels and BMP4 expression, IGF1, ADD1, CEBP $\alpha$  and TGF beta in visceral adipose. The data was analyzed using a normality, homogeneity, and difference test and path analysis using SPSS.

**Results:** A significant decrease in visceral adipose PPARG expression between the treatment groups compared to the K- and K+ groups with  $p=0,00$ . The most prominent PPARG expression inhibition occurred within the FTIT group ( $3.67\pm 1,63$ ;  $p<0.01$ ). A decrease also occurred towards the levels of BMP4 in blood ( $37.68\pm 5.95$  ng/mL;  $p<0.01$ ) and the expression in visceral adipose ( $5.50\pm 1.87$ ;  $p<0.01$ ). Within the FTIT group, a decrease in visceral IGF1 expression ( $3.00\pm 1.10$ ;  $p<0.01$ ), visceral ADD1 expression ( $6.33\pm 1.86$ ;  $p<0.01$ ), and CEBP $\alpha$  ( $5.67\pm 2.34$ ;  $p<0.01$ ). Using path analysis it was found that EA on ST 40 FTIT inhibits adipogenesis through the ADD1 pathway ( $B = -2030$ ) towards PPARG  $B = 0521$ . PPARG and CEBP $\alpha$  interact reciprocally with  $B = 0521$  and  $B = 0.980$ .

**Conclusion:** EA ST 40 has the ability to inhibit the determination phase and differentiation phase of adipogenesis. Inhibition occurs by lowering blood levels of BMP4 and BMP4 expression in adipocytes, the decreasing of of ADD1 expression, increased levels of TGF beta in the blood and increased expression of TGF Beta in adipocytes which is a potent inhibitor of the differentiation process. The inhibition of adipogenesis that is caused by EA ST40 is best done at a high frequency, and the strength of the effect increases equally to the increase in the intensity of the equivalent electricity used.

**Keyword:** EA ST 40, PPARG, adipogenesis, obesity, high fat diet