Review of: "The obesity-linked human lncRNA AATBC regulates adipocyte plasticity by stimulating mitochondrial dynamics and respiration"

Laurent Vergnes[1]

1 University of California, Los Angeles

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This study characterizes the human lncRNA AATBC in adipocytes. This lncRNA has been previously characterized in cancer models. Here, the authors identified AATBC as a lncRNA enriched in thermogenic adipocytes and induced by beta-adrenergic activation and differentiation. Using loss and gain of function studies, the authors demonstrated that AATBC promote mitochondrial respiration and UCP1 expression. Effects on mitochondrial respiration were associated with a change with fusion/fission remodeling. In humans, AATBC expression was lower in obese female, correlated with UCP1 and PPARGC1A, and inversely correlated with ADIPOQ, LEPTIN, FABP4 and PPARG in visceral fat.

The results are very convincing. The study in mice by overexpressing AATBC was a long shot since the lncRNA is not express there, so mild changes were expected. The inclusion of human studies to determine the effect of AATBC in vivo really elevated the work and demonstrated that the lncRNA may influence thermogenesis and obesity. Further studies in the future could be done in peri-adrenal fat from pheochromocytoma and control patients, for instance, to determine whether AATBC expression is associated with browning in vivo.

About the respirometry experiments, it is not clear whether the assays were normalized (protein or cell count), looking at the y-axis legend or the Method section.

Supplementary figures 3 and 4 seem to be switched.