EXPRESSION OF IGF2R, IGF2, TGFβ, AND uPAR IN A RAT MODEL OF OBESITY

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ABSTRACT

We hypothesized that obesity induction in lean Long Evans Tokushima Otsuka (LETO, L) and obesity-prone Otsuka Long Evans Tokushima Fatty (OLETF, O) would alter insulin growth factor 2 receptor (IGF2R), insulin growth factor 2 (IGF2), transforming growth factor beta (TGFB), and urokinase-plasminogen receptor (uPAR), that IGF2R and TGFB would increase, and IGF2 and uPAR would decrease. LETO and OLETF rats were raised 4 to 32 weeks, subdivided into a control diet (CON) and high-fat diet (HFD) group. We also raised a cohort of O-HFD rats which underwent interventions (exercise training (EX) or calorie restriction (CR)) or did not undergo intervention (sedentary (SED)), from 20 to 32 weeks. Body weight and body fat percentage increased, and capillarity decreased significantly in biceps brachii (BB) and vastus lateralis (VL) muscle, in non-obese versus obese animals ($p < 0.05$). We observed multiple significant changes in skeletal and adipose feed arteries with obesity induction for our four factors, which did not support our hypothesis. Overall, we find this to be a viable model for examining tissue capillarity in obesity, and that our hypothesis does not agree with the data. We propose that the IGF2R system is moderated in a static-to-proangiogenic state during obesity, and decreases in overall capillarity are driven by other mechanisms.