Post-prandial [PP] lipemia is a significant contributor to the development of dyslipidemia and cardiovascular disease [CVD]. It is also evident that PP lipemia is prevalent during conditions of obesity and insulin resistance [IR] and may contribute to increased progression of CVD. Our group has assessed the potential of the obese JCR:LA-cp rat as a model of PP lipaemia in order to explore chylomicron (CM, apoB48) metabolism during the onset and development of IR in the metabolic syndrome. Studies confirm that both fasting plasma and PP apoB48 area under the curve are significantly elevated in the obese JCR:LA-cp phenotype as compared to lean controls. Mechanistic studies have also found that production of lymphatic CM apoB48 and particle size are significantly increased in this model. In addition, PP dyslipidemia in the obese phenotype can be improved with acute dietary supplementation of n-3 polyunsaturated fatty acids. Using a vascular approach, we have hypothesized that arterial remodeling that accompanies IR may explain accelerated entrapment of apoB48-containing particles. Small leucine rich proteoglycans (including biglycan and decorin) have been observed to colocalize with apolipoprotein-B in human tissue. However the potential impact of IR on vascular remodeling, particularly in presence of obesity, remains unclear. Preliminary observations from the JCR:LA-cp model indicate that biglycan protein core content increases both with age and is exacerbated by IR, suggestive of pro-atherogenic remodelling.