Inflammation is not a driving factor in the development of insulin resistance in diet induced obesity in rats

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Obesity and insulin resistance are frequently associated with chronic low grade inflammation, and the presence of inflammation is considered a contributing factor to disease progression. Male Sprague Dawley rats were fed either a high fat diet (36% wt/wt), a high caloric cafeteria-style diet consisting of popular snack food items, or a control diet (5% fat wt/wt) in order to explore the relationship between inflammation and insulin resistance in diet-induced obesity. While both the high fat and cafeteria diets resulted in increased adiposity, the cafeteria diet was more effective at inducing obesity with body weight (292 ± 5 *vs* 346 ± 6g; mean ± SEM) and epididymal fat pad weight (0.49 ± 0.1 *vs* 2.16 ± 0.1g) significantly different between rats fed the cafeteria diet and control animals. Insulin sensitivity was measured by performing hyperinsulinemic euglycaemic clamps (10 mU/min/kg) in anaesthetized rats. Rats received an initial intraperitoneal injection of sodium pentobarbital (50 mg/kg body weight) and anaesthesia was maintained by a continuous infusion of sodium pentobarbital (0.6 mg/min/kg) *via* the jugular vein during the 2 hour experimental procedure before exsanguination at the end of the experiment. Glucose infusion rate during the clamp was found to be impaired in the cafeteria diet-fed rats compared to controls (26.0 ± 0.6 *vs* 21.2 ± 1.3 $mg.min^{-1}.kg^{-1}$) indicating insulin resistance. Despite being associated with obesity and insulin resistance, neither the high fat nor cafeteria diet-fed rats showed signs of inflammation in skeletal muscle or adipose tissue. These findings suggest that inflammation is not always present in obesity, and is not necessary for the development of insulin resistance.