Both adult and childhood obesity rates are continuing to grow in developed countries throughout the world, possibly resulting in future generations with a shorter lifespan than the current population. There are undeniably multiple factors that have lead to the increase in obesity rates including, but not limited to; behavior (activity), food choices, genetics, and the environment. Although it is unlikely to be the sole contributor, an increase in the consumption of foods and beverages sweetened with high fructose corn syrup (HFCS) has been implicated as one cause of the increased obesity rates in adults and children (Bray et al., Am. J. Clin. Nutr, 2004). Although there is still a relative paucity of human subject data testing this hypothesis, there is however a substantial amount of literature using rodents. Historically, diets high in fructose were utilized for the generation of a hypertensive rodent model since excessive fructose metabolism results in significant hyperlipidemia (Frayn et al., Am. J. Clin. Nutr, 1995). Unfortunately, recent studies aiming to determine if fructose (a component in HFCS) is truly a culprit in rising obesity rates commonly utilize amounts of fructose in diets or liquid which are non-physiological (some 60% of daily caloric intake)(Suga et al., Am. J. Physiol. 2000). These studies are inconclusive and raise questions about the relevance to typical human dietary habits. In addition, although HFCS (most commonly used form) is comprised of 55% Fructose and 45% Glucose, many researchers test the above hypothesis by using free fructose diets only. Again, this experimental method is flawed by the omission of glucose and is a commonly used valid argument by beverage companies in defense of their product and its effects on human metabolism. In spite of these challenges, our aim is to test the hypothesis that diets containing high fructose corn syrup result in impaired metabolism and may result in impaired metabolism of subsequent generations.