Interaction of Dietary Carbohydrate and Fat on Glucose Metabolism in Growing Pigs

Paul M. Cline
University of Georgia
Tsung Cheng Tsai
University of Georgia
Clay A. Lents
USDA-ARS Roman L Hruska US Meat Animal Research Center
Alex M. Stelzleni
University of Georgia
C. Robert Dove
University of Georgia
Michael Azain (mazain@uga.edu)
University of Georgia https://orcid.org/0000-0002-5963-7204

Research

Keywords: HFCS, fructose, insulin resistance, leptin resistance, diabetes, metabolic syndrome, grow-finish pigs

Posted Date: August 3rd, 2020

DOI: https://doi.org/10.21203/rs.3.rs-48869/v1

Version of Record: A version of this preprint was published at Domestic Animal Endocrinology on August 1st, 2021. See the published version at https://doi.org/10.1016/j.domaniend.2021.106655.

Abstract

Background: Increased consumption of fructose has been suggested to be a contributing cause of the increased rates of obesity in humans. Rodent studies have shown an increase in de novo lipogenesis and decreased insulin sensitivity in response to feeding high levels of fructose, but it is unclear if these effects occur in the same progression in humans. We aimed to develop a swine model for studying changes in glucose metabolism and insulin resistance resulting from dietary carbohydrate alone or in combination with high dietary fat. Two experiments were conducted to determine if the source of dietary carbohydrate, with or without added fat, had an effect on body weight gain, glucose metabolism, or insulin response in growing pigs.

Results: In the first experiment, pigs (24 barrows, initial body weight 28 kg) were fed one of four diets in which the source of carbohydrate was varied: 1) 20% starch; 2) 10% glucose + 10% starch; 3) 10% fructose + 10% starch; and 4) 20% fructose for 9 weeks. There were no differences in growth rate or glucose clearance observed. Experiment 2 was conducted as a 3 x 2 factorial with the main effects of carbohydrate source (20% starch, glucose, or fructose) and fat level (0 vs 10%). Pigs (24 barrows, initial body weight 71 kg) were fed one of six experimental diets for 9 weeks. Compared to the other dietary treatments, pigs fed fructose with high fat had an elevated glucose area under the curve during the GTT (Carbohydrate x Fat interaction, $P < 0.01$). This same group had a lower insulin response (Carbohydrate x Fat, $P < 0.05$).

Conclusions: This work demonstrates that pigs can be a viable model to assess the long-term effects of dietary carbohydrates on metabolism and body composition. Studies of longer duration are needed to determine if these changes are indicative of insulin resistance.

Background

The increase in the incidence of obesity in the US coincides with the increase in consumption of high fructose corn syrup (HFCS) and has lead researchers to suggest that there may be an association [1]. The average fructose consumption in the united states is 50 g/d or 10–11% of the total daily energy intake [2]. However, most published research on the effect of fructose consumption in rodents focuses on high levels of fructose in the diet, usually greater than 20% of the total daily energy intake. Although these studies may not be directly comparable, they have found that the intake of fructose at high levels leads to elevated hepatic triglyceride (TG) production, fat gain, and insulin resistance [3–5]. When levels of fructose are fed below 15% of the total energy intake, there is no increase in TGs or fat gain, however in some experiments there is an increase in insulin sensitivity [6, 7].

The consumption of fructose has been shown to increase total energy intakes t [8]. There have been two theories proposed on why this may be the case. Fructose is a highly sweet and enjoyable taste, which could make people consume more food when it contains fructose versus the same food without the fructose [9]. The second theory is that fructose does stimulate insulin release and thus there is no satiety signal [8].

It has been suggested that fructose consumption may be beneficial because of it has a low glycemic index [10]. Due to the low glycemic index of fructose (20 ± 5), the consumption of fructose may improve glucose tolerance in individuals with poor glucose tolerance or diabetes [11]. Because fructose is sweeter than sucrose or glucose, less fructose is required to achieve similar sweetness and therefore fewer total calories will be consumed.

The most common approach for investigating the effects of fructose on human health is to add fructose to the diet without adjusting the total caloric intake. Stanhope and Havel [12] conducted a 10 week study where obese men and women were asked not to change their diets, but were asked to consume glucose or fructose daily at 25% of their energy requirement for weight maintenance. Weight gain was similar in the two groups, however, those in the high fructose group had increased fasting glucose and insulin and decreased insulin sensitivity.

Studies of the effects of fructose in rodents may circumvent some of the problems in studies conducted in humans, but may also introduce other issues. The use of rodents as the models for the effects of dietary carbohydrates has limitations that may be circumvented by the use of pigs. While there are differences in the growth rate and life span of pigs compared to humans, they are more similar metabolically than are rodents [14]. More specifically, the metabolism of lipoproteins, the digestion and absorption of

nutrients, the gastro-intestinal tract and organs, and the anatomy of the central nervous system and brain in pigs are similar to humans, which makes them a better model for studying nutrient metabolism and resistance to leptin and insulin [15].

The objective of the following experiments was to understand changes in glucose metabolism and insulin sensitivity in swine consuming diets with high fructose. Studies on the effects of long-term consumption of fructose by swine are limited. The hypothesis that consuming a diet containing purified monosaccharide at 10 or 20% of the diet impairs insulin response to a glucose tolerance test and to a meal was tested in the first experiment. The response of blood metabolites to dietary treatments were monitored. The hypothesis that dietary carbohydrate interacts with fat to affect glucose tolerance and metabolism was tested in the second experiment.

Methods

All experiment procedures used in these studies were approved by the University of Georgia Institutional Animal Care and Use Committee. Experiments were conducted in the Large Animal Research Unit at the University of Georgia. Pigs (PIC 280 sire x C42 dams, PIC Franklin KY) were individually housed in an environmentally controlled room at 25°C (±2) with a 12 hr light/dark cycle (Lights on 0600 to 1800 h) in pens with a woven wire floor (1.83m x 4.27m). All diets met or exceeded the nutrient requirements of the animals [17]

Exp 1

Twenty-four barrows were blocked by initial body weight (28.3 ± 1.3 kg) and randomly allotted to 1 of 4 dietary treatments. The experimental diets were created using the same basal ingredients (corn, soybean meal and distillers dried grains with solubles (DDGS)). The DDGS was added in the diet at 43.87% of the total diet which accounted for 44% of the calories in the diet. The starch content of DDGS is less than 10% as compared to corn, which has greater than 60% starch. The experimental diets were as follows: 1) starch (20% added starch), 2) glucose (10% glucose + 10% starch), 3) low fructose (10% fructose + 10% starch) and 4) high fructose (20% fructose) (**Table 1**). Experimental diets were fed for a total of 9 weeks. Pigs had ad libitum access to feed and water during the first 7 weeks of the experiment and then were meal fed the last 2 weeks to consume two meals at 0600 to 0630 h and 1600 to 1630 h. Digestibility of the diets was determined by the addition of 0.2% titanium oxide as an indigestible marker. Individual body weights and feed intake were recorded weekly during the first 7 weeks. Ultrasound (week 7) was used to provide a non-invasive measure of body composition and change over time and was used to determine subcutaneous fat thickness and loin area at the 10^{th} rib [18]. Ultrasound sound images were collected and interpreted using swine image analysis software (Designer Genes Technologies, Inc, Harrison Ark.).

On week 8 after the start of dietary treatments, pigs were fitted with intravenous jugular catheters [19]. Catheters were checked twice daily for patency and were maintained with heparinized saline (50 units of heparin/ml, 0.018g NaCl/ml, and 1,000 units of penicillin/ml) adjusted to a pH of 7.4. An intravenous glucose tolerance test (i.v.GTT) and a response to meal test (RTM) were completed on week 9 (4 and 7 d after placement of catheters respectively). Pigs were fasted for 16 hr prior to the start of each test. Duration and time intervals between the blood collections were adapted from procedures of Campbell et al. [20]. For the i.v.GTT, baseline samples were drawn 6 and 3 minute prior to glucose infusion (0.5 g glucose/kg body weight). Samples were then obtained every 3 minutes for the first 18 minutes post infusion, then every 5 minutes for the next 30 minutes and then every 10 minutes for the next 20 minutes and finally every 15 minutes for the next 30 minutes, for a total post-infusion collection period of 98 minutes. The RTM test was completed by presenting food to the pigs for 30 minute and then removing the food for the remainder of the blood collection. The amount of the food that was consumed was recorded for each pig. Blood was collected 6 and 3 minutes prior to presentation of food to obtain baseline values. After food was given to the pigs, blood was collected every five minutes for the first 60 minutes, then two 10-minute collections and finally two 15-minute collection for a total blood collection from baseline of 110 minutes. Blood samples were centrifuged (1,200 x *g* at 4°C for 20 min) and serum was decanted and frozen at -20°C for later analysis.

Pigs were maintained on their experimental diet for one week after the removal of the intravenous catheter and slaughtered after an overnight fast in the University of Georgia Meat Science and Technology Center for collection of carcass measurements.

Exp 2

Twenty four, individually penned barrows with an initial body weight of 71.4 (±1.2) kg and were blocked by weight and randomly allotted to 1 of 6 dietary treatments and fed experimental diets for a total of 9 weeks. Pigs had ad libitum access to feed and water during the first 7 weeks of the experiment and then were meal fed the last 2 weeks to consume two meals at 0600 to 0630 h and 1600 to 1630 h. The experimental diets were created using two basal diets containing corn, soybean meal, and distillers dried grains with solubles (DDGS) with or without added fat (**Table 2**). There were 3 carbohydrate sources: 20% starch, 20% glucose, and 20% fructose as a percent of the diet, with or without 10% added fat as a percent of the diet. The starch, glucose, and fructose accounted for 21.8% of the calories in the 0% added fat diet and 19.0% of the calories in the 10% added fat diet. The diet that with the 0% added fat had 12.7% of the calories in diet accounted for by fat and the diet with 10% added fat had 33.5% of the calories in the diet from fat. Individual intakes and body weights were recorded weekly during the first 7 weeks.

Ultrasound was measured on weeks 2 and 7 as described above. On week 8, pigs were fitted with intravenous jugular catheters as described above. Catheters were flushed twice daily using heparinized saline (50 units of heparin/ml, 0.018g NaCl/ml, and 1,000 units of penicillin/ml) adjusted to a pH of 7.4.

An i.v.GTT and a RTM test were completed on week 9 (4 and 7 d after placement of catheters respectively) using the same procedures as in Exp 1.

Pigs were maintained on their experimental diet for one week after the removal of the intravenous catheter and slaughtered after an overnight fast in the University of Georgia Meat Science and Technology Center for collection of carcass measurements.

Sample Analysis

Concentration of glucose, insulin, leptin, triglyceride (TG), cholesterol, nonesterified fatty acids (NEFA), and fructose in serum were determined. Commercial kits were used to measure glucose, cholesterol, TG, and NEFA (WAKO Chemicals USA, INC. Richmond, VA). A commercial kit was used to measure fructose (Bioassays INC., EnzyChrom Fructose Assay Kit, Hayward, CA). Total serum insulin and leptin were determined by RIA using commercially available kits (Millipore, St. Charles, MO).

Fecal samples were obtained at week 6 and week 7 and samples were pooled across weeks. Samples were oven dried at 49°C for 7 d, finely ground (1 mm screen) and stored at -80°C until analysis to determine the apparent total tract digestibility (ATTD) of the diet. Titanium in the diet and feces was determined as described by Titgemeyer et al. [21] for analysis of ATTD. Glucose concentration in the feces was determined using commercially available kit (WAKO Chemicals USA, INC. Richmond, VA). Fructose and starch concentrations were determined using commercially available kits (Bioassays INC., Hayward, CA).

Statistical Analyses

All statistical analyses were performed using the General Linear Models procedure (PROC GLM) of SAS (SAS Institute, Inc., Cary, NC). Exp 1 was analyzed as a randomized block design with 4 treatments. Exp 2 was analyzed as a 3x2 factorial design with 3 different sources of carbohydrates and two levels of fat. Individual pig was the experimental unit in both studies. Least squares means, probabilities of differences, and standard errors of the means were obtained. Differences were considered significant at $P < 0.05$ and were assumed a trend at $P < 0.10$.

Results

Exp 1

The analyzed composition of the diet verified the test ingredients were added to the diet in the correct proportions (**Table 3**). The analyzed composition of the feces indicated that all glucose and fructose was absorbed or metabolized in the GIT (gastro intestinal tract) as they were not detectable in feces. Fecal analysis showed that starch absorption in the GIT of the pig was efficient, with fecal starch content at less than 2% on a dry matter basis.

There was no effect of diet on final body weight or average daily gain ($P > 0.10$, **Table 4**). Pigs fed glucose and high fructose diets tended to consumed 12.3% more feed than pigs fed the starch diet ($P = 0.09$). Pigs fed the starch diet tended to have the best feed efficiency ($P = 0.08$).

Loin area as measured by ultrasound averaged 31.5 cm^2 and did not differ with treatment. Pigs fed the glucose diet had a greater ultrasound estimate of fat depth (1.30 cm) compared to the other dietary treatments (1.05 cm; $P < 0.05$). However, there was no difference in subcutaneous fat thickness observed when pigs were slaughtered after 9 weeks on the test diets ($P > 0.10$). Similarly, there was no effect observed due to dietary treatment on loin eye area measured on the carcass ($P > 0.10$).

i.v.GTT

The only observed effect for blood parameters was that the pigs fed the low fructose diet tended to have the lowest value for the Glucose AUC return to baseline, which is the area under the curve from baseline glucose to when glucose returned to the baseline value ($P = 0.07$, **Table 5**). No significant effect was seen due to dietary treatments on basal glucose, basal insulin, peak glucose, peak insulin, Insulin AUC (all parameters measured), or Glucose AUC (0-9 min, 30-98 min, 0-98 min; $P > 0.10$). Serum leptin concentration did not vary with time (data not shown), and an average value (0, 9, 18, 30, 40, 60, and 98 minute samples) was determined. There was no effect of diet on serum leptin levels ($P > 0.10$).

RTM test

There was no effect of diet on basal glucose or basal insulin ($P > 0.10$, **Table 6**) in the RTM test. There were no effects of dietary treatment on insulin AUC and glucose AUC for all time periods measured which included fed period (0-30 min), post fed period (30-60 min), and total (0-110 min; $P > 0.10$). There was no effect of dietary treatment on circulating TG, cholesterol, or NEFA (Data not shown, $P > 0.10$). Serum fructose levels averaged 1.6 mg/dL prior to feeding the meal and were not different between treatments. A significant, dose-dependent effect was observed for serum fructose concentrations due to the dietary treatment with peak fructose concentrations occurring 40 minutes after feeding (1.9, 2.4, 3.7, and 8.3 mg/dl for starch, glucose, low fructose and high fructose diets respectively (**Supplemental Figure 1**, $P < 0.01$).

Exp 2

Growth performance and carcass characteristics

There was no effect of diet on final body weight or average daily gain ($P > 0.10$, **Table 7**). There were no differences in intake or feed efficiency observed in pigs fed diets containing different carbohydrates and there was no fat x carbohydrate interaction ($P > 0.10$). However, pigs fed the diets with 10% added fat consumed less feed than pigs fed diets with 0% added fat ($P < 0.05$). Feed efficiency tended to be improved for pigs consuming the 10% added fat diets ($P < 0.10$).

There was no difference in fat depth determined by ultrasound after 7 wk on the test diets. However, independent of carbohydrate source, pigs fed the 10% added fat diets had greater calculated fat accretion based on the change in fat depth from week 2 to 7 compared to pigs fed the 0% added fat diets ($P < 0.05$). Similarly, there was a main effect of dietary fat on 10^{th} rib fat thickness in the carcass after 9 wk with pigs fed 10% fat diets having a 20% increase in 10^{th} rib fat thickness ($P < 0.01$) and no change in carcass weight.

i.v.GTT

Basal glucose concentrations were not affected by diet ($P > 0.10$, **Table 8, Figure 1**). Basal insulin levels were decreased by 27.6% in pigs fed 10% added fat compared to 0% added fat ($P < 0.01$), but was not affected by carbohydrate and there was no fat x carbohydrate interaction ($P > 0.10$). Peak glucose was increased in pigs fed the 10% added fat diets compared to the pigs fed 0% added fat diets ($P < 0.05$). There was an interaction ($P < 0.04$) of fat x carbohydrate on peak glucose. Pigs that were fed diets containing starch and fructose had no differences on peak glucose between the 0% added fat diet and 10% added fat diet. The pigs that were fed the glucose diets had difference between the 10% added fat and 0% added fat diets of 419.70 and 302.38 mg glucose/dl respectively ($P < 0.05$). Peak insulin was decreased for all pigs fed 10% added fat diet compared to pigs fed 0% added fat ($P < 0.05$, **Figure 2**). An interaction ($P < 0.05$) of fat x carbohydrate was also observed for peak insulin. The pigs fed diets containing starch and glucose had no difference in peak insulin between 0% added fat and 10% added fat diets. The pigs fed diets containing fructose had a peak insulin value of 119.75 and 43.15 μU/ml for those consuming the 0% and 10% added fat, respectively ($P < 0.05$). There was a fat x carbohydrate interaction for glucose clearance. Glucose was cleared more rapidly (time to

½ peak = 25.33 min) in pigs fed fructose diets with no added fat as compared to those fed fructose with fat (42.84 min; $P < 0.05$). In contrast, there was no difference in the time to reach ½ peak in pigs fed starch or glucose, with or without added fat ($P > 0.10$).

There was a main effect of dietary fat on glucose AUC, with the addition of 10% fat in the pigs diets resulting in larger AUC 0-98 min, AUC 0-9 min, and AUC 30-98 min ($P < 0.05$). There was also a fat x carbohydrate interaction observed for glucose AUC for time periods 0-98 min and 30-98 min ($P < 0.01$). Pigs fed starch and glucose with or without fat and fructose with 0% added fat diets had the same glucose AUC for time periods 0-98 and 30-98 minutes and pigs fed fructose with 10% added fat had increased glucose AUC of 32.0% and 44.7%, respectively. There was an effect of diet on insulin AUC in pigs fed the 10% added fat diets, resulting in decreased insulin AUC 0-98 min ($P < 0.05$). There was also a fat x carbohydrate interaction observed for insulin AUC 0-9 min. The pigs fed starch and glucose with or without addition of fat and the 0% added fat fructose diet maintained the same insulin AUC 0-9 min. The pigs fed the 10% added fat fructose diet decreased their insulin AUC 0-9 min by 70.4% compared to the 0% added fat fructose fed pigs ($P=0.05$).

A fat x carbohydrate interaction was observed for the length of time for serum glucose and insulin to return to baseline. The pigs fed starch and glucose with or without addition of fat and the 0% added fat fructose diets maintained the same length of time for serum glucose and insulin to return to baseline. The pigs fed the 10% added fat fructose diet took 68.14 and 44.5 min longer for glucose and insulin to return to baseline respectively compared to pigs fed 0% added fat fructose diet ($P<0.05$).

The pigs fed 10% added fat diets decreased their insulinogenic index (Δinsulin/Δglucose) by 38.9% compared to the 0% added fat fed pigs ($P < 0.01$). A fat x carbohydrate interaction was observed with pigs fed 10% added fat fructose diet had the lowest insulinogenic value of 0.16 compared to the pigs fed the 0% added fat fructose diet that had an insulinogenic value of 0.48. No effect of time due to dietary treatments was found on leptin levels (data not shown) so an average value of leptin representing samples taken at times 0, 9, 18, 30, 40, 60, and 98 minutes is reported. There was a trend for carbohydrate in the diet to affect serum leptin concentrations with values of 3.01, 2.78, and 2.97 ng/ml corresponding to starch, glucose, and fructose fed pigs respectively ($P=0.09$).

RTM test

There was no difference in feed intake due to diet during the 30 minute feeding period ($P>0.10$, **Table 9**). Basal glucose concentrations were not affected by diet ($P>0.10$). Basal insulin was decreased by 29.1% in pigs fed 10% added fat compared to 0% added fat ($P=0.08$) but was not affected by carbohydrate or carbohydrate or fat x carbohydrate interaction ($P>0.10$). There was a trend for pigs fed fat to have a greater glucose AUC (0-110 min) as compared to pigs fed diets that were not supplemented with fat ($P=0.09$). No main effect of fat or carbohydrate or interaction was seen for glucose AUC 0-30 min ($P>0.10$). A trend for an interaction was observed for glucose AUC 30-60 min with pigs fed 10% added fat fructose diet achieving the highest glucose AUC ($P=0.08$). For serum insulin AUC 0-110 min no main effect of fat or carbohydrate or interaction was observed ($P>0.10$). A trend for a main effect of carbohydrate was observed for serum insulin AUC 0-30 min. The glucose fed pigs had the highest insulin AUC and no difference between starch and fructose fed pigs was observed ($P=0.07$). A fat effect was observed for serum insulin AUC 30-60 min with pigs fed 10% added fat had decreased AUC by 45.9% compared to the 0% added fat pigs ($P < 0.05$).

Lipid metabolites in the serum were measured during the RTM test including TG, cholesterol, and NEFA. No effects were detectable due to dietary treatments ($P>0.10$,). An effect was seen on serum fructose concentration due to dietary treatment, with peak fructose concentrations of 1.68, 0.70 and 5.56 mg/dl for starch, glucose, and fructose fed pigs respectively ($P < 0.01$, **Figure 3**). Pigs consuming the fructose diets had a peak fructose concentration that was 5.8% of their basal glucose concentration.

Discussion

The objective of the first experiment was to determine if the consumption of a diet high in fructose in pigs would lead to excess lipid deposition and impaired glucose clearance. Multiple reviews have suggested that the increased consumption of high fructose corn syrup in humans can be correlated to the increased obesity epidemic occurring in developed nations [1, 22]. However, at least some epidemiological research has disputed the relationship of sugar consumption to obesity [23]. There is a potential metabolic basis for fructose consumption to result in increased lipid deposition. Basciano et al [24] suggested that the basis for the effects of fructose may be related to differences in how fructose is metabolized relative to glucose, and in particular, the effects on the

phosphofructokinase step in glycolysis. The ability of fructose to by-pass this enzyme allows fructose to be metabolized into glucose, glycogen, lactate, pyruvate, and TG with less regulation than would occur with glucose. While all metabolites were not measured in this study, we observed no changes in baseline glucose, insulin, leptin, TG, cholesterol or NEFA in response to feeding fructose.

There were no differences in final body weight of pigs in this study due to the inclusion of purified monosaccharide in the diets and no evidence of a disruption of glucose metabolism or increased lipid deposition. There are limited studies on the effects of feeding fructose to normal pigs. Waterman et al. [25] examined the effects of high fructose (rats and chicks) or sucrose (pigs) and found that diets with 60% fructose increased liver lipogenesis and circulating TG in rats, but had no effect on the rate of lipogenesis in adipose tissue and no effect on fat pad weights. There were no effects of high fructose diets on body weight gain, feed intake or liver lipogenesis in chicks. There were also no effects of feeding diets with 60% sucrose in growing pigs. Several studies were conducted to examine the effect of high fructose diets in the sow during late gestation and lactation. These studies showed minimal effects of fructose. For example, Coffey et al. [26] reported no effects of the high fructose diet in sows or their progeny. Campbell et al [20] reported no differences in glucose tolerance or insulin release in sows fed diets with 28% HFCS as compared to those fed glucose.

Ossabaw pigs are unique from modern commercial lines of pigs as they have a genotype that predisposes them to diet-induced obesity [27]. Growing Ossabaw pigs fed low fat (10% of calories) diets with 20% of the calories from fructose, had greater feed intake and body weight gain as compared to pigs fed a high starch (chow) diet [28]. Peak insulin levels were 55% greater in the pigs fed the high fructose diet vs the chow group indicating insulin resistance. Fasting glucose, insulin and TG levels were not changed. Although, not reported, it was assumed that the additional gain was body fat. The interpretation of this work is confounded by that differences in daily caloric intake. Pigs on the chow diet consumed 2500 kcal as compared o 6000 kcal in the high fructose diet. In a more recent study [29], Ossabaw pigs fed isocaloric amounts of a high starch (47%) vs high sucrose (47%) for 6 months had no difference in growth rate, body fat, glucose, insulin or TG. There were differences in cholesterol and lipoprotein levels, but these were likely attributed to differences in fat sources between the starch and sucrose diets.

Rodent models, particularly the rat, have been used extensively in the study of the effects of dietary fructose with conflicting results on whether fructose induces obesity. In some studies, rats fed fructose containing diets have been shown to exhibit increased feed intake and body weight gain in the form of adipose tissue, but this is not universally observed. Differences in the level of fructose in the diet, the age, strain and sex of the animals and the duration of the studies all likely contribute to these differences [see 30, 31 for review]. Dietary fructose levels in these studies ranged from 20 to 60% of the calories. In the current pig study, fructose was included at 21.8% of the total kcal in the diet, which more closely approximates consumption of fructose in human diets. Per capita consumption of high fructose corn syrup in the US has declined from 28 kg in 2000 to 17 kg in 2018 [32]. Assuming a total energy intake of 2500 kcal per day, the expected calories from fructose for the average person would be 12.3 to 7.5% of total caloric consumption over this period.

In humans, some studies show that over-consumption of calories associated with sweeteners over a long period of time leads to excess weight gain [9, 33, 34]. In the present work, there was a trend for pigs fed the glucose or high fructose diets to consume more energy than those fed the starch diet ($P = 0.09$), but there were no differences in body weight gain and no consistent evidence of greater fat accumulation. Thus, feeding pigs a diet that included simple sugars at 21.8% of the total kcal of the diet for 9 weeks was not sufficient to change fat deposition or final body weight.

In the second experiment, the effects of high fructose in combination with a high fat diet was investigated. Calories from fat in the diet with 10% added fat were in the range of 33% and thus, more similar to Western diets [9]. As in the first experiment, there were no effects of carbohydrate on growth rates or feed intake. However, as expected [35] the greater caloric density of diets with added fat resulted in reduced feed intake and greater fat accumulation. There were no observed differences in basal glucose, basal insulin, insulin AUC, glucose AUC, leptin, cholesterol, TG, or NEFA serum concentrations in pigs consuming fructose and fat in the ranges described in the current experiments. Pigs fed glucose and high fructose diets took 3 minutes longer for their serum glucose values to return to baseline after glucose bolus. The lack of differences between pigs fed high fructose and starch diets is most likely due to the moderate levels of sugars fed in this study as compared to levels used in rodents [30, 31]. The pigs fed the high fructose diet had peak serum fructose levels of 0.5 mmol/L those fed the low fructose diet had 0.5 mmol/L during the RTM test.

Fructose levels have been measured in portal vein of rats at 1.4 mmol/L when fed a diet with 68% of the total kcal as fructose and humans with a portal vein concentration of 1.0 mmol/L when given a bolus of food containing a large amount of fructose [36] Fructose levels in the peripheral blood are generally low due to the livers ability to remove 70% of fructose from the portal vein [37] and the kidney's ability to remove an additional 20% of circulating fructose [38].

The negative metabolic effects of feeding fructose at high levels (20% of the diet) were not observed in the first experiment. Therefore, the second experiment was performed to determine if adding extra calories in from fat (soybean oil) at 10% of the diet would create the positive energy status needed to see the increased fat deposition, elevated serum triglycerides and impaired GTT shown in some rodent and human studies [39]. No difference in final body weight was observed in pigs fed added fat or fed a different source of carbohydrate. However, there was a difference in average daily feed intake and feed efficiency. The pigs fed the 10% added fat diet consumed less feed and were more feed efficient. This is not surprising a pigs and other animals, eat to meet their caloric requirements to maximize their genetic potential for growth [40]. Therefore, the pigs fed diets with added fat consumed less feed to meet their caloric requirement. Although pigs in the current study that were fed 10% added fat diet, they consumed only 4.7% greater energy per day compared with pigs fed 0% added fat. Consequently, the pigs fed the 10% added fat diets had increased accumulation of adipose tissue. There were no effects of carbohydrate source on ultrasound measurements or carcass measurements. The increased fat deposition seen on the ultrasound estimate and carcass measurement was expected as previous research shows feeding a diet containing added fat above 6% to commercial pigs results in increased lipid deposition [41–43].

Results from the i.v.GTT showed that the pigs fed the 10% added fat diets had lower basal insulin levels, lower peak insulin levels, and higher peak glucose levels. Pigs fed the 10% added fat with fructose diets had peak insulin that was half that of glucose and starch fed pigs on the high fat diet and a third of the 0% added fat-fructose fed pigs. There are other reports of changes in glucose and insulin dynamics in Ossabaw pigs fed high fat diets and fructose, but these studies are confounded by large differences in caloric intake between low fat and high fat groups (28, 44). In rat studies, hyperinsulinemic conditions and decreased glucose clearance were seen after rats were fed a 15% fructose solution for 6 weeks [45] and when high fructose diets (66% of total kcal of diet) were fed for 2 weeks [46]. In the current study, the altered insulin secretion and decreased glucose clearance rate during the GTT in pigs fed the high fructose / high fat diet cannot be definitively described as insulin resistant, but clearly, those animals had a diminished ability to remove glucose from the peripheral circulation. The low insulinogenic index of pigs fed 10% added fat fructose diet suggests that they were the most responsive to insulin. The insulinogenic index value should be interpreted with caution since it is calculated by the change in serum insulin over plasma glucose. If the pigs had a small change in the serum insulin levels, such as that observed in the pigs fed the 10% fat fructose diet, their insulinogenic index value will automatically be larger. The change in serum insulin values need to be considered when discussing insulinogenic index values. In a 20 week Ossabaw pig study [47], it was reported that pigs fed a 46% fat diet without the addition of purified sugars did not exhibit insulin resistance or glucose intolerance. Therefore, the addition of fat alone does not create the detrimental effects on glucose clearance seen in pigs. There was a trend for serum leptin to be elevated in response to pigs fed the starch diets compared to pigs fed the glucose diets. However, the changes in leptin levels were small and not likely of physiological significance.

Results from the RTM test demonstrate the metabolic changes that occur in the pig due to the consumption of fat and different carbohydrates. Previous research has shown that fructose consumption does not result in insulin secretion, and the pancreas has no role in regulating serum fructose levels [37–38]. The inability of the pancreas to respond to fructose is likely accounted for by the lack of GLUT 5 receptors, which would be needed to transport fructose into the cell [48, 49]. This is supported by the observation in the current experiment that the total insulin AUC was 45.5% greater in pigs fed the high fat glucose diets as compared to those fed the high fat fructose diet. However, the pigs that consumed the starch diets had the same total insulin AUC as the pigs that consumed the fructose diets. This was most likely due to the lower glycemic index of starch [10, 50]. There was a spike in insulin secretion in the 0% added fat fructose fed pigs, which may be attributed to the ability of the liver to convert some fructose to glucose and then secrete the glucose into the peripheral blood [37, 51]. The addition of 10% fat to the pigs fed the fructose diet did not result in an increase in peripheral glucose levels or a rise in insulin levels. It can be hypothesized that the addition of fat to the diet of these pigs disrupted the ability of their livers to convert fructose to glucose.

Lipid metabolites were measured in the peripheral blood during the RTM test to determine the metabolic changes that occurred after feeding the experimental diets. No effect of diet on serum TG levels was observed. Serum cholesterol levels at 60 minutes post prandial were affected by diet. Pigs fed the 0% added fat glucose and fructose diets and pigs fed 10% added fat starch diets

had the highest post prandial cholesterol levels compared to the other dietary treatments. In a study that compared changes in onset of metabolic syndrome in Ossabaw, researchers found that cholesterol was elevated in pigs fed a high fat diet as compared to those fed a low fat diet There was no corresponding increase in blood TG [44].

Comparing previous research to the current research needs to be done with caution. In the current experiments, fructose was added to the diet at 20% of the diet and fat at 10% of the diet which is more representative of a human consuming a high fructose, Western diet [1]. The lack of response to in the current studies may be accounted for by the relatively moderate levels of fructose and fat that were included in the diets and also by the short duration that the diets were fed. It is also important to consider feed intake differences in some previous studies. In the present work, feed intake was not affected by the source of carbohydrate.

Conclusion

The high level of fat in Westernized diets, and the consumption of fructose in many hypercaloric foods, creates a detrimental metabolic state that may lead to insulin resistance and type 2 diabetes, which is prevalent in Westernized societies. More studies need to be completed to determine the metabolic changes that occur when pigs are fed the 10% added fat / fructose diet that results in a decrease in insulin secretion after a glucose challenge. More specifically, liver metabolism needs to be more closely examined to determine if hepatic insulin resistance is occurring and if lipid deposition is increased in pigs for the 10% added fat fructose diet. The glucose intolerance seen when a high fat-fructose diet was fed to the pigs warrants further investigation.

This research demonstrates the utility of the pigs as an animal model to study the impact of diet on metabolism and body composition and to investigate the increase occurrence of type 2 diabetes and obesity. The use of pigs allows researchers to obtain a greater number of blood samples that cannot be completed using rodents as the animal model, which would allow for a more in-depth study of metabolism changes that occur during the induction of insulin resistance and leptin resistance. Due to similarities of nutrient metabolism between pigs and humans, the use of the pig is be more relevant in determining metabolism changes that are seen in humans.

Declarations

Declarations

Ethics approval and consent to participate

Studies were conducted at the University of Georgia with the approval of the University of Georgia Institutional Animal Care and Use Committee.

Consent for publication

Not applicable.

Availability of data and material

All data generated or analyzed during this study are available from the corresponding author upon reasonable request

Competing interests

The authors declare that they have no competing interests.

Funding

Funding for this research came from NIFA-Hatch project: GEO00767.

Authors' contributions

The work was mainly conceived and designed by PMC and MA. Experimental data were collected and analyzed by PCM, TCT. Ultrasound images were obtained and analyzed by AMS. Placement of jugular cannulas was done by CAL, PCM, TCT. The manuscript was mainly written by PCM and MA, and revised by AMS, CRD and CAL. All the authors contributed to the writing, reading and approval of the final manuscript.

Acknowledgements

Not applicable.

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Tables

Table 1. Composition of Experimental Diets (As-fed basis) **(Experiment 1)**[1]

Ingredient	Starch	Glucose	Low Fructose	High Fructose
Corn	20.00	20.00	20.00	20.00
DDGS	43.87	43.87	43.87	43.87
Soybean Meal	13.69	13.69	13.69	13.69
Starch	20.00	10.00	10.00	0.00
Glucose	0.00	10.00	0.00	0.00
Fructose	0.00	0.00	10.00	20.00
Limestone	1.79	1.79	1.79	1.79
Salt	0.35	0.35	0.35	0.35
Vitamin premix [a]	0.15	0.15	0.15	0.15
Mineral premix [b]	0.15	0.15	0.15	0.15
Calculated Composition				
ME, Mcal/kg	3.31	3.31	3.31	3.31
CP, %	20.15	20.15	20.15	20.15
Ether Extract, %	5.15	5.15	5.15	5.15
SID Lysine, %	0.81	0.81	0.81	0.81
SID Lys:ME %	1.89	1.89	1.89	1.89
Ca:P	1.68	1.68	1.68	1.68

[1] A total of 24 barrows (Pig Improvement Co., Franklin, KY; 280 x C42) with initial body weight of 28.3 kg were individually housed and fed 1 of 4 dietary treatments for 9 week trial.

[a] The vitamin premix (Animal Science Products, Nacogdoches, TX) provided the following per kilogram of complete diet: 11000 IU vitamin A, 1650 IU vitamin D_3, 44 IU vitamin E, 4.4 mg vitamin K, 9.9 mg riboflavin, 55 mg niacin, 33 mg pantothenic acid, 44 ug

vitamin B_{12}.

[b] The trace mineral premix (Animal Science Products, Nacogdoches, TX) provided the following per kilogram of complete diet: 165 mg Fe ($FeSO_4 \cdot H_2O$), 16.5 mg Cu ($CuSO_4 \cdot 5H_2O$), 39.6 mg Mn ($MnSO_4$), 165 mg Zn (ZnO), 0.3 mg I ($Ca(IO_3)_2$), and 0.3 mg Se (Na_2SeO_3).

Table 2. Composition of Experimental Diets (As-fed basis) **(Experiment 2)** [1]

Ingredient	0% Added Fat	10% Added Fat
Corn	20.00	10.00
DDGS	43.87	43.87
Soybean Meal	13.69	13.54
Fat	0.00	10.00
Carbohydrate[a]	20.00	20.00
L-Lysine	0.00	0.15
Limestone	1.79	1.79
Salt	0.35	0.35
Vitamin [b]	0.15	0.15
Mineral [c]	0.15	0.15
Calculated Composition		
ME, Mcal/kg	3.31	3.79
CP, %	20.15	19.40
Ether Extract, %	5.15	15.50
SID Lysine, %	0.81	0.90
SID Lys:ME %	1.89	1.90
Ca:P	1.68	1.78

[1] A total of 24 barrows (Pig Improvement Co., Franklin, KY; 280 x C42) with initial body weight of 71.4 kg were individually housed and fed 1 of 6 dietary treatments for 9 week trial.

[a] Starch, Glucose, Fructose was added for carbohydrate.

[b] The vitamin premix (Animal Science Products, Nacogdoches, TX) provided the following per kilogram of complete diet: 11000 IU vitamin A, 1650 IU vitamin D_3, 44 IU vitamin E, 4.4 mg vitamin K, 9.9 mg riboflavin, 55 mg niacin, 33 mg pantothenic acid, 44 ug vitamin B_{12}.

[c] The trace mineral premix (Animal Science Products, Nacogdoches, TX) provided the following per kilogram of complete diet: 165 mg Fe ($FeSO_4 \cdot H_2O$), 16.5 mg Cu ($CuSO_4 \cdot 5H_2O$), 39.6 mg Mn ($MnSO_4$), 165 mg Zn (ZnO), 0.3 mg I ($Ca(IO_3)_2$), and 0.3 mg Se (Na_2SeO_3).

Table 3. Analyzed composition of the diets (as-fed), feces, and ATTD of the diet. **(Experiment 1)** [1] [2]

Item	Starch	Glucose	Low Fructose	High Fructose
Feed (as-fed)				
Dry Matter, %	89.5	90.2	90.3	89.3
Glucose, %	1.1	11.1	1.2	1.1
Fructose, %	0.3	0.3	9.1	19.1
Starch, %	28.7	20.1	20.1	12.2
Ash, %	4.8	4.6	4.5	4.6
Titanium, %	0.2	0.2	0.2	0.2
Fecal (dry matter basis)				
Glucose/Fructose	Nd [3]	Nd	Nd	Nd
Starch, %	1.5	1.4	1.5	1.4
Ash, %	9.5	9.7	9.5	8.7
Titanium, %	1.3	1.4	1.4	1.4
ATTD [2]				
Dry Matter, %	94.3	94.6	94.2	94.0
Starch, %	99.2	98.9	98.9	98.2
Glucose/Fructose, %	100.0	100.0	100.0	100.0
Ash, %	69.6	67.7	67.5	70.9

[1] A total of 24 barrows (Pig Improvement Co., Franklin, KY; 280 x C42) with initial body weight of 28.3 kg were individually housed and fed 1 of 4 dietary treatments for 9 week trial. To determine analyzed composition of the diet 3 grab samples were analyzed and compiled together.

[2] ATTD is defined as apparent total tract digestibility.

[3] ND is defined as not detected.

Table 4. Growth performance of pigs fed 4 different diets containing different sources of carbohydrates for 9 weeks. **(Experiment 1)**[1]

Item	Starch	Glucose	Low Fructose	High Fructose	SEM	P-value
Body weight (wk 0), kg	28.8	28.0	28.5	28.0	1.3	0.89
Body weight (wk 9), kg	79.0	80.9	79.6	80.9	3.0	0.25
ADG, (wk0-wk9), gm	800.0	840.0	810.0	840.0	40.0	0.16
ADFI, (wk0-wk9), gm	1850.0	2110.0	1950.0	2110.0	130.0	0.09
Efficiency (wk0-wk9),gm/gm [a]	0.43	0.40	0.42	0.40	0.01	0.08
Ultrasound at 10th rib [b]	1.02 [y]	1.30 [y]	1.08 [y]	1.05 [y]	0.08	0.05
Fat depth, cm	30.03	31.55	32.67	31.66	2.38	0.76
Loin area, cm^2						
Carcass Characteristics [c]	1.27	1.52	1.44	1.35	0.18	0.72
10th Rib Fat thickness, cm	41.29	34.84	37.53	34.41	2.09	0.18
Loin area, cm^2						

[1] A total of 24 barrows (Pig Improvement Co., Franklin, KY; 280 x C42) with initial body weight of 28.3 kg were individually housed and fed 1 of 4 dietary treatments for 9 week trial.

[a] Efficiency is defined as gm gain / gm of feed intake.

[b] Ultrasound images obtained after 7 weeks on test diets.

[c] Carcass characteristics determined after 7 weeks on test diets.

[y z] Superscripts denote significant differences between diets (P<0.05).

Table 5. Change in blood parameters from the i.v.GTT in pigs fed 4 different diets containing different sources of carbohydrates for 9 weeks. **(Experiment 1)**[1]

Item	Starch	Glucose	Low Fructose	High Fructose	SEM	P-value
Basal Glucose, mg/dL	59.8	57.3	62.2	63.9	7.5	0.61
Basal Insulin, µU/mL	6.4	3.6	5.8	6.8	3.0	0.56
Peak Glucose, mg/dL	415.9	458.7	429.9	431.5	51.0	0.41
Peak Insulin, µU/mL	96.3	83.2	99.2	70.9	31.3	0.56
Insulin AUC (µU/mL per min)						
0-9 min	578.6	474.4	636.7	472.9	107.4	0.57
30-98 min	649.1	477.6	505.5	668.0	89.6	0.29
0-98 min	2,460.4	1.872.8	2,190.8	2,020.5	403.8	0.71
Glucose AUC (mg/dL per min)						
0-9 min	3,131.0	3,239.8	3,067.6	3,149.4	113.6	0.73
30-98 min	3,342.9	3,252.5	3,602.9	3,636.3	219.4	0.47
0-98 min	9,625.0	9,265.6	9,322.9	9,715.2	261.2	0.47
Glucose return to baseline (min)	31.5 ^{z}	28.4 ^{yz}	27.9 ^{y}	31.3 ^{z}	0.9	0.07
Insulin return to baseline (min)	44.6	44.1	44.1	43.9	2.6	0.99
Insulinogenic Index (ΔI/ΔG)	0.25	0.19	0.26	0.18	0.1	0.31
Leptin, ng/ml	2.2	2.4	2.4	3.1	0.5	0.59

[1] A total of 24 barrows (Pig Improvement Co., Franklin, KY; 280 x C42) with initial body weight of 28.3 kg were individually housed and fed 1 of 4 dietary treatments for 9 week trial.

$^{x\,y\,z}$ Superscripts denote significant differences between diets (P<0.05).

Table 6. Change in blood parameters from the RTM test in pigs fed 4 different diets containing different sources of carbohydrates for 9 weeks. **(Experiment 1)**[1]

Item	Starch	Glucose	Low Fructose	High Fructose	SEM	P-value
Baseline Glucose, mg/dl	74.6	72.6	75.4	75.7	3.6	0.91
Baseline Insulin, µU/ml	5.6	5.2	6.8	7.0	1.2	0.63
Insulin AUC (µU/ml per min)						
Total (0 – 110 min)	2,038.4	2,284.5	2,338.4	1,717.2	298.9	0.38
Fed (0-30 min)	488.2	948.2	802.4	571.6	154.6	0.15
Post Fed (30-60 min)	801.5	653.0	790.2	541.0	106.7	0.21
Glucose AUC (mg/dl per min)						
Total (0-110 min)	10,334.4	10,683.3	10,144.2	9,889.4	344.9	0.40
Fed (0-30 min)	2,883.5	3,278.8	3,157.9	2,933.1	134.4	0.14
Post fed (30-60 min)	3,334.4	2,964.6	3,241.3	2,964.6	3,150.8	0.20

[1] A total of 24 barrows (Pig Improvement Co., Franklin, KY; 280 x C42) with initial body weight of 28.3 kg were individually housed and fed 1 of 4 dietary treatments for 9 week trial.

Table 7. Growth characteristics of pigs due to feeding 0% added fat or 10% added fat in combination with three different carbohydrate sources to swine for 9 weeks. **(Experiment 2)**[1]

	0% Added Fat			10% Added Fat				P-value		
Parameter	**Starch**	**Glucose**	**Fructose**	**Starch**	**Glucose**	**Fructose**	**SEM**	**Fat**	**Carb[2]**	**Fat x Carb**
Body weight (d 0), kg	71.4	71.6	71.1	71.1	71.5	71.9	1.2	0.91	0.97	0.88
Body weight (wk 7), kg	121.3	118.3	119.2	116.9	117.8	116.4	6.4	0.29	0.89	0.83
ADG (0–7 wk), kg/d	1.02	0.95	0.98	0.93	0.95	0.91	0.07	0.34	0.89	0.83
ADFI (0-7 wk), kg/d	2.74	2.64	2.70	2.38	2.47	2.52	0.14	0.05	0.90	0.76
Efficiency (0-7wk), gm/gm[a]	0.37	0.36	0.36	0.39	0.38	0.39	0.01	0.10	0.12	0.45
Ultrasound at wk 7										
Fat depth, cm	1.17	1.21	1.35	1.38	1.28	1.33	0.13	0.37	0.71	0.60
Change wk 2-7 , cm[b]	0.02	0.08	0.13	0.27	0.22	0.20	0.09	0.05	0.96	0.57
Carcass characteristics[c]										
Carcass weight, kg	97.3	97.4	97.4	99.5	102.1	97.7	3.9	0.41	0.81	0.81
10^{th} Rib fat thickness, cm	2.29	2.00	2.79	2.86	2.98	3.05	0.24	0.01	0.15	0.27

[1] A total of 24 barrows (Pig Improvement Co., Franklin, KY; 280 x C42) with initial body weight of 71.44 kg were individually housed and fed 1 of 6 dietary treatments for 9 week trial. Body weight and intake were recorded for the first 7 weeks.

[2] Carb is defined as the main effect of carbohydrate source in the diet (starch, glucose, fructose).

[a] Efficiency is defined as gm gain / gm of feed intake.

[b] Change in fat thickness determined by the difference between wk7 – wk2.

[c] Carcass characteristics determined after 9 wk on the test diets.

	0% Added Fat			10% Added Fat				P-value		
Parameter	**Starch**	**Glucose**	**Fructose**	**Starch**	**Glucose**	**Fructose**	**SEM**	**Fat**	**Carb[2]**	**Fat x Carb**
Basal Glucose, mg/dL	94.2	91.9	95.1	96.7	96.4	93.3	3.7	0.52	0.91	0.63
Basal Insulin, uU/mL	4.4	4.5	4.6	3.2	3.6	2.9	0.7	0.01	0.84	0.77
Peak Glucose, mg/dL	410.5^{xy}	302.4^{x}	339.2^{xy}	390.3^{yz}	419.7^{z}	372.0^{xyz}	28.3	0.05	0.18	0.04
Peak Insulin, uU/mL	75.0^{x}	88.3^{xy}	119.8^{y}	75.8^{x}	82.3^{xy}	43.2^{x}	18.9	0.04	0.78	0.05
Time ½ peak, min[a]	25.5^{x}	31.4^{xy}	25.3^{x}	25.0^{x}	20.1^{x}	42.8^{y}	5.3	0.63	0.15	0.03
Glucose AUC, mg/mL per min										
0-98 min	$13,272.8^{x}$	$12,485.5^{x}$	$12,148.2^{x}$	$13,034.8^{x}$	$13,729.2^{x}$	17.874.1y	1,015.6	0.01	0.09	0.01
0-9 min	2,127.8	2,111.7	2,257.2	2,699.8	3,196.7	2,145.5	318.7	0.04	0.31	0.14
30-98 min	$7,306.8^{x}$	$7,130.3^{x}$	$6,369.3^{x}$	$6,986.3^{x}$	$7,086.3^{x}$	$11,514.7^{y}$	880.8	0.02	0.06	0.01
Insulin AUC, uU/mL per min										
0-98 min	2,629	3,081	3,215	1,985	2,137	1,949	671	0.04	0.80	0.84
0-9 min	443^{xy}	484^{xy}	674^{y}	469^{xy}	467^{xy}	199^{x}	126	0.07	0.93	0.05
30-98 min	1,241	1,530	1,070	663	702	1,204	445	0.16	0.83	0.42
Glucose return to baseline, min	42.8^{x}	46.5^{x}	35.8^{x}	42.9^{x}	41.2^{x}	103.9^{y}	9.7	0.01	0.01	0.01
Insulin return to baseline, min	51.3^{x}	54.7^{x}	53.5^{x}	66.8^{x}	56.0^{x}	98.0^{y}	9.1	0.01	0.04	0.04
Insulinogenic index ($\Delta I/\Delta G$)[b]	0.23^{xy}	0.38^{yz}	0.48^{z}	0.25^{x}	0.24^{xy}	0.16^{x}	0.08	0.01	0.33	0.04
Leptin, ng/ml	3.0	2.7	2.9	3.0	2.9	3.1	0.1	0.33	0.09	0.61

Table 8. i.v.GTT Change in blood parameters due to feeding 0% added fat or 10% added fat in combination with three different carbohydrate sources to swine for 9 weeks. **(Experiment 2)**[1]

[1] A total of 24 barrows (Pig Improvement Co., Franklin, KY; 280 x C42) with initial body weight of 71.44 kg were individually housed and fed 1 of 6 dietary treatments for 9 week trial.

[2] Carb is defined as the main effect of carbohydrate source in the diet (starch, glucose, fructose).

[a] The amount of time it took for concentration of glucose to return to half peak concentration.

[b] ΔI/ΔG, Change in insulin (peak insulin – basal insulin) ÷ change in glucose (peak glucose – basal glucose).

[x y z] Superscripts denote significant differences between diets (P<0.05).

Table 9. RTM test Change in blood parameters due to feeding 0% added fat or 10% added fat in combination with three different carbohydrate sources to swine for 9 weeks. **(Experiment 2)**[1]

	0% Added Fat			10% Added Fat				P-value		
Parameter	**Starch**	**Glucose**	**Fructose**	**Starch**	**Glucose**	**Fructose**	**SEM**	**Fat**	**Carb[2]**	**Fat x Carb**
Basal Glucose, mg/dL	89.1	91.3	97.7	98.0	98.2	94.6	3.9	0.16	0.77	0.23
Basal Insulin, uU/mL	7.9	8.5	9.1	5.3	7.4	5.3	1.8	0.08	0.71	0.70
Glucose AUC, min*mg*dL										
Fed (0-30 min)	3,121	3,222	3,173	3,093	3,377	3,127	106	0.74	0.13	0.51
Post Fed (30-60 min)	3,191	2,939	2,940	3,055	3,205	3,200	104	0.11	0.82	0.08
Total (0-110 min)	10,295	10,332	10,014	10,160	11,126	10,558	307	0.09	0.17	0.20
Insulin AUC, min*uU*mL										
Fed (0-30 min)	640	1,791	1,438	486	1,519	443	490	0.21	0.07	0.61
Post fed (30-60 min)	1,238	1,440	607	544	696	536	291	0.03	0.19	0.39
Total (0-110 min)	3,143	4,086	2,964	1,658	2,975	1,620	828	0.05	0.21	0.97
TG, mg/dl										
Baseline	26.6	24.3	18.8	14.9	23.3	16.5	4.7	0.17	0.36	0.41
30 min postprandial	23.4	20.4	20.6	15.3	21.1	16.1	4.2	0.23	0.81	0.52
60 min postprandial	30.6	18.5	22.6	21.9	23.7	22.8	5.2	0.78	0.55	0.34
Cholesterol, mg/dl										
Baseline	122.9	120.9	133.5	134.2	112.0	114.9	7.0	0.32	0.18	0.10
30 min postprandial	121.9	122.6	132.5	142.8	111.9	123.6	8.3	0.95	0.14	0.10
60 min postprandial	116.9 [y]	126.1 [yz]	129.4 [yz]	139.5 [z]	114.2 [y]	115.2 [y]	7.4	0.83	0.48	0.03
NEFA, mmol/l										
Baseline	0.14	0.16	0.09	0.14	0.16	0.20	0.04	0.31	0.85	0.31
30 min postprandial	0.08	0.07	0.06	0.10	0.10	0.07	0.02	0.09	0.15	0.83
60 min postprandial	0.07	0.07	0.06	0.13	0.13	0.08	0.02	0.01	0.17	0.12

[1] A total of 24 barrows (Pig Improvement Co., Franklin, KY; 280 x C42) with initial body weight of 71.44 kg were individually housed and fed 1 of 6 dietary treatments for 9 week trial.

[2] Carb is defined as the main effect of carbohydrate source in the diet (starch, glucose, fructose).

Figures

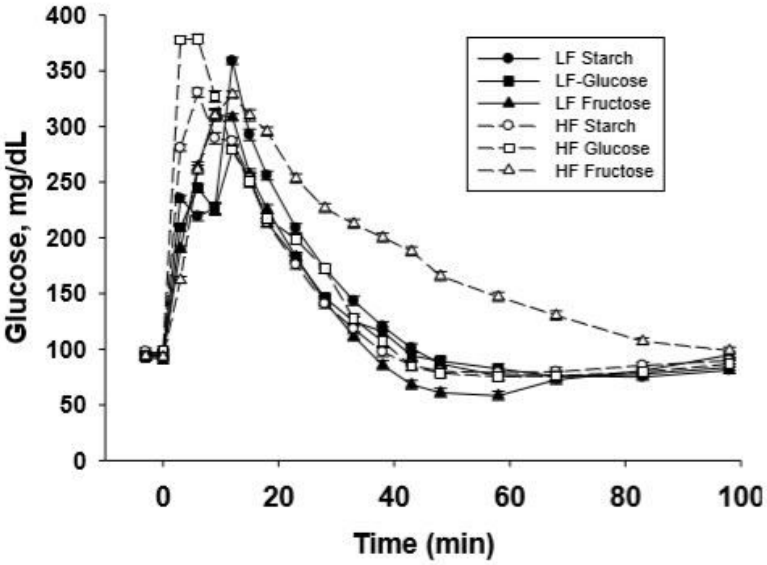

Figure 1

Plasma glucose concentration (mg/dL) in response to the intravenous Glucose Tolerance Test in pigs fed test diets for 9 weeks. Pigs were fasted for 16 h prior to the test. (Experiment 2)

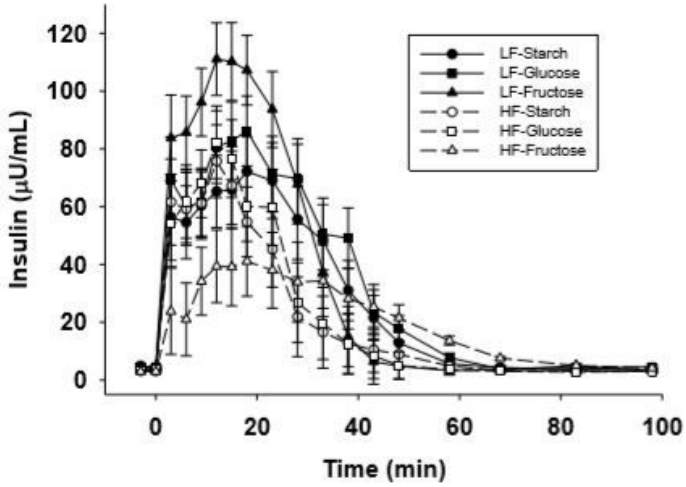

Figure 2

Serum insulin concentration (µU/ml) in response to the intravenous Glucose Tolerance Test in pigs fed test diets for 9 weeks. Pigs were fasted for 16 h prior to the test. (Experiment 2)

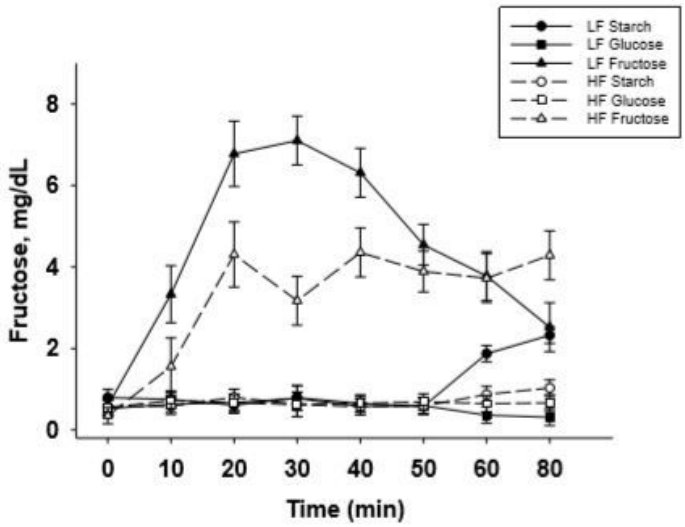

Figure 3

Serum fructose concentration (mg/dl) in response to a meal (RTM test) during week 9 due to feeding 0% added fat or 10% added fat in combination with three different carbohydrate sources to swine for 9 weeks. Pigs had been fasted 16 h prior to the meal. Selected serum samples were analyzed for fructose. (Experiment 2)

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- CHOfeedingcoverletter72020.pdf