

# EFFECTS OF REPLACING SUPPLEMENTAL SUCROSE WITH BEEF ON MATERNAL HEALTH AND FETAL GROWTH AND DEVELOPMENT USING A SOW BIOMEDICAL MODEL

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## I. INTRODUCTION

It is recommended that Americans limit their intake of added sugars to less than 10% of their total daily calories [1]. Added sugars are defined as sugars or syrups that are added to food and beverage items during processing and preparing of that given item [2]. Between 2005 – 2010, American men and women 20 years and older consumed an average of 13% of total daily calories from added sugar [6]. The leading source of added sugars within the American diet is sugar-sweetened beverages including regular, non-sugar free soda; fruit juices; energy drinks; sweetened water; and coffee and tea beverages that contain added sugar [3]. High added sugar consumption levels can lead to obesity and metabolic disorders such as diabetes, cardiovascular disease, and non-alcoholic liver disease [2, 3, 6]. These metabolic disorders may be prevented by replacing added dietary carbohydrate (sucrose) with a healthy protein alternative (beef). The objectives of this study were to investigate the impact of substituting dietary sucrose with beef supplementation on maternal health and fetal development utilizing the sow as a biomedical model. Based upon previous research, it was hypothesized that fetuses from sucrose supplemented sows would have greater body weights and would be more likely to be susceptible to metabolic diseases.

## II. MATERIALS AND METHODS

All procedures were approved by North Dakota State University Institutional Animal Care and Use Committee. Multiparous pregnant sows (Landrace x Yorkshire; BW = 222 kg; n = 21; Reps = 2) were utilized as a biomedical model to investigate substituting supplemental sucrose with beef on maternal health and fetal development. Sows were group housed and fed at North Dakota State University's Animal Nutrition and Physiology Center (ANPC) and bred to common sires utilizing artificial insemination. Pregnancy was confirmed utilizing ultrasound 29 days after breeding. Bred sows were housed in gestation crates in ANPC; provided with enrichment from d 30 to 111 ( $\pm 0.58$ ) of gestation; exposed to 19.4°C ambient temperature; and exposed to light from 700 to 1800 h daily. A complete sow gestation diet (corn-soybean meal-based, CSM; NRC 2012) was fed at one percent of BW on d 30 of gestation at 700 h daily from d 30 to 39 of gestation. Sows were then fed CSM at one percent BW on d 39 of gestation at 700 h daily from d 40 to 110 ( $\pm 0.58$ ) of gestation. Sows were randomly assigned to 1 of 4 isocaloric dietary supplements consisting of 126 g CSM (CON, n = 5); 110 g cooked ground beef (CGB; BEEF, n = 6); 54.8 g CGB and 42.7 g sucrose (B+S, n = 5); or 85.5 g sucrose (SUCROSE, n = 5). Dietary supplements were fed at 1100, 1500, and 1800 h from d 40 to 110 ( $\pm 0.58$ ) of gestation. Sows were euthanized on d 111 ( $\pm 0.58$ ) of gestation. Blood was collected via jugular venipuncture on d 29 and 111 ( $\pm 0.58$ ) of gestation. Blood chemistry was immediately analyzed using iSTAT (Abbot Point of Care, Kansas City, MO) for Na, K, Cl, ionized Ca, total CO<sub>2</sub>, glucose, urea nitrogen, creatinine, hematocrit, hemoglobin, and anion gap. Body weights were measured on d 30, 39, 54, 68, 82, 96, and 111 ( $\pm 0.58$ ) of gestation at 0800 hr. Tenth rib and last rib fat depth were measured on d 35, 70, and 110 ( $\pm 0.58$ ) of gestation utilizing an ALOKA SSD-500V (Hitachi Healthcare, Twinsburg, OH). All sows were provided *ad libitum* access to water. Sows were euthanized on d 111 ( $\pm 0.58$ ) of gestation. Sow pancreas, kidney, liver, heart, heart fat, lung, and semitendinosus weights were recorded. Fetal growth measurements of weight, crown to rump length, crown to nose length, heart girth, and abdominal girth were recorded for all fetuses. Two median weight male and female fetuses were selected from each sow for tissue collections which included pancreas, kidney, liver, heart, heart fat, lung, empty body weight, semimembranosus, and semitendinosus weights. Sow tissue weight data were analyzed using the MIXED procedure of SAS (v. 9.4;

SAS Inst. Inc.; Cary, NC, USA) with compound symmetry. All other data were analyzed using a repeated measures design, with sow as the repeated measure, using the MIXED procedure of SAS with compound symmetry variance covariance matrix. Alpha level was 0.05.

### III. RESULTS AND DISCUSSION

Dietary treatment did not influence blood metabolites of sows on d 29 and 111 of gestation ( $P = 0.09$  and  $P = 0.20$ , respectively). Sow weight throughout gestation was not influenced by dietary treatment ( $P = 0.74$ ). Dietary treatment did not influence tenth rib or last rib fat depth on d 35, 70, or 110 of gestation ( $P = 0.27$ ). Dietary treatment did not influence sow tissue weights ( $P = 0.21$ ). Compared to CON and SUCROSE, BEEF fetuses had larger nose to crown lengths ( $6.03 \pm 0.29$ ,  $6.02 \pm 0.28$ , and  $7.33 \pm 0.36$  cm, respectively;  $P = 0.04$ ). Compared to CON, SUCROSE fetuses had respectively larger BW ( $1296 \pm 73.6$  vs.  $1556 \pm 68.8$  g;  $P = 0.02$ ); heart girths ( $22.90 \pm 0.39$  vs.  $24.13 \pm 0.39$  cm;  $P = 0.03$ ), and liver weights ( $36.48 \pm 2.19$  vs.  $43.44 \pm 2.05$  g;  $P = 0.04$ ). Dietary treatment did not influence other fetal characteristics or organ weights ( $P \geq 0.05$ ).

As hypothesized, SUCROSE fetuses had increased body weights. The observed increase in SUCROSE fetal liver weight could be due to increased amounts of hepatic triglyceride concentrations or inflammation related to non-alcoholic fatty liver disease [4, 5]. Further analysis is required to determine susceptibility of metabolic disorders as a result of fetal programming.

### IV. CONCLUSION

Beef supplementation during pregnancy had minimal effects on maternal health or fetal growth; however, the increase in fetal body weight and liver weight due to sucrose supplementation should be further explored. Potential application of results includes development of dietary guidelines for pregnant women.

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### REFERENCES

#### *Book:*

1. U.S. Department of Agriculture, U.S. Department of Health and Human Services. (2015). Dietary Guidelines for Americans, 2015-2020. 8<sup>th</sup> Edition ed. Washington D.C.: U.S. Government Printing Office.

#### *Web Reference:*

2. Division of Nutrition, Physical Activity, and Obesity, National Center for Chronic Disease Prevention and Health Promotion. (2016). Know Your Limit for Added Sugars. Atlanta, GA. Accessed April 4, 2018. <https://www.cdc.gov/nutrition/data-statistics/know-your-limit-for-added-sugars.html>.
3. Division of Nutrition, Physical Activity, and Obesity, National Center for Chronic Disease Prevention and Health Promotion. (2017). Get the Facts: Sugar-Sweetened Beverages and Consumption. Atlanta, GA. Accessed April 7, 2018. <https://www.cdc.gov/nutrition/data-statistics/sugar-sweetened-beverages-intake.html>.

#### *Paper:*

4. Basaranoglu, M., Basaranoglu, G., & Bugianesi, E. (2015). Carbohydrate intake and nonalcoholic fatty liver disease: fructose as a weapon of mass destruction. *Hepatobiliary Surg Nutr.* 4(2): 109 – 116.
5. DiNicolantonio, J. J., Subramonian, A. M., & O'Keefe, J. H. (2017). Added fructose as a principal driver of non-alcoholic fatty liver disease: a public health crisis. *Open Heart.* 4(2): e000631.
6. Ervin R. B. & Ogden, C. L. (2013). Consumption of added sugars among U.S. adults, 2005 – 2010. *NCHS Data Brief.* (122):1 – 8.