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TASTE AND ENDOCRINE FACTORS IN WOMEN WITH GESTATIONAL
DIABETES MELLITUS

By

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A Dissertation submitted to the
Graduate School-New Brunswick
Rutgers, The State University of New Jersey
in partial fulfillment of the requirements
for the degree of
Doctor of Philosophy

Graduate Program in Nutritional Sciences

written under the direction of

Beverly J. Tepper

and approved by

New Brunswick, New Jersey

January, 2008

ABSTRACT OF THE DISSERTATION

TASTE AND ENDOCRINE FACTORS IN WOMEN WITH GESTATIONAL DIABETES MELLITUS

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Gestational diabetes mellitus (GDM) is hyperglycemia first identified during pregnancy. Many metabolic parameters increase across normal gestation, but may exceed those levels in pregnancies where GDM develops. Studies on the time course and pattern of metabolic factors in GDM are conflicting. Additionally, past research suggests changes in cravings and dietary intake during normal pregnancy and taste alterations in diabetes and pregnancy. Such parameters have been studied only on a limited basis in women with GDM. In this dissertation, we examined metabolic, taste and dietary measures across pregnancy.

The temporal profile of insulin, cortisol and leptin was determined in women who developed GDM and those who remained normal glucose tolerant (NGT). Fasting insulin levels were higher at 24-28 weeks gestation than during postpartum in women with GDM, however it was not until 34-38 weeks that insulin differed from postpartum in

NGT women. Both pregnant groups showed similar increases in cortisol levels across gestation. From 16-20 weeks to 24-28 weeks, leptin rose rapidly in women with GDM and was marginally higher in GDM women relative to NGT women at 24-28 weeks. By 34-38 weeks, leptin values did not differ between pregnant groups.

We also studied pregnancy and GDM effects on sweet taste perception and preference, and examined relationships of taste parameters with circulating hormones. Women with GDM exhibited higher preference for sucrose-sweetened milk as well as positive relationships between liking of glucose solutions and sucrose-sweetened milk and insulin and leptin, respectively. Women with NGT reported higher intake of all sweet food and beverage at 24-28 weeks, primarily as fruit and fruit juice, while women with GDM reported higher frequency of sweet cravings at 34-38 weeks gestation.

These results suggest that women challenged with concurrent pregnancy and diabetes exhibit early rises in insulin and leptin during gestation and such endocrine changes may be related to elements of sweet taste. Additionally, during late gestation, although women with GDM report following a carbohydrate controlled diet, they show a higher preference and craving for sweet taste. In GDM, alterations in sweet taste and differences in sweet cravings and food intake may be metabolically or psychologically based.

DEDICATION

To my sister, Jennifer, for over twenty-six and a half years of support and laughter. Thank you for *always* being by my side, keeping me in good spirits and believing in me.

ACKNOWLEDGEMENTS

I am thankful to *Beverly J Tepper, Ph.D.* for allowing me to be a part of her laboratory at Rutgers University, and for being a wonderful advisor and mentor. I would not be where I am today without her incredible guidance and support.

I would like to thank the members of my dissertation committee for their encouragement and direction. Thank you to *Dr. Sue Shapses*, for her continued advice, support and encouragement. To *Dr. John Smulian*, for providing significant consultation and suggestions, and for being part of my dissertation committee. Many thanks to *Dr. Malcolm Watford* for his unyielding support through my undergraduate *and* graduate school career. He has been a central part of who I am, currently, as a nutrition professional, and the researcher, and person, I will become in the future.

Thank you to *Dr. Dawn Brasaemle* for going above and beyond her role as Graduate Program Director, by providing tremendous academic *and* emotional support.

Thanks to *Natalia Ullrich* for her assistance with the study implementation and for countless other things I am unable to mention for lack of space.

Special thanks to the members of Dr. Tepper's laboratory: *Ivy Koelliker*, *Katherine Nolen* and *Taraja Williams* for being the most unbelievable friends and co-workers over the years and for always being by my side.

Thank you to *Dr. Shou-en Lu* for her remarkable assistance, patience and guidance with the statistical analysis. Her continual thoughtfulness, willingness to help, and interest in the study was enormously appreciated.

I would also like to sincerely thank the following people:

Elizabeth Lutchman, Naveen Kommera, M.D., Raza Ahmed, M.D., and Christian Rodriguez for their assistance in recruiting and testing of subjects during the study.

The *volunteers*, without whom our study would not have been conducted.

Dr. Angela Ranzini and Dr. Joseph Jenci for access to their clinic populations and for their assistance with subject recruitment and screening.

Marian Lake, R.N. and Elaine Vostrovsky, R.N. for their unbelievable help and feedback.

Dr. Louis Amorosa for his assistance on the start-up of the study.

The Faculty and Staff of the Program in Nutritional Sciences for all that I have learned while maturing as a nutrition expert and the support that made all of this possible.

The *phlebotomists and nurses* at Saint Peter's University Hospital for their contribution to the study.

I am especially thankful to my fellow students *Mousumi Bose, Jocilyn Dellava Diana Johnson, William Lagakos, Alexis Rothenberg, and Felicia Stoler* for their amazing friendships throughout my graduate school years. 'Sumi' – I could *not* have done this without you.

Thank you to *Mom and Dad* for supporting me through all of my education.

Thank you to *The Bloom family*, for being a main reason why I survived the toughest summer of my life.

I could not have done any of this without the unbelievably strong network of friends that have loved, encouraged and supported me over the years:

Daniel Benovitz, Tom Canty, John Dacles, Michelle Dacosta, Dianna & Shaun Deignan, Alison Flood, Ines Louro, Kimberly Mark, Tara McGuinness, Alexia Tsakiris, and last, but certainly not least, Jennifer Wolf who is one half of an extraordinary friendship that grows stronger, each second of every day.

A special thanks, once again, to my sister, *Jennifer*, who has brought *the* most love, laughter and support to my life.

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1 INTRODUCTION

1.1 Background and significance

1.1.1 Gestational diabetes mellitus (GDM)

GDM is carbohydrate intolerance first recognized during pregnancy (1). About 7% of U.S. pregnant women develop GDM (2). Depending on the patient demographics and the diagnostic tests used, the overall prevalence rate varies from 1-14% (3, 4). A thorough understanding of risk factors, metabolic changes and treatment is important so as to avoid possible detrimental fetal and maternal outcomes.

There are numerous factors that increase a woman's risk for developing GDM. They include advanced maternal age, high pre-pregnancy weight/BMI, family history of diabetes, non-Caucasian race, history of infertility, prior GDM, first-degree family member with diabetes, significant weight gain in adulthood and cigarette smoking (3, 5, 6). Metabolism is altered in GDM when compared to both normal pregnancy and the nonpregnant state. Although during normal pregnancy, there is significant insulin resistance and hyperlipidemia (7, 8), women with GDM show further dysregulation of glucose and lipid profiles. Triacylglycerols (TG), very low density lipoproteins (VLDL) and free fatty acids (FFA) are the primary lipid metabolites affected by GDM, and along with gestational hormones, such as β -estradiol and progesterone, are especially elevated during the third trimester in women with GDM (9). Overall weight gain may also be affected in GDM pregnancy (10); this lower weight gain may be solely an influence of treatment or may involve other effects of the disease.

1.1.2 Fetal complications from GDM pregnancies

There are a number of fetal or infant morbidities that may arise in circumstances of uncontrolled GDM. There is a higher rate of hyperbilirubinemia and macrosomia in children of women with GDM. Macrosomia increases the risk for operative delivery, shoulder dystocia, and birth trauma (11). Fetal hyperinsulinemia may occur as a result of maternal hyperglycemia, and can cause excess neonatal growth, delayed pulmonary maturation, or even fetal death (12). Elevated postprandial glucose values are more strongly associated with poor perinatal consequences, as opposed to elevation in fasting blood glucose (13). GDM may negatively impact the child's mental development. In children two years of age, there is a significant, inverse correlation between their mental-development-index and Stanford-Binet scores and the mother's levels of plasma β -hydroxybutyrate, a byproduct produced during abnormal glucose management, during the third trimester. The average Stanford-Binet scores also inversely correlate with third-trimester FFA levels (14).

1.1.3 Health implications of GDM in mothers

Poorly managed GDM also affects the mother, both during pregnancy, as well as after giving birth. In women with GDM, preeclampsia or hypertension without proteinuria both exist at twice the rate as what is found in normal pregnancies (15). Women with GDM are also more likely to have a caesarean delivery than those not suffering from diabetes. After adjustments are made for both pregnancy age as well as prior hypertension, the odds ratio for maternal medical complications during GDM pregnancy is 4.3 (16). There is also a much higher likelihood that the mother will

eventually develop GDM in subsequent pregnancies, or eventually develop type 2 diabetes. In follow-up studies of women with prior GDM, the risk of developing type 2 diabetes reaches 50% within 20 – 30 years of the GDM pregnancy (as reviewed in (17)). Some studies indicate that women with previous GDM have a higher rate of developing type 2 diabetes during the first 2.5-5 years postpartum, at a rate of about 10% per year (18). One study specifies factors that are the strongest predictors of future diabetes. The most pronounced include: four abnormal glucose values on the oral glucose tolerance test (OGTT) used for diagnosis and any type of diabetes during pregnancy (19). The increased risk for developing diabetes in the future suggests that women should be monitored and followed with yearly glucose tolerance tests.

1.1.4 Management of GDM

Like in type 2 diabetes, the maintenance of blood glucose within normal limits is the main objective of treatment of gestational diabetes mellitus (20). The first line of defense in management is dietary intervention.

Current medical nutrition therapy for GDM, as indicated by the American Diabetes Association (ADA), strives for achievement of appropriate nutrition for good maternal health and fetal growth and controlling carbohydrate intake within the context of a diet with sufficient calories and nutrients. Goals include the achievement of adequate energy intake for appropriate weight gain, attainment of normoglycemia, as well as the avoidance of blood or urinary ketones. The recommendation is to distribute carbohydrate throughout the day, by consuming three smaller meals plus 2-4 snacks per day, thereby avoiding large fluctuations in blood glucose. To prevent possible overnight

ketosis, an evening snack is recommended. Past studies have shown that the use of hypocaloric diets in obese women with GDM may result in the development of ketosis. A more appropriate caloric restriction may be a 30% reduction in overall estimated energy requirements. This will result in adequately maintained blood glucose values, without the danger of blood or urinary ketones (21). The recurring issue with any kind of diet plan is that it can many times be difficult to follow and/or completely understand. The aforementioned dietary advice gives a woman with GDM guidelines. However, it does not provide such details as importance in type of carbohydrate (simple vs. complex), more specific timing for carbohydrate consumption, and recommended context for carbohydrate intake (can a carbohydrate rich food be eaten on its own, or should it be eaten in the context of a meal?). Most importantly, women with GDM must be concerned with maintaining a healthy pregnancy for themselves and their unborn children in addition to controlling their diabetes. Dietary strategies developed for older populations with type 2 diabetes may not be entirely appropriate for the GDM populace. With a better understanding of taste changes and the relationships of said changes with endocrine parameters, a stronger biologically based foundation may arise from which to work off of, in the development of more fitting and specific dietary guidelines.

Although insulin is commonly used in uncontrolled GDM, physicians occasionally express concern in introducing oral medication during pregnancy due to possible implications on the fetus. However with the increase in prevalence and severity of diabetes during gestation, clinicians have started to incorporate oral hypoglycemic agents into the gestational diabetes management plan when the patient is not successful in maintaining healthy blood glucose through the use of diet alone, but may not yet require

insulin. Research indicates that oral medications, such as metformin and glyburide, can be used successfully, and without harm during pregnancy, to better control blood glucose and reduce fetal morbidities (20).

Crowther et al conducted a controversial, randomized clinical trial to study treatment versus non-treatment in gestational diabetes. The authors suggest that treatment, via dietary counseling, documenting of blood glucose and use of exogenous insulin when appropriate, decreases the rate of serious perinatal morbidities/mortalities. These outcome measures include nerve palsy, bone fracture, shoulder dystocia or death. Treatment group does not, however, predict the rate of caesarian section (22).

1.2 Topics of taste

1.2.1 Taste in type 1 and type 2 diabetes

Past research suggests that diabetes has an effect on taste perception. Schelling et al. demonstrated that people with controlled or uncontrolled diabetes exhibit a higher gustatory threshold for glucose than control subjects (23). The gustatory threshold is described as the lowest concentration of a solution at which that tastant can be perceived (24). In another study, patients with type 2 diabetes and healthy first-degree relatives of diabetics demonstrate significantly higher glucose thresholds than those of their controls. Moreover, the sucrose threshold of these type 2 diabetics is also elevated above that of controls. Another research group reported that newly diagnosed type 2 diabetes patients exhibit a decreased taste response to glucose. The increased thresholds may extend beyond sweet taste, to salty and bitter taste (25). Increased taste threshold may affect food liking and selection (26). In a study from our laboratory, investigators

examined alterations in sweet taste in type 2 diabetics and then observed if those changes were associated with changes in food consumption. Diabetic subjects who consume diets higher in sweetness on a daily basis give higher pleasantness ratings to a sweetened beverage (27). Another study found that acute hypoglycemia, in people with type 1 diabetes, seems to lead to a rise in food cravings, most especially those foods that are higher in carbohydrate (28). One research group followed a diabetic population receiving dietary treatment over a three month period. The researchers found that the people with diabetes report a decreased liking of sweet and fatty foods while receiving dietary treatment (29).

1.2.2 Taste and food intake in pregnancy

A large percentage of pregnant women report changes in taste at some point during their pregnancy (30-32). The most common taste changes are those of sweetness perception (33), although other taste qualities may also be affected (e.g. saltiness, spiciness or bitterness) (34). The basis for these changes has not yet been elucidated, but they may reveal themselves mainly in the form of cravings and aversions (31, 33, 35-37). Research indicates a generalized decrease in gustatory function in all stages of pregnancy, but taste thresholds seem to be highest during the first trimester (38, 39).

Women may consume more calories, in general, as well as more sweet food, during their second trimester (33). Estrogens and progestins may be responsible for such alterations in the intake of sweet foods in this time point in pregnancy. Changes in sweet taste in pregnancy could indicate an increased need for energy (30). Salty foods,

such as peanuts, on the other hand, are consumed significantly more in the third trimester. This may be related to a higher threshold for saltiness since salty foods are rated less salty by pregnant women in the third trimester than during other stages of pregnancy. This alteration in salt consumption and liking may be related to fluid retention and/or possible preeclampsia development during this point in pregnancy (33).

One untested hypothesis suggests that the cortical areas controlling gustatory function and the region controlling uterine function (insula) are located in close proximity to one another. Taste may be affected due to coordinated activation of both areas when the uterine environment is stimulated, either via the menstrual cycle or during pregnancy (40).

1.2.3 Dietary cravings/aversions in normal pregnancy

Food cravings are reported in as many as 97% of nonpregnant females, with chocolate being the highest craved food item (41). Overall, foods that are high in sweetness and fat are shown to comprise most reported cravings, especially in women (42). The biological and psychological bases of cravings remain controversial.

In pregnancy, alterations in taste and smell in addition to metabolic changes may drive cravings and aversions (31, 37). Fruit and fruit juices, as well as dairy products and salty snacks are the most frequently craved foods in pregnancy (43).

1.2.4 Food cravings and taste changes in GDM

Only one study examined taste changes and diet parameters in women with and without GDM (44). Data were collected at one time point during pregnancy (28-32 weeks) and one time point during early postpartum. The findings suggest that women with GDM give higher liking ratings to sucrose-sweetened strawberry milks than women without GDM. However, liking ratings for the milks do not exceed those of nonpregnant controls. As discussed previously, taste changes dynamically across pregnancy, and consequently these data cannot be extrapolated to other time points during pregnancy. Thus, an understanding of the temporal profile of sweet taste changes in GDM is still lacking.

Another key finding is a positive correlation between plasma glucose taken one hour following a glucose load and intake of fruit and fruit juices and sweetness liking ratings of glucose solutions in women with GDM, but not in pregnant women without GDM. The subject population in this research study was mostly made up of women with mild diet-treated GDM. There was, however, a small subset of women who had a more severe form of GDM, thereby requiring the use of insulin during their pregnancies. The hedonic ratings for the glucose solutions were the highest in these women and it was also these women in the GDM group that showed the greatest fruit and fruit juice consumption. These data suggest that the degree of glucose intolerance or some other endocrine or metabolic abnormality underlies the taste changes in GDM. These relationships have never been studied in GDM and are one of the goals of the work proposed here.

1.3 Endocrinology and metabolism

1.3.1 Fuel metabolism during normal pregnancy

During early normal pregnancy, there are substantial increases in maternal fat storage (45) as well as an increase or no change in insulin sensitivity. In the later stages of pregnancy, the transfer of glucose, amino and fatty acids to the fetus increases, promoting accelerated fetal growth. Maternal FFA, TG and cholesterol circulate at very high levels during gestation, providing a source of fatty acids for placental and fetal fuel as well as eventual lactation (7, 45, 46). High estrogen levels may promote increased circulating lipids, as evidenced by its involvement in VLDL production and reduction in the lipase-driven transfer of TG from lipoproteins (47).

Insulin resistance increases during normal pregnancy. The mechanisms behind increased insulin resistance in healthy pregnancy are complicated, but seem to involve increased serum levels of human chorionic somatotropin (hCS), progesterone, estrogen, and cortisol (as reviewed in (48)). During late pregnancy, when insulin resistance is at its peak, maternal glucose utilization decreases, thereby sparing this carbohydrate fuel for use by the growing fetus. Insulin resistance also causes a decrease in lipolysis inhibition. This allows for the mother to primarily use fat for energy (49). The resistance to insulin's effects on lipolysis as well as on oxidation of fat, occurs in the later stages of pregnancy, but then diminishes in the postpartum period (50).

1.3.2 Endocrine changes in normal pregnancy

Pregnancy is a time of continuous metabolic change, affecting many hormones and metabolites. Such parameters include insulin, cortisol, leptin, estrogen,

progesterone, glucose, and various blood lipids. During pregnancy, the hypothalamic-pituitary-adrenal (HPA) axis is activated, causing a rise in various steroids. Glucocorticoids function in the maturation of fetal organs, general preparation of the child for the extra-uterine environment as well as appropriate timing of childbirth (51) and increased circulating levels are found as early as 11 weeks gestation (52). At the start of the second trimester, there is typically a two- to three- fold increase in circulating cortisol. Estrogen stimulates cortisol-binding globulin production, thereby playing a role in the elevation of blood cortisol. There is also an increase in unbound cortisol. Elevations in circulating cortisol are involved in such pregnancy-related manifestations as fluid retention, reddening of the face and stretch marks (51, 53). Increasing cortisol levels are correlated with decreasing glucose tolerance, therefore some researchers suggest that the insulin resistance of pregnancy may be partially due to elevated cortisol levels (54).

Endogenous estrogen and progesterone also play important roles in maintaining the normal function of the fetoplacental unit (53). In the beginning of pregnancy, the corpus luteum and placenta contain the central supplies of progesterone and estrogen and blood levels of both hormones increase at this time. From 6-10 weeks gestation, studies have shown a short-term plateau or possible decrease in progesterone. During the later stages of pregnancy, progesterone and estrogen rise continuously until term (55).

The obesity-related peptide, leptin, is involved in a variety of physiological processes during pregnancy, such as early embryonic and fetal growth, implantation of the egg, fetal bone accumulation and pulmonary development (56).

In addition to maternal adipose tissue, leptin is also secreted by the placenta, endometrium, fetal adipocytes, mammary glands, and the chief cells of rat and human stomach glands. Consequently, leptin concentrations increase 66% during pregnancy, peak during the 2nd trimester and decrease to baseline at delivery (57). During normal pregnancy, a rise in leptin levels occurs concomitantly with significant increases in the levels of reproductive hormones (58). The reproductive hormones seem to be related to the release of leptin. For example, adipocyte incubation with estradiol caused an increase in leptin secretion (59). Additionally, especially during the first trimester and postpartum, normalized leptin concentrations correlate with estradiol levels (56). It may be that the hormonal milieu of pregnancy increases the production of leptin, contributing to leptin resistance and weight gain.

There is an increase in maternal fat stores during pregnancy, which could cause a surge in leptin production (60). Hardie et al showed that at each gestational stage, there is a correlation between leptin levels and pre-pregnancy BMI. Since the pattern of change in circulating leptin parallels the process of maternal fat accumulation and mobilization in pregnancy, leptin may be a marker for these processes (56). Maternal fat stores, however, tend to peak late in the second trimester, and then decrease towards term. This is due to the mobilization of maternal fat stores that helps sustain the quickly developing fetus (61) and provides a source of maternal fuel other than glucose. One study shows significantly higher levels of fasting serum leptin at 36 weeks of pregnancy in comparison to leptin levels measured at 3 and 6 months postpartum. These changes are positively correlated with corresponding changes in serum insulin. In the same study,

both BMI and body weight correlate with leptin levels in all time points of pregnancy as well as in the nonpregnant subjects. (62).

1.3.3 Endocrine and metabolic changes in diabetic pregnancy

Prior work examining insulin resistance in pregnancy shows differences between women with NGT and women with GDM. Some studies suggest that insulin response is reduced in GDM, perhaps due to the deterioration of β -cell capacity in these women (63, 64). Other research groups propose that increased insulin resistance in GDM is more closely related to a progressive deterioration in insulin sensitivity in target cells, and altered hepatic glucose production, both of which occur during gestation (65). Endo et al. showed that insulin sensitivity decreases over time in women with GDM, but remains unchanged in pregnant women with NGT (66). Furthermore, the insulinogenic index (insulin response per unit of glycemic stimulus) is significantly lower in women with GDM than women without the disease (as reviewed in (67)). Whether the GDM subject population shows a lean or obese phenotype, the main foundation for the development of diabetes during pregnancy seems to be a decline in pancreatic β -cell function, not allowing for the mounting of an adequate insulin response to a glucose stimulus (65).

In nonpregnant individuals with type 2 diabetes, plasma leptin is highly correlated with fat mass and plasma insulin, not the degree of insulin resistance (68, 69). These data imply that in diabetes, leptin may reflect the degree of adiposity not the severity of the disease. Whether this is the case in GDM is unknown. Since most women who develop GDM are overweight prior to pregnancy (70), leptin may be elevated in these women as they enter pregnancy. Moreover, the glucose intolerance intrinsic to GDM may

accentuate this hyperleptinemia. However, this is controversial, as studies show higher (71), lower (72) or no change (73) in leptin concentrations in women with GDM relative to pregnant women with normal glucose tolerance.

Except for one study in which all women with GDM were treated by diet (73), previous studies in GDM combined women receiving different treatments into a single study group (71, 74-77), or did not specify treatment mode (72, 78, 79). The absence of clinically well-defined subject groups might have contributed to the lack of consensus in this literature. The largest study to date reported that women with GDM have higher leptin than women without GDM when measured at 28 weeks gestational age (71). Another study measured leptin levels at 33 weeks gestational age also found higher leptin levels in women with GDM when compared to NGT women (78). However, two other studies reported no difference in plasma leptin between women with and without GDM when measured late in pregnancy or at term, respectively (73, 77).

Cortisol levels in women who developed type 2 diabetes before the onset of pregnancy are higher, when the samples are taken nocturnally, than women without diabetes prior to pregnancy (48). As previously discussed, some studies indicate a relationship between blood cortisol levels and glucose intolerance, where those women whose cortisol levels rise most over the course of pregnancy exhibit the highest glucose intolerance. This positive correlation suggests that increased cortisol levels contribute to the insulin resistance of pregnancy (54).

Lipid metabolites are also increased in GDM pregnancy. One research team found higher TG between 26-38 weeks gestation in women with GDM as compared to healthy pregnant controls (10). Another research group found that women with GDM

only display higher blood TG than pregnant type 2 diabetics in the second trimester and higher levels than nonpregnant controls in the postpartum state (48). Some studies indicate that women with GDM have increased FFA during gestation, when compared to women without GDM (10, 80).

Hormones such as estrogen and progesterone may be higher in women with GDM than in women without at certain time points of pregnancy. This is shown in a study following women with diet-treated GDM, who have higher estrogen levels at 37-38 weeks gestation and higher progesterone at 33-38 weeks gestation (10).

1.4 General endocrine and metabolic measures and relationships

1.4.1 HOMA-IR

The homeostasis model assessment (HOMA-IR) is a computer model using fasting glucose and insulin, put forth by Matthews et al as a noninvasive measure of insulin resistance. The calculation for HOMA-IR is: $(\text{fasting insulin } (\mu\text{U/mL}) \times \text{fasting glucose (mmol/L)}) / 22.5$). This model's estimate of insulin resistance correlates with insulin resistance estimates attained from using the euglycemic clamp, hyperglycemic clamp or blood insulin concentrations (81). The HOMA-IR model is also found to correlate well with Bergman's SI, a measure of insulin sensitivity, and the glucose clamp method, which quantifies insulin secretion and resistance, in individuals with type 2 diabetes (82). HOMA-IR is recognized as an appropriate tool for insulin resistance estimation in large population studies, where accuracy for a single subject is not as important as the determination of population tendencies (83). Since insulin resistance is

an eventuality of pregnancy, HOMA-IR is validated for use during gestation both in pregnant women with NGT and women who have GDM (84).

1.4.2 Relationship between leptin and insulin

There seems to be a paradox in the relationship between leptin and insulin secretion and sensitivity. Some past research indicates that high insulin levels may stimulate adipocyte leptin production and secretion (85). The inability to release insulin in response to glucose, may lead to impaired leptin secretion. On the other hand, in nonpregnant individuals, leptin plays a major role in glucose homeostasis and may be involved in the development of insulin resistance (86). It is plausible that leptin may have similar functions in pregnancy.

1.4.3 Influence of glucocorticoids on glucose metabolism and leptin

In diabetes, there are consistently higher levels of circulating glucocorticoids in addition to irregular glucose metabolism. This is most likely due to a disorder in the function of the steroid receptors (87). Additionally, the higher circulation of glucocorticoids may play a role in leptin resistance, as suggested in rodent models (88).

1.5 Endocrine influence on appetite, sweet taste and food intake

1.5.1 Leptin and appetite

Leptin is released from the adipocytes and is an indication of body adiposity. Research suggests that it influences both appetite and metabolism. In normal mice, administration of leptin decreases energy intake and increases metabolic rate (89).

1.5.2 Leptin and sweet taste

One of leptin's target organs is the taste organ, and studies have linked leptin to behavioral and neurophysiological sweet taste responses in rodent models of obesity. Kawai et al found that injection of lean and ob/ob mice with leptin reduces the preference for sweet substances, such as sucrose and saccharin, as well as peripheral taste nerve (chorda tympani and glossopharyngeal) responses (90). Leptin injections do not, however, reduce sweet preference in db/db mice, which are leptin resistant (90, 91). Interestingly, greater chorda tympani nerve responses to sweet taste are observed in two models of leptin resistance: VMH-lesioned obese rats and high-fat fed diet-induced obese rats. Additionally, higher taste nerve responses are exhibited in streptozotocin-diabetic rats, models of type 1 diabetes (92).

There is also evidence that leptin may be involved in taste and food palatability ratings in humans. One study showed that the main predictor for palatability of a high carbohydrate meal is basal serum leptin levels (93), a finding that suggests a relationship between leptin and liking of carbohydrate-containing foods. However, high fasting serum leptin concentrations in obese women has been associated with a trend towards decreased liking for the taste of a high-fat, low-sugar mixture (94).

1.5.3 Influence of insulin on hunger and sweet taste

Hyperinsulinemia may be linked to hunger and sweet taste preference. For instance, one study showed that high insulin levels leads to elevations in hunger, sweetness palatability and overall food intake (95). Another study found that induced hyperglycemia causes satiety, however induced hyperinsulinemia does not. The authors suggest, however, that endogenous insulin may potentiate the satiating effects of the high blood glucose (96). Hence, conditions of dysregulated insulin may influence sweet taste and sweet food eating patterns.

1.5.4 Reproductive hormones and food intake

Reproductive hormones may influence taste and food intake. For example, one study demonstrated that estrogen increases the threshold for gustatory detection of sucrose in rodents (97). In another study, when rats are ovariectomized, there is a significant increase in daily food intake as compared to sham rats. Upon estradiol replacement, the effect on daily intake is reduced (98). Therefore, taste may be affected when reproductive hormones are released upon stimulation of the uterine environment. This situation arises at certain times in the menstrual cycle as well as during pregnancy.

1.6 Rationale

Taste changes have been documented previously in the GDM population, however research has only touched on the magnitude and time course of those changes. Additionally, in women with GDM, there are shifts in numerous metabolic and endocrine factors, either or both of which may correlate with changes in taste perception and preference. Since these relationships remain to be tested in this population, the overall purpose of this research project was to study taste changes in a larger subject population and across more time points in pregnancy, as well as to examine the possible relationships between those taste alterations and shifts in endocrine patterns.

1.7 Literature cited – Introduction

1. Metzger BE, Coustan DR. Summary and recommendations of the fourth international workshop-conference on gestational diabetes mellitus: the organizing committee. *Diabetes Care* 1998;21:B161-B167.
2. American Diabetes Association. Gestational diabetes mellitus. *Diabetes Care* 2004;27:S88-S90.
3. Dooley SL, Metzger BE, Cho NH. Gestational diabetes mellitus. Influence of race on disease prevalence and perinatal outcome in a U.S. population. *Diabetes* 1991;40:25-29.
4. Magee M, Walden C, Benedetti T, Knopp RH. Influence of diagnostic criteria on the incidence of gestational diabetes and perinatal morbidity. *JAMA* 1993;269:609-615.
5. Berkowitz GS, Lapinski RH, Wein R, Lee D. Race/ethnicity and other risk factors for gestational diabetes. *Am J Epidemiol* 1992;135:965-973.
6. Bongain A, Isnard V, Gillet JY. Obesity in obstetrics and gynecology. *Eur J Obstet Gyn R B* 1998;77:217-228.
7. Boyd EM. The lipemia of pregnancy. *J Clin Invest* 1934;13:347-363.
8. Montes A, Walden CE, Knopp RH, Cheung M, Chapman MB, Albers JJ. Physiologic and supraphysiologic increases in lipoprotein lipids and apoproteins in late pregnancy and postpartum. *Atherosclerosis* 1984;4:407-417.
9. Wijendran V, et al. Maternal plasma phospholipid polyunsaturated fatty acids in pregnancy with and without gestational diabetes mellitus: relations with maternal factors. *Am J Clin Nutr* 1999;70:53-61.
10. Couch SC, Philipson EH, Bendel RB, Pujda LM. Elevated lipoprotein lipids and gestational hormones in women with diet-treated gestational diabetes mellitus compared to healthy pregnant controls. *J Diabetes Complicat* 1998;12:1-9.
11. Clinical management guidelines for obstetrician-gynecologists. *ACOG Practice Bulletin* 2001.
12. Diabetes and pregnancy. *ACOG Practice Bulletin* 1994;200.
13. Major CA, Henry MJ, De Veciana M, Morgan MA. The effects of carbohydrate restriction in patients with diet-controlled gestational diabetes. *Obstet Gynecol* 1998;91:600-604.

14. Rizzo T, Metzger BE, Burns WJ, Burns K. Correlations between antepartum maternal metabolism and intelligence of offspring. *N Engl J Med* 1991;325:911-916.
15. Goldman M, Kitzmiller J, Abrams B, Cowan RM, Laros J, R.K. Obstetric complications with GDM: effects of maternal weight. *Diabetes* 1991;40 (Supplement 2):79-82.
16. Saydah SH, Chandra A, Eberhardt MS. Pregnancy experience among women with and without gestational diabetes in the U.S. 1995 National Survey of Family Growth. *Diabetes Care* 2005;28:1035-1040.
17. Lee AJ, Hiscock RJ, Wein P, Walker SP, Permezel M. Gestational diabetes mellitus: Clinical predictors and long-term risk of developing type 2 diabetes. *Diabetes Care* 2007;30:878-883.
18. Gregory KD, Kjos SL, Peters RK. Cost of non-insulin-dependent diabetes in women with a history of gestational diabetes: implications for prevention. *Obstet Gynecol* 1993;81:782-786.
19. Albareda M, et al. Diabetes and abnormal glucose tolerance in women with previous gestational diabetes. *Diabetes Care* 2003;26:1199-1205.
20. Homko CJ, Reece EA. Insulins and oral hypoglycemic agents in pregnancy. *J Matern Fetal Neonatal Med* 2006;19:679-686.
21. American Diabetes Association. Nutrition recommendations and interventions for diabetes. *Diabetes Care*, 2007:S48-S65.
22. Crowther CA, Hiller JE, Moss JR, McPhee AJ, Jeffries WS, Robinson JS. Effect of treatment of gestational diabetes mellitus on pregnancy outcomes. *N Engl J Med* 2005;352:2477-2486.
23. Schelling JL, Tetreault L, Lasagna L, Davis M. Abnormal taste threshold in diabetes. *Lancet* 1965;19:508-512.
24. Middleton RA, Allman-Farinelli MA. Taste sensitivity is altered in patients with chronic renal failure receiving continuous ambulatory peritoneal dialysis. *J Nutr* 1999;129:122-125.
25. Perros P, MacFarlane TW, Counsell C, Frier BM. Altered taste sensation in newly-diagnosed NIDDM. *Diabetes Care* 1996;19:768-770.
26. Hardy SL, Brennand CP, Wyse BW. Taste thresholds of individuals with diabetes mellitus and of control subjects. *J Am Diet Assoc* 1981;79:286-289.

27. Tepper BJ, Hartfiel LM, Schneider SH. Sweet taste and diet in type II diabetes. *Physiol Behav* 1996;60:13-18.
28. Strachan MWJ, Ewing FME, Frier BM, Harper A, Deary IJ. Food cravings during acute hypoglycemia in adults with type 1 diabetes. *Physiol Behav* 2004;80:675-682.
29. Laitinen JH, Tuorila HM, Uusitupa MIJ. Changes in hedonic responses to sweet and fat in recently diagnosed non-insulin-dependent diabetic patients during diet therapy. *Eur J Clin Nutr* 1991;45:393-400.
30. Brown JE, Toma RB. Taste changes during pregnancy. *Am J Clin Nutr* 1986;43:414-418.
31. Hook EB. Dietary cravings and aversions during pregnancy. *Am J Clin Nutr* 1978;31:1355-1362.
32. Worthington-Roberts B, Little RE, Lambert MD, Wu R. Dietary cravings and aversions in the postpartum period. *J Am Diet Assoc.* 1989;89:647-651.
33. Bowen DJ. Taste and food preference changes across the course of pregnancy. *Appetite* 1992;19:233-242.
34. Kölblle N, Hummel T, von Mering R, Huch A, Huch R. Gustatory and olfactory function in the first trimester of pregnancy. *Eur J Obstet Gyn R B* 2001;99:179-183.
35. Bayley TM, Dye L, Jones S, DeBono M, Hill AJ. Food cravings and aversions during pregnancy: relationships with nausea and vomiting. *Appetite* 2002;38:45-51.
36. Crystal SR, Bowen DJ, Bernstein IL. Morning sickness and salt intake, food cravings and food aversions. *Physiol Behav* 1999;67:181-187.
37. Dickens G, Trethowan W. Cravings and aversions during pregnancy. *J Psychosom Res* 1971;15:259-268.
38. Ochsenbein-Kölblle N, von Mering R, Zimmermann R, Hummel T. Changes in gustatory function during the course of pregnancy and postpartum. *BJOG* 2005;112:1636-1640.
39. Kuga M, Ikeda M, Suzuki K, Takeuchi S. Changes in gustatory sense during pregnancy. *Acta Otolaryngol* 2002;Supplement 546:146-153.

40. Persinger MA. Shifting gustatory thresholds and food cravings during pregnancy as expanding uterine-induced steady potential shifts within the insula: a hypothesis. *Percept Mot Skills* 2001;92:50-52.
41. Weingarten HP, Elston D. Food cravings in a college population. *Appetite* 1991;17:167-175.
42. Yanovski S. Sugar and fat: Cravings and aversions. *J Nutr* 2003;133:835S-837S.
43. Hirschberg AL. Hormonal regulation of appetite and food intake. *Ann Med* 1998;30:7-20.
44. Tepper BJ, Seldner AC. Sweet taste and intake of sweet foods in normal pregnancy and pregnancy complicated by gestational diabetes mellitus. *Am J Clin Nutr* 1999;70:277-284.
45. Herrera E, Lasunción MA, Gomez-Coronado D, Aranda P, López-Luna P, Maier I. Role of lipoprotein lipase activity on lipoprotein metabolism and circulating triglycerides in pregnancy. *Am J Obstet Gynecol* 1988;158:1575-1583.
46. Peters JP, Heinemann M, Man EB. The lipids of serum in pregnancy. *J Clin Invest* 1951;30:388-394.
47. Alvarez JJ, Montelongo A, Iglesias A, Lasunción MA, Herrera E. Longitudinal study on lipoprotein profile, high density lipoprotein subclass, and postheparin lipases during gestation in women. *J Lipid Res* 1996;37:299-308.
48. Hollingsworth DR. Alterations of maternal metabolism in normal and diabetic pregnancies: differences in insulin-dependent, non-insulin-dependent, and gestational diabetes. *Am J Obstet Gynecol* 1983;146:417-429.
49. Homko CJ, Sivan E, Reece EA, Boden G. Fuel metabolism during pregnancy. *Semin Reprod Endocrinol* 1999;17:119-125.
50. Sivan E, Homko CJ, Chen X, Reece EA, Boden G. Effect of insulin on fat metabolism during and after normal pregnancy. *Diabetes* 1999;48:834-838.
51. Trainer P. Corticosteroids and pregnancy. *Semin Reprod Med* 2002;20:375-380.
52. Demey-Ponsart E, Foidart JM, Sulon J, Sodoyez JC. Serum CBG, free and total cortisol and circadian patterns of adrenal function in normal pregnancy. *J Steroid Biochem* 1982;16:165-169.
53. Soldin OP, Guo T, Weiderpass MD, Tractenberg RE, Hilakivi-Clarke L, Soldin S. Steroid hormone levels in pregnancy and 1 year postpartum using isotope dilution tandem mass spectrometry. *Fertil Steril* 2005;84:701-710.

54. Hornnes PJ, Kuhl C. Gastrointestinal hormones and cortisol in normal pregnant women and women with gestational diabetes. *Acta Endocrinol Suppl (Copenh)* 1986;277:24-26.
55. Schindler AE. Endocrinology of pregnancy: Consequences for the diagnosis and treatment of pregnancy disorders. *J Steroid Biochem* 2005;97:386-388.
56. Hardie L, Trayhurn P, Abramovich D, Fowler P. Circulating leptin in women: a longitudinal study in the menstrual cycle and during pregnancy. *Clin Endocrinol* 1997;47:101-106.
57. Heini AF, Lara-Castro C, Kirk KA, Considine RV, Caro JF, Weinsier RL. Association of leptin and hunger-satiety ratings in obese women. *Int J Obes Relat Metab Disord* 1998;22:1084-1087.
58. Masuzaki H, et al. Nonadipose tissue production of leptin: Leptin as a novel placenta-derived hormone in humans. *Nat Med* 1997;3:1029-1033.
59. Sivan E, Whittaker PG, Sinha D, Homko CJ. Leptin in human pregnancy: the relationship with gestational hormones. *Am J Obstet Gynecol* 1998;179:1128-1132.
60. Lepercq J, et al. Overexpression of placental leptin in diabetic pregnancy: A critical role for insulin. *Diabetes* 1998;47:847-850.
61. Sattar N, Greer IA, Pirwani I, Gibson J. Leptin levels in pregnancy: marker for fat accumulation and mobilization? *Acta Obstet Gyn Scan* 1998;77:278-283.
62. Butte NF, Hopkinson JM, Nicolson MA. Leptin in human reproduction: serum leptin levels in pregnant and lactating women. *J Clin Endocr Metab* 1997;82:585-589.
63. Buchanan TA, Metzger BE, Freinkel N, Bergman RN. Insulin sensitivity and β -cell responsiveness to glucose during pregnancy in lean and moderately obese women with normal glucose tolerance or mild gestational diabetes. *Am J Obstet Gynecol* 1990;162:1008-1014.
64. Catalano PM, et al. Carbohydrate metabolism during pregnancy in control subjects and women with gestational diabetes. *Am J Physiol Endocrinol Metab* 1993;264:60-67.
65. Catalano PM, Huston L, Amini SB, Kalhan SC. Longitudinal changes in glucose metabolism during pregnancy in obese women with normal glucose tolerance and gestational diabetes mellitus. *Am J Obstet Gynecol* 1999;180:903-916.

66. Endo S, et al. Differences in insulin sensitivity in pregnant women with overweight and gestational diabetes mellitus. *Gynecol Endocrinol* 2006;22:343-349.
67. Kuhl C, Hornnes PJ, Andersen OJ. Etiology and pathophysiology of gestational diabetes mellitus. *Diabetes* 1985;34:66-70.
68. Mohamed-Ali V, Pinkney JH, Panahloo A, Goodrick S. Relationships between plasma leptin and insulin concentrations, but not insulin resistance, in non-insulin-dependent (type 2) diabetes mellitus. *Diabetes Medicine* 1997;14:376-380.
69. Tasaka Y, Yanagisawa K, Iwamoto Y. Human plasma leptin in obese subjects and diabetics. *Endocrine Journal* 1997;44:671-676.
70. Jovanovic-Peterson L, Peterson CM. Review of gestational diabetes mellitus and low-calorie diet and physical exercise as therapy. *Diabetes Metabolism Reviews* 1996;12:287-308.
71. Kautzky-Willer A, Pacini G, Tura A, et al. Increased plasma leptin in gestational diabetes. *Diabetologia* 2001;44:164-172.
72. Festa A, Shnawa N, Krugluger W, Hopmeier P, Schernthaner G, Haffner SM. Relative hypoleptinaemia in women with mild gestational diabetes mellitus. *Diabetes Medicine* 1999;16:656-662.
73. Simmons D, Breier BH. Fetal Overnutrition in Polynesian Pregnancies and in Gestational Diabetes May Lead to Dysregulation of the Adipoinular Axis in Offspring. *Diabetes Care* 2002;25:1539-1544.
74. McLachlan KA, O'Neal D, A. J, Alford FP. Do adiponectin, TNF α , leptin and CRP relate to insulin resistance in pregnancy? Studies in women with and without gestational diabetes, during and after pregnancy. *Diabetes/Metabolism Research and Reviews* 2006;22:131-138.
75. Verhaeghe J, van Bree R, Lambin S, Caluwaerts S. Adipokine profile and c-reactive protein in pregnancy: Effects of glucose challenge response versus body mass index. *Journal of The Society For Gynecologic Investigation* 2005;12:330-334.
76. Meller M, Qiu C, Vadachkoria S, Abetew DF, Luthy DA, Williams MA. Changes in Placental Adipocytokine Gene Expression Associated with Gestational Diabetes Mellitus. *Physiological Research* 2006;55:501-512.

77. Lappas M, Yee K, Permezel M, Rice GE. Release and regulation of leptin, resistin and adiponectin from human placenta, fetal membranes, and maternal adipose tissue and skeletal muscle from normal and gestational diabetes mellitus-complicated pregnancies. *Journal of Endocrinology* 2005;186:457-465.
78. Buhling KJ, Harder T, Sehoul J, Nanz J, Plagemann A, Dudenhausen JW. Independent association between leptin and blood pressure during third trimester in normal and gestational diabetic pregnancies. *European Journal of Obstetrics & Gynecology and Reproductive Biology* 2005;119:180-184.
79. Ategbro JM, Grissa O, Yessoufou A, et al. Modulation of Adipokines and Cytokines in Gestational Diabetes and Macrosomia. *The Journal of Clinical Endocrinology & Metabolism* 2006;91:4137-4143.
80. Montelongo A, Lasunción MA, Pallardo LF, Herrera E. Longitudinal study of plasma lipoproteins and hormones during pregnancy in normal and diabetic women. *Diabetes* 1992;41:1651-1659.
81. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: Insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985;28:412-419.
82. Lansang MC, Williams GH, Carroll JS. Correlation between the glucose clamp technique and the homeostasis model assessment in hypertension. *Am J Hypertens* 2001;14:51-53.
83. Cohen O, Epstein GS, Weisz B, Homko CJ, Sivan E. Longitudinal assessment of insulin sensitivity in pregnancy. Validation of the homeostasis model assessment. *Clin Endocrinol* 2006;2006:640-644.
84. Kirwan JP, Huston-Presley L, Kalhan SC, Catalano PM. Clinically useful estimates of insulin sensitivity during pregnancy: Validation studies in women with normal glucose tolerance and gestational diabetes mellitus. *Diabetes Care* 2001;24:1602-1607.
85. Zimmet PZ, et al. Is there a relationship between leptin and insulin sensitivity independent of obesity? A population-based study in the Indian Ocean nation of Mauritius. *Int J Obes* 1998;22:171-177.
86. Ceddia RB, Koistinen HA, Zierath JR, Sweeney G. Analysis of paradoxical observations on the association between leptin and insulin resistance. *FASEB Journal* 2002;16:1163-1176.

87. Tempel DL, Leibowitz SF. Adrenal steroid receptors: interactions with brain neuropeptide systems in relation to nutrient intake and metabolism. *J Neuroendocrinol* 1994;6:479-501.
88. Zakrzewska KE, Cusin I, Sainsbury A, Rohner-Jeanrenaud F, B. J. Glucocorticoids as counterregulatory hormones of leptin: toward an understanding of leptin resistance. *Diabetes* 1997;46:717-719.
89. Halaas JL, et al. Weight-reducing effects of the plasma protein encoded by the obese gene. *Science* 1995;269:543-546.
90. Kawai K, Sugimoto K, Nakashima K, Miura H, Ninomiya Y. Leptin as a modulator of sweet taste sensitivities in mice. *PNAS* 2000;97:11044-11049.
91. Shigemura N, et al. Leptin modulates behavioral responses to sweet substances by influencing peripheral taste structures. *Endocrinology* 2004;145:839-847.
92. Shimizu Y, et al. Enhanced responses of the chorda tympani nerve to sugars in the ventromedial hypothalamic obese rat. *J Neurophysiol* 2003;90:128-133.
93. Raynaud E, et al. Serum leptin is associated with the perception of palatability during a standard high-carbohydrate breakfast test. *Clin Sci* 1999;96:343-348.
94. Karhunen LJ, Lappalainen RI, Haffner SM, et al. Serum leptin, food intake and preferences for sugar and fat in obese women. *Int J Obesity* 1998;22:819-821.
95. Rodin J, Wack J, Ferrannini E, DeFronzo RA. Effect of insulin and glucose on feeding behavior. *Metabolism* 1985;34:826-831.
96. Gielkens HAJ, Verkijk M, Lam WF, Lamers CBHW, Masclee AAM. Effects of hyperglycemia and hyperinsulinemia on satiety in humans. *Metabolism* 1998;47:321-324.
97. Curtis KS, Stratford JM, Contreras RJ. Estrogen increases the taste threshold for sucrose in rats. *Physiol Behav* 2005;86:281-286.
98. Latour MG, Shinoda M, Lavoie J-M. Metabolic effects of physical training in ovariectomized and hyperestrogenic rats. *J Appl Physiol* 2001;90:235-241.

2 GOALS OF THE DISSERTATION

This dissertation will be organized in three sections:

- a) Endocrine and dietary patterns
- b) Relationships between taste changes and endocrine factors
- c) Patterns of sweet cravings and sweet food intake

With the following goals:

1. ***To compare reported dietary intake and temporal pattern of hormones and metabolites in women with GDM to those of women with NGT across pregnancy stages.***

The primary hormones of interest are insulin, leptin and cortisol due to their involvement in GDM pregnancy. The primary metabolite of interest is glucose. Other parameters include estradiol and progesterone, as well as TG, VLDL and FFA.

Specific Aim 1. Document changes in hormones and metabolites over the course of pregnancy.

Hypotheses to be tested.

There will be a serial rise and subsequent fall in fasting serum concentrations of insulin, cortisol and leptin in normal pregnancy. Peak fasting serum hormone levels in women with GDM will exceed those observed in women with NGT.

Insulin resistance assessed by an oral glucose challenge and calculated by HOMA-IR will be elevated in women with GDM at the time of diagnosis. These parameters will improve with medical management.

Specific Aim 2. Document reported dietary intake across gestation in women with GDM and women with NGT.

Hypotheses to be tested.

Women with NGT will show increased intake of sweet foods in mid pregnancy relative to other time points during pregnancy.

Women with GDM will show higher intake of sweet foods at mid-pregnancy than women with NGT, which will extend through the end of gestation.

2. *To document taste changes found in normal pregnancy and GDM pregnancy, and to relate these changes to alterations in gestational hormone and metabolic profiles. Two types of sweet stimuli will be investigated: glucose solutions and sucrose-sweetened milks.*

Specific Aim 3. Document taste changes across gestation in women with GDM and women with NGT.

Specific Aim 4. Assess relationships between hormonal and metabolic parameters and changes in sweet taste.

Hypotheses to be tested.

Changes in sweet taste in women with GDM will be correlated with peak concentrations of insulin, cortisol or leptin, either singly or in combination. Since these hormones are expected to peak mid-to-late pregnancy, correlations between taste parameters and hormones are expected to occur at this time.

3. *To compare the temporal pattern of reported dietary intake of sweet foods and cravings in women with GDM to those of women with NGT across pregnancy stages.*

Specific Aim 5. Document alterations in patterns of sweet food consumption over the course of pregnancy.

Hypotheses to be tested.

In all pregnant women, sweet food cravings and consumption will peak mid-pregnancy, fall during later pregnancy and return to pre-pregnancy values following the birth of the baby.

In women with GDM, the peak in sweet cravings and consumption of sweet foods will exceed the levels observed in women with NGT.

3 MAIN EXPERIMENTS

SECTION A. Temporal Profile of Insulin Resistance, Leptin and Other Metabolic Hormones Across Pregnancy in Women with Diet-treated, Mild Gestational Diabetes Mellitus

ABSTRACT

Background: Gestational diabetes mellitus (GDM) is glucose intolerance diagnosed during pregnancy. Leptin is elevated in normal pregnancy and may be further elevated in GDM, but the latter has been poorly documented.

Objectives: To compare the temporal profiles of circulating insulin, leptin and cortisol across gestation in women who developed GDM to those with normal glucose tolerance (NGT).

Design: A prospective design was used. Subjects included 93 pregnant women with NGT, 15 pregnant women who developed GDM and 19 nonpregnant controls. At 16-20 weeks, 24-28 weeks, and 34-38 weeks gestation, and 6-10 weeks postpartum, fasting insulin, leptin and cortisol levels were measured. Insulin resistance (IR) was assessed by HOMA-IR and ROC analysis determined if IR was a predictor of GDM. A 50-gram oral glucose challenge was administered during the latter 3 test sessions.

Results: From 16-20 weeks to 24-28 weeks gestation, leptin rose rapidly in women who developed GDM compared to nonpregnant controls ($p=0.002$), and was marginally higher in the GDM group relative to the NGT group (35.0 ± 3.4 vs. 26.8 ± 1.2 ng/mL; $p=0.06$) at 24-28 weeks gestation. By 34-38 weeks gestation, leptin values did not differ between pregnant groups. IR peaked in women with GDM at 24-28 weeks gestation and IR at 16-20 weeks gestation was a moderate predictor of GDM development.

Conclusions: Leptin rises earlier in GDM pregnancy but approximates levels of NGT pregnancy later in gestation. IR at 16-20 weeks gestation may predict the future development of GDM.

KEY WORDS: Gestational Diabetes, Pregnancy, Leptin, Insulin

INTRODUCTION

Gestational Diabetes Mellitus (GDM) is a form of carbohydrate intolerance of variable severity that is first recognized during pregnancy (1). Its metabolic profile and management is similar to that of type 2 diabetes (2). GDM affects 7% of all pregnancies in the U.S. (3). GDM is of primary clinical importance because it increases the risk of obstetric and perinatal complications and is often associated with metabolic changes in the offspring (4, 5). The exact pathophysiology of GDM is unknown, but some contributors include pronounced insulin resistance and diminished β -cell secretion of insulin (6).

Pregnancy is a unique metabolic state designed to meet the increasing fetal demand for fuels as gestation progresses to term. In addition to the reproductive hormones, metabolic hormones such as insulin and cortisol are also elevated during pregnancy (7). Insulin needs increase 4-fold during pregnancy and insulin release reaches a plateau by 30 weeks gestational age (8). Insulin resistance begins to develop at mid-pregnancy and persists throughout the remainder of the gestational period (9, 10). Despite this ongoing insulin resistance, most women maintain normal glucose tolerance. Leptin, a protein involved in energy homeostasis and primarily secreted by adipose tissue, also increases during pregnancy, peaks during the 2nd trimester and decreases to baseline at delivery (11, 12). Since the pattern of change in circulating leptin parallels the process of maternal fat accumulation and mobilization in pregnancy, leptin may be a marker for these processes (11, 13).

GDM is most commonly detected at 24-28 weeks gestational age, when many endocrine parameters are reaching their peak and insulin demand outstrips secretory

capacity (7). The hallmarks of GDM include postprandial hyperinsulinemia and hyperglycemia (7, 14), as well as disruptions in lipid metabolism, including elevations in triacylglycerols and free fatty acids (15). Fasting glucose may be normal in GDM, particularly when the disease is mild (16).

The role of leptin in GDM is poorly understood. Leptin is normally secreted in direct proportion to the size of the adipose mass (13). Thus, plasma leptin is higher in women who are overweight at the beginning of pregnancy and also rises more sharply in women who gain more weight during pregnancy (11, 12). In nonpregnant individuals with type 2 diabetes, plasma leptin is highly correlated with fat mass and plasma insulin, not the degree of insulin resistance (17, 18). These data imply that in type 2 diabetes, leptin reflects the degree of adiposity not the severity of diabetes. Whether this is the case in GDM is unknown. Since most women who develop GDM are overweight (8), leptin may already be elevated in these women as they enter pregnancy. Moreover, the glucose intolerance intrinsic to GDM may accentuate this hyperleptinemia. However, this is controversial; as studies have shown higher (19), lower (20) or no difference (21) in leptin concentrations in women with GDM relative to pregnant women with normal glucose tolerance (NGT).

The objective of this study was to determine, in normal and overweight women, if the development of GDM is associated with an alteration in the temporal profile of serum insulin, leptin and cortisol across gestation and into early postpartum as compared to pregnancy with NGT. We hypothesized that by late pregnancy, patterns of all three hormones would be disrupted in women with GDM.

SUBJECTS AND METHODS

Subjects and recruitment. Healthy pregnant and nonpregnant women, 18-45 years of age were recruited from the Women's Ambulatory Clinic at Saint Peter's University Hospital, New Brunswick, NJ. Recruitment was carried out and data was collected on a continuous, rolling basis over a 3-year period. Only normal weight (BMI: 19.8-24.9 kg/m²) and overweight (BMI: 25.0-30.0 kg/m²) were included. Exclusion criteria for all women included pre-existing medical conditions (including type 1 or type 2 diabetes, hypertension or impaired renal function), GDM in a previous pregnancy, and use of medications that interfere with appetite (22). Furthermore, nonpregnant women had to be weight stable during the 3 months prior to the study, not following dietary restrictions (e.g., weight-loss or low-sodium diets), have regular menstrual cycles, and be free of disordered eating (23). This study was approved by the Institutional Review Boards of Rutgers University and Saint Peter's Medical Center. All subjects gave written consent and received monetary compensation for their participation.

Study design. This research was conducted as a single, prospective study. This specific study is a subanalysis of a larger investigation on alterations in sweet taste preferences and food cravings in GDM and their hormonal correlates.

The study design is shown in **Table 1**. Pregnant women completed 3 test sessions during pregnancy (16-20 weeks, 24-28 weeks, and 34-38 weeks gestational age) and 1 test session during early postpartum (6-10 weeks after delivery). Nonpregnant controls were tested at similar intervals. All test sessions were conducted at the hospital clinic the morning after an overnight fast. At all test sessions, body weight was measured with an electronic scale (to the nearest 0.5 kg), and a food frequency questionnaire was

completed during a personal interview with a registered dietitian. The food frequency questionnaire was validated against 3-day diet records ($r=0.87$) and was used in a previous study on GDM (24). Latino foods were included on the questionnaire and interviews were conducted in Spanish by request. At the beginning of each session a blood sample was collected by venipuncture by trained personnel. Blood was collected in SST tubes and kept on ice for no longer than 1-hour. Samples were then centrifuged and serum was stored at -70°C for later assay.

All pregnant women at the clinic are routinely screened for GDM at 24-28 weeks gestation with a 1-hour, 50-gram oral glucose challenge. Women with a positive screen (glucose >7.7 mmol/L) undergo a 3-hour, 100-gram oral glucose tolerance test (OGTT) to confirm their diagnosis (25). Thus, women who developed GDM during the course of this study were identified at 24-28 weeks gestational age. Also, women with GDM were referred to nutritional counseling at the time of their diagnosis and received diet therapy until the end of their pregnancies. A diabetic exchange diet plan was followed (26). Women without GDM received standard nutritional guidance for pregnancy.

Since a major risk factor for GDM is overweight prior to pregnancy (8), we oversampled overweight pregnant women, relative to normal weight pregnant women in order to obtain a sufficient number of women who would eventually develop GDM.

As part of the study protocol, 2 additional 50-gram glucose challenge tests were administered; one at 34-38 weeks gestation and one at 6-10 weeks postpartum (see **Table 1**). Nonpregnant women underwent a total of 3, 50-gram glucose challenges at the same time intervals as the pregnant women. A second blood sample was collected at the end of each glucose challenge. The administration of multiple glucose challenge tests permitted

the monitoring of postprandial glucose and insulin responses across pregnancy. At the end of the study, obstetric history and neonatal birthweight was obtained from the medical charts.

Analytical methods. Serum insulin was measured in duplicate using commercially available double-antibody radioimmunoassays (RIAs) purchased from Linco Research (St. Charles, MO). Inter- and intraassay variations for insulin in pregnant subjects was 6.42% and 6%, respectively. Inter- and intraassay variations for insulin in nonpregnant subjects were 13.6% and 9.54%, respectively. Serum leptin was measured with RIAs for human leptin with reagents from Linco Research (St. Charles, MO). Inter- and intraassay variations for leptin in pregnant subjects were 8.43% and 7.54%, respectively. Inter- and intraassay variations for leptin in nonpregnant subjects were 15.1% and 14.1%, respectively. Serum cortisol was measured with RIAs with reagents from Linco Research (St. Charles, MO). Inter- and intraassay variations for cortisol in pregnant subjects were 7.4% and 4.8%, respectively. Inter- and intraassay variations for leptin in nonpregnant subjects were 12.8% and 6.23%, respectively. Serum estrogen and progesterone were measured with RIAs with reagents from Linco Research (St. Charles, MO). RIAs were performed in duplicate by the Diabetes Research Center of the University of Pennsylvania. Serum glucose was analyzed with a Hitachi 717 analyzer using hexokinase reagents from Amresco (Solon, OH). Analysis of glucose was performed by Accumed Diagnostic Laboratory (South Amboy, NJ). All technicians were blind to the conditions of the experiment.

Homeostatic model assessment of insulin resistance (HOMA-IR) was calculated from basal insulin and glucose values. HOMA-IR is a noninvasive measure of insulin

resistance that has been validated against direct physiological measurement by euglycemic clamp (27).

Data analysis. Dietary data were compiled using NUTRITIONIST PRO software (N-squared Computing, Salem, OR). Mixed model analysis with exchangeable intra-person correlation structure, determined by Akaike's Information Criterion (AIC), was used to assess the temporal trend of the fasting hormone profiles. The fasting hormone profiles and HOMA-IR data were modeled as a function of subject group (GDM, NGT and nonpregnant control), gestational age (16-20 weeks, 24-28 weeks and 34-38-weeks), their interactions, and BMI at entry. Separate analysis of variance (ANOVA) was performed at each time point to probe for differences among groups. Glucose challenge and dietary data were also analyzed by mixed model analysis. Linear contrasts were constructed to compare group differences during pregnancy (the average of the last two pregnancy sessions, 24-28 weeks and 34-38 weeks gestation) and the postpartum session, and between experimental periods (pregnancy vs. postpartum) within each subject group. This was done to simplify the data presentation since initial analyses revealed that the groups did not differ in these measures at 16-20 weeks gestational age, and that group differences observed at 24-28 weeks gestation were maintained at 34-38 weeks gestation. BMI at entry was used as a covariate in all analyses. Statistical analyses were conducted using SAS version 9.1 for the personal computer (SAS Institute Inc, Cary, NC). Statistical significance was set at $\alpha=0.05$. Bonferroni corrections were applied for multiple testing, as appropriate. The criterion, after correction, was set at $p<0.017$.

Receiver operating characteristic (ROC) analysis was used to evaluate the performance of HOMA-IR to detect increased risk of GDM. Sensitivity values were graphed against 1-specificity for all available values in order to obtain ROC plots. ROC analysis, as described by Zweig and Campbell (28), was used to summarize the potential of HOMA-IR to differentiate between the presence and the absence of GDM. The values for AUC/ROC range from 0.5 (indicating no predictive value) to 1 (implying perfect diagnostic accuracy). In general, a value for AUC/ROC of 0.8–0.9 designates moderate diagnostic accuracy, whereas a value for AUC/ROC of 0.9–1.0 signifies very good diagnostic accuracy (29). The ROC analysis was conducted on all survey participants.

RESULTS

Subjects. One hundred and twenty-seven women participated in the study including 108 pregnant women and 19 nonpregnant controls. The majority of pregnant women were recruited into the study at 16-20 weeks gestational age, as planned. A small number of pregnant women (n=15) entered the study at 24-28 weeks gestation. Pregnant women who were overweight at entry (n=78; mean BMI: 28.4 ± 0.3 kg/m²) participated at more than twice the rate of pregnant women who were normal weight at entry (n=30; mean BMI: 23.0 ± 0.7 kg/m²). In total, 15 women developed GDM (BMI= 28.8 ± 2.6 kg/m²), and all of these women were overweight at entry into the study. Thus, 19% of overweight women (14% of all pregnant women) developed GDM during the course of the study. Samples sizes at each stage of the study are depicted in **Figure 1**.

Subject characteristics are shown in **Table 2**. The majority of the participants were Hispanic and the study groups were similar in race/ethnicity. Women with GDM had good pregnancy outcomes. Maternal weight gain was lower in the GDM group relative to the NGT group (p= 0.006) and infant birthweight and the rate of cesarean delivery did not differ between the two groups of women. There were no multiple births in any subject. Two women in the NGT group experienced miscarriages and their data were excluded from the analyses.

Fasting hormone profiles, HOMA-IR and AUC/ROC analysis. Mixed model analysis revealed a main effect of group on leptin and cortisol (p=0.0024 and p<0.0001, respectively). A main effect of gestational age was found for leptin and cortisol (p< 0.001), but not insulin or HOMA-IR. **Figure 2** illustrates the temporal (within-group) pattern in the serum measures as well as between-group differences at each time point.

Fasting insulin remained stable across gestation in women with GDM, and then fell after delivery. Fasting insulin was higher in the GDM group at 24-28 weeks gestation than it was during postpartum ($p=0.004$). In contrast, fasting insulin rose steadily in women with NGT, reached a peak at 34-38 weeks gestation, and then decreased after delivery. Fasting insulin was higher in the NGT group at 34-38 weeks gestation than it was at 16-20 weeks gestation or 6-10 weeks postpartum ($p=0.0002$ and 0.0006). Between-group comparisons showed that at 24-28 weeks gestational age, fasting insulin was higher in women with GDM compared to nonpregnant controls ($p=0.014$). However, women with NGT did not differ in fasting insulin from either women with GDM or nonpregnant controls at this time ($p=0.0989$ and $p=0.0739$, respectively).

Fasting leptin was elevated in both groups of pregnant women at 24-28 weeks and 34-38 weeks gestation relative to the other time points (all p -values <0.01). At 24-28 weeks gestation, leptin was elevated in women with GDM relative to nonpregnant controls ($p=0.022$) and was marginally higher in the GDM group compared to the NGT group (35.0 ± 3.4 ng/mL vs. 26.8 ± 1.2 ng/mL; $p=0.06$). However, fasting leptin did not differ between the NGT group and the other groups of women at 24-28 weeks gestation. By 34-38 weeks gestation, leptin did not differ between the two groups of pregnant women and leptin was higher in both groups of pregnant women relative to nonpregnant controls (both, $p<0.01$).

Fasting cortisol followed a similar pattern in the two groups of pregnant women such that cortisol was elevated in both groups of pregnant women relative to nonpregnant controls at both 24-28 weeks and 34-38 weeks gestation (both, $p<0.0001$). However,

women with GDM and women with NGT did not differ from each other at these time points. After delivery, cortisol was lower in formerly pregnant women than in nonpregnant controls ($p=0.0012$).

HOMA-IR is shown in **Figure 3**. Since HOMA-IR and fasting insulin were highly correlated in this mostly pregnant and Hispanic study population (Pearson Partial Correlation Coefficient: $r=0.98$; $p<0.0001$), the interpretation of the HOMA-IR data was the same as that for fasting insulin. That is, women with GDM experienced peak insulin resistance at 24-28 weeks gestation ($p=0.008$ for the GDM group at 24-28 weeks gestation vs. postpartum), whereas women with NGT experienced peak insulin resistance at 34-38 weeks gestation ($p=0.0005$ for the NGT group at 34-38 weeks gestation vs. 16-20 weeks gestation and $p=0.0124$ for the NGT group at 34-38 weeks gestation vs. 6-10 weeks postpartum). Of importance is that by 34-38 weeks gestation, the level of insulin resistance in NGT women had reached the same level as for GDM women.

Figure 4 shows AUC/ROC for the four different time points of the study. For 16-20 weeks gestation, AUC/ROC curve for HOMA-IR to predict GDM was 0.792 and significantly different than 0.5 ($p=0.0042$). For all other time points of gestation as well as during the postpartum period, the AUC was not significantly different from 0.5. These results indicate that HOMA-IR at 16-20 weeks may be a moderate predictor of the development of GDM.

Oral glucose challenge tests. Fasting, and post-challenge serum insulin and glucose are shown in **Table 3**. During pregnancy, there were no differences in fasting insulin amongst the groups. Women with NGT exhibited lower fasting serum glucose

than controls ($p=0.0027$), but women with GDM did not differ from the other two groups. All fasting glucose values, however, were within the normal range (30)

One hour after oral glucose, serum insulin was elevated in all pregnant women relative to controls ($p<0.0001$), and as expected, values for women with GDM exceeded those of women with NGT ($p=0.0062$). Serum glucose was elevated in women with GDM relative to the other groups ($p<0.0001$), consistent with the presence of mild glucose intolerance in these women (31) (**Table 3**). After delivery, post-challenge, serum insulin fell in both groups of formerly pregnant women such that values did not differ amongst any of the groups. However, mild hyperglycemia persisted into the postpartum period in women with prior GDM as indicated by higher post-challenge serum glucose in these women in comparison to the other groups (both, $p<0.01$).

Diet variables. **Table 4** shows the reported energy and macronutrient intakes of the women during pregnancy and postpartum. During pregnancy, reported energy intake was lower in women with GDM than in women with NGT ($p=0.0006$). In addition, women with GDM reportedly consumed more protein ($p=0.002$), but less carbohydrate ($p=0.0004$) (as % of energy) than women with NGT. Simple sugars as a percentage of carbohydrate energy was also lower in women with GDM than in women with NGT or controls (both, $p<0.01$).

Few between-group differences were observed during the postpartum period, except that reported intake of simple sugars (as % of carbohydrate energy) was lower in women with previous GDM than the controls ($p=0.003$). Within-group dietary parameters did not differ between pregnancy and post-partum.

DISCUSSION

This study documented the temporal profile of serum insulin, leptin, cortisol and insulin resistance across pregnancy and early postpartum in women who developed GDM compared to pregnant women with NGT. As expected, women with NGT showed hormone profiles typical of healthy pregnancy where values rose progressively during gestation then fell to baseline after delivery (7). Contrary to our expectations, the temporal profile of these hormones did not differ markedly in women with GDM relative to pregnant women with NGT. The most likely explanation for this finding is that the women studied here developed mild GDM that did not lead to large fluctuations in these hormones. The determination of mild GDM in these women was based on several indicators including the presence normal fasting glucose values, modest insulin resistance (according to HOMA-IR) and moderate hyperglycemia after oral glucose. Nevertheless, serum leptin tended to rise more sharply in women who developed GDM and was marginally higher in women with GDM relative to women with NGT at 24-28 weeks gestational age. Serum leptin did not rise further in women with GDM, and by 34-38 weeks gestational age, values did not differ between the two groups of pregnant women.

Previous studies have raised doubts about whether or not leptin is elevated in pregnancy complicated by GDM. Two studies reported that women with GDM had higher leptin than women without GDM when measured at 28 weeks and 33 weeks gestational age, respectively (19, 32). However, two other studies reported no difference in plasma leptin between women with and without GDM when measured in late pregnancy (21, 33). The present results are consistent with these seemingly contradictory findings, in that women with GDM had marginally higher leptin than women with NGT

at 24-28 weeks gestation, but this difference disappeared by late pregnancy. Because we measured leptin at multiple time points during pregnancy, we were able to establish these temporal changes in leptin that were missed in previous studies. A noteworthy feature of our study was that the women with GDM received the same therapeutic care – all received diet therapy. Except for one other study in which all women with GDM received diet therapy (21), previous studies on leptin combined women receiving different treatments into a single study group (34), or did not specify treatment mode (20, 35). The absence of clinically well-defined subject groups might have complicated the interpretation of these studies and contributed to the lack of consensus in this literature.

A large epidemiological study suggested that higher leptin early in pregnancy was associated with the development of GDM later in pregnancy (36). In that study, women in the highest tertile for leptin at gestational weeks 13 had a 4.7-fold higher risk of developing GDM relative to women in the lowest tertile, after controlling for confounding variables including body weight. In our study, leptin was not elevated at 16-20 weeks gestation in women who later developed GDM, although leptin rose rapidly in these women after this time point. However, their baseline time point was earlier than ours (13 weeks vs. 16-20 weeks) and women in our study were only mildly diabetic; both circumstances could have possibly contributed to this disparity.

In nonpregnant individuals with type 2 diabetes, leptin reflects the degree of adiposity not the severity of glucose intolerance (17, 18). This relationship may not be as clear in GDM. Indeed, women who developed GDM in our study were of comparable weight at entry to those who did not develop GDM. However, we controlled for BMI at entry in the data analyses, and serum leptin still tended to rise more sharply in the GDM

group relative to the other groups at 24-28 weeks gestational age. On the other hand, women with GDM gained less weight during pregnancy than women with NGT which could have exerted a dampening effect on leptin concentrations in the GDM group. It is recognized that pregnancy weight gain is only a surrogate of maternal adiposity, and variation in the rate of maternal fat gain across pregnancy might also play a role. We cannot rule out the possibility that maternal adiposity might have had a greater impact on serum leptin if more obese pregnant women had been studied.

Obstetric outcomes in the women with GDM indicated that they maintained good diabetic control and successfully managed their disease by diet alone. Pregnancy weight gain in women with GDM was in the recommended range for women who are overweight at conception (37). Also, infant birthweight and the incidence of caesarean delivery did not differ between the two groups of women. Women with GDM reported lower energy intakes as well as lower carbohydrate and simple sugar intake (as a percentage of carbohydrate energy) than women with NGT. These parameters were in accord with the dietary guidance the women with GDM received, and were comparable to values from our previous work in a similar cohort of women (24). Although food intake might have been underreported by women with GDM, their energy intakes were reasonable in light of their pregnancy weight gain. To our knowledge, studies documenting the self-reported intakes and pregnancy outcomes of women receiving conventional diet therapy for GDM are rare. Although the usefulness of diet therapy in GDM has recently been questioned (38, 39), our findings suggest that dietary counseling is important in the overall medical management of mild GDM.

This study had several strengths and weaknesses. The study population was primarily Hispanic (52-79%, depending on the group); therefore conclusions arising from the study are mainly applicable to a predominantly Hispanic population. The overall prevalence rate for GDM in our study was 14%, twice the reported rate for the general obstetric population (7%) (3). However, sample size in the GDM group was low which could have compromised the power to detect differences in some of the statistical tests. Although attrition rates varied among the groups (see **Figure 1**), results from women who left the study prematurely and those who remained did not differ (data not shown). The overall attrition rate for the study was modest (33%).

The etiological significance of early hyperleptinemia in GDM pregnancy is presently unknown. In nonpregnant individuals, leptin plays a role in glucose homeostasis and may be involved in the development of insulin resistance (40). It is plausible that leptin may have similar functions in pregnancy. Indeed, leptin is positively correlated with the pregnancy rise in insulin and cortisol that is followed by a progressive decline in insulin sensitivity with advancing gestation. In pregnancy without GDM, serum leptin reflects the pattern of maternal fat deposition and mobilization as gestation progresses to term. However, factors other than adiposity modulate leptin in pregnancy. Principal among these are the steroid hormones, cortisol and estrogen, which are potent stimulators of leptin release (12). The emergence of insulin resistance and disordered glucose metabolism early in the gestational period may shift the time course of leptin release in GDM pregnancy. Whether leptin serves as a marker or predictor of GDM is an important question that deserves further investigation.

Finally, the observation that the degree of insulin resistance of NGT reaches that of GDM near term is a novel finding. GDM is considered a condition of enhanced insulin resistance, which we confirmed at an earlier gestational age. However, it appears that normal pregnancies achieve a similar level of insulin resistance by the time they reach term. Insulin resistance, therefore, seems to be important for all pregnancies in later gestation, perhaps reflecting the needs of the maturing fetus. Earlier onset of insulin resistance may be the feature of importance for GDM, and could be a predictor for the development of GDM, as evidenced by the current study's ROC analysis. Targeting the early identification of insulin resistance may be better than current strategies for predicting adverse outcomes relevant to GDM, especially if insulin resistance is a feature of normal gestation near or at term.

LITERATURE CITED

1. Metzger BE, Coustan DR. Summary and recommendations of the fourth international workshop-conference on gestational diabetes mellitus: the organizing committee. *Diabetes Care* 1998;21:B161-B167.
2. American Diabetes Association. Nutrition recommendations and interventions for diabetes. *Diabetes Care*, 2007:S48-S65.
3. American Diabetes Association. Gestational diabetes mellitus. *Diabetes Care* 2004;27:S88-S90.
4. Goldman M, Kitzmiller J, Abrams B, Cowan RM, Laros J, R.K. Obstetric complications with GDM: effects of maternal weight. *Diabetes* 1991;40 (Supplement 2):79-82.
5. Cousins L. Obstetric complications. In: *Diabetes mellitus and pregnancy: principles and practice*. 2nd ed. New York: Churchill Livingstone, 1995.
6. Langer O, Hod M. Management of gestational diabetes mellitus. *Obstet Gynecol Clin North Am* 1996;23:137-159.
7. Boden G. Fuel metabolism in pregnancy and in gestational diabetes mellitus. *Obstet Gynecol Clin North Am* 1996;23:1-10.
8. Jovanovic-Peterson L, Peterson CM. Review of gestational diabetes mellitus and low-calorie diet and physical exercise as therapy. *Diabetes Metab Res* 1996;12:287-308.
9. Stanley K, Fraster R, Bruce C. Physiological changes in insulin resistance in human pregnancy: longitudinal study with the hyperinsulinaemic euglycaemic clamp technique. *Brit J Obstet Gynaec* 1998;105:756-759.
10. Di Cianni G, Miccoli R, Volpe L, Lencioni C, Del Prato S. Intermediate metabolism in normal pregnancy and in gestational diabetes. *Diabetes Metab Res* 2003;19:259-270.
11. Zakrzewska KE, Cusin I, Sainsbury A, Rohner-Jeanrenaud F, B. J. Glucocorticoids as counterregulatory hormones of leptin: toward an understanding of leptin resistance. *Diabetes* 1997;46:717-719.
12. Henson MC, Castracane VD. Leptin in pregnancy: an update. *Biol Reprod* 2006;74:218-229.

13. Highman TJ, Friedman JE, Huston LP, Wong WW. Longitudinal changes in maternal serum leptin concentrations, body composition, and resting metabolic rate in pregnancy. *Am J Obstet Gynecol* 1998;178:1010-1015.
14. Catalano PM, Huston L, Amini SB, Kalhan SC. Longitudinal changes in glucose metabolism during pregnancy in obese women with normal glucose tolerance and gestational diabetes mellitus. *Am J Obstet Gynecol* 1999;180:903-916.
15. Catalano PM, Kirwan JP, Haugel-de Mouzon S, King J. Gestational diabetes and insulin resistance: role in short- and long-term implications for mother and fetus. *J Nutr* 2003;133:1674S-1683S.
16. Butte NF. Carbohydrate and lipid metabolism in pregnancy: normal compared with gestational diabetes mellitus. *Am J Clin Nutr* 2000;71:1256S-1261S.
17. Mohamed-Ali V, Pinkney JH, Panahloo A, Goodrick S. Relationships between plasma leptin and insulin concentrations, but not insulin resistance, in non-insulin-dependent (type 2) diabetes mellitus. *Diabet Med* 1997;14:376-380.
18. Tasaka Y, Yanagisawa K, Iwamoto Y. Human plasma leptin in obese subjects and diabetics. *Endocr J* 1997;44:671-676.
19. Kautzky-Willer A, Pacini G, Tura A, et al. Increased plasma leptin in gestational diabetes. *Diabetologia* 2001;44:164-172.
20. Festa A, Shnawa N, Krugluger W, Hopmeier P, Schernthaner G, Haffner SM. Relative hypoleptinaemia in women with mild gestational diabetes mellitus. *Diabet Med* 1999;16:656-662.
21. Simmons D, Breier BH. Fetal overnutrition in polynesian pregnancies and in gestational diabetes may lead to dysregulation of the adipoinsular axis in offspring. *Diabetes Care* 2002;25:1539-1544.
22. Schiffman SS. Drugs influencing taste and smell perception. In: *Smell and taste in health and disease*. New York: Raven Press, 1991.
23. Garner DM, Olmsted MP, Polivy J. Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *Int J Eat Disorder* 1983;2:15-33.
24. Tepper BJ, Seldner AC. Sweet taste and intake of sweet foods in normal pregnancy and pregnancy complicated by gestational diabetes mellitus. *Am J Clin Nutr* 1999;70:277-284.
25. Carpenter MW, Coustan DR. Criteria for screening tests for gestational diabetes. *Am J Obstet Gynecol* 1982;144:768-773.

26. American Diabetes Association. Meal planning exchange lists. <http://www.diabetes.org/nutrition-and-recipes/nutrition/exchangelist.jsp>.
27. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985;28:412-419.
28. Zweig MH, Campbell G. Receiver-operating characteristic (ROC) plots: A fundamental evaluation tool in clinical medicine. *Clin Chem* 1993;39:561-577.
29. Hanley JA, McNeil BJ. The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology* 1982;143:29-36.
30. Piché M-E, Arcand-Bossé J-F, Després J-P, Pérusse L, Lemieux S, Weisnagel SJ. What is a normal glucose value? *Diabetes Care* 2004;27:2470-2477.
31. American Diabetes Association. Standards of medical care in diabetes. *Diabetes Care* 2007;30:S4-S41.
32. Buhling KJ, Harder T, Sehouli J, Nanz J, Plagemann A, Dudenhausen JW. Independent association between leptin and blood pressure during third trimester in normal and gestational diabetic pregnancies. *Eur J Obstet Gyn R B* 2005;119:180-184.
33. Lappas M, Yee K, Permezel M, Rice GE. Release and regulation of leptin, resistin and adiponectin from human placenta, fetal membranes, and maternal adipose tissue and skeletal muscle from normal and gestational diabetes mellitus-complicated pregnancies. *J Endocrinol* 2005;186:457-465.
34. McLachlan KA, O'Neal D, A. J, Alford FP. Do adiponectin, TNF α , leptin and CRP relate to insulin resistance in pregnancy? Studies in women with and without gestational diabetes, during and after pregnancy. *Diabetes Metab Res* 2006;22:131-138.
35. Ategbo JM, Grissa O, Yessoufou A, et al. Modulation of adipokines and cytokines in gestational diabetes and macrosomia. *J Clin Endocr Metab* 2006;91:4137-4143.
36. Qiu C, Williams MA, Vadachkoria S, Frederick IO, Luthy DA. Increased maternal plasma leptin in early pregnancy and risk of gestational diabetes mellitus. *Obstet Gynecol* 2004;103:519-525.
37. Food and Nutrition Board. Nutrition during pregnancy. Part 1: Weight gain. Washington, D.C.: Institute of Medicine, National Academy of Sciences, 1990.

38. Landon MB, Thom E, Spong CY, et al. A planned randomized clinical trial of treatment for mild gestational diabetes mellitus. *J Matern Fetal Neonatal Med* 2002;11:226-231.
39. Turok DK, Ratcliff SD, Baxley EG. Management of gestational diabetes mellitus. *Am Fam Physician* 2003;68:1775-1776.
40. Ceddia RB, Koistinen HA, Zierath JR, Sweeney G. Analysis of paradoxical observations on the association between leptin and insulin resistance. *FASEB Journal* 2002;16:1163-1176.

TABLE 1

Study Protocol

	Pregnancy			Postpartum
	16-20 wk	24-28 wk ¹	34-38 wk	6-10 wk
Procedure				
Fasting blood sample, diet assessment & body weight measurement	X	X	X	X
Blood sample 1-h after 50-g oral glucose		X	X	X

¹ All pregnant women undergo a routine 1-h, 50-g glucose screen for GDM. Women with a positive screen proceed to a 3-h oral glucose tolerance test to confirm the diagnosis.

TABLE 2

Subject characteristics

	GDM	NGT	Nonpregnant controls
Total Enrollment	15	93	19
Age at entry (yr) ¹	29.2 ± 3.1	26.2 ± 0.7	27.1 ± 1.3
BMI at entry (kg/m ²) ¹	28.8 ± 2.6 ^a	27.0 ± 3.5 ^a	24.5 ± 0.5 ^b
Total pregnancy weight gain (kg) ¹	8.3 ± 1.5 ^a	13.9 ± 0.6 ^b	—
Ethnicity (%)			
Hispanic	60	52	79
Caucasian	20	20	11
African American	13	20	5
Asian	0	5	0
Other	7	3	5
Cesarean Delivery (%) ¹	27	30	—
Gestational age (wk) ¹	40.0 ± 0.6	39.4 ± 0.2	—
Infant birthweight (g) ¹	3321 ± 125	3325 ± 53	—
Breastfeeding (%) ¹	73	77	—

¹ Values are means ± SEM. Values in the same row with different superscripts are different at p<0.05.

TABLE 3Serum insulin and glucose during fasting and one-hour following a 50-g glucose challenge^{1,2}

	Pregnancy ³			Postpartum (6-10 wk pp)		
	GDM (n = 15)	NGT (n = 93)	Control (n = 19)	GDM (n = 12)	NGT (n = 61)	Control (n = 12)
Insulin (μ U/mL)						
Fasting	26.1 \pm 2.6	21.7 \pm 1.0	13.8 \pm 2.4	12.9 \pm 3.8 ⁴	15.9 \pm 1.6 ⁴	15.2 \pm 4.0
Post-glucose challenge	116.9 \pm 8.8 ^a	88.8 \pm 3.5 ^b	58.3 \pm 7.7 ^c	54.1 \pm 12.9 ⁴	44.4 \pm 5.1 ⁴	41.5 \pm 13.7
Glucose (mmol/L)						
Fasting	4.4 \pm 0.1 ^{a,b}	4.3 \pm 0.1 ^a	4.7 \pm 0.1 ^b	5.0 \pm 0.2 ⁴	4.8 \pm 0.1 ⁴	4.6 \pm 0.2
Post-glucose challenge	8.1 \pm 0.3 ^a	6.1 \pm 0.1 ^b	5.6 \pm 0.3 ^b	7.3 \pm 0.5 ^a	5.2 \pm 0.2 ^{b,4}	5.1 \pm 0.5 ^b

¹ Values are means (\pm SE) estimated by the mixed model analysis, adjusted for BMI at entry.² Between-group differences were separately examined during pregnancy and postpartum using linear contrasts with mixed model analysis. Values within each experimental period with different superscripts are different at $p < 0.017$ (with Bonferroni corrections for multiple testing).³ Values during pregnancy were averaged across the 2 glucose challenge tests (24-28 wk and 34-38 wk gestation).⁴ Different from pregnancy time point at $p < 0.017$.

TABLE 4Reported daily energy and macronutrient intake based on a food frequency questionnaire^{1,2,3}

	Pregnancy ⁴			Postpartum (6-10 wk pp)			
	GDM (n = 15)	NGT (n = 93)	Control (n = 19)	GDM (n = 12)	NGT (n = 61)	Control (n = 12)	
Energy							
	(kJ/d)	7880 ± 831 ^a	10711 ± 331 ^b	7143 ± 791 ^a	9594 ± 1141	9535 ± 510	6622 ± 1141
	(kcal/d)	1883 ± 199 ^a	2560 ± 79 ^b	1707 ± 189 ^a	2293 ± 273	2279 ± 121	1583 ± 273
Protein (% energy)		17.5 ± 0.7 ^a	15.0 ± 0.3 ^b	15.9 ± 0.7 ^{a,b}	16.7 ± 1.0	15.9 ± 0.4	16.1 ± 1.0
Fat (% energy)		39.5 ± 1.6 ^a	35.2 ± 0.6 ^b	36.2 ± 1.1 ^{a,b}	38.3 ± 2.2	37.0 ± 0.8	32.8 ± 1.6
Carbohydrate (% energy)		44.8 ± 1.7 ^a	51.6 ± 0.7 ^b	48.3 ± 1.6 ^{a,b}	46.7 ± 2.4	48.6 ± 1.1	51.7 ± 2.4
Simple sugar (% CHO energy) ⁵		31.1 ± 2.6 ^a	41.4 ± 1.0 ^b	42.2 ± 2.5 ^b	32.8 ± 3.6 ^a	40.8 ± 1.6 ^{a,b}	48.8 ± 3.6 ^b

¹ Values are means (± SE) estimated by the mixed model analysis, adjusted for BMI at entry.² Between-group differences were separately examined during pregnancy and postpartum using linear contrasts with mixed model analysis.

Values within each experimental period with different superscripts are different at p<0.017 (with Bonferroni corrections for multiple testing).

³ Furthermore, within group comparison between experimental periods were made. There were no within group differences.⁴ Values during pregnancy were averaged across the last two sessions (24-28 wk and 34-38 wk gestation).⁵ Simple sugars exclude lactose, galactose and maltose.

FIGURE LEGENDS

Figure 1 Diagram indicating sample sizes at each stage of the study.

Figure 2 Fasting serum insulin, leptin and cortisol (mean \pm SE) across pregnancy and postpartum in women with GDM (\blacktriangle), women with NGT (\blacksquare) and nonpregnant controls (\circ). Mixed model analysis revealed a main effect of group on leptin and cortisol ($p=0.0024$ and $p<0.0001$, respectively). A main effect of gestational age was found for leptin and cortisol ($p<0.001$), but not for insulin. Within-group effects across sessions are indicated by lower-case letters. Values with different lower case letters are significantly different. Between-group effects were tested with ANOVA at each session. Circled values are significantly different from each other at each session. The significance level for all comparisons was set at $p=0.017$, after adjusting for the Bonferroni correction. Sample sizes at each session are indicated in **Figure 1**.

Figure 3 Homeostatic assessment of insulin resistance (HOMA-IR = (fasting insulin ($\mu\text{U/mL}$) x fasting glucose (mmol/L) / 22.5)) (mean \pm SE) across pregnancy and postpartum in women with GDM (\blacktriangle), women with NGT (\blacksquare) and nonpregnant controls (\circ). A main effect of gestational age was not found for HOMA-IR. Within-group effects across sessions are indicated by lower-case letters. Values with different lower case letters are significantly different. Between-group effects were tested with ANOVA at each session. Circled values are significantly different from each other at each session. The significance level for all comparisons was set at $p=0.017$, after adjusting for the Bonferroni correction. Sample sizes at each session are indicated in **Figure 1**.

Figure 4 AUC/ROC for the four time points of the study. At 16-20 weeks gestation, AUC/ROC curve for HOMA-IR to predict GDM was 0.792 and significantly different than 0.5 ($p=0.0042$). For all other time points of gestation as well as during the postpartum period, the AUC was not significantly different from 0.5. These results indicate that HOMA-IR at 16-20 weeks was a moderate predictor of the development of GDM.

Figure 1

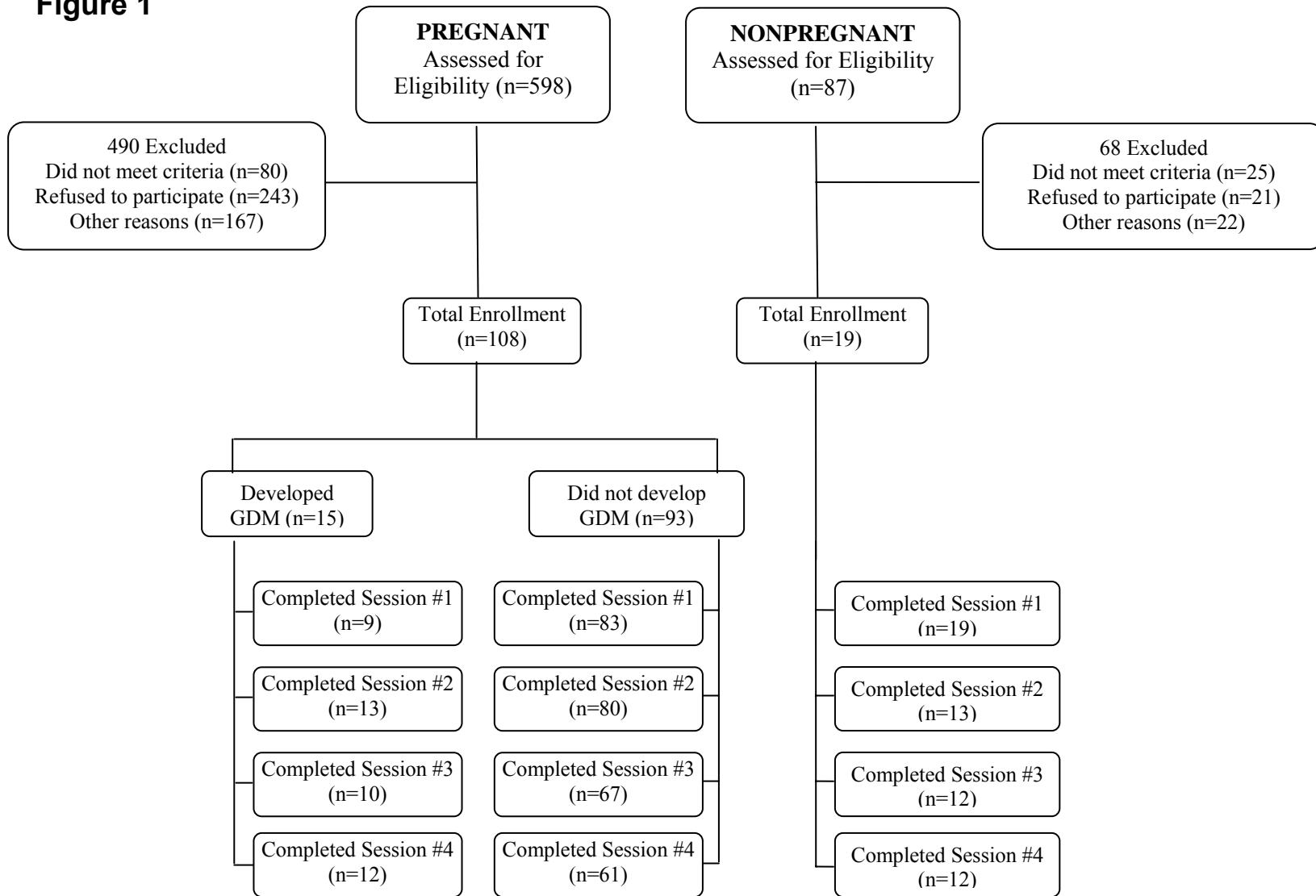


Figure 2

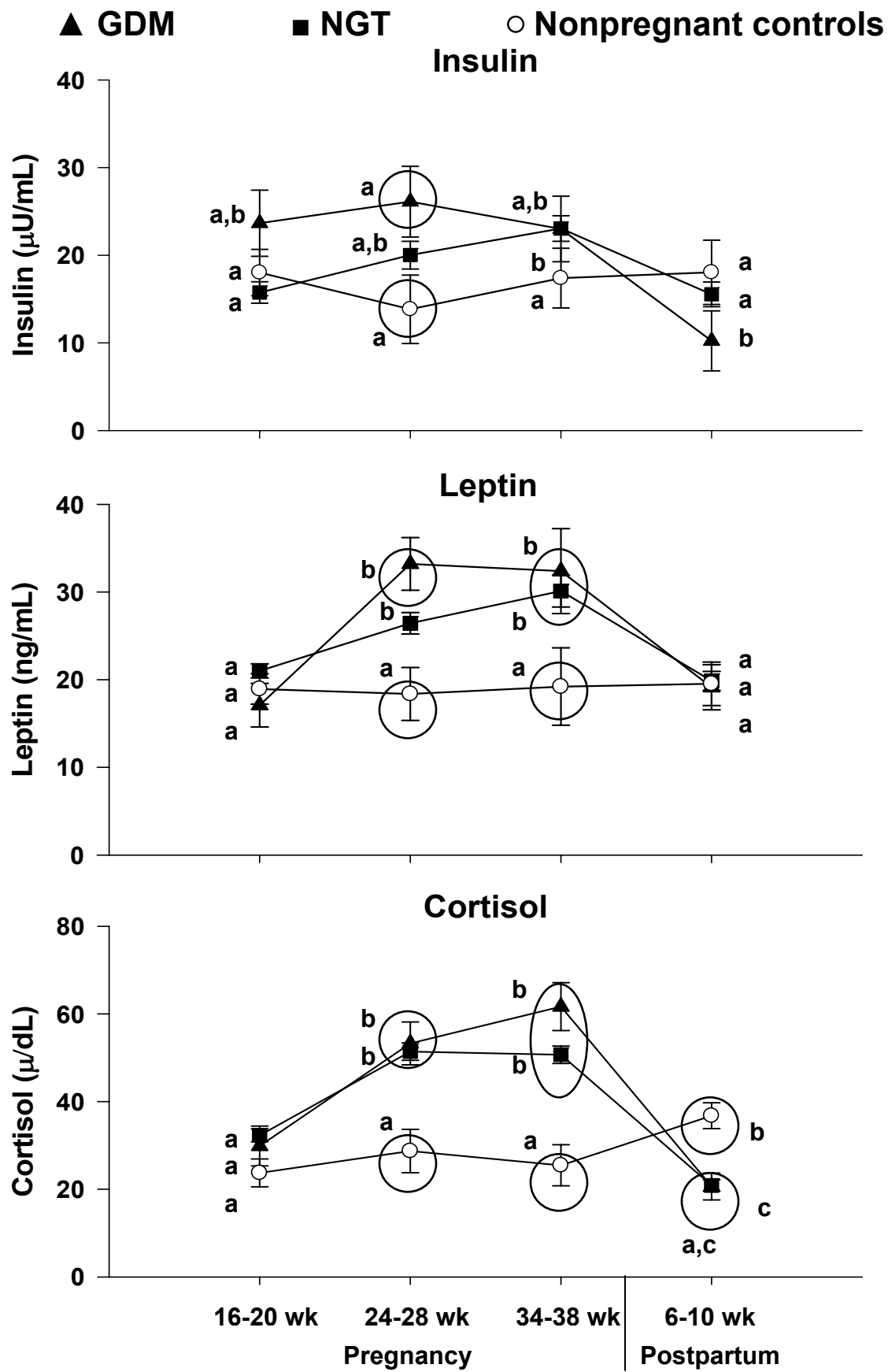


Figure 3

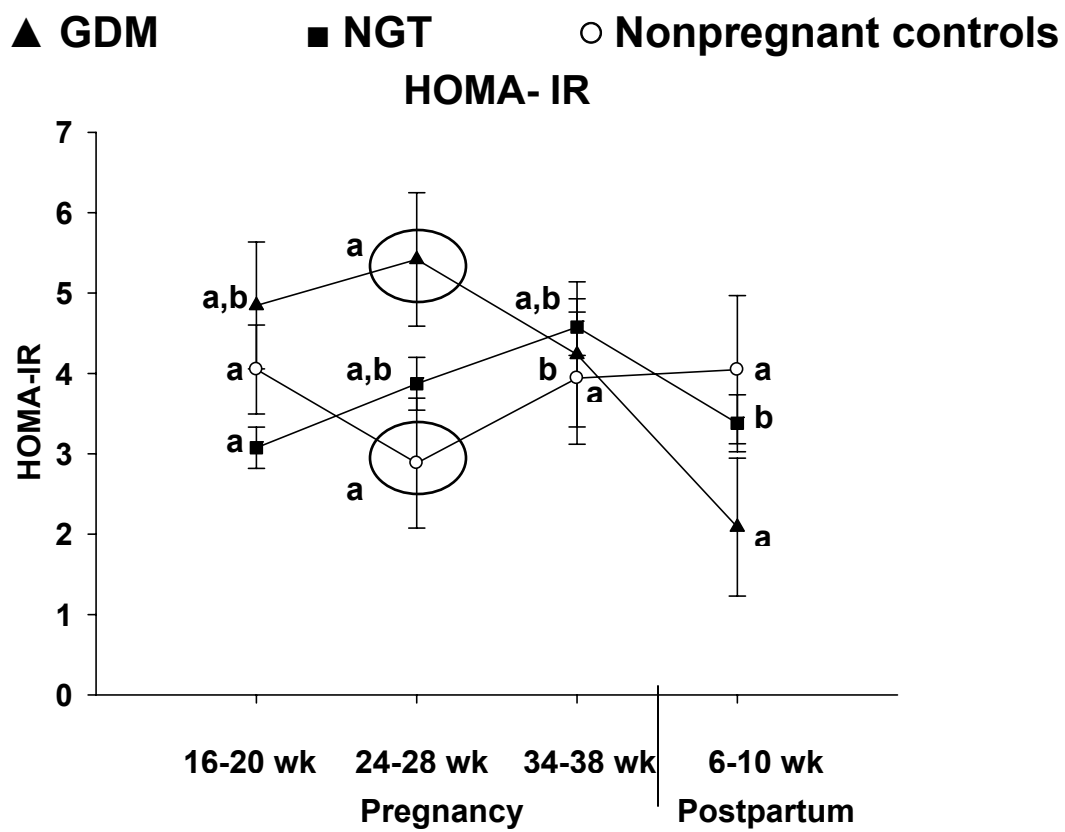
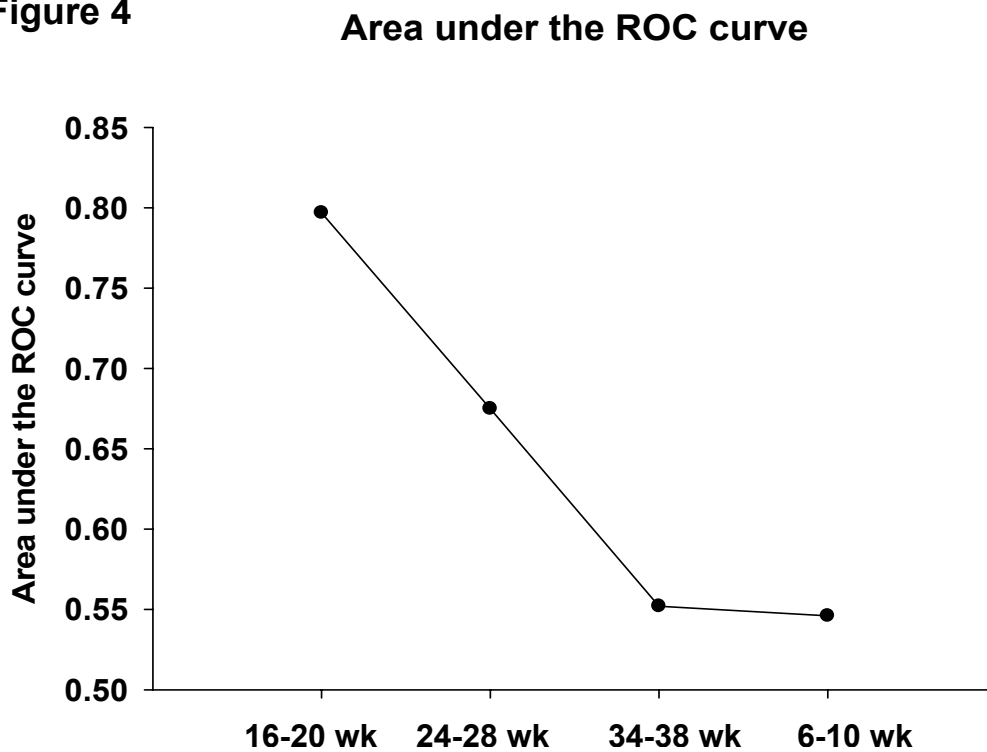


Figure 4



**SECTION B. Changes in Sweet Taste Across Pregnancy and Its Relationship to
Endocrine Parameters in Women with and Without Mild Gestational Diabetes
Mellitus**

ABSTRACT

Background: Gestational diabetes mellitus (GDM) is glucose intolerance diagnosed during pregnancy. Changes in sweet taste in GDM could alter the acceptability of sweet foods and influence dietary intake. The time course of these changes has not been clearly documented and the relationships of the taste changes to endocrine and metabolic parameters in GDM have not been studied.

Objectives: To compare the effects of pregnancy and GDM on perception and preference of sweet taste and relate these alterations to endocrine and metabolic profiles.

Design: A prospective design was used. Subjects included 93 pregnant women with NGT, 15 pregnant women who developed GDM and 19 nonpregnant controls. Subjects evaluated sweetened milks, with 0-20% sucrose and 0-10% fat, and glucose solutions (10–160 mmol/L) at 16-20 weeks, 24-28 weeks, and 34-38 weeks gestation, and 6-10 weeks postpartum. Fasting insulin and leptin were measured at these same time points.

Results: GDM did not influence the perceived intensity of the sweetened milks or the glucose solutions although pregnancy increased the liking of the glucose solutions at 24-28 weeks and 34-38 weeks gestation relative to nonpregnant controls. At 34-38 weeks gestation, women with GDM liked the 5% sucrose-sweetened milk more than nonpregnant controls for all attributes (p-values 0.0054-0.0147) as well as the creaminess and flavor of the 5% sucrose-sweetened milk more than women with NGT (p=0.008 and 0.0067, respectively). Women with GDM also liked the creaminess of 10% sucrose-sweetened milk, more than women with NGT (p=0.0129). In women with GDM, fasting insulin was positively correlated with the averaged sweetness liking rating of the glucose solutions ($R^2=0.63$, p=0.0037) and fasting leptin was positively correlated with the

sweetness liking rating of the 10% sucrose-sweetened milk ($R^2=0.42$, $p=0.0166$). These relationships were observed in women with GDM at 24-28 weeks gestation, but not at any other time points. No correlations involving these measures were observed in women with NGT or nonpregnant controls.

Conclusions: Women with GDM exhibited increased liking for sucrose-sweetened milks at moderate sweetness concentrations (5-10% sucrose) that occur late in pregnancy. However, correlations between sweetness liking ratings and serum leptin and insulin in GDM occur earlier in gestation.

KEY WORDS: Gestational Diabetes, Sweet Taste, Insulin, Leptin

INTRODUCTION

Gestational Diabetes Mellitus (GDM) is hyperglycemia first recognized during pregnancy (1) that affects 7% of pregnant women (2). It is a risk factor for adverse fetal outcomes, and is highly correlated with the development of type 2 diabetes later on in life (3, 4). Dietary management of GDM follows the same strategies as that for type 2 diabetes, i.e. controlling of total calories and carbohydrates and maintenance of normal blood sugar levels (5, 6). However, the optimum diet for pregnant women with GDM has yet to be identified, and compliance with traditional diet therapies is low (7).

Since diabetes is a condition of abnormal glucose metabolism, it could affect the perception of simple carbohydrates and sweetened foods and beverages and alter dietary compliance. Studies suggest that type 2 diabetes influences sweet taste function and liking of sweet foods. For example, Perros et al showed that newly diagnosed type 2 diabetic individuals had a reduced taste response to glucose (8). In another study, subjects with type 2 diabetes and their healthy first-degree relatives had decreased sensitivity to glucose and sucrose (i.e. higher thresholds) than non-diabetic controls (9). These findings suggest that sweet taste changes in type 2 diabetes may be present, or precede, the development of diabetic symptoms. Another study investigating perception and liking of beverages in established type 2 diabetes, showed no alterations in perception or pleasantness of these beverages, but diabetic subjects who gave higher ratings of pleasantness to sweetened beverages, reportedly consumed more dietary sweetness than non-diabetic controls (10). Since women with GDM are also pregnant, some of the taste changes in GDM may be related to pregnancy. Overall gustatory function is lower in pregnant women during the first trimester, relative to other parts of

pregnancy (11, 12). However, bitter and salt taste detection (at threshold) and preference may be more affected than sweet taste (13, 14). One study failed to show a pregnancy effect on taste perception of higher concentrations of sweetness. However, that same study reported that pregnant women consumed more sweet foods during their second trimester in contrast to women at any other pregnancy time point (15). This is in agreement with the cravings literature showing that cravings for sweets are elevated at this same time point (16, 17). Thus, there is evidence that healthy pregnant women have an increased desire for sweetness in mid-gestation.

Nevertheless, the literature on taste perception changes in pregnancy has several weaknesses. Frequently, rather than following the same women through gestation, they compare one group of women at one time point in pregnancy to a separate group of women, at another time point in pregnancy (14, 15, 18). Other studies depend on postpartum, retrospective reports of taste or dietary measures (19, 20). These methodological weaknesses make these findings difficult to interpret.

If GDM reflects the combined effect of diabetes and pregnancy, then one would expect sweet taste to be elevated in women with NGT at mid-pregnancy, and these values to be further elevated in women with GDM due to their diabetic state. Only one previous study has examined both taste and diet in GDM (21). Tepper & Seldner examined taste and dietary measures early in the third trimester (within one month of diagnostic testing) and 12 weeks postpartum, in women with GDM and in women with NGT. Contrary to expectations, women with NGT showed a lower preference for 10% sucrose-sweetened milk during pregnancy than in the postpartum period, a finding that concurs with some prior research (18), but conflicts with other (15). In contrast, women with GDM showed

higher liking for the 10% sucrose-sweetened milk relative to NGT women during pregnancy, although values in women with GDM did not exceed those observed in nonpregnant controls at any time point during the study. Women with GDM did not differ from women with NGT in intensity or liking of glucose solutions. These results suggest that GDM moderately increases the liking of a sweet-fat beverage relative to women without GDM, but does not influence ratings of simple sugar in solution. The reasons for this discrepancy are presently unknown.

Tepper & Seldner examined pregnant women at 28-32 weeks gestation. Thus it is possible that the peak sweetness preference, which is thought to accompany the onset of GDM, was missed. One objective of the current study was to track taste changes across pregnancy, to determine when these taste changes arise. It is possible that sweet taste changes develop around the time of diagnosis when disease is untreated and symptoms are at their greatest.

The relationship between endocrine factors and changes in sweet taste in normal pregnancy are poorly understood. Some past work suggests that overall taste changes serve a protective function, or promote the ingestion of nutrients required by the mother or developing fetus (19). In normal pregnancy, levels of insulin, leptin, cortisol, estrogen and progesterone all rise, typically peak late in gestation, and then fall after delivery (22, 23). It is possible that reported taste changes in pregnancy may be linked with this change in endocrine profile. Studies in rodents have suggested that estrogen increases the threshold for gustatory detection of sucrose (24). Human research has not yet provided evidence for such a relationship (25, 26), although one study showed a lower preference for sweet taste in pregnant women as compared to nonpregnant women. The authors

focused on the role of progesterone and estrogen in this discrepancy, suggesting that high levels of progesterone may decrease estrogen's effectiveness to activate taste mechanisms (18).

Both leptin and insulin may be involved in sweet taste. Some studies indicate that hyperinsulinemia may be related to hunger and sweet taste preference (27, 28). Human research has demonstrated associations between high leptin levels and decreased preference for chocolate and a high-fat, low sugar mixture in obese women (29), but associations between leptin and increased palatability of a high carbohydrate diet in subjects exhibiting a wide range of BMI (30). Two obese mouse models (ob/ob and db/db) show increased behavioral responses to sweet taste. Leptin injections have shown to attenuate sweet taste responses in lean and ob/ob mice, but not db/db mice that are leptin resistant (31). Thus, it is possible that these hormones contribute to sweet taste differences found in models of insulin and/or leptin resistance.

Another key finding in the Tepper & Seldner study (21) was a positive correlation between plasma glucose one hour following a glucose load and sweetness liking ratings of glucose solutions and intake of fruit and fruit juices in women with GDM but not NGT (21). This discovery suggests that the degree of glucose intolerance or other underlying metabolic factors is related to preference for sweet taste and sweet food intake in GDM. The possibility of associations between other measures of metabolism and taste changes during a GDM pregnancy prompted further evaluation of these relationships.

This study followed women who developed GDM and who did not develop GDM over the courses of gestation. The objectives were two-fold: 1) To identify the time course and magnitude of taste changes in the two groups of pregnancy women, and 2) To

relate these taste changes to endocrine and metabolic profiles associated with pregnancy and GDM, focusing primarily on insulin and leptin. Women with NGT would have a higher preference for sweetness at mid-pregnancy than at other time points, and the sweet preference in GDM would exceed that found in normal pregnancy at 24-28 weeks gestation (time of GDM diagnosis) when diabetes symptoms were at their peak. Additionally, in women with GDM, sweet preference would be correlated with elevations in insulin and leptin, hormones which are elevated in pregnancy and diabetes.

SUBJECTS AND METHODS

Subjects and recruitment. This sub-study is a part of a larger, prospective study designed to investigate changes in sweet taste, endocrine and metabolic correlates during gestation in pregnant women with normal glucose tolerance and women who developed GDM, therefore the same subjects were tested throughout. Please refer to **Chapter 2** for recruitment procedures, subject characteristics and general methods.

Study design. The sub-study was designed to prospectively document taste changes and related endocrine parameters during gestation and early postpartum in pregnant women with normal glucose tolerance and women who developed GDM.

All pregnant women at the clinic are routinely screened for GDM at 24-28 weeks gestation using a 1-hour, 50-gram oral glucose challenge. Women with a positive screen (glucose >140mg/dL) undergo a 3-hour, 100-gram oral glucose tolerance test to confirm their diagnosis (32). Thus, women who developed GDM during the course of this study were identified at 24-28 weeks gestational age. Also, women with GDM were referred to nutritional counseling at the time of their diagnosis and received diet therapy until the end of their pregnancies. A diabetic exchange diet plan was followed, which incorporated carbohydrate control within the context of sufficient calories for proper maternal health and fetal growth (33). Women without GDM received standard nutritional guidance for pregnancy.

Taste Stimuli. The two primary taste stimuli were glucose solutions and strawberry milks that were used in our previous study in GDM (21). Five glucose solutions prepared with dextrose dissolved in spring water (0.01-0.16M dextrose) (Fisher Scientific, Fair Lawn, NJ). Glucose was tasted because it is a simple carbohydrate, is the

primary fuel for the body and previous studies in type 2 diabetes showed taste changes with this stimulus (8,9). Sucrose-sweetened strawberry-flavored milk was used as a tastant because it is a sweet-fat stimulus that simulates a real beverage. Strawberry milks were prepared using nonfat dry milk (Carnation) reconstituted in spring water according to packaged directions. Twelve samples were prepared by substituting 0%, 5%, or 10% (wt:vol) bland vegetable oil (Hunt-Wesson Inc, Fullerton, CA), and 0%, 5%, 10% or 20% (wt:vol) sucrose (Fisher Scientific, Fair Lawn, NJ) in for the appropriate volume of the nonfat milk. Strawberry flavor (International Flavor & Fragrances, Union Beach, NJ) and red food coloring (McCormick & Co Inc, Hunt Valley, MD) were added. The samples were mixed in a blender until homogenized. Previous studies showed that these samples are well suited for sensory testing because they are visually similar but perceptually different in flavor and texture (34).

Taste Testing. Both beverage stimuli were rated for sweetness intensity and liking using 15-cm line scales where 0=none and 15=very strong (for intensity) and 0=dislike extremely and 15=like extremely (for liking). Additionally, sucrose-sweetened milk samples were rated for intensity and liking of creaminess and flavor.

Each test session was conducted after an overnight fast. See **Figure 1** for a graphical representation of each test session. Each session began with a fasting blood sample. Subjects were then seated in a separate room to evaluate the taste samples. The subjects were offered twenty milliliters of the drink samples along with directions to expectorate each tastant after tasting and completely rinse their mouths with water in between each beverage. All samples were identified with 3-digit code numbers and were presented randomly within stimulus type. In order to prevent sensory fatigue, the subjects

were given approximately a 5-min rest period between each class of stimulus. In total, the sessions took subjects between 1 and 2 hours to complete.

Blood was collected in SST containers and kept on ice for no longer than 1-hour. Samples were then centrifuged and serum was stored at -70°C for later assay. Serum was assayed for insulin, leptin, cortisol and glucose.

Analytical methods. Serum insulin was measured in duplicate using commercially available double-antibody radioimmunoassays (RIAs) purchased from Linco Research (St. Charles, MO). Inter- and intraassay variations for insulin in pregnant subjects was 6.42% and 6%, respectively. Inter- and intraassay variations for insulin in nonpregnant subjects were 13.6% and 9.54%, respectively. Serum leptin was measured with RIAs for human leptin with reagents from Linco Research (St. Charles, MO). Inter- and intraassay variations for leptin in pregnant subjects were 8.43% and 7.54%, respectively. Inter- and intraassay variations for leptin in nonpregnant subjects were 15.1% and 14.1%, respectively. Serum cortisol was measured with RIAs with reagents from Linco Research (St. Charles, MO). Inter- and intraassay variations for cortisol in pregnant subjects were 7.4% and 4.8%, respectively. Inter- and intraassay variations for leptin in nonpregnant subjects were 12.8% and 6.23%, respectively. Serum estrogen and progesterone were measured with RIAs with reagents from Linco Research (St. Charles, MO). RIAs were performed in duplicate by the Diabetes Research Center of the University of Pennsylvania. Serum glucose was analyzed with a Hitachi 717 analyzer using hexokinase reagents from Amresco (Solon, OH). Analysis of glucose was performed by Accumed Diagnostic Laboratory (South Amboy, NJ). All technicians were blind to the conditions of the experiment.

Data analysis. Mixed model analysis with exchangeable intra-person correlation structure, determined by Akaike's Information Criterion (AIC), was used to assess the temporal trend across sessions, of the taste ratings of the glucose solutions and sucrose-sweetened milks. All data were modeled as a function of subject group (GDM, NGT and nonpregnant control), gestational age (16-20 weeks, 24-28 weeks and 34-38-weeks), and their interactions. Linear contrasts were constructed to compare group differences during pregnancy and the postpartum session, and between experimental periods within each subject group. BMI at entry was used as a covariate in all analyses. Statistical analyses were conducted using SAS version 9.1 for the personal computer (SAS Institute Inc, Cary, NC). Statistical significance was set at $\alpha=0.05$. Bonferroni corrections were applied for multiple testing, as appropriate. The criterion, after correction, was set at $p<0.017$.

Initial analysis revealed no differences in taste ratings across sweetness concentration of the glucose solutions; therefore data were collapsed across the 5 concentrations to make one single measure. Review of the sucrose-sweetened milk data also revealed no differences in taste ratings as a function of fat. Consequently, the data were collapsed across the 3 fat concentrations for final data analyses. This approach was also used in our previous study (21). Thus, one across-sucrose concentration curve was generated for all taste parameters, for each subject group at all sessions.

Added variable plots (35) were used to graphically examine the linear associations between the taste ratings and fasting endocrine concentrations, adjusted for BMI at entry. The partial correlation coefficients were calculated to assess the strength of linear association.

RESULTS

Subjects. Samples sizes at each stage of the study are depicted in **Figure 2**. Subject characteristics are shown in **Table 1**. Please see **Section A** for any additional description of the subject population.

Ratings of sucrose-sweetened milks. Intensity ratings for sweetness, creaminess, and overall flavor in sucrose-sweetened milk (collapsed across fat concentrations) are shown in **Figure 3**. Mixed model analysis was conducted to determine if the overall pattern of taste perception varied as a function of GDM or pregnancy. The analysis revealed that all groups showed a significant curvilinear relationship between the taste intensity ratings and increasing sucrose concentration, after controlling for intake BMI ($p < 0.0001$). The shapes of these functions did not differ between sessions or by subject group. Thus, neither pregnancy nor GDM altered the general pattern of taste perception during gestation.

Liking ratings for sweetness, creaminess, and overall flavor in sucrose-sweetened milk, averaged across fat concentration are shown in **Figure 4**. Similar to the intensity ratings, mixed model analysis revealed that all groups showed a significant curvilinear relationship between the liking ratings and increasing sucrose concentration of the samples, after controlling for intake BMI ($p < 0.0001$). The shapes of these functions did not differ between sessions or by subject group. Thus, neither pregnancy nor GDM altered the overall pattern of liking of the samples during gestation.

However, at 34-38 weeks gestation, linear contrasts revealed group differences in the liking of sweetness, creaminess and flavor of the samples. For all three attributes, women with GDM gave significantly higher ratings than nonpregnant controls for the 5%

sucrose-sweetened milk (p-values 0.0054-0.0147). Also at 5% sucrose concentration, women with GDM rated creaminess liking and flavor liking significantly higher than women with NGT (p=0.008 and 0.0067, respectively). Women with GDM also gave higher creaminess liking ratings for the 10% sucrose-sweetened milk, as compared to women with NGT (p=0.0129). There were no group differences at any other time point.

Ratings of glucose solutions. **Figure 5** shows the group ratings of both the averaged sweetness intensity and averaged sweetness liking of the glucose solutions across sessions (gestational age). Mixed model analysis revealed that averaged sweetness intensity ratings declined across sessions (p<0.0001), but did not vary by group. Conversely, averaged sweetness liking varied by group (p=0.0018), but not across sessions. There were also no significant interactions between group and session for averaged sweetness intensity or liking of the glucose solutions.

To assess the general effect of pregnancy, data for the pregnant groups were combined and compared to the nonpregnant group. See **Figure 6** for the graphical representation of these results. There were within group differences over gestation for the averaged sweetness intensity as well as between group differences at 24-28 weeks and 34-38 weeks gestation for the averaged sweetness liking. In the combined pregnant group, averaged sweetness intensity ratings at 16-20 weeks were higher than at all other sessions (p=0.02, p<0.001, p<0.01, when this session was compared to 24-28 weeks, 34-38 weeks and 6-10 weeks postpartum, respectively). In the control group, averaged sweetness intensity at 16-20 weeks differed from that same measure at 24-28 weeks (p=0.02) and 34-38 weeks (p<0.01). The differences found between 16-20 weeks and other sessions may reveal a training effect in the entire subject population. The pregnant

group showed higher ratings for the averaged sweetness liking of the glucose solutions than the nonpregnant control group at 24-28 weeks ($p=0.03$) and 34-38 weeks ($p=0.02$) gestation.

Correlations between endocrine and taste parameters.

Linear associations were examined between all endocrine and taste parameters at all sessions. No relationships were observed between the taste intensity measures and endocrine values, however, two sets of relationships with the liking ratings emerged.

Relationship between leptin and sweetness liking of sucrose-sweetened milk.

Figure 7 shows scatterplots of the relation between leptin and the sweetness liking of 10% sucrose-sweetened milk. At 24-28 weeks gestation, fasting leptin and the sweetness liking rating of the sucrose-sweetened milk were positively correlated in women with GDM ($R^2=0.42$, $p=0.0166$). There was no association between these two parameters in women with GDM at any other time point. Also, no significant correlations between these two parameters were found in the other two groups at 24-28 weeks ($R^2=0.00002$ for NGT women and $R^2=0.0173$ for nonpregnant controls, both:NS), or at any other time point in the study.

Relationship between insulin and averaged sweetness liking of glucose solutions

Figure 8 shows scatterplots of the relation between insulin and average liking of sweetness of the glucose solutions. At 24-28 weeks gestation only, fasting insulin and sweetness liking of the glucose solutions were positively correlated in women with GDM ($R^2=0.63$, $p=0.0037$). No positive correlations involving these measures were observed

in women with NGT or nonpregnant controls at this time point ($R^2=0.0036$ and 0.0235 , respectively, NS), or any other time point in the study.

DISCUSSION

The first objective of this study was to examine changes in sweet taste in women with normal pregnancy and women who developed GDM. At 34-38 weeks, women with GDM liked 5% sucrose-sweetened milk more than women with NGT and nonpregnant controls. These results support previous findings, showing that women with GDM liked similarly-sweetened milks more than women with NGT (21). An important feature of these responses was that women with GDM did not like the highly-sweetened milks more than the other two groups, but rather they gave higher liking ratings to the moderately sweetened milks. These concentrations are typical of sweetener concentrations of commercial sweetened milk beverages, which is approximately 6.6% (36). Thus, these results have relevance to real-world beverages. In this study, intensity ratings did not drive preference, since there was no group difference in intensity; however there were group differences in liking ratings.

Moreover, the elevated liking ratings for sweetened milks by women with GDM occurred late in pregnancy (at 34-38 weeks gestation), but not at earlier time points. We had anticipated that women with GDM would show higher liking ratings than women with NGT at 24-28 weeks gestation, when the endocrine profiles of both groups of pregnant women are most divergent, and GDM is first diagnosed. In view of Tepper & Seldner's data showing that liking for sucrose-sweetened milk was elevated at 28-32 weeks, and the present data showed that liking for that same stimulus was elevated at 34-38 weeks, but not at 24-28 weeks, we can now suggest that the onset of the changes in sweet taste occur after 28 weeks gestational age.

Women with GDM receive substantial dietary intervention following the time of diagnosis, starting around 28 weeks gestation, thus, we speculate that higher preference for sweet taste after that time point (i.e. 34-38 weeks gestation) may be more strongly related to dietary restriction rather than metabolic changes associated with the untreated diabetes. Whether intense dietary management contributes to sweet preference changes warrants further substantiating research.

Our findings related to the taste responses of the glucose solutions differed from those found in our previous study (21). In the earlier study, there was no pregnancy or GDM effect on sweetness intensity or liking of glucose solutions. Alternatively, in the current study, pregnancy contributed to higher sweetness liking of glucose at 24-28 weeks and 34-38 weeks gestation, however GDM did not generate higher sweetness liking than normal pregnancy. The present data suggest that pregnancy increases liking for the sweetness of glucose, but GDM does not elevate these levels further.

It appears that GDM increases the liking of sweetened milk, but not a pure carbohydrate, like glucose, in solution. This difference could reside in the physical and chemical differences between the stimuli. Sweetened milks emulate a real beverage whereas sugar solution is not typically consumed as a beverage. The milks are a sweet-fat stimulus and higher preference may be limited to this type of food rather than simple carbohydrate. Some past studies have shown lower glucose detection in people with type 2 diabetes (8, 9) but have not linked this change with differences in sweet preference or intake.

Interestingly, we found no difference in the perception of either the glucose solutions or the sweetened milks between women with GDM and women with NGT or

relative to nonpregnant controls. Thus, differences in perception do not seem to be motivating the differences in liking observed here. Our previous studies in women with GDM and type 2 diabetes have also reported differences in liking in the absence of perceived intensity at supra-threshold concentrations (10, 21).

The second objective of this study was to relate the taste changes to endocrine parameters in women with and without GDM, and two relationships arose. Liking of the sweetened milks was highly correlated with fasting serum leptin, and averaged sweetness liking of the glucose solutions was highly correlated with fasting serum insulin. These relationships were solely observed in women with GDM, and only at 24-28 weeks. The present results are novel and suggest that the degree of liking of sweet taste in GDM may be related to metabolism of leptin and insulin when diabetes is first emerging during pregnancy, but not when the disease is stabilized by diet, later on in pregnancy. The specific mechanisms are unclear at present but we can speculate based on current knowledge about taste and appetite related to these two hormones.

This study provided evidence that part of the sweet taste changes that occur both in pregnancy and in GDM may be related to endocrine factors. For example, there is accumulating evidence for the involvement of leptin in taste function. The leptin receptor has been found in taste receptor cells, and studies have linked leptin to behavioral and neurophysiological sweet taste responses in rodent models of obesity. Kawai et al found that *ob/ob* and *db/db* mice show increased responses to sweet taste. Additionally, leptin injections reduced the preference for sweet substances, such as sucrose and saccharin, as well as peripheral taste nerve (chorda tympani and glossopharyngeal) responses in *ob/ob* mice, and lean mice (31). However, exogenous leptin did not reduce sweet preference in

db/db mice, a model that is resistant to the effects of leptin. This suggests that leptin resistance may interfere with the ability of leptin to regulate sweet taste. Other rodent models of leptin resistance also seem to be impervious to leptin's effects on sweet taste. For example, VMH-lesioned obese rats, and high-fat diet-induced obese rats both showed larger chorda tympani nerve responses to sweet taste (37).

The potential relationship between leptin and sweet taste has never been investigated in a human model of diabetes or leptin resistance, but there is evidence for a role for leptin in general taste and palatability in humans. For example, in one human study, the main predictor for palatability of a high-carbohydrate breakfast was basal serum leptin levels (30), a finding that suggests a relationship between leptin and liking of carbohydrate-containing foods. However, high fasting serum leptin concentrations in obese humans have also been associated with a trend towards decreased liking for the taste of a high-fat, low-sugar mixture (29). However, while the aforementioned study examined obese women, who presumably were leptin resistant but not diabetic or pregnant, our research group studied overweight pregnant women with GDM. GDM may be a valuable model for studying the relationship between leptin resistance and sweet taste, since the time of onset of the disease is known and secondary complications (e.g. neuropathy, kidney disease) are absent.

Hyperinsulinemia also may be linked to hunger and sweet taste preference. For instance, one study showed that high insulin levels led to elevations in hunger, sweetness palatability and overall food intake (27). Another study found that induced hyperglycemia induced satiety, however induced hyperinsulinemia did not. The authors

did suggest, however, that endogenous insulin may be potentiating the satiating effects of the high blood glucose (28).

Although estrogen has been recognized in the past as a potential mechanism (24, 25), the estrogen patterns in both pregnant groups followed the normal course of gestation and did not differ significantly from one another (data not shown).

The higher liking ratings of the sucrose-sweetened milk, in women with GDM, coincide with prior work done in this laboratory (21). These results help support the idea that women with GDM may have sweet taste alterations similar to those found in nonpregnant individuals with diabetes. This study also suggested that there are relationships between insulin and leptin, two critical hormones in metabolism, and the sweet taste of glucose and sucrose in women with GDM, as compared to women with NGT and nonpregnant women.

In summary, this study demonstrated that women with GDM showed increased preference for a sweet-fat beverage, at 34-38 weeks gestation, while they were under dietary management and under good diabetic control. Future studies should seek to determine if the dietary restrictions and taste changes are coincidental or the former promote the latter. We also discovered novel relationships between endocrine parameters and taste measures, which may be related to hyperinsulinemia and hyperleptinemia that develops early in GDM pregnancy. Additional research should investigate the underlying mechanisms.

LITERATURE CITED

1. Metzger BE, Coustan DR. Summary and recommendations of the fourth international workshop conference on gestational diabetes mellitus. *Diabetes Care* 1998;21:B161-7.
2. American Diabetes Association. Gestational diabetes mellitus. *Diabetes Care* 2004;27:S88-S90.
3. Lee AJ, Hiscock RJ, Wein P, Walker SP, Permezel M. Gestational diabetes mellitus: clinical predictors and long-term risk of developing type 2 diabetes. *Diabetes Care* 2007;30:878-883.
4. Hunger-Dathe W, Mosebach N, Sämann A, Wolf G, Müller UA. Prevalence of impaired glucose tolerance 6 years after gestational diabetes. *Exp Clin Endocrinol Diabetes* 2006;114:11-17.
5. American Diabetes Association. Meal planning exchange lists. <http://www.diabetes.org/nutrition-and-recipes/nutrition/exchangelist.jsp>.
6. American Diabetes Association. Nutrition recommendations and interventions for diabetes: A position statement of the American Diabetes Association. *Diabetes Care* 2007;30:S48-S65.
7. Armstrong CL, Brown LP, York R, Robbins D, Swank A. From diagnosis to home management: nutritional considerations for women with gestational diabetes. *Diabetes Educ* 1991;17.
8. Perros P, MacFarlane TW, Counsell C, Frier BM. Altered taste sensation in newly-diagnosed NIDDM. *Diabetes Care* 1996;19:768-770.
9. Lawson WB, Zeidler A, Rubenstein A. Taste detection and preference in diabetics and their relatives. *Psychosom Med* 1979;41:219-227.
10. Tepper BJ, Hartfiel LM, Schneider SH. Sweet taste and diet in type II diabetes. *Physiol Behav* 1996;60:13-18.
11. Ochsenbein-Kolble N, von Mering R, Zimmermann R, Hummel T. Changes in gustatory function during the course of pregnancy and postpartum. *BJOG* 2005;112:1636-1640.
12. Kuga M, Ikeda M, Suzuki K, Takeuchi S. Changes in gustatory sense during pregnancy. *Acta Otolaryngol* 2002;Supplement 546:146-153.

13. Kölble N, Hummel T, von Mering R, Huch A, Huch R. Gustatory and olfactory function in the first trimester of pregnancy. *Eur J Obstet Gyn R B* 2001;99:179-183.
14. Brown JE, Toma RB. Taste changes during pregnancy. *Am J Clin Nutr* 1986;43:414-418.
15. Bowen DJ. Taste and food preference changes across the course of pregnancy. *Appetite* 1992;19:233-242.
16. Pope JF, Skinner JD, Carruth BR. Cravings and aversions of pregnant adolescents. *J Am Diet Assoc.* 1992;92:1479-1482.
17. Bayley TM, Dye L, Jones S, DeBono M, Hill AJ. Food cravings and aversions during pregnancy: relationships with nausea and vomiting. *Appetite* 2002;38:45-51.
18. Dippel RL, Elias JW. Preferences for sweet in relationship to use of oral contraceptives and pregnancy. *Horm Behav* 1980;14:1-6.
19. Hook EB. Dietary cravings and aversions during pregnancy. *Am J Clin Nutr* 1978;31:1355-1362.
20. Worthington-Roberts B, Little RE, Lambert MD, Wu R. Dietary cravings and aversions in the postpartum period. *J Am Diet Assoc* 1989;89:647-651.
21. Tepper BJ, Seldner AC. Sweet taste and intake of sweet foods in normal pregnancy and pregnancy complicated by gestational diabetes mellitus. *Am J Clin Nutr* 1999;70:277-284.
22. Boden G. Fuel metabolism in pregnancy and in gestational diabetes mellitus. *Obstet Gynecol Clin North Am* 1996;23:1-10.
23. Henson MC, Castracane VD. Leptin in pregnancy: an update. *Biol Reprod* 2006;74:218-229.
24. Curtis KS, Stratford JM, Contreras RJ. Estrogen increases the taste threshold for sucrose in rats. *Physiol Behav* 2005;86:281-286.
25. Duffy VB, Bartoshuk L, Striegel-Moore L, Rodin J. Taste changes across pregnancy. *Ann NY Acad Sci* 1998;855:805-809.
26. Frye CA, Crystal S, Ward KD, Kanarek RB. Menstrual cycle and dietary restraint influence taste preferences in young women. *Physiol Behav* 1994;55:561-567.

27. Rodin J, Wack J, Ferrannini E, DeFronzo RA. Effect of insulin and glucose on feeding behavior. *Metabolism* 1985;34:826-831.
28. Gielkens HAJ, Verkijk M, Lam WF, Lamers CBHW, Masclee AAM. Effects of hyperglycemia and hyperinsulinemia on satiety in humans. *Metabolism* 1998;47:321-324.
29. Karhunen LJ, Lappalainen RI, Haffner SM, et al. Serum leptin, food intake and preferences for sugar and fat in obese women. *Int J Obesity* 1998;22:819-821.
30. Raynaud E, et al. Serum leptin is associated with the perception of palatability during a standard high-carbohydrate breakfast test. *Clin Sci* 1999;96:343-348.
31. Kawai K, Sugimoto K, Nakashima K, Miura H, Ninomiya Y. Leptin as a modulator of sweet taste sensitivities in mice. *PNAS* 2000;97:11044-11049.
32. Carpenter MW, Coustan DR. Criteria for screening tests for gestational diabetes. *American Journal of Obstetrics and Gynecology* 1982;144:768-773.
33. American Diabetes Association. Standards of Medical Care in Diabetes. *Diabetes Care* 2007;30:S4-S41.
34. Tepper BJ, Shaffer SE, Shearer CM. Sensory perception of fat in common foods using two scaling methods. *Food Qual Pref* 1994;5:245-251.
35. Weisberg S. *Applied linear regression*. 2nd ed. New York: Wiley, 1985.
36. Brand-Miller J, Holt SHA, de Jong V, Petocz P. Cocoa powder increases postprandial insulinemia in lean young adults. *J Nutr* 2003;133:3149-3152.
37. Shimizu Y, et al. Enhanced responses of the chorda tympani nerve to sugars in the ventromedial hypothalamic obese rat. *J Neurophysiol* 2003;90:128-133.

TABLE 1

Subject characteristics

	GDM	NGT	Nonpregnant controls
Total Enrollment	15	93	19
Age at entry (yr) ¹	29.2 ± 3.1	26.2 ± 0.7	27.1 ± 1.3
BMI at entry (kg/m ²) ¹	28.8 ± 2.6 ^a	27.0 ± 3.5 ^a	24.5 ± 0.5 ^b
Total pregnancy weight gain (kg) ¹	8.3 ± 1.5 ^a	13.9 ± 0.6 ^b	—
Ethnicity (%)			
Hispanic	60	52	79
Caucasian	20	20	11
African American	13	20	5
Asian	0	5	0
Other	7	3	5
Cesarean Delivery (%) ¹	27	30	—
Gestational age (wk) ¹	40.0 ± 0.6	39.4 ± 0.2	—
Infant birthweight (g) ¹	3321 ± 125	3325 ± 53	—
Breastfeeding (%) ¹	73	77	—

¹ Values are means ± SEM. Values in the same row with different superscripts are different at p<0.05.

FIGURE LEGENDS

Figure 1 Flow diagram showing individual session progression.

Figure 2 Diagram indicating sample sizes at each stage of the study.

Figure 3 Sweetness, creaminess and flavor intensity ratings of sucrose-sweetened milks. There were no significant group differences for any sucrose concentration at any session.

Figure 4 Sweetness, creaminess and flavor liking ratings of sucrose-sweetened milks. At 34-38 weeks gestation, GDM women showed higher ratings for sweetness liking of 5% sucrose-sweetened milk, when compared to nonpregnant controls, and higher creaminess liking ratings of 10% sucrose-sweetened milk when compared to women with NGT. Also, women with GDM exhibited higher creaminess and flavor liking of 5% sucrose-sweetened milk as compared to both women with NGT and nonpregnant women.

Figure 5 Sweetness intensity and liking ratings of glucose solutions in the three test groups. There were no significant differences between groups for either taste parameter at any session.

Figure 6 Pregnancy effect in sweetness intensity and liking ratings of glucose solutions.

Pregnant women at 16-20 weeks gestation showed higher sweetness intensity ratings than all other sessions ($p < 0.05$ for all comparisons). Control women at 16-20 weeks gestation showed higher sweetness intensity ratings than at 24-28 weeks and 34-38 weeks ($p < 0.05$ for both comparisons). The pregnant group exhibited higher sweetness liking ratings of the glucose solutions at 24-28 weeks gestation ($p < 0.05$ for both comparisons).

Figure 7 Added variable plot of liking rating of sucrose-sweetened milk against fasting serum leptin with the effects of initial BMI removed. The association was positive and significant in women with GDM at 24-28 weeks. There were no other significant associations in any group at any other time point.

Figure 8 Added variable plot of liking rating of glucose solutions against fasting serum insulin with the effects of initial BMI removed. The association was positive and significant in women with GDM at 24-28 weeks. There were no other significant associations in any group at any other time point.

Figure 1

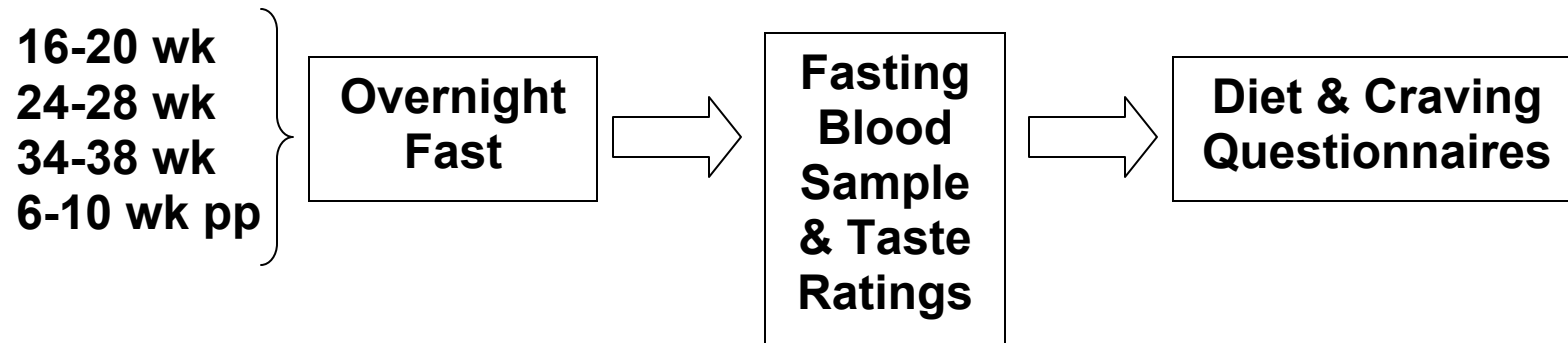


Figure 2

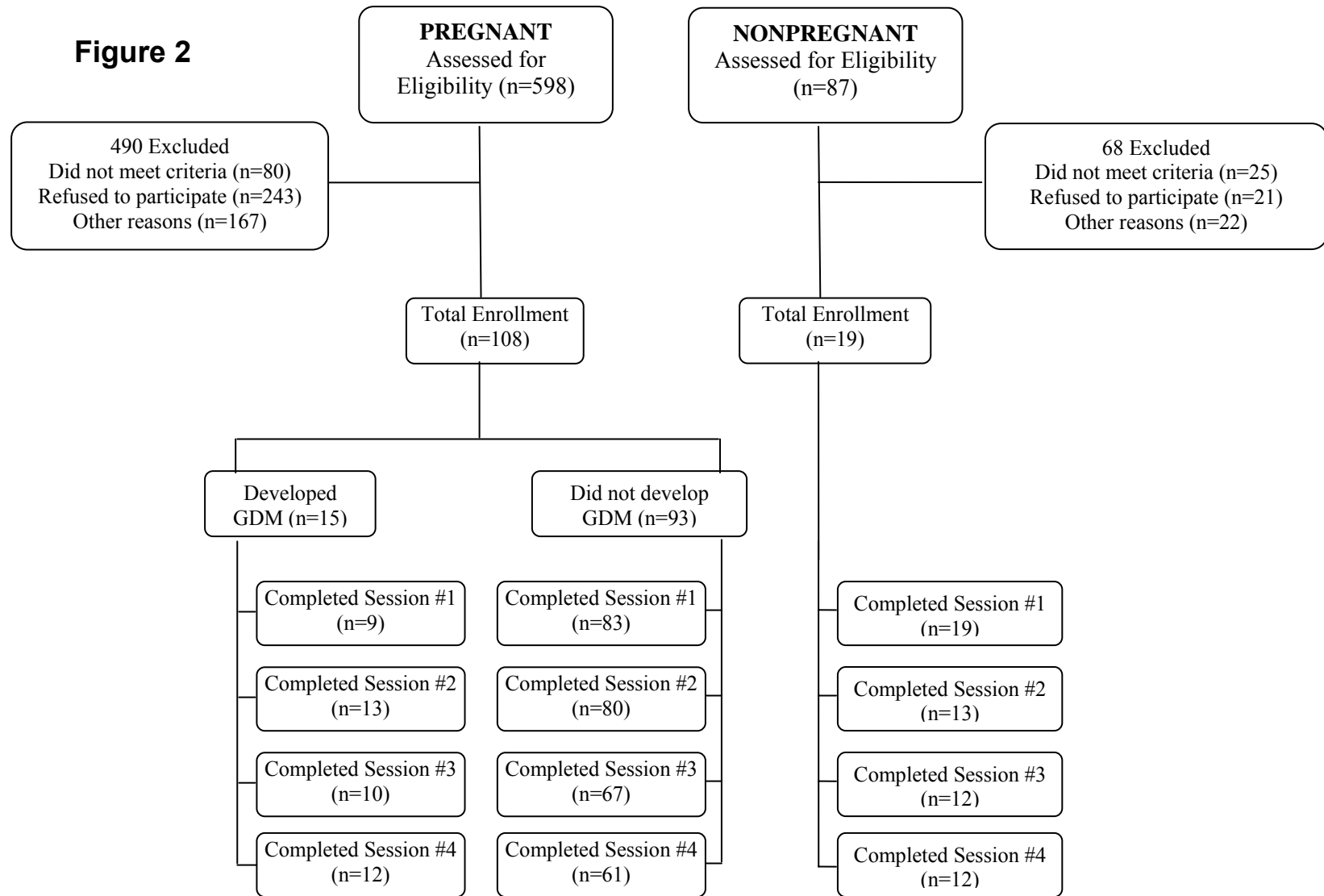


Figure 3

▲ GDM

■ NGT

○ Nonpregnant controls

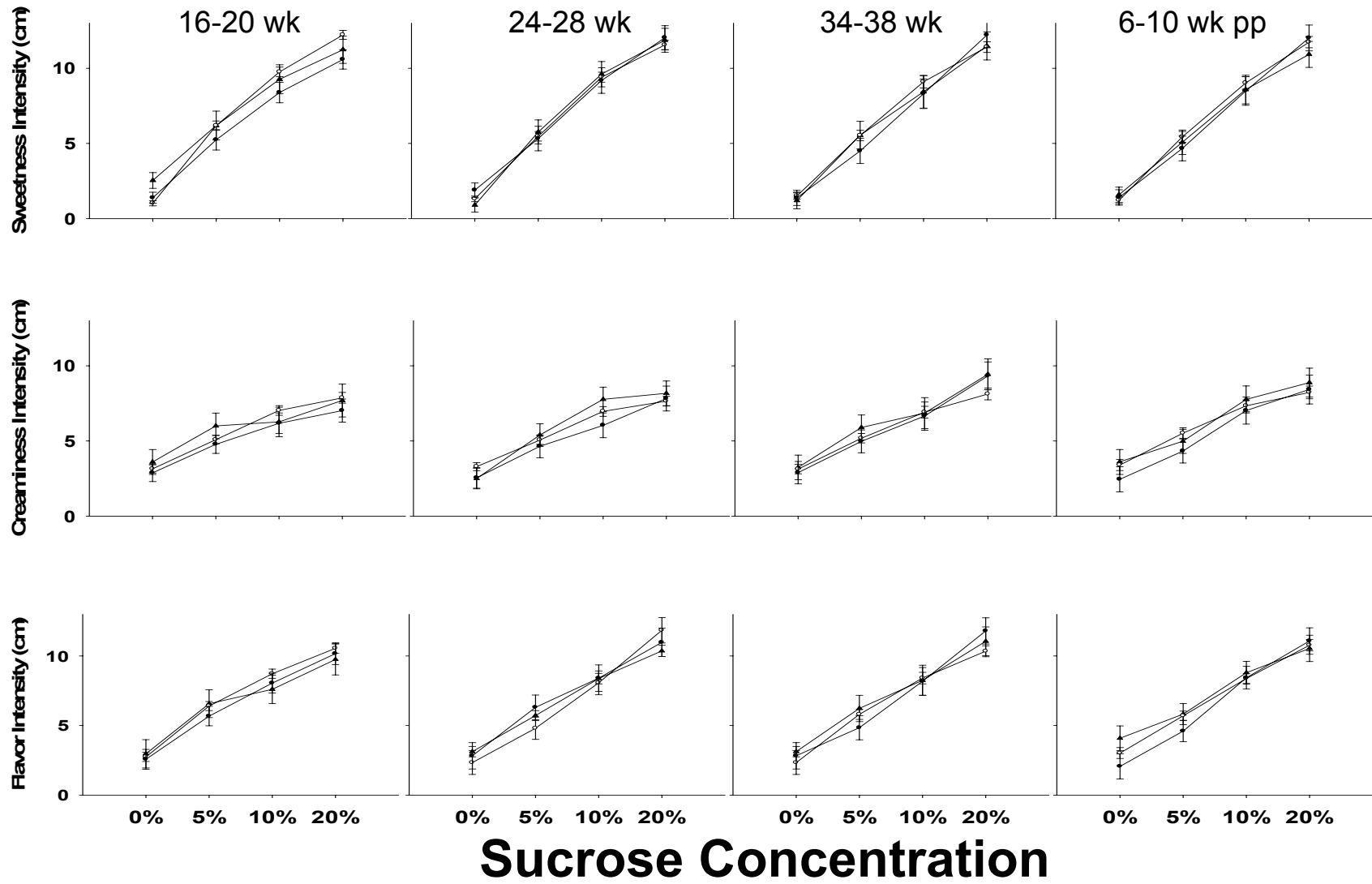


Figure 4

▲ GDM

■ NGT

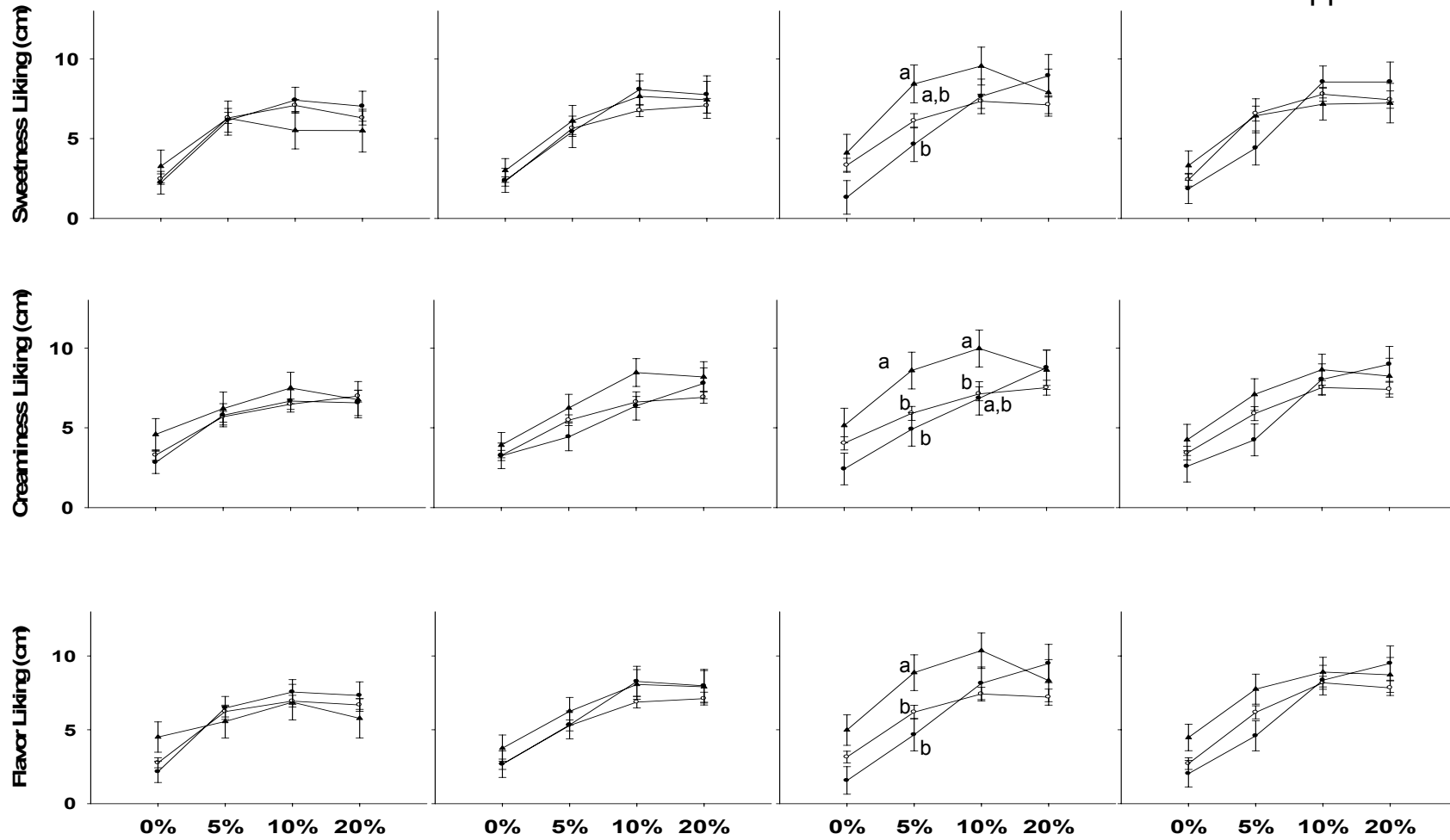
○ Nonpregnant controls

16-20 wk

24-28 wk

34-38 wk

6-10 wk pp



Points with different letters are significantly different from each other at $p < 0.017$

Sucrose Concentration

Figure 5

■ GDM □ NGT ■ Nonpregnant controls

Sweetness Intensity

Sweetness Liking

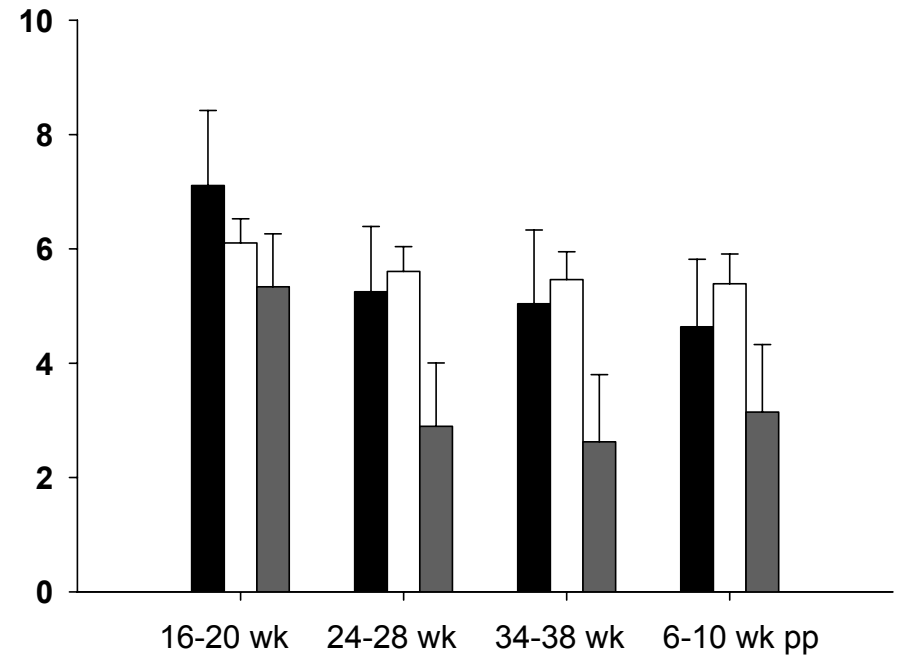
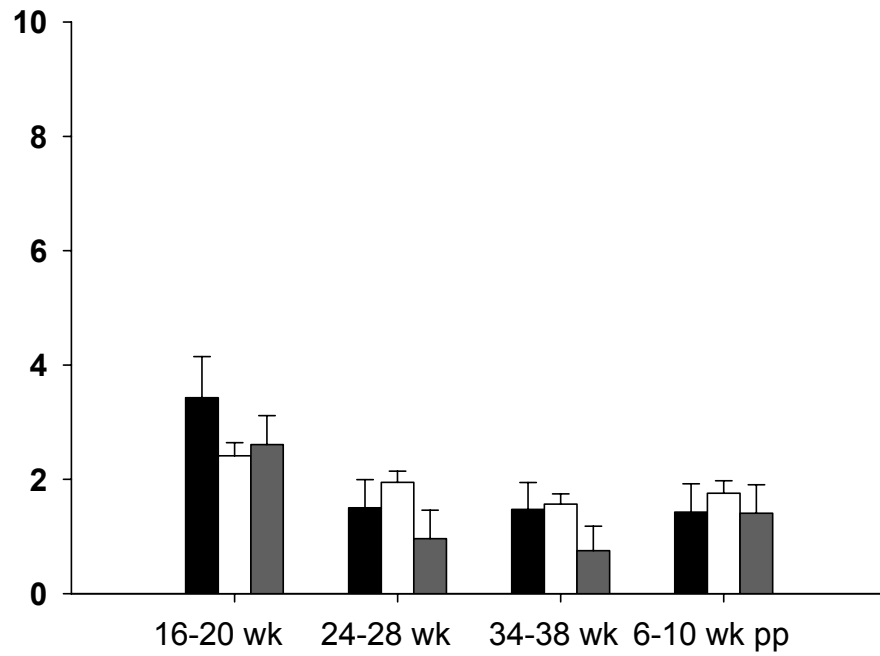


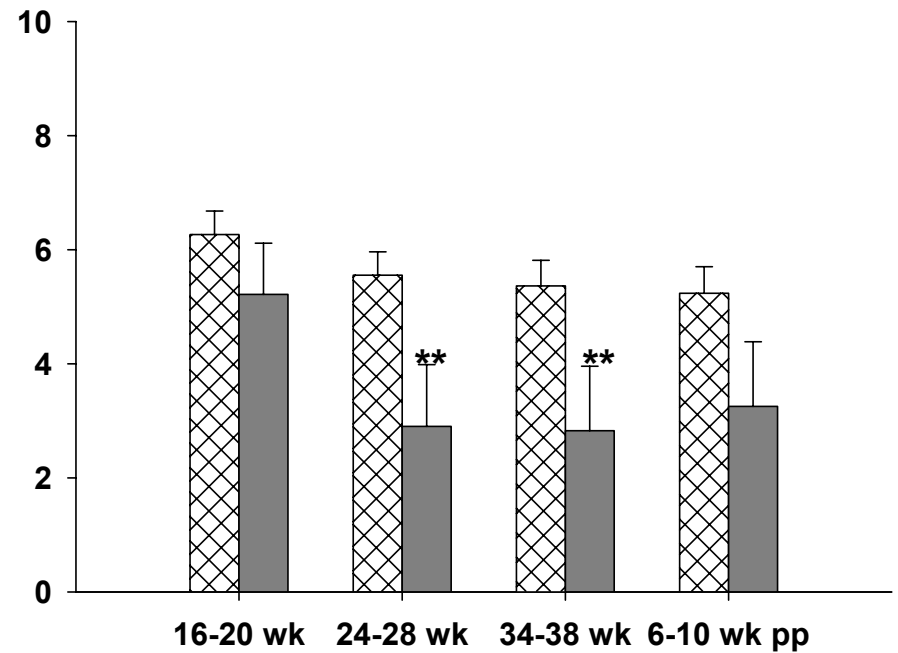
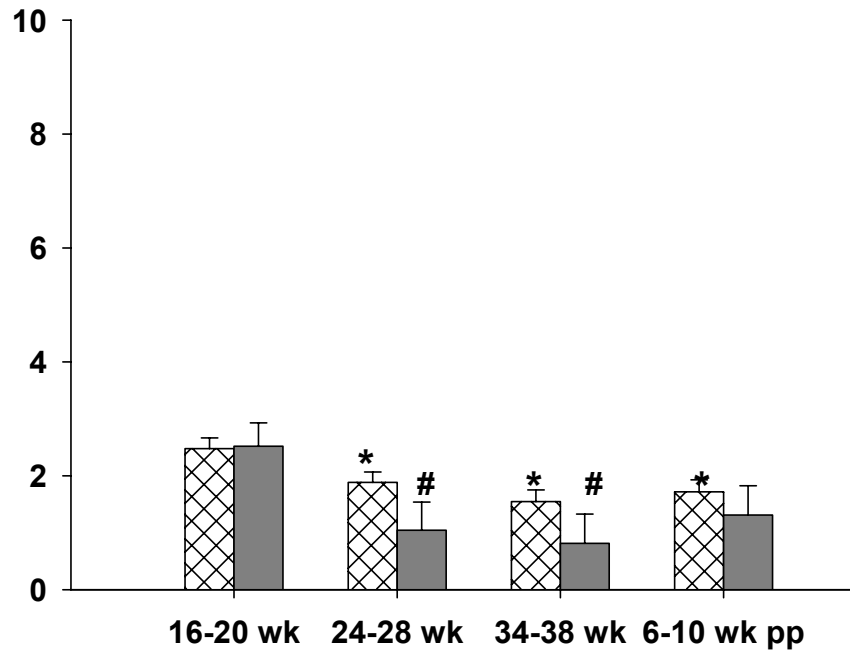
Figure 6

☒ Pregnant

■ Nonpregnant controls

Sweetness Intensity

Sweetness Liking



* Differs from 16-20 wk in pregnant group ($p < 0.05$)
Differs from 16-20 wk in control group ($p < 0.05$)
** Differs from pregnant group at that time point ($p < 0.05$)

Figure 7

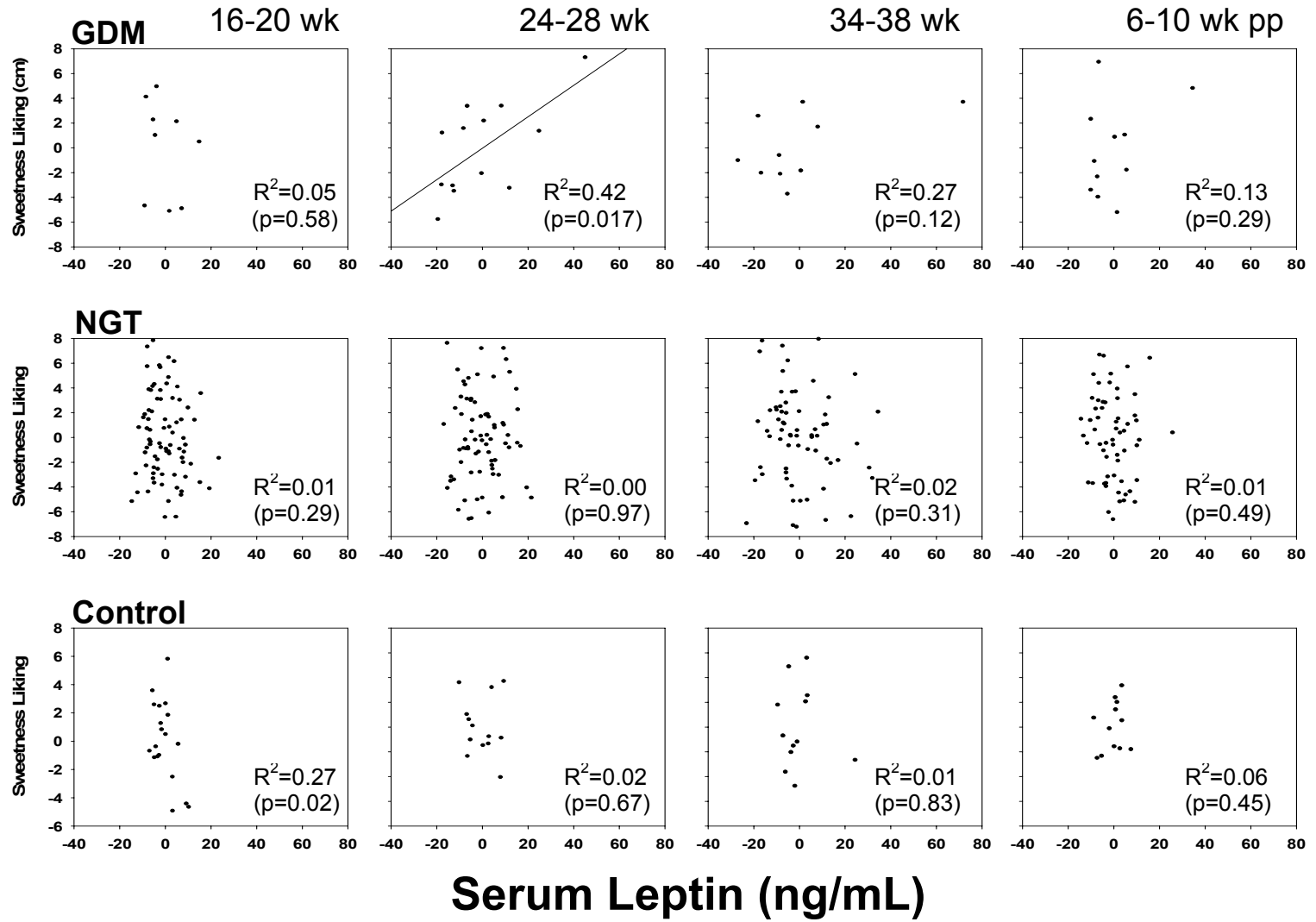
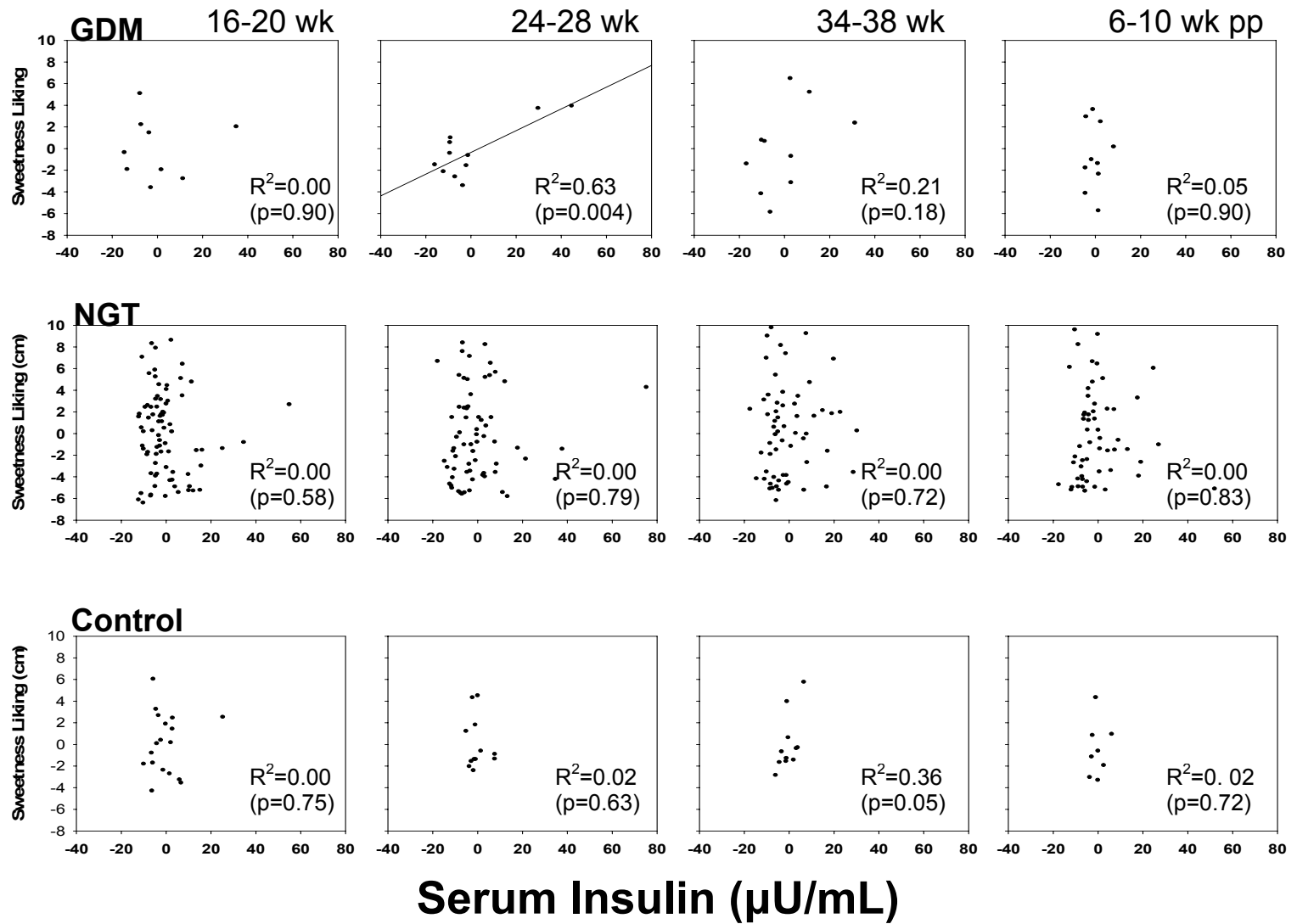


Figure 8



**SECTION C. Food Cravings in Pregnancy and Their Relationship to Diet: A
Prospective Study in Women with Mild Gestational Diabetes Mellitus**

ABSTRACT

Background: Gestational diabetes mellitus (GDM) is diabetes first recognized during pregnancy. Pregnancy is associated with increased food cravings, especially for sweet foods, and higher dietary intake of sweets. The altered glucose metabolism of GDM could elevate sweet cravings and intake, especially around the time of diagnosis.

Objective: To investigate reported sweet food and beverage consumption and food cravings in women who exhibit NGT during their pregnancies, and those who develop GDM.

Design: A prospective design was used. Subjects included 93 pregnant women with NGT, 15 pregnant women who developed GDM and 19 nonpregnant controls. A 141-item food frequency questionnaire and craving survey were completed at 16-20 weeks, 24-28 weeks, and 34-38 weeks gestation, and 6-10 weeks postpartum.

Results: Eighty percent of all pregnant women reported food cravings at 16-20 weeks and the prevalence of cravings decreased over gestation (time trend: $p=0.0004$ for women with NGT and $p=0.0166$ for women with GDM). The prevalence of sweet cravings was higher in women with NGT at 24-28 weeks gestation than at other time points, and was coordinated with higher reported sweet food and beverage consumption, especially fruit and fruit juice. The percentage of women with GDM reporting sweet cravings was not higher at any time point, nor was their sweet food and beverage intake. However, at 34-38 weeks, the subset of women with GDM who did report sweet cravings ($n=4$) experienced approximately twice the frequency of sweet cravings as did women with NGT and nonpregnant controls ($p=0.015$ and 0.009 , respectively).

Conclusions: Although the overall prevalence of sweet cravings was low in women with GDM, a subset of women with GDM experienced a higher frequency of sweet cravings for sweets later in pregnancy. That coincided with intensive dietary management. Sweet cravings in GDM may be related to dietary restriction as opposed to glucose intolerance.

KEY WORDS: Gestational diabetes, Sweet food cravings, Sweet food intake

INTRODUCTION

A food craving is the intense desire for food and or drink. Fifty to ninety percent of pregnant women have reported experiencing food cravings (1-4). Cravings during pregnancy usually arise later in the first trimester, peak during the second trimester then taper off by the last semester of gestation (2, 4). The most frequent targets of cravings are dairy and sweet foods, including chocolate and fruit and fruit juice. Though not as common, pregnant women also report craving salty and savory foods (2, 4, 5).

There is some evidence that cravings are associated with increased food consumption, although data documenting this relationship are sparse. In pregnant adolescents, those who craved sweets during pregnancy had higher sugar and energy intake than those without sweet cravings (2). Bowen et al found that pregnant women consumed more sweet foods during their second trimester and more salty foods during the third trimester when compared to other time points of pregnancy (6). This dietary intake pattern seems to mimic the type and time course of pregnancy cravings.

The basis for pregnancy cravings is poorly understood, and recent research in this area is lacking. Impaired sense of taste during pregnancy may increase preference and/or cravings for certain food and beverage, thus promoting the intake of those items. There is evidence that pregnant women have decreased sensitivity to salt, sour, bitter and sweet solution sensitivity throughout pregnancy, but perception may be especially blunted during the first trimester (7-9). Also, pregnant women may simply have a higher feeling of pleasure or reward from certain foods. This may be related to opioid levels that continuously rise over the gestational period in rodent, monkey and human models (10). Specific cravings for dairy and sweet foods may arise from the increased requirement for

calcium and calories throughout gestation (5, 7). Pregnancy results in a rise in gestational hormones, some of which may be involved in the experience of pregnancy cravings. In nonpregnant animals, the administration of estrogen and progesterone, which are naturally elevated during pregnancy, produced increased salt appetite (7). Furthermore, rodent studies have also suggested that estrogen may increase the taste threshold for sucrose (11).

Most cravings research has been retrospective (5), which relies on the memory of subjects in the postpartum period, or cross-sectional (3, 4, 6, 7, 12), which compares different groups of people rather than one, single group across gestation. To our knowledge, only one other research group has prospectively studied reported food and beverage intake and dietary cravings, concurrently. The researchers found that the percentage of women who experienced cravings was higher in the pregnant group than in the nonpregnant control group; however the incidence of cravings did not change across pregnancy (12). This contrasts with other studies reporting that cravings are higher at certain time points of pregnancy (2, 4).

Gestational diabetes mellitus (GDM), a form of glucose intolerance that is first detected during pregnancy (13), occurs in approximately 7% of pregnant women (14). Since GDM increases the risk for obstetric and perinatal complications, treatment of the disease is of primary clinical importance. GDM has many of the same metabolic features as type 2 diabetes, thus, dietary management strategies are similar to those followed by individuals with type 2 diabetes (15, 16).

Type 2 diabetes may influence sweet perception (17, 18) and increase the liking (19) of sweet foods, both of which may result in increased cravings and intake of sweets.

Only one study has examined these relations in GDM (20), when measured approximately one month after diagnostic testing (28-32 weeks gestation). Women with GDM showed a higher preference for sucrose-sweetened milk, than women with NGT, although liking ratings did not exceed those observed in nonpregnant controls. Plasma glucose levels were related to higher preference for glucose solutions and greater reported fruit and fruit juice intake in women with GDM but not women without GDM. These data suggest that preference and intake of sweets heighten with increasing glucose intolerance in GDM. However, reported intake of simple sugars was not different in women with GDM as compared to women with NGT, and sweet cravings were not elevated in women with GDM relative to women without GDM. These data provide limited evidence for increased preferences for sweets in GDM. It is possible that the timing of the single measurement in the Tepper & Seldner study, in the third trimester (28-32 weeks) may have missed the elevation in sweet cravings, especially if sweet cravings are more prevalent when the diabetes first arises during early- or mid-pregnancy, as opposed to when the disease is stabilized by late pregnancy.

The objective of this study was to prospectively examine food cravings and dietary intake of sweet foods in women with GDM and women with NGT at multiple gestational time points and during the post partum period.

We hypothesized that, in women with NGT, the highest sweet cravings and dietary intake of sweet foods would be found at mid-pregnancy, consistent with past literature. However, we anticipated that women with GDM would report higher sweet food intake and cravings during mid-pregnancy relative to women with NGT.

SUBJECTS AND METHODS

Subjects and recruitment. The women studied here were part of a larger, prospective study designed to investigate changes in sweet taste and their endocrine and metabolic correlates during gestation in pregnant women with normal glucose tolerance and women who developed GDM. Please refer to **Section A** for recruitment procedures, subject characteristics and general methods.

Study design. The objective of this experiment was to document changes in reported dietary intake of sweet foods and cravings during 16-20 weeks, 24-28 weeks and 34-38 weeks gestation and 6-10 weeks postpartum in pregnant women with normal glucose tolerance and women who developed GDM. The study design is shown in **Figure 1**.

All pregnant women at the clinic are routinely screened for GDM at 24-28 weeks gestation using a 1-hour, 50-gram oral glucose challenge. Women with a positive screen (glucose >140mg/dL) undergo a 3-hour, 100-gram oral glucose tolerance test to confirm their diagnosis (21). Thus, women who developed GDM during the course of this study were identified at 24-28 weeks gestational age. Women with GDM were referred to nutritional counseling at the time of their diagnosis and received diet therapy until the end of their pregnancies. A diabetic exchange diet plan was used, which incorporated carbohydrate control within the context of sufficient calories for proper maternal health and fetal growth (15). Women without GDM received standard nutritional guidance for pregnancy.

All testing was completed at Saint Peter's Hospital in New Brunswick, NJ in the same session as when taste ratings were collected. Each session was conducted the

morning after an overnight fast, in a small office within the Women's Ambulatory Clinic, in coordination with an obstetrics visit. Subjects were interviewed regarding dietary behaviors and cravings in an office adjacent to the clinic.

Food craving and food frequency questionnaires. Food craving information was collected during each session, by a questionnaire (22) that highlighted cravings during the past month. Although the Tepper & Seldner study used that same time frame (20), the current study inquired about cravings within the past week, to capture the dynamic changes in appetite found in pregnancy. The questionnaire asked whether or not the woman was experiencing cravings, to report the first, second and third strongest cravings and to specify the frequency of stated cravings. The first time point of data collection in this study was at 16-20 weeks gestation, well after 12 weeks gestation, when reported frequency of nausea and/or food aversions decline, and cravings tend to emerge (4).

Dietary data were collected using a 141-item food frequency questionnaire (FFQ). This questionnaire determined habitual consumption of foods by asking the subject to estimate the number of standard servings consumed daily, weekly or monthly for 141 common foods or food groups. The latter questionnaire had been validated in women against 3-d diet records for energy, macronutrient intake and frequency of consumption ($r=0.87$) and was used in our previous study in GDM (20). Latino foods were included on the questionnaire and interviews were conducted in Spanish by request. Frequency of consumption of food groups was also derived from the FFQ, and our focus was especially on frequency of intake of sweet food groups and sub-groups. All questionnaires were completed with the assistance of the research investigator.

Data analysis. Cravings were grouped into categories motivated by both nutritional content and predominant taste category (see **Table 1** below). Some foods were categorized according to their dominant taste quality (i.e. soup: salty food) and other foods were categorized mainly by their nutritional composition (i.e. cheese: non-sweet dairy food). Cravings during pregnancy were examined using chi-square tests and marginal logistic regression models with the generalized estimating equations (GEE) method (23). PROC freq and PROC genmod were used to carry out these analyses.

TABLE 1
Categorization of cravings

Sweet	Candy, cookies, frozen desserts, fruit & fruit juice, pastries, pudding, soda, sweetened cereal, sweetened milk, sweetened yogurt
Salty	Pickles, salsa, salty snacks (i.e. chips, crackers, pretzels), soup
Savory	Eggs, French fries, meat, mixed dishes (i.e. ethnic dishes, pizza, sandwiches), seafood
Starchy	Bagels, beans, bread, corn, non-fried potatoes, rice, tortillas
Non-sweet dairy	Cheese, milk, plain yogurt
Other	Ice, vegetables, water

Dietary data were compiled using NUTRITIONIST PRO software (N-squared Computing, Salem, OR), and analyzed for energy, macro-and micronutrient composition. Mixed model analysis with exchangeable intra-person correlation structure, determined

by Akaike's Information Criterion (AIC), was used to assess temporal trends in the mean frequency of sweet and savory cravings. Reported intake of sweet food and beverage was analyzed similarly. Analysis of sweet food intake included all sweetened food and beverage, and 2 sub-groups: fruit and fruit juice and other sweet food and beverage. All data were modeled as a function of subject group (GDM, NGT and nonpregnant control), gestational age (16-20 weeks, 24-28 weeks and 34-38-weeks), and their interactions. Linear contrasts were constructed to compare group differences during pregnancy and the postpartum session, and between experimental periods within each subject group. BMI at entry was used as a covariate in all analyses. Statistical analyses were conducted using SAS version 9.1 for the personal computer (SAS Institute Inc, Cary, NC). Statistical significance was set at $\alpha = 0.05$. Bonferroni corrections were applied for multiple testing, and resulted in a final criterion of $p < 0.017$.

RESULTS

Subjects. Samples sizes at each stage of the study are depicted in **Figure 1**. Subject characteristics are shown in **Table 2**. Please see **Section A** for any additional description of the subject population.

Cravings analysis.

Cravings data were analyzed for total cravings followed by each individual category. Since sufficient responses were reported only for total, sweet and savory cravings, we did not proceed with the analyses of any of the other craving categories (i.e. salty, starchy).

Percent of women with any type of craving

Approximately 80% of all pregnant women reported cravings at 16-20 weeks gestation (see **Table 3**). Logistic regression analysis revealed that the percentage of women with cravings across sessions in both pregnant groups declined ($p=0.0004$ for women with NGT and $p=0.0166$ for women with GDM). The percent of women with GDM who reported cravings fell in the postpartum period, and was lower than observed in both previously pregnant women and controls. ($\chi^2 = 11.82$, $p=0.0027$). This decline was not observed in either of the other groups. The percentage of nonpregnant women with cravings ranged from 50-69%, but did not vary in a systematic way.

Percent of women with sweet cravings

The percentage of women with sweet cravings rose then fell significantly for women with NGT (time trend $p=0.0004$), with 55% of women in this group reporting sweet cravings at this time, and 29% reporting sweet cravings during postpartum (See **Table 3**). Although a lower percentage of women with GDM reported sweet cravings

during postpartum than the other groups, the differences were not significant.

Furthermore, there were no across-session differences for the nonpregnant group.

Percentage of women with savory cravings

There was no group effect or session trend in the reporting of savory cravings.

These results are also depicted in **Table 3**.

Sweet craving frequency

Figure 3 provides a graphic representation of the analysis of sweet craving frequency. For those women who had sweet cravings, reported frequencies of those cravings were compared both across sessions and between groups. Mixed model analysis revealed no overall session trends or group effects for mean sweet craving frequency.

However, pairwise comparisons, using linear contrasts, revealed that, among the women with GDM who did crave sweet food and beverage, the frequency of sweet cravings was higher at 34-38 weeks than at 24-28 weeks gestation ($p=0.0146$). Furthermore, at 34-38 weeks gestation, linear contrasts revealed that women with GDM exhibited a significantly higher mean sweet craving frequency than both the NGT women ($p=0.015$) and nonpregnant controls ($p=0.009$). Although only 40-45% of all pregnant women reported sweet cravings at 34-38 weeks gestation, women with GDM reported twice the number of sweet cravings/week than women with NGT (12 vs. 6, respectively).

Savory craving frequency

For those women who had savory cravings, reported frequencies of those cravings were compared both across sessions and between groups. Craving frequency did not vary across sessions or between groups. **Figure 3** also provides a graphic representation of the analysis of savory craving frequency.

Reported sweet food intake. Reported intake of sweet food and beverage is shown in **Figure 4**. Mixed model analysis demonstrated no overall session effect on sweet food intake in any of the subject groups. When linear contrasts were used to make pairwise comparisons, however, women with NGT consumed significantly more servings of sweet food and beverage, of all types, at 24-28 weeks gestation when compared to both women with GDM and nonpregnant control subjects ($p=0.0149$ and 0.0133 , respectively). These differences stemmed from lower fruit and fruit juice intake, where women with NGT consumed about 3 servings/ day, as compared to about 1 serving/ day in the other two groups ($p=0.003$, NGT vs. GDM and $p=0.0099$, NGT vs. controls). There were no group differences in the consumption of other types of sweet food and beverage, a category which included soda and desserts. Women with GDM did not show any increase in sweet food intake at any time point.

At 34-38 weeks gestation, there were directional trends for women with NGT to consume more servings/day of sweet food and beverage of all types than women with GDM ($p=0.0573$). Also at this session, women with GDM consumed a marginally lower number of servings of sweet food and beverage intake other than fruit and fruit juice, as compared to women with NGT ($p=0.02$). Neither pregnant group differed from nonpregnant controls in any of these measures at this session.

DISCUSSION

This study investigated food cravings and reported sweet food and beverage intake in women with and without GDM. Eighty percent of all pregnant women reported cravings during pregnancy when measured at 16-20 weeks (mid-pregnancy) with a steady decline to the end of gestation. This further substantiates past research regarding the typical pattern of cravings during pregnancy (2, 4). Although savory cravings have been a focus in previous research (2, 4, 5), these cravings were not very common in the current subject population.

There was some evidence that the percent of women in the NGT group with sweet cravings rose early in the third trimester, and then fell as pregnancy progressed, though this pattern was not particularly robust. Particularly at 24-28 weeks gestation, the majority of women with NGT who had cravings had sweet cravings (55% sweet cravings, from 69% total), consistent with previous literature (2, 4, 5).

Contrary to expectations, the prevalence of sweet cravings in women with GDM was not higher at 24-28 weeks gestation than other time points, or in comparison to the other subject groups. However, within the subset of women with GDM who did have sweet cravings, there was a higher reported frequency of sweet cravings later in pregnancy (at 34-38 weeks gestation) than in either of the other subject groups. Women with GDM reported approximately twice the sweet craving frequency/week than the other groups. We are aware that the number of women with GDM represented in the analysis of sweet craving frequency was low; therefore, these findings warrant confirmation in a larger study population.

All pregnant women are provided with diet therapy as a part of their overall medical management, but intense counseling begins in women with GDM after diagnosis, at around 28 weeks gestation. The guidelines were to control carbohydrate intake within the context of a diet with sufficient calories and nutrients appropriate for maternal health and fetal growth (16). Women with GDM may begin craving carbohydrate-rich foods of which they are told to limit their intake. Additionally, the high frequency of sweet cravings observed in later pregnancy was not seen in early pregnancy, when the GDM group was untreated. The possibility that, in women with GDM, dietary restriction may be generating the sweet cravings could have important implications for the medical and dietary management of GDM.

As mentioned previously, we examined all types of cravings, but there was not sufficient data from the other categories (i.e. salty, starchy) to further analyze group or session differences. It is notable that the demographic make-up in this study differs from that from other studies (1, 2, 4, 22, 24), and demographics may play a role in the inclination to report cravings. The majority of the women studied here were Hispanic, and data on cravings are sparse in this group.

Our results indicate that, at 24-28 weeks gestation, women with NGT consumed more daily servings of sweet foods than both other groups, and this difference stemmed from higher intake of fruit and fruit juice. This increase is consistent with past literature, which showed that women with normal pregnancies consumed more sweet foods during their second trimester (6). Our study showed no evidence that women with GDM consumed an excess of sweet foods. In fact, at 34-38 weeks, women with GDM tended to consume less sweet food and beverage overall, and less of other sweet food and

beverage than women with NGT. This dietary pattern was consistent with the dietary guidance they were given and also agrees with our previous diet analysis of these same women. Dietary assessment revealed that, at 34-38 weeks gestation, women with GDM consumed less carbohydrate as a percentage of total calories, and less simple sugar as a percentage of total carbohydrate (see **Section A**). The possibility exists that sweet food intake was underreported by women with GDM, however, their energy intakes were consistent with their overall weight gain (which was lower than women with NGT), and they successfully controlled their disease (see **Section A**).

In conclusion, despite a subset of women with GDM exhibiting a higher frequency of sweet cravings, women with GDM as a group showed proper disease management, solely through the use of diet. However, this may not always be the case. There may be women with GDM that are more susceptible to sweet cravings, and this could undermine their dietary adherence. It has already been reported that compliance with traditional diet therapies is low (25). A better understanding of the psychology and metabolic basis of sweet cravings in GDM could lead to innovative management strategies for this disease. It is our understanding that this is the first prospective study that linked dietary intake with cravings, and provides a foundation for future research linking these two influential factors of dietary management.

LITERATURE CITED

1. Dickens G, Trethowan W. Cravings and aversions during pregnancy. *J Psychosom Res* 1971;15:259-268.
2. Pope JF, Skinner JD, Carruth BR. Cravings and aversions of pregnant adolescents. *J Am Diet Assoc*. 1992;92:1479-1482.
3. Worthington-Roberts B, Little RE, Lambert MD, Wu R. Dietary cravings and aversions in the postpartum period. *J Am Diet Assoc*. 1989;89:647-651.
4. Bayley TM, Dye L, Jones S, DeBono M, Hill AJ. Food cravings and aversions during pregnancy: relationships with nausea and vomiting. *Appetite* 2002;38:45-51.
5. Hook EB. Dietary cravings and aversions during pregnancy. *Am J Clin Nutr* 1978;31:1355-1362.
6. Bowen DJ. Taste and food preference changes across the course of pregnancy. *Appetite* 1992;19:233-242.
7. Brown JE, Toma RB. Taste changes during pregnancy. *Am J Clin Nutr* 1986;43:414-418.
8. Kölblle N, Hummel T, von Mering R, Huch A, Huch R. Gustatory and olfactory function in the first trimester of pregnancy. *Eur J Obstet Gyn R B* 2001;99:179-183.
9. Kuga M, Ikeda M, Suzuki K, Takeuchi S. Changes in gustatory sense during pregnancy. *Acta Otolaryngol* 2002;Supplement 546:146-153.
10. Mercer ME. Food cravings, endogenous opioid peptides, and food intake: a review. *Appetite* 1997;29:325-352.
11. Curtis KS, Stratford JM, Contreras RJ. Estrogen increases the taste threshold for sucrose in rats. *Physiol Behav* 2005;86:281-286.
12. Dippel RL, Elias JW. Preferences for sweet in relationship to use of oral contraceptives and pregnancy. *Horm Behav* 1980;14:1-6.
13. Metzger BE, Coustan DR. Summary and recommendations of the fourth international workshop conference on gestational diabetes mellitus. *Diabetes Care* 1998;21:B161-7.
14. American Diabetes Association. Gestational diabetes mellitus. *Diabetes Care* 2004;27:S88-S90.

15. American Diabetes Association. Meal planning exchange lists. <http://www.diabetes.org/nutrition-and-recipes/nutrition/exchangelist.jsp>.
16. American Diabetes Association. Nutrition recommendations and interventions for diabetes: A position statement of the American Diabetes Association. *Diabetes Care* 2007;30:S48-S65.
17. Hardy SL, Brennand CP, Wyse BW. Taste thresholds of individuals with diabetes mellitus and of control subjects. *J Am Diet Assoc* 1981;79:286-289.
18. Perros P, MacFarlane TW, Counsell C, Frier BM. Altered taste sensation in newly-diagnosed NIDDM. *Diabetes Care* 1996;19:768-770.
19. Tepper BJ, Hartfiel LM, Schneider SH. Sweet taste and diet in type II diabetes. *Physiol Behav* 1996;60:13-18.
20. Tepper BJ, Seldner AC. Sweet taste and intake of sweet foods in normal pregnancy and pregnancy complicated by gestational diabetes mellitus. *Am J Clin Nutr* 1999;70:277-284.
21. Carpenter MW, Coustan DR. Criteria for screening tests for gestational diabetes. *American Journal of Obstetrics and Gynecology* 1982;144:768-773.
22. Weingarten HP, Elston D. Food cravings in a college population. *Appetite* 1991;17:167-175.
23. Liang K-Y, Zeger SL. Longitudinal data analysis using generalized linear models. *Biometrika* 1986;73:13-22.
24. Crystal SR, Bowen DJ, Bernstein IL. Morning sickness and salt intake, food cravings and food aversions. *Physiol Behav* 1999;67:181-187.
25. Armstrong CL, Brown LP, York R, Robbins D, Swank A. From diagnosis to home management: nutritional considerations for women with gestational diabetes. *Diabetes Educ* 1991;17: 455-459.

TABLE 2

Subject characteristics

	GDM	NGT	Nonpregnant controls
Total Enrollment	15	93	19
Age at entry (yr) ¹	29.2 ± 3.1	26.2 ± 0.7	27.1 ± 1.3
BMI at entry (kg/m ²) ¹	28.8 ± 2.6 ^a	27.0 ± 3.5 ^a	24.5 ± 0.5 ^b
Total pregnancy weight gain (kg) ¹	8.3 ± 1.5 ^a	13.9 ± 0.6 ^b	—
Ethnicity (%)			
Hispanic	60	52	79
Caucasian	20	20	11
African American	13	20	5
Asian	0	5	0
Other	7	3	5
Cesarean Delivery (%) ¹	27	30	—
Gestational age (wk) ¹	40.0 ± 0.6	39.4 ± 0.2	—
Infant birthweight (g) ¹	3321 ± 125	3325 ± 53	—
Breastfeeding (%) ¹	73	77	—

¹ Values are means ± SEM. Values in the same row with different superscripts are different at p<0.05.

TABLE 3*

All, Sweet and Savory cravings - % women with cravings (n)

ALL				
	16-20 wk	24-28 wk	34-38 wk	6-10 wk pp
GDM [#]	77.8 (7)	61.5 (8)	60.0 (6)	16.7 (4) ^a
NGT [#]	81.0 (68)	68.8 (53)	57.4 (39)	55.2 (64) ^b
Control	57.9 (11)	69.2 (9)	50.0 (6)	50.0 (12) ^b
SWEET				
	16-20 wk	24-28 wk	34-38 wk	6-10 wk pp
GDM	22.2 (2)	38.5 (5)	40.0 (4)	16.7 (2)
NGT [#]	44.0 (37)	54.6 (42)	45.6 (31)	29.3 (34)
Control	36.8 (7)	30.8 (4)	41.7 (5)	33.3 (8)
SAVORY				
	16-20 wk	24-28 wk	34-38 wk	6-10 wk pp
GDM [#]	66.7 (6)	38.5 (5)	30.0 (3)	8.3 (2) ^a
NGT [#]	46.4 (39)	28.6 (22)	20.6 (14)	29.3 (17) ^b
Control	31.6 (6)	38.5 (5)	16.7 (2)	8.3 (2) ^a

[#] Between session difference as assessed by random effect logistic regression model.^{*} Values within each experimental period with different superscripts are different at $p < 0.017$ (with Bonferroni corrections for multiple testing).

FIGURE LEGENDS

Figure 1 Flow diagram showing individual session progression.

Figure 2 Diagram indicating sample sizes at each stage of the study.

Figure 3 Mean sweet and savory craving frequency across pregnancy sessions. At 34-38 weeks, women with GDM exhibited a higher mean frequency of sweet cravings than both women with NGT and nonpregnant controls ($p=0.015$ and 0.009 , respectively). Women with GDM also showed a significantly higher mean sweet craving frequency at 34-38 weeks than at 24-28 weeks gestation ($p=0.0146$). There were no group differences at any session for mean savory craving frequency.

Figure 4 Daily sweet food and beverage intake across pregnancy sessions. At 24-28 weeks gestation, women with NGT showed a significantly higher intake of total sweet food and beverage than both women with GDM and nonpregnant controls ($p=0.015$ and 0.013 , respectively). Once separating the overall sweet food and beverage group into fruit/fruit juice and other sweet food and beverage intake, it appears that women with NGT consumed more servings of fruit/fruit juice than the other groups ($p<0.01$ for both comparisons). At 34-38 weeks gestation there was a trend ($p=0.0573$) for women with NGT to consume more daily servings of all sweet food and beverage than women with GDM. Women with NGT exhibited marginally higher daily intake of fruit/fruit juice when compared to the nonpregnant controls ($p=0.036$), and the higher daily intake of other sweet food and beverage neared significance ($p=0.02$) when women with NGT women were compared to women with GDM.

Figure 1

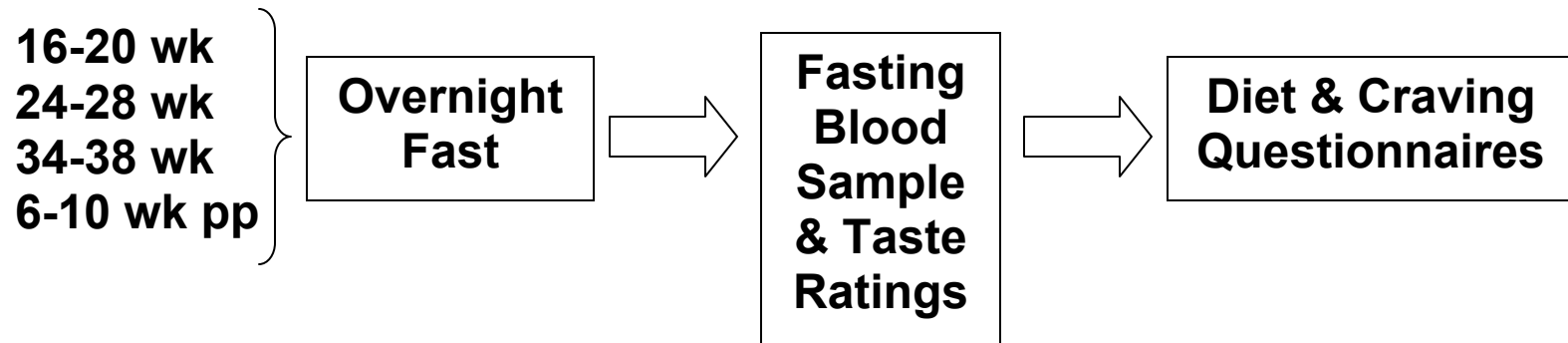


Figure 2

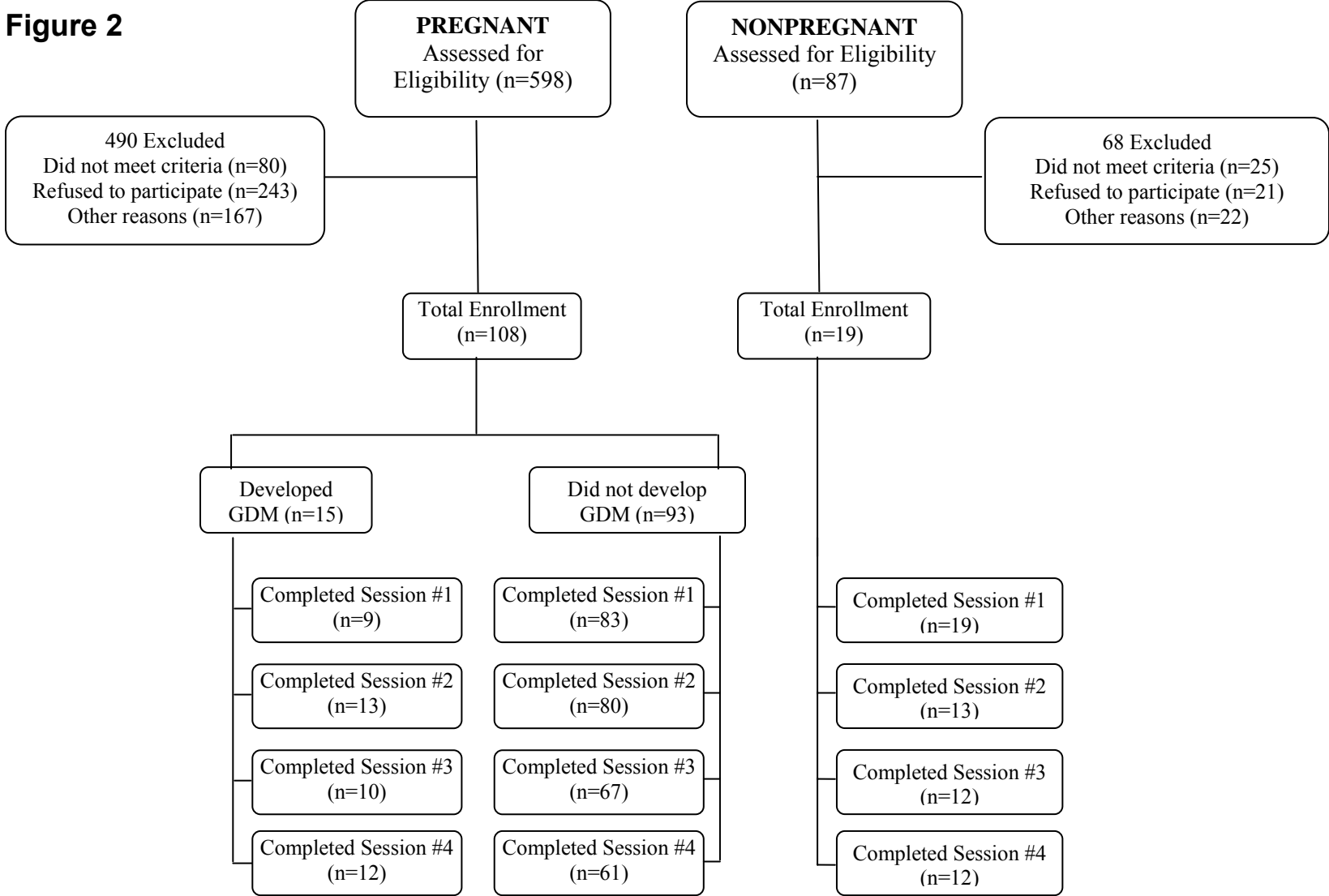
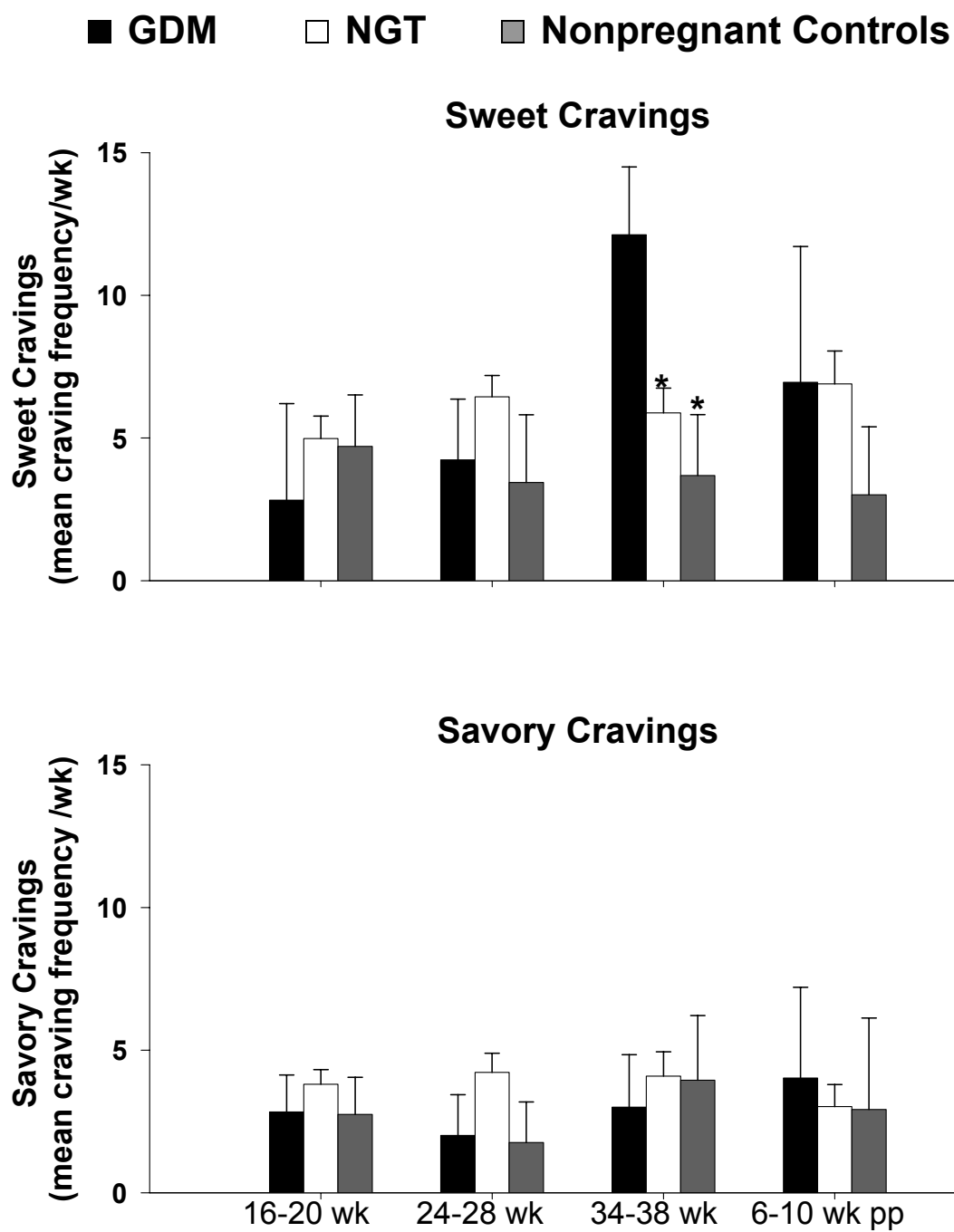


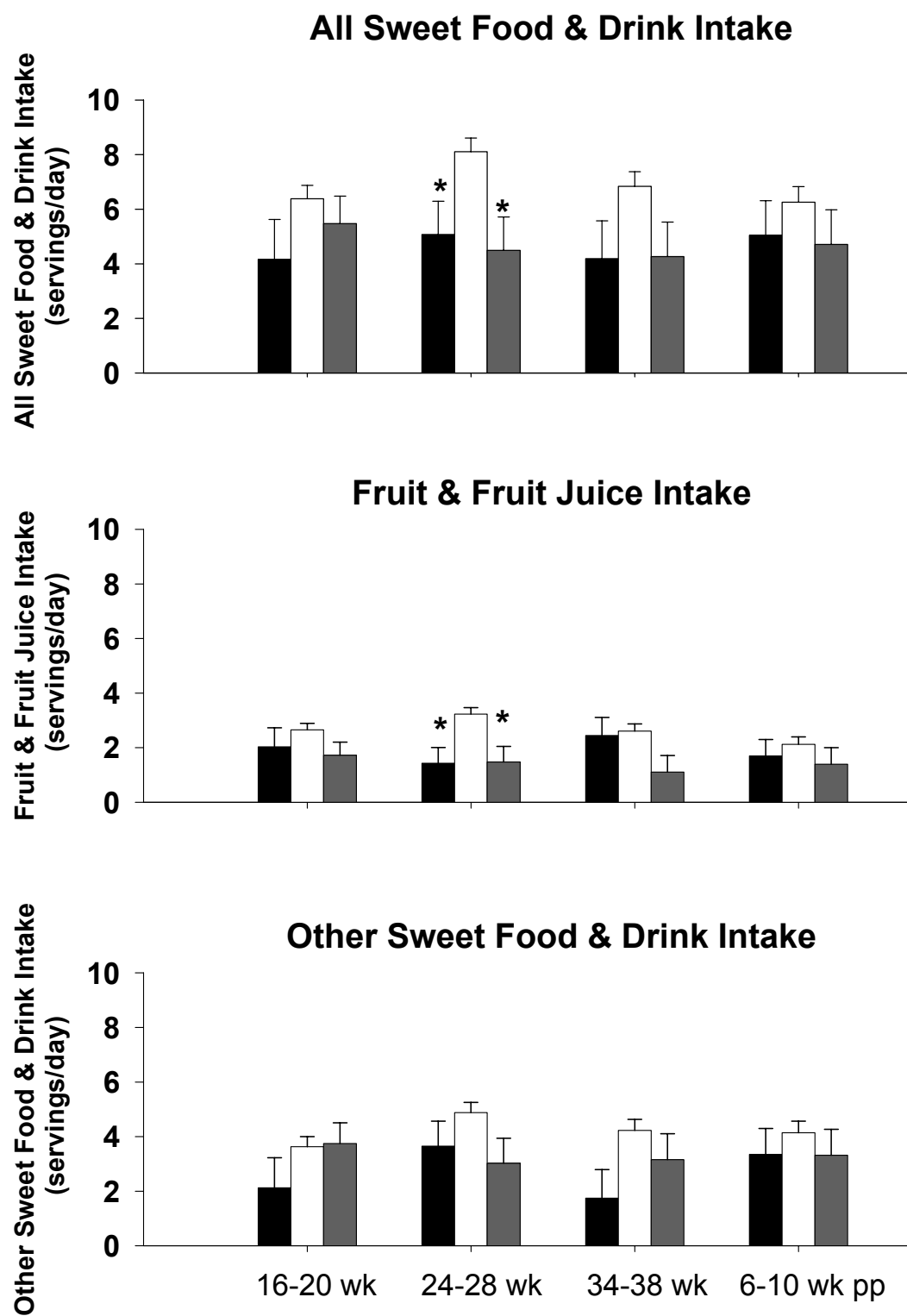
Figure 3



* Differs from GDM group ($p < 0.017$)

Figure 4

■ GDM □ NGT ■ Nonpregnant controls



* Differs from NGT group ($p < 0.017$)

4 CONCLUSION

This research project had three overall objectives in the study of women who did and did not develop diabetes during pregnancy: a) To document the temporal profile of serum insulin, leptin, cortisol and a measure of insulin resistance, HOMA-IR, across pregnancy and early postpartum; b) To test and investigate differences in taste parameters and relate any alterations to the dynamic endocrine measures found in diabetes and pregnancy; and c) To collect and analyze data regarding food cravings and sweet food intake during and following gestation.

In this study, women with NGT followed the metabolic and endocrine courses characteristic of healthy pregnancy. Parameters such as insulin, leptin, cortisol, estrogen, progesterone and triglycerides all showed elevations across gestation and returned to a baseline level following delivery. Surprisingly, the hormone profile found in women with GDM did not differ as considerably as anticipated from the pattern found in women with NGT. However this may have been due to the fact that the study participants with GDM had a mild form of the disease, suggested by the fact that fasting glucose levels remained normal and glucose levels were only moderately elevated following a glucose challenge. Also, infant birthweights and cesarean delivery rates were both similar between the two pregnant groups. Serum leptin had a tendency to more sharply increase from 16-20 weeks to 24-28 weeks in women with GDM, as compared to women with NGT, and therefore showed a marginally higher leptin level at 24-28 weeks. However, by 34-38 weeks, leptin levels in NGT women rose in such a way that the two pregnant groups did not differ at that time.

Appropriate weight of the infants at birth and normal rates of cesarean delivery, amongst other factors, may also provide evidence that the women with GDM successfully controlled their diabetes solely with the use of dietary management. Other such elements included the fact that women with GDM showed appropriate weight gain, based on recommendations for women overweight prior to pregnancy. Dietary compliance seemed high, since women in the GDM group reported lower energy and carbohydrate intake than women with NGT at 34-38 weeks. This pattern follows typical dietary recommendations provided to women with GDM. These results suggest that, in mild GDM, dietary counseling is an important part of medical management.

In women with GDM, the averaged sweetness liking rating for glucose solutions was positively correlated with fasting insulin levels at 24-28 weeks. At this same time point, there was also a relationship seen between the averaged sweetness liking rating of 10% sucrose-sweetened milk and fasting leptin levels. These relationships were not seen in any other group, or at any other session. Later in pregnancy, at 34-38 weeks gestation, women with GDM preferred the sweetness, creaminess and flavor of 5% sucrose-sweetened milk and the creaminess of the 10% sucrose-sweetened milk more than women with NGT. These data indicate that both pregnancy as well as GDM may affect sweet taste. Moreover, differences in sweet taste preference may be linked to alterations in endocrine parameters in women with GDM.

Most pregnant women in this study reported at least one craving during pregnancy. The highest number of cravings was reported at mid-pregnancy (16-20 weeks), with reported cravings diminishing from that point to the end of gestation. Women with NGT seemed to show a small peak in reported sweet cravings at 24-28

weeks, and this pattern was not seen in women with GDM. However, within the sample of women who reported sweet cravings, women with GDM reported a higher frequency of this type of craving than both women with NGT and nonpregnant controls. Women with GDM also differed from themselves, in that sweet craving frequency was significantly higher at 34-38 weeks than 24-28 weeks gestation.

At 24-28 weeks, women with NGT reported a higher daily consumption of sweet food and beverage than women with GDM and nonpregnant controls. When all sweet food and beverage was segmented, it appears that the food group that contributed to the overall sweet food and beverage differences was fruit and fruit juice. At 34-38 weeks, women with NGT continued to show a higher reported sweet food and beverage intake than the other two groups, though this difference was only marginally significant. Upon separation into more specific groups, women with GDM seem to report a lower daily intake of other sweet food and beverage, exclusive of fruit and fruit juice.

Women with GDM in this study, upon assessment of endocrine and dietary parameters, exhibited appropriate disease management, through the use of diet alone. The higher dietary compliance in our population may not be typical, and other groups of women with GDM may not adhere as well to the provided dietary advice. Furthermore, other women who develop GDM during pregnancy may succumb to sweet cravings, thereby detracting from dietary management. Further comprehension of metabolic and/or psychological factors that drive differences in sweet taste and cravings in women with GDM may lead to novel disease management methods.

Overall, women with GDM seem to show differences in endocrine, taste and dietary parameters, each of which have the potential to affect treatment methods and

success. The pattern of leptin across gestation may serve as a marker of future GDM development, and, in women with GDM, appears to show a relationship with sweet taste of sweetened milks early in the third trimester of pregnancy. Moreover, since dietary treatment is integral in the management of GDM, this study has provided a foundation for the further investigation into the relationship between sweet cravings and dietary intake, both of which, if uncontrolled, could undermine proper disease management methods.

This study strengthens past research regarding the contrast between women with GDM and women with NGT, regarding metabolism, taste and dietary intake. Continued research on other potential hormone or metabolic markers, and other factors affecting treatment methods for GDM remains important, since the disease is one of the main risk factors for the development of type 2 diabetes in the future.

5 APPENDIX

Appendix A: Data Tables¹

¹ Values are estimates \pm SE according to mixed procedure analysis.
Values in the same row with different superscripts are different at $p < 0.017$.

TABLE 5.1 – OTHER SUBJECT CHARACTERISTICS

		GDM		NGT		Control	
Body weight at entry (kg)		70.18	± 3.3	69.91	± 1.3	62.37	± 2.9
Height at entry (m)		1.60	± 0.0	1.60	± 0.0	1.60	± 0.0
BMI at entry (kg/m ²)		28.77	± 2.6 ^a	26.95	± 3.5 ^a	24.12	± 0.5 ^b
Smoking status (% nonsmoker)		84.62	. .	93.41	. .	100.00	. .
Ethnicity (%)							
	White	20.00	. .	20.43	. .	10.53	. .
	Black	13.33	. .	20.43	. .	5.26	. .
	Hispanic	60.00	. .	51.61	. .	78.95	. .
	Asian	0.00	. .	4.30	. .	0.00	. .
	Other	6.67	. .	3.32	. .	5.26	. .
Income (%)							
	<\$15,000	6.67	. .	13.19	. .	10.53	. .
	\$15,000-\$24,000	60.00	. .	49.45	. .	47.37	. .
	\$25,000-\$34,000	33.33	. .	32.97	. .	36.84	. .
	\$35,000-\$44,000	0.00	. .	4.40	. .	5.26	. .
	≥ \$45,000	0.00	. .	12.35	. .	5.88	. .
Highest education achieved (%)							
	Elementary school	6.67	. .	13.19	. .	10.53	. .
	High school	60.00	. .	49.45	. .	47.37	. .
	College	33.33	. .	32.97	. .	36.84	. .
	Graduate school	0.00	. .	4.40	. .	5.26	. .
Baby gender (% male)		60.00	. .	39.76
Feeding (% breastfed)		60.00	. .	53.25
Caesarean delivery (%)		26.67	. .	30.38

TABLE 5.2 – FASTING BLOOD PARAMETERS

		GDM			NGT			Control				
			±			±			±			
Insulin (μ U/mL)	16-20 wk	25.21	±	3.9		16.12	±	1.3		16.05	±	2.8
	24-28 wk	27.53	±	3.4	^a	20.15	±	1.4	^{a,b}	11.48	±	3.4
	34-38 wk	24.81	±	3.7		23.51	±	1.5		13.07	±	3.5
	6-10 wk pp	12.94	±	3.7		16.01	±	1.6		13.36	±	4.1
Cortisol (μ g/M)	16-20 wk	28.96	±	4.9		32.06	±	1.6		25.49	±	3.4
	24-28 wk	50.88	±	4.0	^a	51.17	±	1.7	^a	32.52	±	4.2
	34-38 wk	57.57	±	4.9	^a	50.20	±	1.8	^a	29.93	±	4.4
	6-10 wk pp	19.22	±	4.4	^{a,b}	20.48	±	1.9	^a	37.84	±	4.4
Leptin (ng/mL)	16-20 wk	20.43	±	3.6		21.59	±	1.2		15.26	±	2.5
	24-28 wk	34.98	±	3.0	^a	26.80	±	1.2	^{a,b}	15.78	±	3.1
	34-38 wk	35.14	±	3.4	^a	30.87	±	1.3	^a	16.55	±	3.2
	6-10 wk pp	22.60	±	3.2		20.54	±	1.4		15.54	±	3.2
Estrogen (pg/mL)	16-20 wk	2515.45	±	1396.4	^{a,b}	3197.54	±	462.6	^a	104.69	±	1015.8
	24-28 wk	9682.95	±	1161.8	^a	10308.00	±	477.5	^a	193.50	±	1161.8
	34-38 wk	13118.00	±	1324.8	^a	14593.00	±	513.5	^a	230.43	±	1209.6
	6-10 wk pp	-33.14	±	1209.9		-34.72	±	541.8		239.53	±	1209.6
Progesterone (ng/mL)	16-20 wk	48.50	±	17.6	^a	57.15	±	5.8	^a	5.86	±	13.6
	24-28 wk	128.16	±	14.6	^a	133.26	±	6.0	^a	1.34	±	16.7
	34-38 wk	295.28	±	16.7	^a	261.84	±	6.4	^a	6.64	±	15.3
	6-10 wk pp	3.77	±	16.7		2.27	±	7.9		12.63	±	16.7
Glucose (mg/dL)	16-20 wk	83.19	±	3.4	^{a,b}	77.61	±	1.1	^a	88.98	±	2.4
	24-28 wk	83.11	±	2.7		77.00	±	1.2		81.51	±	3.0
	34-38 wk	76.25	±	3.2		79.18	±	1.2		83.58	±	3.1
	6-10 wk pp	90.47	±	3.0		85.57	±	1.3		80.49	±	3.1
Tri- acylglycerol (mg/dL)	16-20 wk	164.42	±	23.3		139.37	±	7.8		104.15	±	16.0
	24-28 wk	211.95	±	18.7	^a	201.73	±	8.0	^a	93.22	±	19.4
	34-38 wk	246.91	±	22.1	^a	250.01	±	8.5	^a	89.07	±	20.2
	6-10 wk pp	159.17	±	20.2		134.62	±	9.0		85.32	±	20.2
VLDL (mg/dL)	16-20 wk	32.99	±	4.7		27.85	±	1.6		20.95	±	3.2
	24-28 wk	42.32	±	3.7	^a	40.36	±	1.6	^a	18.60	±	3.9
	34-38 wk	49.42	±	4.4	^a	50.00	±	1.7	^a	17.85	±	4.0
	6-10 wk pp	31.93	±	4.0		26.93	±	1.8		17.01	±	4.0
FFA (mEq/L)	16-20 wk	0.48	±	0.1		0.40	±	0.0		0.39	±	0.1
	24-28 wk	0.39	±	0.1		0.33	±	0.0		0.34	±	0.1
	34-38 wk	0.44	±	0.1		0.41	±	0.0		0.40	±	0.1
	6-10 wk pp	0.40	±	0.1		0.45	±	0.0		0.32	±	0.1
HOMA-IR	16-20 wk	5.16	±	0.9		3.15	±	0.3		3.57	±	0.6
	24-28 wk	5.68	±	0.7	^a	3.89	±	0.3	^{a,b}	2.31	±	0.7
	34-38 wk	4.71	±	0.8		4.68	±	0.3		2.75	±	0.8
	6-10 wk pp	2.75	±	0.8		3.49	±	0.3		2.64	±	0.9

TABLE 5.3 – POST-GLUCOLA BLOOD PARAMETERS

		GDM			NGT			Control		
Insulin (μ U/mL)	24-28 wk	112.96	\pm 11.2	^a	79.65	\pm 4.7	^b	54.15	\pm 10.7	^b
	34-38 wk	121.04	\pm 13.7	^a	98.48	\pm 5.4	^a	62.51	\pm 11.2	^b
	6-10 wk pp	54.13	\pm 12.9		44.68	\pm 5.1		41.55	\pm 13.7	
Cortisol (μ g/M)	24-28 wk	44.83	\pm 3.8	^a	43.48	\pm 1.5	^a	27.40	\pm 3.6	^b
	34-38 wk	40.60	\pm 4.1	^a	46.99	\pm 1.7	^a	26.68	\pm 3.9	^b
	6-10 wk pp	13.64	\pm 3.8	^a	15.50	\pm 1.7	^a	32.23	\pm 3.8	^b
Glucose (mg/dL)	24-28 wk	143.20	\pm 7.8	^a	105.16	\pm 3.2	^b	96.25	\pm 7.5	^b
	34-38 wk	148.40	\pm 9.6	^a	115.99	\pm 3.7	^b	104.17	\pm 7.8	^b
	6-10 wk pp	131.94	\pm 8.6	^a	93.90	\pm 3.6	^b	91.14	\pm 9.6	^b
Triacylglycerol (mg/dL)	24-28 wk	218.29	\pm 20.5	^a	217.04	\pm 8.5	^a	95.44	\pm 20.5	^b
	34-38 wk	250.41	\pm 23.4	^a	267.38	\pm 9.1	^a	89.06	\pm 21.3	^b
	6-10 wk pp	156.93	\pm 21.4		133.81	\pm 9.6		88.89	\pm 21.3	
VLDL (mg/dL)	24-28 wk	43.61	\pm 4.1	^a	43.37	\pm 1.7	^a	18.83	\pm 4.1	^b
	34-38 wk	50.22	\pm 4.7	^a	53.48	\pm 1.8	^a	17.85	\pm 4.3	^b
	6-10 wk pp	31.26	\pm 4.3		26.73	\pm 1.9		17.77	\pm 4.3	
FFA (mEq/L)	24-28 wk	0.25	\pm 0.0	^{a,b}	0.25	\pm 0.0	^a	0.13	\pm 0.0	^b
	34-38 wk	0.31	\pm 0.0	^a	0.26	\pm 0.0	^a	0.13	\pm 0.0	^b
	6-10 wk pp	0.16	\pm 0.0		0.17	\pm 0.0		0.14	\pm 0.0	

TABLE 5.4 – FASTING GLUCOSE SOLUTION RATINGS

<u>Glucose Solution Concentration</u>	<u>Taste Quality</u>		<u>GDM</u>		<u>NGT</u>		<u>Control</u>	
0.01M	Sweetness Intensity	16-20 wk	2.24	± 0.6	1.27	± 0.2	1.61	± 0.4
		24-28 wk	0.57	± 0.5	1.06	± 0.2	0.56	± 0.5
		34-38 wk	0.75	± 0.6	0.69	± 0.2	0.46	± 0.6
		6-10 wk pp	1.06	± 0.6	1.04	± 0.3	0.82	± 0.6
	Sweetness Liking	16-20 wk	7.83	± 1.5	6.21	± 0.5	4.24	± 1.1
		24-28 wk	5.54	± 1.3	5.72	± 0.5	2.45	± 1.3
		34-38 wk	5.51	± 1.5	5.56	± 0.6	2.37	± 1.3
		6-10 wk pp	5.17	± 1.3	5.28	± 0.6	3.61	± 1.3
0.02M	Sweetness Intensity	16-20 wk	2.30	± 0.7	1.49	± 0.2	1.62	± 0.5
		24-28 wk	1.68	± 0.6	1.09	± 0.2	0.65	± 0.6
		34-38 wk	0.89	± 0.6	0.92	± 0.2	0.57	± 0.6
		6-10 wk pp	0.90	± 0.6	0.98	± 0.3	0.98	± 0.6
	Sweetness Liking	16-20 wk	7.83	± 1.6	6.24	± 0.5	5.32	± 1.1
		24-28 wk	5.00	± 1.4	5.73	± 0.5	2.71	± 1.3
		34-38 wk	5.70	± 1.5	5.63	± 0.6	3.41	± 1.4
		6-10 wk pp	3.57	± 1.4	5.32	± 0.6	3.19	± 1.4
0.04M	Sweetness Intensity	16-20 wk	2.54	± 0.7	1.31	± 0.2	1.99	± 0.5
		24-28 wk	0.88	± 0.6	1.43	± 0.2	0.96	± 0.6
		34-38 wk	1.03	± 0.7	1.13	± 0.3	0.35	± 0.6
		6-10 wk pp	0.92	± 0.6	1.30	± 0.3	0.83	± 0.6
	Sweetness Liking	16-20 wk	7.33	± 1.5	6.14	± 0.5	5.47	± 1.0
		24-28 wk	5.69	± 1.3	5.29	± 0.5	2.71	± 1.3
		34-38 wk	3.86	± 1.4	5.45	± 0.6	2.48	± 1.3
		6-10 wk pp	3.61	± 1.3	5.38	± 0.6	2.59	± 1.3
0.08M	Sweetness Intensity	16-20 wk	4.19	± 0.8	2.76	± 0.3	2.18	± 0.6
		24-28 wk	1.26	± 0.7	2.01	± 0.3	1.08	± 0.7
		34-38 wk	0.87	± 0.8	1.92	± 0.3	0.94	± 0.7
		6-10 wk pp	1.11	± 0.7	1.87	± 0.3	1.52	± 0.7
	Sweetness Liking	16-20 wk	7.28	± 1.5	6.07	± 0.5	5.04	± 1.0
		24-28 wk	5.92	± 1.3	5.41	± 0.5	3.37	± 1.2
		34-38 wk	4.72	± 1.4	5.62	± 0.5	2.73	± 1.3
		6-10 wk pp	4.67	± 1.3	5.53	± 0.6	3.47	± 1.3
0.16M	Sweetness Intensity	16-20 wk	6.34	± 1.2	4.96	± 0.4	5.20	± 0.8
		24-28 wk	2.98	± 1.0	4.19	± 0.4	1.95	± 1.0
		34-38 wk	3.40	± 1.1	3.18	± 0.4	1.75	± 1.0
		6-10 wk pp	3.60	± 1.0	3.59	± 0.5	2.40	± 1.0
	Sweetness Liking	16-20 wk	5.93	± 1.4	6.11	± 0.5	6.01	± 1.0
		24-28 wk	4.08	± 1.2	5.85	± 0.5	3.25	± 1.2
		34-38 wk	4.17	± 1.3	4.99	± 0.5	3.15	± 1.2
		6-10 wk pp	5.63	± 1.2	5.40	± 0.6	3.41	± 1.2
AVERAGE	Sweetness Intensity	16-20 wk	3.52	± 0.6	2.36	± 0.2	2.52	± 0.4
		24-28 wk	1.48	± 0.5	1.95	± 0.2	1.04	± 0.5
		34-38 wk	1.39	± 0.6	1.57	± 0.2	0.81	± 0.5
		6-10 wk pp	1.52	± 0.5	1.76	± 0.2	1.31	± 0.5
	Sweetness Liking	16-20 wk	7.24	± 1.3	6.16	± 0.4	5.22	± 0.9
		24-28 wk	5.25	± 1.1	5.60	± 0.4	2.90	± 1.1
		34-38 wk	4.79	± 1.2	5.45	± 0.5	2.83	± 1.1
		6-10 wk pp	4.53	± 1.1	5.38	± 0.5	3.25	± 1.1

TABLE 5.5 – FASTING STRAWBERRY MILK RATINGS

<u>Strawberry</u> <u>Milk</u> <u>Concentration</u>	<u>Taste</u> <u>Quality</u>		GDM			NGT			Control		
0% Fat 0% Sucrose	Sweetness	16-20 wk	2.66	± 0.8	1.30	± 0.3	1.93	± 0.6			
		24-28 wk	0.73	± 0.7	1.20	± 0.3	2.56	± 0.7			
		34-38 wk	1.18	± 0.8	1.61	± 0.3	1.60	± 0.7			
		6-10 wk pp	1.09	± 0.7	1.13	± 0.3	1.54	± 0.7			
	Sweetness	16-20 wk	3.13	± 1.2	2.43	± 0.4	2.18	± 0.8			
		24-28 wk	3.22	± 1.0	2.44	± 0.4	2.48	± 1.0			
		34-38 wk	4.05	± 1.1	3.26	± 0.4	1.68	± 1.0			
		6-10 wk pp	3.63	± 1.0	2.30	± 0.5	2.10	± 1.0			
	Liking	16-20 wk	2.88	± 1.0	2.49	± 0.3	2.51	± 0.7			
		24-28 wk	2.17	± 0.8	2.24	± 0.3	2.19	± 0.8			
		34-38 wk	3.08	± 0.9	2.59	± 0.4	2.98	± 0.9			
		6-10 wk pp	3.54	± 0.9	2.73	± 0.4	2.16	± 0.9			
	Creaminess	16-20 wk	3.66	± 1.2	3.21	± 0.4	2.79	± 0.8			
		24-28 wk	3.87	± 1.0	2.95	± 0.4	3.66	± 1.0			
		34-38 wk	4.36	± 1.1	3.98	± 0.4	2.74	± 1.0			
		6-10 wk pp	4.65	± 1.0	3.13	± 0.5	2.43	± 1.0			
	Liking	16-20 wk	2.66	± 1.2	2.91	± 0.4	3.49	± 0.8			
		24-28 wk	2.31	± 1.0	3.03	± 0.4	2.83	± 1.0			
		34-38 wk	1.88	± 1.1	3.19	± 0.4	2.58	± 1.0			
		6-10 wk pp	3.32	± 1.0	3.00	± 0.5	2.50	± 1.0			
Flavor	16-20 wk	5.13	± 1.2	2.46	± 0.4	2.14	± 0.8				
	24-28 wk	3.97	± 1.0	2.25	± 0.4	3.34	± 1.0				
	34-38 wk	4.82	± 1.1	3.23	± 0.4	1.89	± 1.0				
	6-10 wk pp	4.85	± 1.0	2.83	± 0.5	2.07	± 1.0				
0% Fat 5% Sucrose	Sweetness	16-20 wk	5.76	± 1.3	6.03	± 0.4	5.39	± 0.8			
		24-28 wk	5.02	± 1.0	5.67	± 0.4	5.52	± 1.0			
		34-38 wk	4.02	± 1.1	4.93	± 0.4	3.93	± 1.0			
		6-10 wk pp	3.93	± 1.0	5.54	± 0.5	5.01	± 1.0			
	Sweetness	16-20 wk	5.84	± 1.5	5.80	± 0.5	5.81	± 1.0			
		24-28 wk	5.28	± 1.2	5.73	± 0.5	4.77	± 1.2			
		34-38 wk	7.71	± 1.3	6.21	± 0.5	4.02	± 1.2			
		6-10 wk pp	5.06	± 1.2	5.90	± 0.5	4.14	± 1.2			
	Liking	16-20 wk	5.16	± 1.1	4.51	± 0.4	4.38	± 0.7			
		24-28 wk	4.83	± 0.9	4.51	± 0.4	4.44	± 0.9			
		34-38 wk	4.68	± 1.0	4.53	± 0.4	3.46	± 0.9			
		6-10 wk pp	3.75	± 0.9	5.02	± 0.4	3.70	± 0.9			
	Creaminess	16-20 wk	5.16	± 1.3	5.44	± 0.4	5.52	± 0.9			
		24-28 wk	6.10	± 1.1	5.51	± 0.4	4.82	± 1.1			
		34-38 wk	7.64	± 1.2	5.77	± 0.5	4.05	± 1.1			
		6-10 wk pp	5.82	± 1.1	5.10	± 0.5	3.95	± 1.1			
	Liking	16-20 wk	6.00	± 1.3	6.22	± 0.4	6.45	± 0.8			
		24-28 wk	5.91	± 1.0	5.44	± 0.4	5.23	± 1.0			
		34-38 wk	5.61	± 1.1	5.56	± 0.4	4.26	± 1.0			
		6-10 wk pp	4.12	± 1.0	5.59	± 0.4	4.94	± 1.0			
Flavor	16-20 wk	5.70	± 1.4	5.95	± 0.5	5.58	± 1.0				
	24-28 wk	6.55	± 1.2	5.49	± 0.5	4.88	± 1.2				
	34-38 wk	8.66	± 1.3	6.14	± 0.5	4.64	± 1.2				
	6-10 wk pp	6.45	± 1.2	5.61	± 0.5	4.95	± 1.2				

0% Fat 10% Sucrose	Sweetness Intensity	16-20 wk	9.79 ^{a,b}	± 1.3	9.30 ^a	± 0.4	7.12 ^b	± 0.9
		24-28 wk	8.61	± 1.0	9.22	± 0.4	9.37	± 1.0
		34-38 wk	8.64	± 1.2	8.69	± 0.5	7.85	± 1.1
		6-10 wk pp	8.48	± 1.1	8.16	± 0.5	8.02	± 1.1
	Sweetness Liking	16-20 wk	6.79	± 1.4	7.25	± 0.5	7.40	± 1.0
		24-28 wk	8.35	± 1.2	6.20	± 0.5	7.60	± 1.2
		34-38 wk	8.14	± 1.3	7.21	± 0.5	6.96	± 1.2
		6-10 wk pp	7.13	± 1.2	7.31	± 0.5	8.17	± 1.2
	Creaminess Intensity	16-20 wk	5.40	± 1.2	6.07	± 0.4	4.92	± 0.8
		24-28 wk	6.36	± 1.0	6.52	± 0.4	5.17	± 1.0
		34-38 wk	6.25	± 1.1	6.27	± 0.4	5.62	± 1.0
		6-10 wk pp	6.91	± 1.0	6.17	± 0.5	6.57	± 1.0
	Creaminess Liking	16-20 wk	7.38	± 1.3	6.26	± 0.4	6.44	± 0.9
		24-28 wk	9.00 ^a	± 1.1	6.18 ^b	± 0.4	5.74 ^b	± 1.1
		34-38 wk	9.49 ^a	± 1.2	7.01 ^{a,b}	± 0.5	6.07 ^b	± 1.1
		6-10 wk pp	7.38	± 1.1	7.14	± 0.5	7.72	± 1.1
	Flavor Intensity	16-20 wk	7.39	± 1.2	8.46	± 0.4	6.64	± 0.8
		24-28 wk	9.83	± 1.0	8.18	± 0.4	7.93	± 1.0
		34-38 wk	7.63	± 1.1	8.11	± 0.4	6.93	± 1.0
		6-10 wk pp	8.91	± 1.0	7.61	± 0.5	8.47	± 1.0
	Flavor Liking	16-20 wk	6.84	± 1.4	7.26	± 0.5	7.79	± 1.0
		24-28 wk	9.20	± 1.2	6.30	± 0.5	7.31	± 1.2
		34-38 wk	9.66	± 1.4	7.09	± 0.5	7.43	± 1.2
		6-10 wk pp	8.17	± 1.2	8.14	± 0.6	8.01	± 1.2
0% Fat 20% Sucrose	Sweetness Intensity	16-20 wk	11.85	± 1.1	11.92	± 0.4	11.00	± 0.8
		24-28 wk	12.19	± 0.9	11.61	± 0.4	12.68	± 0.9
		34-38 wk	10.52	± 1.0	11.40	± 0.4	11.26	± 0.9
		6-10 wk pp	10.48	± 0.9	11.51	± 0.4	11.87	± 0.9
	Sweetness Liking	16-20 wk	6.36	± 1.5	6.24	± 0.5	5.62	± 1.1
		24-28 wk	7.25	± 1.3	6.92	± 0.5	8.55	± 1.3
		34-38 wk	8.17	± 1.5	6.89	± 0.6	8.99	± 1.3
		6-10 wk pp	6.71	± 1.3	7.89	± 0.6	9.67	± 1.3
	Creaminess Intensity	16-20 wk	6.87	± 1.3	6.70	± 0.4	6.26	± 0.9
		24-28 wk	7.29	± 1.1	7.25	± 0.4	7.48	± 1.1
		34-38 wk	9.26	± 1.3	7.27	± 0.5	8.27	± 1.2
		6-10 wk pp	8.10	± 1.2	6.84	± 0.5	8.47	± 1.2
	Creaminess Liking	16-20 wk	6.79	± 1.4	6.45	± 0.5	6.37	± 1.0
		24-28 wk	8.10	± 1.1	6.78	± 0.5	8.02	± 1.1
		34-38 wk	9.61	± 1.3	7.52	± 0.5	8.16	± 1.2
		6-10 wk pp	7.05	± 1.2	7.48	± 0.5	9.37	± 1.2
	Flavor Intensity	16-20 wk	10.07	± 1.2	10.47	± 0.4	10.17	± 0.9
		24-28 wk	9.47	± 1.0	10.25	± 0.4	10.78	± 1.0
		34-38 wk	10.58	± 1.2	10.12	± 0.5	11.07	± 1.1
		6-10 wk pp	9.47	± 1.1	10.76	± 0.5	11.27	± 1.1
	Flavor Liking	16-20 wk	6.36	± 1.5	6.62	± 0.5	6.53	± 1.1
		24-28 wk	9.51	± 1.3	7.07	± 0.5	7.99	± 1.3
		34-38 wk	8.82	± 1.5	7.00	± 0.6	9.37	± 1.3
		6-10 wk pp	7.82	± 1.3	7.93	± 0.6	9.91	± 1.3
5% Fat 0% Sucrose	Sweetness Intensity	16-20 wk	2.72 ^a	± 0.7	0.82 ^b	± 0.2	1.13 ^b	± 0.4
		24-28 wk	0.71	± 0.5	1.39	± 0.2	2.41	± 0.5
		34-38 wk	0.98	± 0.6	1.37	± 0.2	1.27	± 0.6
		6-10 wk pp	1.61	± 0.6	1.04	± 0.2	2.04	± 0.6
	Sweetness Liking	16-20 wk	4.67	± 1.1	2.70	± 1.5	2.66	± 0.8
		24-28 wk	2.91	± 0.9	2.37	± 0.4	2.74	± 0.9
		34-38 wk	3.99	± 1.1	3.04	± 0.4	1.57	± 1.0
		6-10 wk pp	2.00	± 1.0	2.42	± 0.4	2.00	± 1.0

5% Fat 0% Sucrose (cont.)	Creaminess	16-20 wk	4.00	± 1.1	3.02	± 0.4	3.44	± 0.8	
		Intensity	24-28 wk	2.12	± 0.9	3.79	± 0.4	3.99	± 0.9
			34-38 wk	2.39	± 1.0	3.24	± 0.4	3.25	± 1.0
			6-10 wk pp	3.41	± 1.0	3.15	± 0.4	3.12	± 1.0
	Creaminess	16-20 wk	6.27	^a ± 1.2	3.13	^b ± 0.4	3.37	^{a,b} ± 0.8	
		Liking	24-28 wk	4.02	± 1.0	3.75	± 0.4	3.66	± 1.0
			34-38 wk	5.13	± 1.1	3.74	± 0.4	2.74	± 1.0
			6-10 wk pp	3.07	± 1.0	3.30	± 0.4	2.86	± 1.0
	Flavor	16-20 wk	1.99	± 1.2	2.44	± 0.4	3.07	± 0.8	
		Intensity	24-28 wk	2.00	± 0.9	3.15	± 0.4	2.41	± 0.9
			34-38 wk	1.99	± 1.0	3.38	± 0.4	2.72	± 1.0
			6-10 wk pp	3.37	± 1.0	2.82	± 0.4	2.42	± 1.0
	Flavor	16-20 wk	4.47	± 1.2	3.19	± 0.4	2.57	± 0.8	
		Liking	24-28 wk	3.86	± 1.0	3.05	± 0.4	2.92	± 1.0
			34-38 wk	4.90	^a ± 1.1	2.85	^{a,b} ± 0.4	1.68	^b ± 1.0
			6-10 wk pp	3.67	± 1.0	2.35	± 0.5	2.43	± 1.0
5% Fat 5% Sucrose	Sweetness	16-20 wk	6.36	± 1.2	6.16	± 0.4	5.96	± 0.8	
		Intensity	24-28 wk	6.86	± 1.0	4.84	± 0.4	5.29	± 1.0
			34-38 wk	6.54	± 1.1	5.18	± 0.4	3.69	± 1.0
			6-10 wk pp	6.40	± 1.0	5.47	± 0.5	4.84	± 1.0
	Sweetness	16-20 wk	6.90	± 1.4	6.51	± 0.5	6.12	± 0.9	
		Liking	24-28 wk	7.07	± 1.1	5.55	± 0.5	5.83	± 1.1
			34-38 wk	9.17	^a ± 1.3	5.88	^{a,b} ± 0.5	3.61	^b ± 1.2
			6-10 wk pp	7.98	± 1.2	6.65	± 0.5	3.99	± 1.2
	Creaminess	16-20 wk	5.54	± 1.1	4.99	± 0.4	5.08	± 0.8	
		Intensity	24-28 wk	4.67	± 0.9	4.88	± 0.4	5.77	± 0.9
			34-38 wk	7.36	± 1.1	4.82	± 0.4	4.28	± 1.0
			6-10 wk pp	5.51	± 1.0	5.27	± 0.4	4.26	± 1.0
	Creaminess	16-20 wk	6.34	± 1.3	5.59	± 0.4	5.74	± 0.9	
		Liking	24-28 wk	5.19	± 1.1	5.34	± 0.4	5.35	± 1.1
			34-38 wk	8.98	^a ± 1.2	5.70	^b ± 0.5	4.34	^b ± 1.1
			6-10 wk pp	7.75	^a ± 1.1	5.77	^{a,b} ± 0.5	3.90	^b ± 1.1
Flavor	16-20 wk	6.19	± 1.2	6.07	± 0.4	5.76	± 0.8		
	Intensity	24-28 wk	6.65	± 1.0	5.06	± 0.4	5.09	± 1.0	
		34-38 wk	7.69	^a ± 1.1	5.57	^{a,b} ± 0.4	3.88	^b ± 1.0	
		6-10 wk pp	6.83	± 1.0	5.82	± 0.4	4.38	± 1.0	
Flavor	16-20 wk	5.87	± 1.4	6.21	± 0.5	6.04	± 0.9		
	Liking	24-28 wk	6.37	± 1.1	5.14	± 0.5	5.47	± 1.1	
		34-38 wk	9.01	± 1.3	6.14	± 0.5	3.94	± 1.2	
		6-10 wk pp	9.02	± 1.2	6.16	± 0.5	4.18	± 1.2	
5% Fat 10% Sucrose	Sweetness	16-20 wk	8.83	± 1.3	9.81	± 0.4	8.84	± 0.9	
		Intensity	24-28 wk	10.50	± 1.1	9.17	± 0.4	8.63	± 1.1
			34-38 wk	8.35	± 1.2	8.90	± 0.5	8.49	± 1.1
			6-10 wk pp	8.30	± 1.1	9.56	± 0.5	8.96	± 1.1
	Sweetness	16-20 wk	6.30	± 1.4	6.90	± 0.5	7.54	± 1.0	
		Liking	24-28 wk	7.29	± 1.1	7.00	± 0.5	7.92	± 1.1
			34-38 wk	10.04	± 1.3	7.44	± 0.5	7.91	± 1.2
			6-10 wk pp	6.96	± 1.2	8.12	± 0.5	8.04	± 1.2
	Creaminess	16-20 wk	6.88	± 1.2	6.92	± 0.4	6.31	± 0.9	
		Intensity	24-28 wk	7.80	± 1.0	6.82	± 0.4	6.53	± 1.0
			34-38 wk	7.49	± 1.2	6.80	± 0.5	7.43	± 1.1
			6-10 wk pp	7.28	± 1.1	7.24	± 0.5	6.96	± 1.1
	Creaminess	16-20 wk	7.85	± 1.3	6.40	± 0.4	7.52	± 0.9	
		Liking	24-28 wk	8.39	± 1.1	7.16	± 0.4	6.00	± 1.1
			34-38 wk	9.19	± 1.2	6.87	± 0.5	7.87	± 1.1
			6-10 wk pp	8.54	± 1.1	7.58	± 0.5	8.18	± 1.1

5% Fat 10% Sucrose (cont.)	Flavor Intensity	16-20 wk	8.35	± 1.2	8.50	± 0.4	7.77	± 0.8
		24-28 wk	9.18	± 1.0	8.40	± 0.4	8.34	± 1.0
		34-38 wk	8.81	± 1.1	8.44	± 0.4	8.92	± 1.1
		6-10 wk pp	9.13	± 1.1	8.79	± 0.5	8.45	± 1.1
	Liking	16-20 wk	7.09	± 1.4	6.45	± 0.5	7.83	± 1.0
		24-28 wk	6.80	± 1.2	7.02	± 0.5	8.91	± 1.2
		34-38 wk	10.06	± 1.3	7.29	± 0.5	9.28	± 1.2
		6-10 wk pp	8.10	± 1.2	8.24	± 0.5	8.48	± 1.2
5% Fat 20% Sucrose	Sweetness Intensity	16-20 wk	12.30	^{a,b} ± 1.1	12.32	^a ± 0.4	10.20	^b ± 0.8
		24-28 wk	11.23	± 0.9	11.51	± 0.4	11.11	± 0.9
		34-38 wk	11.70	± 1.1	11.69	± 0.4	12.64	± 1.0
		6-10 wk pp	11.02	± 1.0	11.72	± 0.4	12.16	± 1.0
	Sweetness Liking	16-20 wk	4.36	± 1.6	6.41	± 0.5	7.48	± 1.1
		24-28 wk	6.70	± 1.3	6.83	± 0.5	7.98	± 1.3
		34-38 wk	6.55	± 1.5	7.53	± 0.6	8.77	± 1.4
		6-10 wk pp	6.45	± 1.4	7.10	± 0.6	8.18	± 1.4
	Creaminess Intensity	16-20 wk	9.39	± 1.3	8.05	± 0.4	6.51	± 0.9
		24-28 wk	8.22	± 1.1	7.85	± 0.4	7.60	± 1.1
		34-38 wk	9.15	± 1.2	8.24	± 0.5	9.25	± 1.1
		6-10 wk pp	8.05	± 1.1	8.17	± 0.5	8.07	± 1.1
	Creaminess Liking	16-20 wk	6.71	± 1.4	7.26	± 0.5	6.44	± 0.9
		24-28 wk	8.18	± 1.1	6.71	± 0.5	8.06	± 1.1
		34-38 wk	7.08	± 1.3	7.63	± 0.5	9.10	± 1.2
		6-10 wk pp	8.44	± 1.2	7.32	± 0.5	8.19	± 1.2
	Flavor Intensity	16-20 wk	9.84	± 1.2	10.31	± 0.4	9.49	± 0.9
		24-28 wk	9.01	± 1.0	10.05	± 0.4	10.38	± 1.0
		34-38 wk	10.94	± 1.2	10.77	± 0.5	11.94	± 1.1
		6-10 wk pp	10.96	± 1.1	10.85	± 0.5	10.68	± 1.1
	Flavor Liking	16-20 wk	5.75	± 1.5	6.86	± 0.5	7.54	± 1.0
		24-28 wk	6.63	± 1.2	6.85	± 0.5	8.95	± 1.2
		34-38 wk	7.22	± 1.4	7.56	± 0.6	9.68	± 1.3
		6-10 wk pp	8.55	± 1.3	7.77	± 0.6	9.19	± 1.3
10% Fat 0% Sucrose	Sweetness Intensity	16-20 wk	2.73	^a ± 0.7	0.86	^b ± 0.2	1.25	^{a,b} ± 0.4
		24-28 wk	0.66	± 0.5	1.34	± 0.2	1.50	± 0.5
		34-38 wk	1.07	± 0.6	1.29	± 0.2	1.64	± 0.6
		6-10 wk pp	1.37	± 0.6	1.35	± 0.2	1.37	± 0.6
	Sweetness Liking	16-20 wk	3.36	± 1.2	2.21	± 0.4	2.04	± 0.8
		24-28 wk	2.60	± 0.9	2.18	± 0.4	2.30	± 0.9
		34-38 wk	3.04	± 1.1	3.52	± 0.4	1.76	± 1.0
		6-10 wk pp	3.19	± 1.0	2.48	± 0.4	1.75	± 1.0
	Creaminess Intensity	16-20 wk	4.37	± 1.3	3.82	± 0.4	2.84	± 0.8
		24-28 wk	2.24	± 1.0	3.86	± 0.4	2.68	± 1.0
		34-38 wk	3.38	± 1.1	3.21	± 0.4	3.24	± 1.0
		6-10 wk pp	3.06	± 1.0	4.14	± 0.5	2.90	± 1.0
	Creaminess Liking	16-20 wk	4.89	± 1.3	3.49	± 0.4	2.52	± 0.8
		24-28 wk	3.57	± 1.0	3.12	± 0.4	2.77	± 1.0
		34-38 wk	4.70	± 1.2	4.13	± 0.5	2.85	± 1.1
		6-10 wk pp	4.40	± 1.1	3.68	± 0.5	3.08	± 1.1
	Flavor Intensity	16-20 wk	4.11	± 1.2	2.71	± 0.4	1.98	± 0.8
		24-28 wk	2.25	± 1.0	3.13	± 0.4	2.36	± 1.0
		34-38 wk	2.76	± 1.1	3.37	± 0.4	2.84	± 1.0
		6-10 wk pp	4.81	^a ± 1.0	3.02	^{a,b} ± 0.5	2.01	^b ± 1.0
	Flavor Liking	16-20 wk	4.36	± 1.3	2.62	± 0.4	1.88	± 0.8
		24-28 wk	3.07	± 1.0	2.72	± 0.4	2.23	± 1.0
		34-38 wk	4.13	± 1.1	3.20	± 0.4	2.05	± 1.0
		6-10 wk pp	4.11	± 1.1	2.81	± 0.5	2.05	± 1.0

10% Fat 5% Sucrose	Sweetness	16-20 wk	6.48	± 1.2	6.37	± 0.4	4.67	± 0.8
	Intensity	24-28 wk	5.42	± 1.0	5.94	± 0.4	5.16	± 1.0
		34-38 wk	5.83	± 1.2	6.34	± 0.4	6.12	± 1.1
		6-10 wk pp	4.49	± 1.1	5.23	± 0.5	4.58	± 1.1
		Sweetness	16-20 wk	6.55	± 1.4	6.57	± 0.5	6.46
	Liking	24-28 wk	5.92	± 1.2	5.66	± 0.5	5.77	± 1.2
		34-38 wk	7.96	± 1.4	6.16	± 0.5	6.69	± 1.2
		6-10 wk pp	5.99	± 1.2	7.15	± 0.6	5.38	± 1.2
		Creaminess	16-20 wk	7.50	± 1.2	5.78	± 0.4	4.71
	Intensity	24-28 wk	6.20	± 1.0	5.77	± 0.4	4.35	± 1.0
		34-38 wk	5.13	± 1.2	6.14	± 0.4	7.63	± 1.1
		6-10 wk pp	5.40	± 1.1	6.21	± 0.5	5.33	± 1.1
		Creaminess	16-20 wk	7.64	± 1.3	6.11	± 0.4	5.68
	Liking	24-28 wk	7.19	^a ± 1.1	5.54	^{a,b} ± 0.4	3.43	^b ± 1.1
		34-38 wk	7.93	± 1.3	6.10	± 0.5	7.34	± 1.2
		6-10 wk pp	7.18	± 1.2	6.73	± 0.5	5.41	± 1.2
		Flavor	16-20 wk	6.74	± 1.2	6.92	± 0.4	5.19
	Intensity	24-28 wk	5.39	± 1.0	5.18	± 0.4	5.02	± 1.0
		34-38 wk	5.18	± 1.1	6.18	± 0.4	6.56	± 1.0
		6-10 wk pp	6.16	± 1.0	5.65	± 0.5	4.87	± 1.0
Flavor		16-20 wk	5.76	± 1.4	6.66	± 0.5	7.24	± 1.0
Liking	24-28 wk	6.03	± 1.2	5.30	± 0.5	5.45	± 1.2	
	34-38 wk	8.21	± 1.3	6.25	± 0.5	6.04	± 1.2	
	6-10 wk pp	7.46	± 1.2	6.76	± 0.6	5.01	± 1.2	
	10% Fat 10% Sucrose	Sweetness	16-20 wk	9.41	± 1.4	10.13	± 0.4	9.22
Intensity	24-28 wk	9.37	± 1.1	9.80	± 0.4	10.00	± 1.1	
	34-38 wk	7.90	± 1.2	9.62	± 0.5	8.95	± 1.1	
	6-10 wk pp	8.69	± 1.1	9.29	± 0.5	8.80	± 1.1	
	Sweetness	16-20 wk	4.87	± 1.5	7.22	± 0.5	6.31	± 1.0
Liking	24-28 wk	7.53	± 1.2	7.13	± 0.5	8.43	± 1.2	
	34-38 wk	9.82	± 1.4	7.37	± 0.5	8.59	± 1.2	
	6-10 wk pp	7.23	± 1.2	7.92	± 0.6	9.61	± 1.2	
	Creaminess	16-20 wk	8.00	± 1.4	8.19	± 0.4	6.41	± 0.9
Intensity	24-28 wk	8.28	± 1.1	7.59	± 0.4	7.52	± 1.1	
	34-38 wk	6.32	± 1.2	7.59	± 0.5	7.37	± 1.1	
	6-10 wk pp	8.53	± 1.1	8.53	± 0.5	8.12	± 1.1	
	Creaminess	16-20 wk	6.35	± 1.4	6.71	± 0.5	6.28	± 0.9
Liking	24-28 wk	7.65	± 1.1	6.51	± 0.5	7.82	± 1.1	
	34-38 wk	9.99	± 1.3	7.32	± 0.5	7.65	± 1.2	
	6-10 wk pp	9.52	± 1.2	7.71	± 0.5	8.66	± 1.2	
	Flavor	16-20 wk	8.53	± 1.3	9.27	± 0.4	9.24	± 0.9
Intensity	24-28 wk	9.40	± 1.0	8.46	± 0.4	9.14	± 1.0	
	34-38 wk	8.53	± 1.2	8.64	± 0.5	8.51	± 1.1	
	6-10 wk pp	8.04	± 1.1	8.71	± 0.5	8.71	± 1.1	
	Flavor	16-20 wk	6.57	± 1.5	7.18	± 0.5	6.46	± 1.0
Liking	24-28 wk	8.60	± 1.2	7.36	± 0.5	8.13	± 1.2	
	34-38 wk	10.38	± 1.3	7.85	± 0.5	8.55	± 1.2	
	6-10 wk pp	10.05	± 1.2	8.12	± 0.5	9.04	± 1.2	

10% Fat 20% Sucrose	Sweetness	16-20 wk	9.94	± 1.1	12.39	± 0.4	10.80	± 0.8
	Intensity	24-28 wk	11.97	± 0.9	11.49	± 0.4	12.54	± 0.9
		34-38 wk	11.92	± 1.0	11.12	± 0.4	12.98	± 1.0
		6-10 wk pp	10.89	± 1.0	11.93	± 0.4	12.40	± 1.0
		Sweetness	16-20 wk	6.03	± 1.6	6.21	± 0.5	7.78
	Liking	24-28 wk	7.47	± 1.3	7.38	± 0.5	7.84	± 1.3
		34-38 wk	8.11	± 1.5	6.73	± 0.6	9.80	± 1.4
		6-10 wk pp	7.49	± 1.4	7.15	± 0.6	8.88	± 1.4
		Creaminess	16-20 wk	7.26	± 1.3	8.86	± 0.4	8.21
	Intensity	24-28 wk	9.03	± 1.1	7.84	± 0.4	8.34	± 1.1
		34-38 wk	9.92	± 1.2	8.83	± 0.5	10.44	± 1.1
		6-10 wk pp	9.60	± 1.1	9.66	± 0.5	9.77	± 1.1
		Creaminess	16-20 wk	6.93	± 1.4	7.22	± 0.5	7.19
	Liking	24-28 wk	7.64	± 1.1	7.16	± 0.5	8.09	± 1.1
		34-38 wk	8.22	± 1.3	7.19	± 0.5	9.88	± 1.2
		6-10 wk pp	8.39	± 1.2	7.29	± 0.5	10.23	± 1.2
		Flavor	16-20 wk	9.57	± 1.2	10.90	± 0.4	10.76
	Intensity	24-28 wk	9.81	± 1.0	10.24	± 0.4	10.74	± 1.0
		34-38 wk	11.56	± 1.1	10.17	± 0.4	12.45	± 1.0
		6-10 wk pp	11.27	± 1.0	10.75	± 0.5	11.22	± 1.0
Flavor		16-20 wk	5.58	± 1.5	6.57	± 0.5	7.60	± 1.0
Liking	24-28 wk	6.95	± 1.3	7.34	± 0.5	7.80	± 1.3	
	34-38 wk	8.18	± 1.4	6.91	± 0.6	10.10	± 1.3	
	6-10 wk pp	9.10	± 1.3	7.68	± 0.6	10.11	± 1.3	

TABLE 5.6 – FASTING COMMERCIAL FOOD RATINGS

Commercial Food	Taste Quality			GDM		NGT		Control	
Canned Fruit	Sweetness Intensity	16-20 wk	3.85	± 1.2	4.47	± 0.4	5.11	± 0.9	
		24-28 wk	5.66	± 1.1	4.12	± 0.4	5.51	± 1.0	
		34-38 wk	4.49	± 1.2	4.49	± 0.5	6.13	± 1.1	
		6-10 wk pp	5.21	± 1.1	4.97	± 0.5	6.64	± 1.1	
	Sweetness	16-20 wk	8.70	± 1.4	7.60	± 0.5	5.28	± 1.1	
		24-28 wk	7.86	± 1.3	6.54	± 0.5	7.35	± 1.2	
	Liking	34-38 wk	6.98	± 1.4	7.15	± 0.6	7.04	± 1.3	
		6-10 wk pp	8.67	± 1.3	7.97	± 0.6	7.16	± 1.3	
	Flavor Intensity	16-20 wk	7.27	± 1.3	7.30	± 0.5	6.87	± 1.0	
		24-28 wk	7.00	± 1.2	7.23	± 0.5	7.24	± 1.1	
		34-38 wk	8.24	± 1.3	8.26	± 0.5	8.48	± 1.2	
		6-10 wk pp	7.90	± 1.2	7.92	± 0.5	9.76	± 1.2	
	Flavor Liking	16-20 wk	8.11	± 1.5	8.35	± 0.5	6.38	± 1.1	
		24-28 wk	9.71	± 1.3	7.85	± 0.5	7.18	± 1.3	
		34-38 wk	9.18	± 1.5	8.12	± 0.6	8.05	± 1.3	
		6-10 wk pp	10.96	± 1.4	8.91	± 0.6	8.13	± 1.3	
	Salted Peanuts	Saltiness Intensity	16-20 wk	5.87	± 1.5	8.33	± 0.4	6.77	± 0.9
			24-28 wk	8.01	± 1.1	8.69	± 0.4	8.13	± 1.1
			34-38 wk	7.95	± 1.3	8.60	± 0.5	9.50	± 1.1
			6-10 wk pp	7.18	± 1.1	9.21	± 0.5	10.30	± 1.1
Saltiness Liking		16-20 wk	10.37	± 1.4	10.40	± 0.4	9.86	± 0.9	
		24-28 wk	11.20	± 1.1	10.44	± 0.4	10.20	± 1.0	
		34-38 wk	10.22	± 1.3	10.27	± 0.5	11.10	± 1.0	
		6-10 wk pp	11.01	± 1.1	10.04	± 0.5	11.90	± 1.1	
Crunchiness Intensity		16-20 wk	9.54	± 1.5	9.48	± 0.4	8.82	± 0.9	
		24-28 wk	9.69	± 1.2	9.98	± 0.4	10.07	± 1.1	
		34-38 wk	9.03	± 1.3	10.20	± 0.5	10.92	± 1.1	
		6-10 wk pp	11.16	± 1.2	10.53	± 0.5	10.51	± 1.2	
Crunchiness Liking		16-20 wk	11.78	± 1.3	11.38	± 0.4	10.50	± 0.8	
		24-28 wk	10.96	± 1.0	11.15	± 0.4	11.16	± 0.9	
		34-38 wk	10.58	± 1.1	11.14	± 0.4	13.10	± 1.0	
		6-10 wk pp	11.87	± 1.0	10.66	± 0.4	11.83	± 1.0	
Oiliness Intensity		16-20 wk	4.03	± 1.5	5.95	± 0.4	5.62	± 0.9	
		24-28 wk	5.95	± 1.1	7.38	± 0.4	6.27	± 1.1	
		34-38 wk	6.39	± 1.3	7.98	± 0.5	7.54	± 1.1	
		6-10 wk pp	6.46	± 1.1	8.20	± 0.5	8.41	± 1.1	
Oiliness Liking	16-20 wk	6.16	± 1.6	8.20	± 0.5	7.09	± 1.0		
	24-28 wk	8.17	± 1.2	7.50	± 0.5	8.18	± 1.2		
	34-38 wk	8.03	± 1.4	7.82	± 0.5	9.25	± 1.2		
	6-10 wk pp	8.81	± 1.2	7.56	± 0.5	7.89	± 1.2		
Flavor Intensity	16-20 wk	6.56	± 1.4	8.74	± 0.4	8.21	± 0.9		
	24-28 wk	7.64	± 1.1	8.90	± 0.4	10.75	± 1.1		
	34-38 wk	8.71	± 1.3	9.47	± 0.5	8.83	± 1.1		
	6-10 wk pp	8.66	± 1.1	10.19	± 0.5	11.18	± 1.1		
Flavor Liking	16-20 wk	9.77	± 1.3	11.40	± 0.4	10.74	± 0.8		
	24-28 wk	11.65	± 1.0	10.87	± 0.4	11.45	± 1.0		
	34-38 wk	10.79	± 1.2	10.92	± 0.4	11.77	± 1.0		
	6-10 wk pp	10.95	± 1.0	10.99	± 0.5	12.24	± 1.0		

Vanilla Pudding	Sweetness Intensity	16-20 wk	9.70	± 1.2	9.71	± 0.4	9.34	± 0.9
		24-28 wk	9.38	± 1.1	9.23	± 0.4	9.50	± 1.0
		34-38 wk	7.69	± 1.2	9.37	± 0.5	10.57	± 1.1
		6-10 wk pp	8.49	± 1.1	9.91	± 0.5	11.18	± 1.1
	Sweetness Liking	16-20 wk	9.21	± 1.4	8.88	± 0.5	7.95	± 1.0
		24-28 wk	8.96	± 1.3	9.51	± 0.5	9.73	± 1.2
		34-38 wk	8.80	± 1.4	9.60	± 0.5	10.13	± 1.3
		6-10 wk pp	9.76	± 1.3	9.73	± 0.6	9.80	± 1.3
	Creaminess Intensity	16-20 wk	11.35	± 1.1	12.00	± 0.4	11.45	± 0.8
		24-28 wk	11.26	± 1.0	11.92	± 0.4	13.25	± 0.9
		34-38 wk	10.59	± 1.1	11.84	± 0.4	12.96	± 1.0
		6-10 wk pp	11.82	± 1.0	11.80	± 0.4	12.38	± 1.0
	Creaminess Liking	16-20 wk	9.87	± 1.4	10.08	± 0.5	8.65	± 1.0
		24-28 wk	10.49	± 1.2	10.50	± 0.5	11.27	± 1.2
		34-38 wk	10.46	± 1.4	10.18	± 0.5	9.97	± 1.2
		6-10 wk pp	10.78	± 1.3	10.32	± 0.6	9.82	± 1.2
	Flavor Intensity	16-20 wk	8.34	± 1.2	10.30	± 0.4	8.68	± 0.9
		24-28 wk	9.54	± 1.1	9.40	± 0.4	10.54	± 1.0
		34-38 wk	8.04	± 1.2	10.03	± 0.5	10.74	± 1.1
		6-10 wk pp	9.10	± 1.1	10.40	± 0.5	10.01	± 1.1
	Flavor Liking	16-20 wk	8.18	± 1.4	9.56	± 0.5	8.80	± 1.0
		24-28 wk	10.56	± 1.3	10.10	± 0.5	9.06	± 1.2
		34-38 wk	10.34	± 1.4	9.94	± 0.5	10.06	± 1.3
		6-10 wk pp	10.82	± 1.3	10.13	± 0.6	9.58	± 1.3
Snack Chips	Saltiness Intensity	16-20 wk	8.19	± 1.3	9.28	± 0.5	8.83	± 0.9
		24-28 wk	7.70	± 1.1	9.75	± 0.5	10.10	± 1.1
		34-38 wk	9.63	± 1.3	9.74	± 0.5	10.85	± 1.1
		6-10 wk pp	9.11	± 1.2	10.06	± 0.5	11.83	± 1.2
	Saltiness Liking	16-20 wk	9.89	± 1.3	10.51	± 0.4	9.01	± 0.9
		24-28 wk	11.01	± 1.0	10.60	± 0.4	10.29	± 1.0
		34-38 wk	11.74	± 1.2	10.85	± 0.5	11.12	± 1.0
		6-10 wk pp	11.19	± 1.1	11.01	± 0.5	11.49	± 1.1
	Spiciness Intensity	16-20 wk	3.93	± 1.5	5.41	± 0.5	5.59	± 1.0
		24-28 wk	5.75	± 1.2	6.06	± 0.5	5.44	± 1.2
		34-38 wk	5.39	± 1.4	6.54	± 0.5	5.89	± 1.2
		6-10 wk pp	5.32	± 1.3	6.99	± 0.6	5.84	± 1.3
	Spiciness Liking	16-20 wk	10.25	± 1.5	8.51	± 0.5	7.10	± 1.0
		24-28 wk	9.76	± 1.2	9.15	± 0.5	8.91	± 1.2
		34-38 wk	10.33	± 1.4	8.91	± 0.5	9.89	± 1.2
		6-10 wk pp	9.90	± 1.3	8.76	± 0.6	9.74	± 1.3
	Crunchiness Intensity	16-20 wk	10.75	± 1.1	11.74	± 0.4	10.83	± 0.8
		24-28 wk	10.06	± 0.9	11.89	± 0.4	12.03	± 0.9
		34-38 wk	11.84	± 1.0	12.05	± 0.4	12.51	± 0.9
		6-10 wk pp	12.17	± 0.9	11.90	± 0.4	12.73	± 0.9
	Crunchiness Liking	16-20 wk	11.90	± 1.2	11.51	± 0.4	10.40	± 0.8
		24-28 wk	11.54	± 1.0	11.31	± 0.4	11.80	± 1.0
		34-38 wk	11.34	± 1.2	11.84	± 0.4	11.99	± 1.0
		6-10 wk pp	11.09	± 1.0	11.96	± 0.5	12.39	± 1.0
	Flavor Intensity	16-20 wk	7.55	± 1.3	9.85	± 0.4	9.36	± 0.9
		24-28 wk	8.39	± 1.1	9.79	± 0.4	10.13	± 1.2
		34-38 wk	8.22	± 1.3	9.90	± 0.5	10.13	± 1.1
		6-10 wk pp	8.41	± 1.2	10.32	± 0.5	10.38	± 1.1
	Flavor Liking	16-20 wk	11.15	± 1.2	11.20	± 0.4	10.26	± 0.8
		24-28 wk	11.56	± 1.0	11.31	± 0.4	11.74	± 1.1
		34-38 wk	11.03	± 1.2	11.25	± 0.5	12.88	± 1.0
		6-10 wk pp	10.90	± 1.1	11.29	± 0.5	12.15	± 1.0

Sharp Cheddar Cheese										
Saltiness Intensity	16-20 wk	6.70	± 1.6	6.73	± 0.5	5.83	± 1.0			
	24-28 wk	6.99	± 1.3	6.74	± 0.5	6.56	± 1.3			
	34-38 wk	8.71	± 1.4	7.21	± 0.5	7.17	± 1.2			
	6-10 wk pp	6.50	± 1.3	7.07	± 0.6	9.20	± 1.3			
Saltiness Liking	16-20 wk	6.49	± 1.7	7.47	± 0.5	6.26	± 1.1			
	24-28 wk	7.43	± 1.4	7.44	± 0.5	9.16	± 1.4			
	34-38 wk	10.05	± 1.5	7.12	± 0.6	8.33	± 1.3			
	6-10 wk pp	7.49	± 1.4	7.09	± 0.6	8.75	± 1.4			
Bitterness Intensity	16-20 wk	8.01	± 1.7	7.41	± 0.5	6.87	± 1.1			
	24-28 wk	8.43	± 1.4	7.68	± 0.5	7.72	± 1.4			
	34-38 wk	7.40	± 1.5	7.91	± 0.6	8.80	± 1.3			
	6-10 wk pp	8.38	± 1.4	8.81	± 0.6	9.76	± 1.4			
Bitterness Liking	16-20 wk	6.13	± 1.7	6.47	± 0.5	6.37	± 1.1			
	24-28 wk	8.17	± 1.4	6.43	± 0.5	8.98	± 1.4			
	34-38 wk	9.70	± 1.5	6.41	± 0.6	7.03	± 1.3			
	6-10 wk pp	6.84	± 1.4	7.14	± 0.6	8.30	± 1.4			
Flavor Intensity	16-20 wk	8.44	± 1.6	10.02	± 0.5	10.13	± 1.0			
	24-28 wk	9.17	± 1.3	9.61	± 0.5	9.53	± 1.3			
	34-38 wk	8.48	± 1.4	9.54	± 0.6	10.93	± 1.2			
	6-10 wk pp	9.08	± 1.3	9.83	± 0.6	10.84	± 1.3			
Flavor Liking	16-20 wk	6.15	± 1.9	6.71	± 0.6	6.72	± 1.2			
	24-28 wk	9.10	± 1.5	7.45	± 0.6	9.81	± 1.5			
	34-38 wk	10.02	± 1.7	6.80	± 0.7	7.90	± 1.4			
	6-10 wk pp	5.13	± 1.5	7.45	± 0.7	8.38	± 1.5			

TABLE 5.7 – POST-GLUCOLA GLUCOSE SOLUTION RATINGS

<u>Glucose Solution Concentration</u>	<u>Taste Quality</u>		GDM			NGT			Control		
0.01M	Sweetness	24-28 wk	0.78	±	0.4	1.04	±	0.2	0.93	±	0.4
		34-38 wk	0.88	±	0.5	1.00	±	0.2	1.00	±	0.4
		6-10 wk pp	0.85	±	0.4	0.95	±	0.2	1.24	±	0.4
	Liking	24-28 wk	5.27	±	1.3	4.76	±	0.5	4.02	±	1.3
		34-38 wk	4.93	±	1.4	5.30	±	0.6	3.27	±	1.3
		6-10 wk pp	3.91	±	1.3	4.81	±	0.6	1.95	±	1.3
0.02M	Sweetness	24-28 wk	0.79	±	0.4	1.12	±	0.2	0.93	±	0.4
		34-38 wk	1.03	±	0.5	1.05	±	0.2	0.84	±	0.5
		6-10 wk pp	1.36	±	0.5	0.92	±	0.2	1.00	±	0.5
	Liking	24-28 wk	5.55	±	1.2	5.11	±	0.5	3.79	±	1.2
		34-38 wk	5.31	±	1.4	5.06	±	0.6	3.21	±	1.3
		6-10 wk pp	6.00	±	1.3	4.27	±	0.6	2.57	±	1.3
0.04M	Sweetness	24-28 wk	0.74	±	0.4	1.22	±	0.2	0.59	±	0.4
		34-38 wk	1.22	±	0.5	1.17	±	0.2	0.86	±	0.4
		6-10 wk pp	1.12	±	0.4	0.96	±	0.2	1.30	±	0.4
	Liking	24-28 wk	4.08	±	1.2	5.02	±	0.5	3.53	±	1.2
		34-38 wk	5.68	±	1.4	4.57	±	0.5	3.47	±	1.3
		6-10 wk pp	4.26	±	1.3	4.70	±	0.6	3.12	±	1.3
0.08M	Sweetness	24-28 wk	2.47	±	0.7 ^{a,b}	2.78	±	0.3 ^a	0.77	±	0.7 ^b
		34-38 wk	1.45	±	0.8	1.78	±	0.3	1.27	±	0.8
		6-10 wk pp	1.47	±	0.8	1.74	±	0.3	1.92	±	0.8
	Liking	24-28 wk	3.62	±	1.2	4.72	±	0.5	3.89	±	1.2
		34-38 wk	5.60	±	1.3	4.81	±	0.5	3.01	±	1.2
		6-10 wk pp	5.01	±	1.2	4.71	±	0.5	2.93	±	1.2
0.16M	Sweetness	24-28 wk	3.41	±	0.9	4.33	±	0.4 ^a	2.17	±	0.9 ^b
		34-38 wk	2.31	±	1.0	4.33	±	0.4 ^a	2.19	±	0.9 ^b
		6-10 wk pp	3.23	±	0.9	3.71	±	0.4	3.46	±	0.9
	Liking	24-28 wk	3.89	±	1.1	4.17	±	0.4	3.70	±	1.1
		34-38 wk	6.03	±	1.2	4.39	±	0.5	3.09	±	1.1
		6-10 wk pp	4.62	±	1.1	4.45	±	0.5	2.74	±	1.1
AVERAGE	Sweetness	24-28 wk	1.64	±	0.5	2.10	±	0.2	1.08	±	0.5
		34-38 wk	1.38	±	0.5	1.86	±	0.2	1.23	±	0.5
		6-10 wk pp	1.61	±	0.5	1.66	±	0.2	1.78	±	0.5
	Liking	24-28 wk	4.48	±	1.1	4.76	±	0.4	3.78	±	1.1
		34-38 wk	5.51	±	1.2	4.83	±	0.5	3.21	±	1.1
		6-10 wk pp	4.76	±	1.1	4.59	±	0.5	2.66	±	1.1

TABLE 5.8 – POST-GLUCOLA STRAWBERRY MILK RATINGS

<u>Strawberry Milk Concentration</u>	<u>Taste Quality</u>		GDM			NGT			Control		
0% Fat 0% Sucrose	Sweetness	24-28 wk	1.35	±	0.6	1.16	±	0.2	1.25	±	0.6
	Intensity	34-38 wk	0.98	±	0.7	1.66	±	0.3	1.55	±	0.6
		6-10 wk pp	1.10	±	0.6	1.34	±	0.3	2.38	±	0.6
		Sweetness	24-28 wk	1.97	±	1.0	2.57	±	0.4	2.13	±
	Liking	34-38 wk	2.95	±	1.1	3.16	±	0.4	1.75	±	1.0
		6-10 wk pp	2.93	±	1.1	3.02	±	0.5	2.68	±	1.0
		Creaminess	24-28 wk	3.43	±	0.8	2.53	±	0.3	3.37	±
	Intensity	34-38 wk	2.71	±	1.0	2.65	±	0.4	3.25	±	0.8
		6-10 wk pp	3.68	±	0.9	2.76	±	0.4	3.07	±	0.9
		Creaminess	24-28 wk	4.11	±	0.9	3.07	±	0.4	2.97	±
	Liking	34-38 wk	4.25	±	1.1	3.84	±	0.4	2.38	±	0.9
		6-10 wk pp	2.75	±	1.0	3.18	±	0.4	3.21	±	1.0
Flavor		24-28 wk	3.95	±	1.0	2.97	±	0.4	2.51	±	1.0
Intensity	34-38 wk	2.62	±	1.2	3.32	±	0.5	2.59	±	1.0	
	6-10 wk pp	4.22	±	1.1	3.54	±	0.5	4.26	±	1.1	
	Flavor	24-28 wk	2.76	±	0.9	2.41	±	0.4	2.78	±	0.9
Liking	34-38 wk	4.48	±	1.1	3.40	±	0.4	2.01	±	0.9	
	6-10 wk pp	3.45	±	1.0	2.85	±	0.4	2.96	±	1.0	
	Sweetness	24-28 wk	6.18	±	1.0	4.53	±	0.4	6.77	±	1.0
Intensity		34-38 wk	4.94	±	1.1	4.97	±	0.5	4.51	±	1.0
		6-10 wk pp	4.34	±	1.0	4.85	±	0.5	6.26	±	1.0
	Sweetness	24-28 wk	5.51	±	1.2	5.09	±	0.5	6.01	±	1.2
Liking		34-38 wk	7.05	±	1.3	5.75	±	0.5	4.31	±	1.2
		6-10 wk pp	7.00	±	1.2	6.20	±	0.5	5.18	±	1.3
	Creaminess	24-28 wk	5.36	±	0.9	3.95	±	0.4	5.33	±	0.9
Intensity		34-38 wk	4.21	±	1.0	4.30	±	0.4	3.41	±	0.9
		6-10 wk pp	5.29	±	0.9	4.73	±	0.4	4.33	±	0.9
	Creaminess	24-28 wk	6.36	±	1.1	4.34	±	0.4	4.62	±	1.1
Liking		34-38 wk	8.04 ^a	±	1.2	5.47 ^{a,b}	±	0.5	3.82 ^b	±	1.1
		6-10 wk pp	7.02	±	1.1	6.00	±	0.5	5.19	±	1.1
	Flavor	24-28 wk	6.83	±	0.9	5.38	±	0.4	5.61	±	0.9
Intensity		34-38 wk	5.17	±	1.1	5.01	±	0.4	4.71	±	0.9
		6-10 wk pp	7.39	±	1.0	5.23	±	0.4	5.07	±	1.0
	Flavor	24-28 wk	6.32	±	1.2	5.09	±	0.5	4.98	±	1.2
Liking		34-38 wk	7.59	±	1.3	5.55	±	0.5	4.36	±	1.2
		6-10 wk pp	8.05 ^a	±	1.2	6.01 ^{a,b}	±	0.5	4.32 ^b	±	1.2

0% Fat 10% Sucrose	Sweetness	24-28 wk	8.40	± 1.1	9.06	± 0.5	9.58	± 1.1	
	Intensity	34-38 wk	7.47	± 1.3	8.30	± 0.5	7.71	± 1.1	
		6-10 wk pp	8.35	± 1.2	9.17	± 0.5	9.83	± 1.2	
		Sweetness	24-28 wk	9.26	± 1.2	6.89	± 0.5	8.37	± 1.2
	Liking	34-38 wk	9.40	± 1.3	7.36	± 0.5	8.31	± 1.2	
		6-10 wk pp	7.95	± 1.2	8.01	± 0.5	7.37	± 1.2	
		Creaminess	24-28 wk	7.64	± 1.0	6.12	± 0.4	6.82	± 1.0
	Intensity	34-38 wk	5.41	± 1.1	5.89	± 0.4	5.23	± 1.0	
		6-10 wk pp	6.02	± 1.0	6.45	± 0.5	6.54	± 1.0	
		Creaminess	24-28 wk	9.37	± 1.1	6.63	± 0.4	8.00	± 1.1
	Liking	34-38 wk	9.02 ^a	± 1.2	6.69 ^b	± 0.5	7.05 ^{a,b}	± 1.1	
		6-10 wk pp	7.93	± 1.1	7.63	± 0.5	6.52	± 1.1	
		Flavor	24-28 wk	8.67	± 1.1	8.26	± 0.4	9.70	± 1.1
	Intensity	34-38 wk	7.77	± 1.2	7.75	± 0.5	6.89	± 1.1	
		6-10 wk pp	8.43	± 1.1	8.72	± 0.5	8.57	± 1.1	
		Flavor	24-28 wk	9.24 ^a	± 1.1	6.77 ^b	± 0.5	8.40 ^{a,b}	± 1.1
	Liking	34-38 wk	9.54	± 1.3	7.24	± 0.5	7.76	± 1.1	
		6-10 wk pp	9.76	± 1.2	8.11	± 0.5	7.35	± 1.2	
Sweetness		24-28 wk	12.28	± 0.9	11.76	± 0.4	12.92	± 0.9	
	Intensity	34-38 wk	11.30	± 1.0	11.41	± 0.4	12.26	± 0.9	
		6-10 wk pp	11.09	± 0.9	12.27	± 0.4	12.02	± 0.9	
Sweetness		24-28 wk	8.42	± 1.3	6.77	± 0.5	7.55	± 1.3	
	Liking	34-38 wk	6.46	± 1.5	7.33	± 0.6	7.87	± 1.3	
		6-10 wk pp	8.31	± 1.4	7.93	± 0.6	8.33	± 1.4	
Creaminess		24-28 wk	9.22	± 1.1	7.00	± 0.5	6.59	± 1.1	
	Intensity	34-38 wk	8.89	± 1.3	7.64	± 0.5	8.37	± 1.1	
		6-10 wk pp	8.70	± 1.1	7.98	± 0.5	8.55	± 1.1	
Creaminess		24-28 wk	8.42	± 1.1	6.55	± 0.5	6.80	± 1.1	
	Liking	34-38 wk	6.72	± 1.3	7.95	± 0.5	9.08	± 1.1	
		6-10 wk pp	8.85	± 1.2	8.04	± 0.5	8.63	± 1.2	
Flavor		24-28 wk	11.68	± 1.0	9.87	± 0.4	10.81	± 1.0	
	Intensity	34-38 wk	10.29	± 1.2	10.26	± 0.5	10.20	± 1.0	
		6-10 wk pp	10.90	± 1.1	11.10	± 0.5	10.53	± 1.1	
Flavor		24-28 wk	9.28	± 1.2	6.33	± 0.5	8.26	± 1.2	
	Liking	34-38 wk	7.51	± 1.4	7.46	± 0.6	9.08	± 1.2	
		6-10 wk pp	8.93	± 1.3	8.14	± 0.6	7.95	± 1.3	
5% Fat 0% Sucrose		Sweetness	24-28 wk	1.92	± 0.6	1.22	± 0.2	2.39	± 0.6
	Intensity		34-38 wk	0.86	± 0.7	1.32	± 0.3	1.62	± 0.6
			6-10 wk pp	1.65	± 0.6	1.25	± 0.3	1.16	± 0.6
		Sweetness	24-28 wk	2.48	± 1.0	2.33	± 0.4	3.02	± 1.0
	Liking		34-38 wk	2.83	± 1.1	3.06	± 0.5	1.63	± 1.0
			6-10 wk pp	3.45	± 1.0	3.16	± 0.5	1.65	± 1.0
		Creaminess	24-28 wk	2.67	± 0.9	3.11	± 0.4	4.93	± 0.9
	Intensity		34-38 wk	2.76	± 1.0	3.22	± 0.4	2.94	± 0.9
			6-10 wk pp	2.86	± 1.0	3.38	± 0.4	4.71	± 1.0
		Creaminess	24-28 wk	3.26	± 1.0	3.51	± 0.4	3.23	± 1.0
	Liking		34-38 wk	5.58	± 1.2	3.80	± 0.5	2.73	± 1.0
			6-10 wk pp	4.45	± 1.1	3.75	± 0.5	3.46	± 1.1
		Flavor	24-28 wk	3.20	± 1.0	2.98	± 0.4	3.34	± 1.0
	Intensity		34-38 wk	2.55	± 1.1	3.13	± 0.4	2.04	± 1.0
			6-10 wk pp	3.76	± 1.0	2.95	± 0.5	3.28	± 1.0
		Flavor	24-28 wk	2.96	± 1.0	2.31	± 0.4	3.45	± 1.0
	Liking		34-38 wk	5.32	± 1.1	2.94	± 0.4	1.81	± 1.0
			6-10 wk pp	4.72	± 1.0	3.40	± 0.5	2.15	± 1.0

5% Fat 5% Sucrose	Sweetness	24-28 wk	4.62	± 1.0	5.93	± 0.4	6.18	± 1.0	
	Intensity	34-38 wk	7.60	^a ± 1.2	5.43	^{a,b} ± 0.5	4.34	^b ± 1.0	
		6-10 wk pp	4.50	± 1.1	5.00	± 0.5	5.00	± 1.1	
		Sweetness	24-28 wk	6.75	± 1.2	5.06	± 0.5	5.25	± 1.2
	Liking	34-38 wk	7.33	± 1.3	5.78	± 0.5	5.13	± 1.2	
		6-10 wk pp	5.66	± 1.2	6.46	± 0.5	4.72	± 1.2	
		Sweetness	24-28 wk	4.50	± 1.0	5.76	± 0.4	5.66	± 1.0
	Intensity	34-38 wk	8.33	^a ± 1.1	5.36	^b ± 0.4	5.00	^b ± 1.0	
		6-10 wk pp	5.08	± 1.0	5.63	± 0.5	5.76	± 1.0	
		Sweetness	24-28 wk	6.40	± 1.2	5.48	± 0.5	4.99	± 1.2
	Liking	34-38 wk	9.16	± 1.3	6.07	± 0.5	5.39	± 1.2	
		6-10 wk pp	6.65	± 1.2	6.42	± 0.6	4.98	± 1.2	
		Flavor	24-28 wk	5.49	± 1.0	5.49	± 0.4	5.77	± 1.0
	Intensity	34-38 wk	7.06	± 1.1	5.90	± 0.5	5.23	± 1.0	
		6-10 wk pp	6.53	± 1.0	5.77	± 0.5	4.45	± 1.0	
		Flavor	24-28 wk	6.97	± 1.2	5.47	± 0.5	6.02	± 1.2
	Liking	34-38 wk	8.20	± 1.4	5.69	± 0.5	5.54	± 1.2	
		6-10 wk pp	7.63	± 1.2	6.08	± 0.6	5.50	± 1.2	
Sweetness		24-28 wk	9.32	± 1.1	9.28	± 0.4	10.77	± 1.1	
	Intensity	34-38 wk	8.89	± 1.2	8.95	± 0.5	8.28	± 1.1	
		6-10 wk pp	7.63	± 1.1	9.28	± 0.5	9.21	± 1.1	
Sweetness		24-28 wk	7.88	± 1.2	7.20	± 0.5	9.39	± 1.2	
	Liking	34-38 wk	8.27	± 1.3	7.74	± 0.5	8.32	± 1.2	
		6-10 wk pp	6.41	± 1.2	7.93	± 0.5	7.44	± 1.2	
Creaminess		24-28 wk	6.71	± 1.1	7.10	± 0.4	7.33	± 1.1	
	Intensity	34-38 wk	7.49	± 1.2	7.78	± 0.5	7.58	± 1.1	
		6-10 wk pp	5.80	± 1.1	7.91	± 0.5	7.83	± 1.1	
Creaminess		24-28 wk	6.35	± 1.1	6.78	± 0.5	7.58	± 1.1	
	Liking	34-38 wk	9.62	± 1.3	7.59	± 0.5	7.75	± 1.1	
		6-10 wk pp	6.25	± 1.2	7.67	± 0.5	8.68	± 1.1	
Flavor		24-28 wk	8.97	± 1.1	8.50	± 0.4	9.42	± 1.1	
	Intensity	34-38 wk	7.93	± 1.2	8.46	± 0.5	7.82	± 1.1	
		6-10 wk pp	7.40	± 1.1	9.07	± 0.5	8.25	± 1.1	
Flavor		24-28 wk	7.53	± 1.2	6.84	± 0.5	8.56	± 1.2	
	Liking	34-38 wk	9.29	± 1.3	7.76	± 0.5	8.18	± 1.2	
		6-10 wk pp	6.42	± 1.2	8.45	± 0.5	7.47	± 1.2	
5% Fat 20% Sucrose		Sweetness	24-28 wk	11.79	± 0.9	12.18	± 0.4	13.12	± 0.9
	Intensity		34-38 wk	11.39	± 1.0	11.64	± 0.4	12.33	± 0.9
			6-10 wk pp	9.79	^a ± 0.9	12.41	^b ± 0.4	12.29	^{a,b} ± 0.9
		Sweetness	24-28 wk	7.03	± 1.3	6.86	± 0.5	7.96	± 1.3
	Liking		34-38 wk	7.46	± 1.5	6.95	± 0.6	8.11	± 1.3
			6-10 wk pp	6.58	± 1.4	7.90	± 0.6	8.34	± 1.4
		Creaminess	24-28 wk	8.54	± 1.1	8.21	± 0.5	8.35	± 1.1
	Intensity		34-38 wk	7.99	± 1.3	8.30	± 0.5	7.33	± 1.1
			6-10 wk pp	8.06	± 1.1	9.47	± 0.5	8.98	± 1.1
		Creaminess	24-28 wk	7.77	± 1.2	7.04	± 0.5	7.77	± 1.2
	Liking		34-38 wk	8.23	± 1.4	7.58	± 0.5	7.91	± 1.2
			6-10 wk pp	7.33	± 1.2	8.10	± 0.6	10.37	± 1.2
		Flavor	24-28 wk	10.78	± 1.0	10.17	± 0.4	10.84	± 1.0
	Intensity		34-38 wk	11.06	± 1.2	10.74	± 0.5	10.46	± 1.0
			6-10 wk pp	9.50	± 1.1	11.27	± 0.5	11.71	± 1.1
		Flavor	24-28 wk	7.35	± 1.3	6.99	± 0.5	7.90	± 1.3
	Liking		34-38 wk	9.21	± 1.5	7.55	± 0.6	8.72	± 1.3
			6-10 wk pp	8.42	± 1.3	7.95	± 0.6	10.14	± 1.3

10% Fat 0% Sucrose	Sweetness	24-28 wk	2.10	± 0.6	1.15	± 0.2	1.62	± 0.6	
	Intensity	34-38 wk	1.17	± 0.7	1.35	± 0.3	1.36	± 0.6	
		6-10 wk pp	1.47	± 0.6	1.52	± 0.3	2.17	± 0.6	
	Sweetness	24-28 wk	3.40	± 1.1	2.30	± 0.4	2.80	± 1.1	
	Liking	34-38 wk	4.35	± 1.2	3.82	± 0.5	1.63	± 1.1	
		6-10 wk pp	4.31	± 1.1	3.06	± 0.5	2.24	± 1.1	
	Creaminess	24-28 wk	2.56	± 1.0	3.35	± 0.4	3.82	± 1.0	
	Intensity	34-38 wk	5.60	± 1.2	3.92	± 0.5	3.92	± 1.0	
		6-10 wk pp	5.62	± 1.1	3.79	± 0.5	4.37	± 1.1	
	Creaminess	24-28 wk	3.95	± 1.1	3.17	± 0.5	4.09	± 1.1	
	Liking	34-38 wk	6.71	± 1.3	4.58	± 0.5	3.78	± 1.1	
		6-10 wk pp	5.02	± 1.2	4.04	± 0.5	3.62	± 1.2	
	Flavor	24-28 wk	2.95	± 1.0	3.05	± 0.4	2.45	± 1.0	
	Intensity	34-38 wk	3.07	± 1.2	3.76	± 0.5	2.30	± 1.0	
		6-10 wk pp	4.79	± 1.1	3.55	± 0.5	3.38	± 1.1	
	Flavor	24-28 wk	4.28	± 1.1	2.40	± 0.4	2.64	± 1.1	
	Liking	34-38 wk	4.89	± 1.2	3.92	± 0.5	2.01	± 1.1	
		6-10 wk pp	5.30	± 1.1	3.34	± 0.5	2.40	± 1.1	
	10% Fat 5% Sucrose	Sweetness	24-28 wk	6.37	± 1.0	5.62	± 0.4	5.79	± 1.0
		Intensity	34-38 wk	5.02	± 1.2	5.31	± 0.5	5.68	± 1.0
			6-10 wk pp	5.02	± 1.1	5.65	± 0.5	5.84	± 1.1
		Sweetness	24-28 wk	5.95	± 1.2	5.67	± 0.5	5.86	± 1.2
		Liking	34-38 wk	7.04	± 1.3	6.30	± 0.5	4.68	± 1.2
			6-10 wk pp	8.19	± 1.2	6.43	± 0.5	5.29	± 1.2
Creaminess		24-28 wk	5.78	± 1.0	6.56	± 0.4	6.33	± 1.0	
Intensity		34-38 wk	7.28	± 1.2	6.16	± 0.5	5.62	± 1.0	
		6-10 wk pp	5.09	± 1.1	6.10	± 0.5	7.61	± 1.1	
Creaminess		24-28 wk	6.25	± 1.1	5.55	± 0.4	5.09	± 1.1	
Liking		34-38 wk	9.65 ^a	± 1.3	6.84 ^{a,b}	± 0.5	5.52 ^b	± 1.1	
		6-10 wk pp	7.91	± 1.1	6.07	± 0.5	6.86	± 1.1	
Flavor		24-28 wk	6.85	± 1.0	5.86	± 0.4	5.83	± 1.0	
Intensity		34-38 wk	7.27	± 1.1	5.61	± 0.5	6.63	± 1.0	
		6-10 wk pp	6.29	± 1.0	6.32	± 0.5	6.02	± 1.0	
Flavor		24-28 wk	6.44	± 1.2	5.75	± 0.5	5.68	± 1.2	
Liking		34-38 wk	8.73 ^a	± 1.3	6.12 ^{a,b}	± 0.5	4.78 ^b	± 1.2	
		6-10 wk pp	8.47	± 1.2	5.91	± 0.5	5.33	± 1.2	
10% Fat 10% Sucrose		Sweetness	24-28 wk	10.62	± 1.1	9.70	± 0.4	10.30	± 1.1
		Intensity	34-38 wk	9.88	± 1.2	9.25	± 0.5	9.01	± 1.1
			6-10 wk pp	9.41	± 1.1	9.94	± 0.5	11.05	± 1.1
		Sweetness	24-28 wk	6.89	± 1.2	7.83	± 0.5	6.69	± 1.2
		Liking	34-38 wk	9.67	± 1.4	7.74	± 0.5	6.43	± 1.2
			6-10 wk pp	7.48	± 1.2	8.38	± 0.6	9.03	± 1.2
	Creaminess	24-28 wk	7.65	± 1.1	7.31	± 0.5	7.72	± 1.1	
	Intensity	34-38 wk	7.37	± 1.3	7.90	± 0.5	7.40	± 1.1	
		6-10 wk pp	6.32	± 1.2	8.06	± 0.5	8.43	± 1.2	
	Creaminess	24-28 wk	8.38	± 1.1	7.22	± 0.5	6.55	± 1.1	
	Liking	34-38 wk	9.74	± 1.3	7.28	± 0.5	7.28	± 1.1	
		6-10 wk pp	8.27	± 1.2	7.89	± 0.5	8.21	± 1.2	
	Flavor	24-28 wk	9.97	± 1.1	8.54	± 0.4	9.76	± 1.1	
	Intensity	34-38 wk	9.56	± 1.2	8.64	± 0.5	7.92	± 1.1	
		6-10 wk pp	8.27	± 1.1	8.91	± 0.5	9.92	± 1.1	
	Flavor	24-28 wk	9.43	± 1.2	7.71	± 0.5	7.32	± 1.2	
	Liking	34-38 wk	10.36	± 1.4	7.68	± 0.5	7.06	± 1.2	
		6-10 wk pp	9.35	± 1.3	8.42	± 0.6	8.63	± 1.3	

10% Fat 20% Sucrose	Sweetness	24-28 wk	11.93	± 0.9	11.97	± 0.4	12.45	± 0.9
	Intensity	34-38 wk	11.79	± 1.0	11.81	± 0.4	11.09	± 0.9
		6-10 wk pp	11.55	± 0.9	12.04	± 0.4	12.50	± 0.9
		Sweetness	24-28 wk	7.18	± 1.4	7.33	± 0.6	7.57
	Liking	34-38 wk	7.00	± 1.6	7.43	± 0.6	9.08	± 1.4
		6-10 wk pp	6.83	± 1.4	7.69	± 0.6	7.61	± 1.4
		Creaminess	24-28 wk	8.31	± 1.1	8.92	± 0.5	8.91
	Intensity	34-38 wk	8.52	± 1.3	9.18	± 0.5	9.71	± 1.1
		6-10 wk pp	9.53	± 1.2	9.63	± 0.5	9.27	± 1.2
		Creaminess	24-28 wk	8.25	± 1.2	6.73	± 0.5	7.64
	Liking	34-38 wk	7.47	± 1.4	8.26	± 0.6	9.62	± 1.2
		6-10 wk pp	8.43	± 1.3	7.85	± 0.6	9.68	± 1.3
		Flavor	24-28 wk	11.16	± 1.0	9.85	± 0.4	10.66
	Intensity	34-38 wk	11.15	± 1.2	11.03	± 0.5	9.73	± 1.0
		6-10 wk pp	11.35	± 1.1	11.11	± 0.5	11.26	± 1.1
		Flavor	24-28 wk	8.57	± 1.3	6.81	± 0.5	8.16
	Liking	34-38 wk	8.36	± 1.5	7.82	± 0.6	8.83	± 1.3
		6-10 wk pp	8.05	± 1.4	8.15	± 0.6	8.14	± 1.4

TABLE 5.9 – POST-GLUCOLA COMMERCIAL FOOD RATINGS

Commercial Food	Taste Quality		GDM			NGT			Control		
Canned Fruit	Sweetness	24-28 wk	7.20	±	1.1 ^a	4.14	±	0.5 ^b	5.68	±	1.1 ^{a,b}
		34-38 wk	4.91	±	1.3	4.76	±	0.5	5.82	±	1.1
		6-10 wk pp	4.87	±	1.2	4.70	±	0.5	6.89	±	1.1
	Liking	24-28 wk	9.90	±	1.3	7.06	±	0.5	5.77	±	1.3
		34-38 wk	7.07	±	1.4	6.96	±	0.6	6.17	±	1.3
		6-10 wk pp	8.24	±	1.4	7.14	±	0.6	7.42	±	1.3
	Flavor	24-28 wk	7.91	±	1.2	7.56	±	0.5	7.70	±	1.2
		34-38 wk	6.60	±	1.4	8.01	±	0.6	7.70	±	1.2
		6-10 wk pp	8.28	±	1.3	8.25	±	0.6	9.42	±	1.2
	Liking	24-28 wk	9.88	±	1.3	7.65	±	0.6	6.38	±	1.3
		34-38 wk	8.66	±	1.5	8.43	±	0.6	6.07	±	1.3
		6-10 wk pp	10.13	±	1.5	8.42	±	0.6	9.19	±	1.4
Salted Peanuts	Saltiness	24-28 wk	7.84	±	1.1	9.15	±	0.4	8.67	±	1.1
		34-38 wk	8.26	±	1.3	9.22	±	0.5	9.72	±	1.1
		6-10 wk pp	8.21	±	1.1	9.95	±	0.5	10.84	±	1.1
	Liking	24-28 wk	10.96	±	1.0	10.36	±	0.4	9.83	±	1.0
		34-38 wk	10.30	±	1.3	10.43	±	0.5	10.80	±	1.0
		6-10 wk pp	11.21	±	1.1	10.75	±	0.5	12.17	±	1.1
	Crunchiness	24-28 wk	9.38	±	1.0	10.09	±	0.4	10.42	±	1.0
		34-38 wk	10.44	±	1.2	10.58	±	0.5	9.75	±	1.0
		6-10 wk pp	11.23	±	1.1	10.83	±	0.5	10.62	±	1.1
	Liking	24-28 wk	11.12	±	1.0	11.22	±	0.4	10.05	±	1.0
		34-38 wk	10.87	±	1.2	11.49	±	0.4	10.88	±	1.0
		6-10 wk pp	12.08	±	1.0	11.09	±	0.5	11.78	±	1.0
	Oiliness	24-28 wk	5.03	±	1.1	6.86	±	0.5	6.23	±	1.1
		34-38 wk	6.44	±	1.4	8.55	±	0.5	7.81	±	1.1
		6-10 wk pp	6.87	±	1.2	8.06	±	0.5	9.01	±	1.2
	Liking	24-28 wk	8.65	±	1.2	7.26	±	0.5	7.59	±	1.2
		34-38 wk	7.92	±	1.4	7.87	±	0.5	8.22	±	1.2
		6-10 wk pp	9.09	±	1.2	7.59	±	0.6	9.24	±	1.2
	Flavor	24-28 wk	8.16	±	1.1	9.34	±	0.4	9.79	±	1.1
		34-38 wk	8.24	±	1.3	10.63	±	0.5	9.59	±	1.1
		6-10 wk pp	8.79	±	1.1	9.69	±	0.5	9.94	±	1.1
	Liking	24-28 wk	10.72	±	1.0	10.64	±	0.4	11.30	±	1.0
		34-38 wk	10.94	±	1.2	11.05	±	0.5	11.46	±	1.0
		6-10 wk pp	12.41	±	1.1	10.42	±	0.5	12.45	±	1.1
Vanilla Pudding	Sweetness	24-28 wk	8.33	±	1.0	9.42	±	0.4	9.19	±	1.0
		34-38 wk	7.74	±	1.2	9.23	±	0.5	8.99	±	1.0
		6-10 wk pp	8.60	±	1.1	9.84	±	0.5	10.05	±	1.1
	Liking	24-28 wk	7.08	±	1.2	9.15	±	0.5	7.44	±	1.2
		34-38 wk	10.18	±	1.4	10.06	±	0.5	9.19	±	1.2
		6-10 wk pp	8.91	±	1.3	10.32	±	0.6	9.63	±	1.3
	Creaminess	24-28 wk	10.15	±	1.0	12.13	±	0.4	11.75	±	1.0
		34-38 wk	10.60	±	1.1	11.61	±	0.4	11.18	±	1.0
		6-10 wk pp	11.38	±	1.0	11.76	±	0.4	12.71	±	1.0
	Liking	24-28 wk	9.16	±	1.3	9.66	±	0.5	9.20	±	1.3
		34-38 wk	10.40	±	1.4	10.45	±	0.6	9.68	±	1.3
		6-10 wk pp	9.72	±	1.3	10.79	±	0.6	10.00	±	1.3
	Flavor	24-28 wk	8.95	±	1.0	9.94	±	0.4	9.83	±	1.0
		34-38 wk	8.96	±	1.1	10.24	±	0.5	10.11	±	1.0
		6-10 wk pp	7.53	±	1.0	10.24	±	0.5	10.85	±	1.0

Vanilla Pudding (cont.)	Flavor	24-28 wk	10.08	± 1.3	9.48	± 0.5	7.61	± 1.3
	Liking	34-38 wk	10.19	± 1.5	9.51	± 0.6	9.23	± 1.3
		6-10 wk pp	10.15	± 1.3	10.26	± 0.6	9.68	± 1.3
Snack Chips	Saltiness Intensity	24-28 wk	6.99	± 1.1	9.52	± 0.4	9.45	± 1.1
		34-38 wk	8.55	± 1.2	9.98	± 0.5	10.17	± 1.1
		6-10 wk pp	10.00	± 1.1	10.23	± 0.5	12.12	± 1.1
	Saltiness Liking	24-28 wk	10.47	± 1.0	10.71	± 0.4	10.82	± 1.0
		34-38 wk	10.91	± 1.1	10.69	± 0.5	11.81	± 1.0
		6-10 wk pp	10.49	± 1.0	11.16	± 0.5	12.17	± 1.0
	Spiciness Intensity	24-28 wk	5.21	± 1.2	6.23	± 0.5	4.20	± 1.2
		34-38 wk	5.22	± 1.4	7.14	± 0.5	6.22	± 1.2
		6-10 wk pp	5.48	± 1.2	7.54	± 0.6	5.83	± 1.2
	Spiciness Liking	24-28 wk	10.05	± 1.2	9.06	± 0.5	7.82	± 1.2
		34-38 wk	9.61	± 1.4	9.16	± 0.6	9.31	± 1.2
		6-10 wk pp	9.53	± 1.3	9.10	± 0.6	10.93	± 1.3
	Crunchiness Intensity	24-28 wk	9.95	± 0.9	11.66	± 0.4	12.41	± 0.9
		34-38 wk	11.76	± 1.0	11.98	± 0.4	11.99	± 0.9
		6-10 wk pp	11.91	± 0.9	11.93	± 0.4	12.79	± 0.9
	Crunchiness Liking	24-28 wk	11.67	± 1.0	11.83	± 0.4	11.96	± 1.0
		34-38 wk	11.12	± 1.1	11.80	± 0.4	12.48	± 0.9
		6-10 wk pp	10.68	± 1.0	11.95	± 0.4	12.87	± 1.0
Flavor Intensity	24-28 wk	8.42	± 1.1	10.18	± 0.4	9.23	± 1.1	
	34-38 wk	8.49	± 1.3	10.45	± 0.5	9.68	± 1.1	
	6-10 wk pp	10.11	± 1.1	10.46	± 0.5	10.73	± 1.1	
Flavor Liking	24-28 wk	11.63	± 1.0	11.31	± 0.4	12.26	± 1.0	
	34-38 wk	11.57	± 1.2	11.03	± 0.5	12.27	± 1.0	
	6-10 wk pp	10.32	± 1.1	11.63	± 0.5	12.65	± 1.0	
Sharp Cheddar Cheese	Saltiness Intensity	24-28 wk	5.40	± 1.2	6.01	± 0.5	7.19	± 1.2
		34-38 wk	7.28	± 1.4	7.55	± 0.5	6.45	± 1.2
		6-10 wk pp	7.69	± 1.3	7.78	± 0.6	10.03	± 1.3
	Saltiness Liking	24-28 wk	8.39	± 1.3	6.92	± 0.5	7.03	± 1.3
		34-38 wk	8.54	± 1.5	6.85	± 0.6	8.61	± 1.3
		6-10 wk pp	7.49	± 1.3	6.66	± 0.6	9.81	± 1.3
	Bitterness Intensity	24-28 wk	7.32	± 1.3	7.89	± 0.5	7.46	± 1.3
		34-38 wk	7.51	± 1.5	8.41	± 0.6	7.82	± 1.3
		6-10 wk pp	8.30	± 1.3	9.40	± 0.6	9.70	± 1.3
	Bitterness Liking	24-28 wk	7.37	± 1.3	6.06	± 0.5	6.37	± 1.3
		34-38 wk	6.71	± 1.5	6.24	± 0.6	7.26	± 1.3
		6-10 wk pp	5.42	± 1.4	6.94	± 0.6	9.31	± 1.4
	Flavor Intensity	24-28 wk	10.76	± 1.2	9.49	± 0.5	8.40	± 1.2
		34-38 wk	9.37	± 1.4	9.79	± 0.5	9.21	± 1.2
		6-10 wk pp	8.24	± 1.3	10.22	± 0.6	9.63	± 1.3
	Flavor Liking	24-28 wk	6.26	± 1.5	7.34	± 0.6	7.72	± 1.5
		34-38 wk	8.28	± 1.7	6.53	± 0.7	8.20	± 1.5
		6-10 wk pp	8.08	± 1.5	7.42	± 0.7	10.03	± 1.5

TABLE 5.10 – DIET ANALYSIS OF FOOD FREQUENCY QUESTIONNAIRE

Diet Parameters: FFQ		GDM		NGT		Control	
Kcal	16-20 wk	2241.09	± 313.1	2419.03	± 103.8	2186.34	± 215.5
	24-28 wk	1891.86	± 260.5	2661.05	± 107.8	1794.06	± 260.5
	34-38 wk	1873.32	± 297.1	2473.35	± 115.2	1628.96	± 271.3
	6-10 wk pp	2289.41	± 271.3	2285.75	± 121.5	1587.80	± 271.3
Protein (g)	16-20 wk	97.13	± 11.8	91.04	± 3.9	75.43	± 8.1
	24-28 wk	82.51	± 9.9	95.80	± 4.1	73.00	± 9.9
	34-38 wk	81.78	± 11.2	93.28	± 4.4	64.92	± 10.3
	6-10 wk pp	93.91	± 10.3	88.19	± 4.6	64.43	± 10.3
Protein/ 1000kcal (g/1000kcal)	16-20 wk	44.00	± 2.7	38.06	± 0.9	35.20	± 1.9
	24-28 wk	43.61	± 2.2	36.94	± 0.9	40.40	± 2.2
	34-38 wk	43.71	± 2.6	38.38	± 1.0	38.86	± 2.3
	6-10 wk pp	41.50	± 2.3	39.88	± 1.0	39.98	± 2.3
Carbohydrate (g)	16-20 wk	263.24	± 44.1	309.32	± 14.6	287.46	± 30.3
	24-28 wk	219.09	± 36.7	344.87	± 15.2	219.54	± 36.7
	34-38 wk	194.04	± 41.8	317.51	± 16.2	194.96	± 38.2
	6-10 wk pp	259.16	± 38.2	282.82	± 17.1	204.56	± 38.2
Carbohydrate/ 1000kcal (g/1000kcal)	16-20 wk	118.74	± 6.8	128.96	± 2.2	129.35	± 4.7
	24-28 wk	118.08	± 5.6	129.07	± 2.3	121.41	± 5.6
	34-38 wk	106.38	± 6.4	128.98	± 2.5	120.41	± 5.9
	6-10 wk pp	116.39	± 5.9	121.55	± 2.6	129.26	± 5.9
Fat (g)	16-20 wk	92.76	± 14.5	96.05	± 4.8	84.62	± 10.0
	24-28 wk	81.93	± 12.1	105.31	± 5.0	70.20	± 12.1
	34-38 wk	88.22	± 13.7	96.88	± 5.3	67.04	± 12.6
	6-10 wk pp	101.62	± 12.6	93.45	± 5.6	57.85	± 12.6
Fat/ 1000kcal (g/1000kcal)	16-20 wk	40.50	± 2.4	39.11	± 0.8	39.36	± 1.7
	24-28 wk	42.07	± 2.0	39.50	± 0.8	39.36	± 2.0
	34-38 wk	45.82	± 2.3	38.67	± 0.9	41.38	± 2.1
	6-10 wk pp	42.66	± 2.1	41.14	± 0.9	36.54	± 2.1
Protein (% Kcal)	16-20 wk	17.53	± 1.1	15.22	± 0.4	14.1	± 0.7
	24-28 wk	17.56	± 0.9	14.82	± 0.4	16.21	± 0.9
	34-38 wk	17.43	± 1.0	15.33	± 0.4	15.55	± 0.9
	6-10 wk pp	16.71	± 0.9	15.94	± 0.4	16.05	± 0.9
Carbohydrate (% Kcal)	16-20 wk	47.38	± 2.7	51.58	± 0.9	51.79	± 1.9
	24-28 wk	47.2	± 2.3	51.69	± 0.9	48.5	± 2.3
	34-38 wk	42.35	± 2.6	51.48	± 1.0	48.14	± 2.4
	6-10 wk pp	46.68	± 2.4	48.53	± 1.1	51.72	± 2.4
Fat (% Kcal)	16-20 wk	36.43	± 2.2	35.21	± 0.7	35.42	± 1.5
	24-28 wk	38.01	± 1.8	35.56	± 0.8	35.29	± 1.8
	34-38 wk	41.32	± 2.1	34.75	± 0.8	37.14	± 1.9
	6-10 wk pp	38.36	± 1.9	36.99	± 0.9	32.81	± 1.9
Cholesterol (mg)	16-20 wk	308.38	± 54.2	345.38	± 18.0	245.20	± 37.3
	24-28 wk	321.93	± 45.1	371.66	± 18.7	241.70	± 45.1
	34-38 wk	305.11	± 51.4	345.88	± 19.9	195.19	± 47.0
	6-10 wk pp	331.48	± 47.0	340.05	± 21.0	232.64	± 47.0
Cholesterol/ 1000kcal (mg/1000kcal)	16-20 wk	138.12	± 17.1	142.48	± 5.7	115.01	± 11.7
	24-28 wk	163.31	± 14.2	141.04	± 5.9	135.84	± 14.2
	34-38 wk	160.91	± 16.2	143.20	± 6.3	121.68	± 14.8
	6-10 wk pp	146.79	± 14.8	159.54	± 6.6	143.40	± 14.8
Saturated Fat (g)	16-20 wk	32.89	± 5.9	34.12	± 2.0	27.22	± 4.1
	24-28 wk	30.01	± 4.9	38.07	± 2.0	24.05	± 4.9
	34-38 wk	30.07	± 5.6	36.18	± 2.2	23.26	± 5.1
	6-10 wk pp	33.36	± 5.1	33.57	± 2.3	19.61	± 5.1

Saturated Fat/ 1000kcal (g/1000kcal)	16-20 wk	14.65	±	1.2	13.67	±	0.4	12.50	±	0.9
	24-28 wk	15.42	±	1.0	14.19	±	0.4	13.46	±	1.0
	34-38 wk	15.85	±	1.2	14.28	±	0.5	14.46	±	1.1
	6-10 wk pp	14.37	±	1.1	14.69	±	0.5	12.32	±	1.1
Mono- unsaturated Fat (g)	16-20 wk	34.01	±	5.0	30.94	±	1.6	27.10	±	3.4
	24-28 wk	26.67	±	4.1	34.92	±	1.7	23.13	±	4.1
	34-38 wk	27.66	±	4.7	31.12	±	1.8	23.29	±	4.3
	6-10 wk pp	37.57	±	4.3	30.55	±	1.9	19.32	±	4.3
Mono- unsaturated Fat/1000kcal (g/1000kcal)	16-20 wk	14.41	±	1.0	12.69	±	0.3	12.97	±	0.7
	24-28 wk	13.67	±	0.8	13.17	±	0.3	12.99	±	0.8
	34-38 wk	14.49	±	0.9	12.46	±	0.4	14.44	±	0.9
	6-10 wk pp	14.96	±	0.9	13.49	±	0.4	12.37	±	0.9
Poly- unsaturated Fat (g)	16-20 wk	17.38	±	3.5	20.04	±	1.2	18.18	±	2.4
	24-28 wk	17.00	±	2.9	20.72	±	1.2	14.51	±	2.9
	34-38 wk	22.09	±	3.3	17.98	±	1.3	13.05	±	3.0
	6-10 wk pp	20.36	±	3.0	18.48	±	1.4	11.93	±	3.0
Poly- unsaturated Fat/1000kcal (g/1000kcal)	16-20 wk	7.73	±	1.1	8.36	±	0.4	8.77	±	0.7
	24-28 wk	8.74	±	0.9	7.87	±	0.4	8.04	±	0.9
	34-38 wk	11.05	±	1.0	7.26	±	0.4	7.85	±	0.9
	6-10 wk pp	8.87	±	0.9	8.16	±	0.4	7.40	±	0.9
Fiber (g)	16-20 wk	21.62	±	3.1	22.73	±	1.0	19.05	±	2.1
	24-28 wk	17.00	±	2.6	21.37	±	1.1	17.30	±	2.6
	34-38 wk	15.90	±	2.9	20.58	±	1.1	13.91	±	2.7
	6-10 wk pp	19.89	±	2.7	18.40	±	1.2	11.72	±	2.7
Fiber/ 1000kcal (g/1000kcal)	16-20 wk	10.23	±	1.1	9.92	±	0.4	8.58	±	0.8
	24-28 wk	9.53	±	0.9	8.57	±	0.4	9.16	±	0.9
	34-38 wk	8.95	±	1.0	8.83	±	0.4	8.77	±	1.0
	6-10 wk pp	9.08	±	1.0	8.13	±	0.4	7.15	±	1.0
Total Sugar (g)	16-20 wk	114.45	±	27.5	140.05	±	9.1	143.76	±	18.9
	24-28 wk	99.40	±	22.9	171.26	±	9.5	101.06	±	22.9
	34-38 wk	82.39	±	26.1	152.79	±	10.1	88.66	±	23.8
	6-10 wk pp	111.06	±	23.8	133.09	±	10.7	106.99	±	23.8
Total Sugar/ 1000kcal (g/1000kcal)	16-20 wk	49.89	±	6.6	57.15	±	2.2	63.51	±	4.6
	24-28 wk	52.33	±	5.5	62.67	±	2.3	56.19	±	5.5
	34-38 wk	42.50	±	6.3	61.45	±	2.4	54.79	±	5.8
	6-10 wk pp	47.66	±	5.8	57.16	±	2.6	68.83	±	5.8
Vitamin C (mg)	16-20 wk	177.87	±	68.7	211.74	±	22.7	210.21	±	47.2
	24-28 wk	80.38	±	57.1	288.48	±	23.6	140.90	±	57.1
	34-38 wk	134.06	±	65.1	225.98	±	25.3	112.19	±	59.5
	6-10 wk pp	140.96	±	59.5	205.32	±	26.6	177.43	±	59.5
Vitamin C/ 1000kcal (mg/1000kcal)	16-20 wk	73.43	±	18.6	88.22	±	6.2	90.25	±	12.8
	24-28 wk	42.75	±	15.5	101.81	±	6.4	74.28	±	15.5
	34-38 wk	61.17	±	17.7	91.81	±	6.9	65.68	±	16.1
	6-10 wk pp	60.44	±	16.1	86.49	±	7.2	116.92	±	16.1
Vitamin A (IU)	16-20 wk	11581.00	±	1642.3	8895.89	±	544.1	6799.66	±	1130.1
	24-28 wk	4752.29	±	1366.3	8230.74	±	565.3	6926.76	±	1366.4
	34-38 wk	5861.38	±	1558.1	7760.80	±	604.1	4291.80	±	1422.6
	6-10 wk pp	5593.80	±	1422.9	7316.99	±	637.2	4155.84	±	1422.6
Vitamin A/ 1000kcal (IU/1000kcal)	16-20 wk	4555.93	±	789.1	3839.37	±	261.5	3210.90	±	543.0
	24-28 wk	2444.85	±	656.5	3334.40	±	271.6	3707.34	±	656.6
	34-38 wk	3130.91	±	748.7	3322.47	±	290.3	2713.92	±	683.6
	6-10 wk pp	2379.31	±	683.7	3655.07	±	306.2	2654.86	±	683.6

Beta								
Carotene (µg)	16-20 wk	2590.98	±	488.2	1948.02	±	161.7	1597.54 ± 335.9
	24-28 wk	1031.47	±	406.1	1820.28	±	168.0	1580.93 ± 406.2
	34-38 wk	1470.68	±	463.1	1722.61	±	179.6	847.71 ± 422.9
	6-10 wk pp	1416.19	±	422.9	1841.89	±	189.4	875.71 ± 422.9
Beta								
Carotene/ 1000kcal (µg/1000kcal)	16-20 wk	981.99	±	304.4	870.79	±	100.9	757.83 ± 209.5
	24-28 wk	513.28	±	253.2	770.93	±	104.8	850.99 ± 253.3
	34-38 wk	798.34	±	288.8	765.98	±	112.0	539.73 ± 263.7
	6-10 wk pp	601.13	±	263.7	993.86	±	118.1	559.24 ± 263.7
Sodium (mg)	16-20 wk	2959.81	±	406.0	2908.59	±	134.5	2142.69 ± 279.4
	24-28 wk	2156.31	±	337.8	2884.66	±	139.8	1886.67 ± 337.8
	34-38 wk	2096.10	±	385.2	2782.07	±	149.4	1681.52 ± 351.7 ^b
	6-10 wk pp	2360.90	±	351.8	2375.96	±	157.6	1567.38 ± 351.7
Sodium/ 1000kcal (mg/1000kcal)	16-20 wk	1356.72	±	102.8	1201.54	±	34.1	989.29 ± 70.7 ^b
	24-28 wk	1110.57	±	85.5	1102.77	±	35.4	1004.75 ± 85.5
	34-38 wk	1129.60	±	97.5	1129.47	±	37.8	1025.21 ± 89.1
	6-10 wk pp	1007.75	±	89.1	1066.56	±	39.9	982.91 ± 99.1
Iron (mg)	16-20 wk	18.35	±	25.4	19.26	±	8.4	92.70 ± 17.5
	24-28 wk	14.70	±	21.1	19.82	±	8.8	33.59 ± 21.2
	34-38 wk	13.63	±	24.1	18.69	±	9.4	10.11 ± 22.0 ^b
	6-10 wk pp	16.95	±	22.0	15.39	±	9.9	11.26 ± 22.0
Iron/ 1000kcal (mg/1000kcal)	16-20 wk	7.95	±	7.9	8.07	±	2.6	30.60 ± 5.5
	24-28 wk	8.65	±	6.6	7.68	±	2.7	19.77 ± 6.6
	34-38 wk	8.14	±	7.5	7.73	±	2.9	6.21 ± 6.9
	6-10 wk pp	8.10	±	6.9	6.78	±	3.1	6.97 ± 6.9
Calcium (mg)	16-20 wk	1281.82	±	199.1	1208.42	±	66.0	960.76 ± 137.0
	24-28 wk	1037.03	±	165.7	1269.19	±	68.5	808.56 ± 165.7 ^b
	34-38 wk	1097.08	±	188.9	1285.61	±	73.2	680.49 ± 172.5 ^b
	6-10 wk pp	1053.27	±	172.5	1056.49	±	77.3	656.71 ± 172.5
Calcium/ 1000kcal (mg/1000kcal)	16-20 wk	543.89	±	49.3	486.94	±	16.3	448.64 ± 33.9
	24-28 wk	545.74	±	41.0	483.81	±	17.0	455.73 ± 41.0
	34-38 wk	579.22	±	46.8	519.74	±	18.1	435.96 ± 42.7 ^b
	6-10 wk pp	472.14	±	42.7	460.69	±	19.1	397.13 ± 42.7
Folate (µg)	16-20 wk	455.14	±	77.8	488.28	±	25.8	434.03 ± 53.5
	24-28 wk	370.14	±	64.7	526.00	±	26.8	409.29 ± 64.7
	34-38 wk	378.11	±	73.8	470.55	±	28.6	327.31 ± 67.4
	6-10 wk pp	443.66	±	67.4	443.49	±	30.2	325.21 ± 67.4
Folate/ 1000kcal (µg/1000kcal)	16-20 wk	205.02	±	21.0	206.72	±	6.9	198.29 ± 14.4
	24-28 wk	206.70	±	17.4	199.58	±	7.2	217.93 ± 17.4
	34-38 wk	204.16	±	19.9	199.18	±	7.7	202.40 ± 18.2
	6-10 wk pp	208.36	±	18.2	191.83	±	8.1	208.97 ± 18.2

TABLE 5.11 – FREQUENCY OF FOOD CONSUMPTION (X/WK)

Food/Drink (servings/wk)		GDM			NGT			Control		
Fruit	16-20 wk	8.91	± 2.7	a,b	11.74	± 0.9	a	5.01	± 1.9	b
	24-28 wk	6.81	± 2.3	a,b	10.98	± 0.9	a	3.92	± 2.3	b
	34-38 wk	12.28	± 2.6	a,b	11.57	± 1.0	a	4.26	± 2.3	b
	6-10 wk pp	8.94	± 2.3		7.75	± 1.1		2.89	± 2.3	
Fruit Juice	16-20 wk	5.33	± 3.7		7.54	± 1.2		7.05	± 2.5	
	24-28 wk	3.18	± 3.1	a	11.62	± 1.3	b	6.38	± 3.1	a,b
	34-38 wk	4.84	± 3.5		6.70	± 1.4		3.50	± 3.2	
	6-10 wk pp	2.93	± 3.2		7.11	± 1.4		6.86	± 3.2	
Fruit Drink	16-20 wk	2.85	± 1.8		2.09	± 0.6		2.53	± 1.3	
	24-28 wk	0.26	± 1.5		2.21	± 0.6		2.01	± 1.5	
	34-38 wk	0.03	± 1.7		2.66	± 0.7		1.52	± 1.6	
	6-10 wk pp	3.19	± 1.6		1.20	± 0.7		1.91	± 1.6	
Bread	16-20 wk	9.65	± 4.7		15.25	± 1.6		7.35	± 3.2	
	24-28 wk	8.03	± 3.9		15.40	± 1.6		4.54	± 3.9	
	34-38 wk	9.52	± 4.4		13.20	± 1.7		10.54	± 4.1	
	6-10 wk pp	16.56	± 4.1		12.78	± 1.8		3.48	± 4.1	
Tortillas	16-20 wk	3.26	± 3.7		5.10	± 1.2		4.93	± 2.6	
	24-28 wk	3.36	± 3.1		5.36	± 1.3		1.13	± 3.1	
	34-38 wk	4.72	± 3.5		6.40	± 1.4		0.64	± 3.2	
	6-10 wk pp	8.13	± 3.2		6.97	± 1.5		0.87	± 3.2	
Cakes	16-20 wk	2.53	± 2.2		4.41	± 0.7		4.04	± 1.5	
	24-28 wk	2.92	± 1.8		7.11	± 0.8		3.63	± 1.8	
	34-38 wk	0.97	± 2.1	a	6.53	± 0.8	b	4.59	± 1.9	a,b
	6-10 wk pp	2.64	± 1.9		5.92	± 0.9		2.91	± 1.9	
Low-fat Cakes	16-20 wk	0.11	± 0.2		0.14	± 0.1		0.13	± 0.1	
	24-28 wk	0.32	± 0.2		0.13	± 0.1		0.08	± 0.2	
	34-38 wk	0.20	± 0.2		0.17	± 0.1		0.08	± 0.2	
	6-10 wk pp	0.33	± 0.2		0.17	± 0.1		0.11	± 0.2	
Fat-free Cakes	16-20 wk	0.00	± 0.0		0.00	± 0.0		0.00	± 0.0	
	24-28 wk	0.00	± 0.0		0.01	± 0.0		0.00	± 0.0	
	34-38 wk	0.00	± 0.0		0.00	± 0.0		0.00	± 0.0	
	6-10 wk pp	0.00	± 0.0		0.00	± 0.0		0.00	± 0.0	
Cereal	16-20 wk	4.99	± 2.2		5.99	± 0.7		3.94	± 1.5	
	24-28 wk	5.10	± 1.8		6.21	± 0.7		3.24	± 1.8	
	34-38 wk	4.29	± 2.0		6.46	± 0.8		1.64	± 1.9	
	6-10 wk pp	5.29	± 1.9		4.60	± 0.8		2.81	± 1.9	
Rice	16-20 wk	6.65	± 1.9		4.90	± 0.6		7.40	± 1.3	
	24-28 wk	5.19	± 1.6		4.73	± 0.7		8.16	± 1.6	
	34-38 wk	5.36	± 1.8		4.43	± 0.7		7.43	± 1.7	
	6-10 wk pp	5.94	± 1.7		5.67	± 0.8		6.98	± 1.7	
Pasta	16-20 wk	5.24	± 1.6		3.00	± 0.5		1.48	± 1.1	
	24-28 wk	2.78	± 1.3		2.84	± 0.6		1.11	± 1.3	
	34-38 wk	2.65	± 1.5		4.03	± 0.6		0.84	± 1.4	
	6-10 wk pp	3.81	± 1.4		2.31	± 0.6		1.06	± 1.4	
Beans	16-20 wk	2.46	± 1.5		3.06	± 0.5		3.20	± 1.0	
	24-28 wk	3.50	± 1.3		2.22	± 0.5		5.34	± 1.3	
	34-38 wk	2.32	± 1.4		2.30	± 0.6		3.14	± 1.3	
	6-10 wk pp	3.09	± 1.3		2.37	± 0.6		3.30	± 1.3	
Refried Beans	16-20 wk	0.34	± 0.4		0.44	± 0.1		1.17	± 0.3	
	24-28 wk	0.77	± 0.4		0.57	± 0.2		0.31	± 0.4	
	34-38 wk	0.39	± 0.4		0.29	± 0.2		0.17	± 0.4	
	6-10 wk pp	-0.01	± 0.4		0.38	± 0.2		0.26	± 0.4	

Plantains	16-20 wk	0.43	± 0.7	^a	0.86	± 0.2	^a	2.40	± 0.5	^b
	24-28 wk	0.56	± 0.6		0.60	± 0.2		0.96	± 0.6	
	34-38 wk	0.46	± 0.6		0.74	± 0.2		0.65	± 0.6	
	6-10 wk pp	0.27	± 0.6		0.61	± 0.3		0.56	± 0.6	
Vegetables	16-20 wk	14.50	± 2.9		12.45	± 1.0		9.34	± 2.0	
	24-28 wk	8.66	± 2.4		10.38	± 1.0		10.14	± 2.4	
	34-38 wk	6.52	± 2.8		10.56	± 1.1		9.40	± 2.5	
	6-10 wk pp	9.20	± 2.5		10.44	± 1.1		8.75	± 2.5	
Potatoes	16-20 wk	2.40	± 0.7		2.29	± 0.2		1.39	± 0.5	
	24-28 wk	1.44	± 0.6		1.81	± 0.3		0.66	± 0.6	
	34-38 wk	1.25	± 0.7		1.56	± 0.3		0.72	± 0.6	
	6-10 wk pp	2.01	± 0.6		1.87	± 0.3		0.64	± 0.6	
French Fries	16-20 wk	0.82	± 0.6		1.19	± 0.2		0.87	± 0.4	
	24-28 wk	0.77	± 0.5		1.23	± 0.2		0.99	± 0.5	
	34-38 wk	0.23	± 0.5		0.97	± 0.2		0.61	± 0.5	
	6-10 wk pp	0.66	± 0.5		0.71	± 0.2		0.69	± 0.5	
Starchy Vegetables	16-20 wk	0.78	± 0.7		1.61	± 0.2		1.54	± 0.5	
	24-28 wk	1.29	± 0.6		1.92	± 0.2		0.97	± 0.6	
	34-38 wk	0.39	± 0.7		1.57	± 0.3		0.64	± 0.6	
	6-10 wk pp	3.47	± 0.6	^a	1.51	± 0.3	^b	0.45	± 0.6	^b
Oils	16-20 wk	6.69	± 2.2		6.68	± 0.7		7.54	± 1.5	
	24-28 wk	4.36	± 1.8		5.59	± 0.8		4.56	± 1.8	
	34-38 wk	10.43	± 2.1	^a	4.62	± 0.8	^b	3.51	± 1.9	^{a,b}
	6-10 wk pp	9.64	± 1.9		5.38	± 0.9		5.88	± 1.9	
Alcohol	16-20 wk	0.00	± 0.5	^a	0.00	± 0.2	^a	1.39	± 0.3	^b
	24-28 wk	0.00	± 0.4	^a	0.00	± 0.2	^a	1.63	± 0.4	^b
	34-38 wk	0.00	± 0.4	^a	0.00	± 0.2	^a	1.52	± 0.4	^b
	6-10 wk pp	0.11	± 0.4		0.33	± 0.2		0.86	± 0.4	
Meat	16-20 wk	13.72	± 2.4		13.22	± 0.8		9.49	± 1.7	
	24-28 wk	11.79	± 2.0		12.69	± 0.8		9.57	± 2.0	
	34-38 wk	13.33	± 2.3		11.81	± 0.9		9.47	± 2.1	
	6-10 wk pp	15.41	± 2.1		12.21	± 0.9		8.61	± 2.1	
Eggs	16-20 wk	2.43	± 1.0		4.00	± 0.3		2.25	± 0.7	
	24-28 wk	3.51	± 0.8		4.05	± 0.3		2.28	± 0.8	
	34-38 wk	3.27	± 0.9		3.60	± 0.4		1.25	± 0.9	
	6-10 wk pp	3.49	± 0.9		3.74	± 0.4		1.95	± 0.9	
Nuts	16-20 wk	1.18	± 1.5		1.55	± 0.4		2.04	± 0.9	
	24-28 wk	1.20	± 1.1		2.04	± 0.4		0.78	± 1.1	
	34-38 wk	1.36	± 1.2		1.21	± 0.5		0.83	± 1.1	
	6-10 wk pp	2.78	± 1.1		1.34	± 0.5		0.51	± 1.1	
Dairy	16-20 wk	14.46	± 5.3		20.17	± 1.8		10.82	± 3.6	
	24-28 wk	16.15	± 4.4		20.62	± 1.8		9.63	± 4.4	
	34-38 wk	12.63	± 5.0		19.94	± 2.0		9.21	± 4.6	
	6-10 wk pp	15.27	± 4.6		15.58	± 2.1		12.75	± 4.6	
Low-fat Dairy	16-20 wk	4.99	± 1.9		3.24	± 0.6		0.90	± 1.3	
	24-28 wk	7.02	± 1.6		3.41	± 0.7		1.45	± 1.6	
	34-38 wk	6.04	± 1.8		2.76	± 0.7		1.53	± 1.7	
	6-10 wk pp	1.83	± 1.7		1.68	± 0.8		2.38	± 1.7	
Fat-free Dairy	16-20 wk	4.21	± 1.4		1.50	± 0.5		2.32	± 1.0	
	24-28 wk	0.66	± 1.2		1.12	± 0.5		1.67	± 1.2	
	34-38 wk	1.56	± 1.4		1.92	± 0.5		0.35	± 1.3	
	6-10 wk pp	0.98	± 1.2		0.90	± 0.6		1.97	± 1.2	

Pizza	16-20 wk	0.93	± 0.6	1.43	± 0.2	1.14	± 0.4
	24-28 wk	1.45	± 0.5	1.37	± 0.2	0.91	± 0.5
	34-38 wk	0.99	± 0.6	1.37	± 0.2	0.88	± 0.5
	6-10 wk pp	1.44	± 0.5	1.31	± 0.2	0.59	± 0.5
Salty Snacks	16-20 wk	1.53	± 0.7	1.86	± 0.2	1.01	± 0.5
	24-28 wk	0.59	± 0.6	1.71	± 0.2	1.12	± 0.6
	34-38 wk	0.42	± 0.7	1.08	± 0.3	1.01	± 0.6
	6-10 wk pp	0.70	± 0.6	1.22	± 0.3	0.49	± 0.6
Low-fat Salty Snacks	16-20 wk	0.17	± 0.1	0.05	± 0.0	0.03	± 0.1
	24-28 wk	0.00	± 0.1	0.07	± 0.0	0.00	± 0.1
	34-38 wk	0.30	± 0.1	0.10	± 0.0	0.00	± 0.1
	6-10 wk pp	0.08	± 0.1	0.02	± 0.0	0.00	± 0.1
Tomato Sauce	16-20 wk	2.32	± 0.8	1.85	± 0.3	1.66	± 0.6
	24-28 wk	1.39	± 0.7	1.76	± 0.3	0.37	± 0.7
	34-38 wk	1.44	± 0.8	1.58	± 0.3	0.94	± 0.7
	6-10 wk pp	1.28	± 0.7	1.94	± 0.3	1.45	± 0.7
Condiments	16-20 wk	5.68	± 0.9	1.98	± 0.3	1.84	± 0.6
	24-28 wk	0.96	± 0.8	1.80	± 0.3	1.64	± 0.8
	34-38 wk	1.21	± 0.9	0.96	± 0.3	0.51	± 0.8
	6-10 wk pp	2.88	± 0.8	1.01	± 0.4	0.28	± 0.8
Pickles	16-20 wk	0.56	± 1.3	2.22	± 0.4	0.09	± 0.9
	24-28 wk	0.17	± 1.1	1.09	± 0.4	0.31	± 1.1
	34-38 wk	0.00	± 1.2	1.12	± 0.5	0.10	± 1.1
	6-10 wk pp	0.00	± 1.1	0.24	± 0.5	0.02	± 1.1
Candy	16-20 wk	1.90	± 2.8	4.58	± 0.9	4.89	± 1.9
	24-28 wk	2.19	± 2.4	4.77	± 1.0	8.97	± 2.4
	34-38 wk	1.97	± 2.7	3.44	± 1.0	2.07	± 2.5
	6-10 wk pp	1.41	± 2.5	4.35	± 1.1	4.43	± 2.5
Sugar Free Candy	16-20 wk	2.73	± 1.2	0.79	± 0.4	0.92	± 0.8
	24-28 wk	0.55	± 1.0	0.61	± 0.4	0.85	± 1.0
	34-38 wk	0.84	± 1.1	0.79	± 0.4	6.59	± 1.0
	6-10 wk pp	-0.01	± 1.0	0.76	± 0.5	1.10	± 1.0
Chocolate	16-20 wk	0.55	± 1.0	1.23	± 0.3	1.48	± 0.7
	24-28 wk	0.68	± 0.8	1.70	± 0.3	1.03	± 0.8
	34-38 wk	0.28	± 0.9	1.67	± 0.4	1.65	± 0.8
	6-10 wk pp	1.99	± 0.8	1.72	± 0.4	1.08	± 0.8
Clear Soups	16-20 wk	1.72	± 0.8	1.95	± 0.3	1.28	± 0.6
	24-28 wk	0.72	± 0.7	1.62	± 0.3	2.15	± 0.7
	34-38 wk	1.03	± 0.8	1.72	± 0.3	0.78	± 0.7
	6-10 wk pp	1.22	± 0.7	1.68	± 0.3	0.67	± 0.7
Creamy Soups	16-20 wk	0.28	± 0.2	0.24	± 0.1	0.27	± 0.1
	24-28 wk	0.02	± 0.1	0.19	± 0.1	0.04	± 0.1
	34-38 wk	0.09	± 0.2	0.18	± 0.1	0.29	± 0.2
	6-10 wk pp	0.07	± 0.2	0.06	± 0.1	0.01	± 0.2
Gravy	16-20 wk	0.22	± 0.3	0.51	± 0.1	0.39	± 0.2
	24-28 wk	0.10	± 0.3	0.36	± 0.1	0.00	± 0.3
	34-38 wk	0.21	± 0.3	0.33	± 0.1	0.24	± 0.3
	6-10 wk pp	0.09	± 0.3	0.54	± 0.1	0.07	± 0.3
Added Sugar	16-20 wk	2.19	± 3.8	4.59	± 1.3	5.03	± 2.6
	24-28 wk	4.08	± 3.2	7.41	± 1.3	5.28	± 3.2
	34-38 wk	1.23	± 3.6	5.74	± 1.4	3.51	± 3.3
	6-10 wk pp	5.20	± 3.3	5.33	± 1.5	4.14	± 3.3

Sugar Substitute	16-20 wk	-0.02	± 2.5		0.28	± 0.8		0.84	± 1.7
	24-28 wk	8.25	± 2.1	^a	1.42	± 0.8	^b	0.54	± 2.1
	34-38 wk	5.03	± 2.3		0.47	± 0.9		1.97	± 2.1
	6-10 wk pp	3.68	± 2.1		1.31	± 1.0		1.46	± 2.1
Combined Fats	16-20 wk	5.78	± 1.5	^a	3.54	± 0.5	^{a,b}	1.27	± 1.0
	24-28 wk	4.99	± 1.3		3.50	± 0.5		2.26	± 1.3
	34-38 wk	3.02	± 1.4		3.08	± 0.6		1.15	± 1.3
	6-10 wk pp	3.09	± 1.3		3.25	± 0.6		1.16	± 1.3
Combined Low-Fats	16-20 wk	0.00	± 0.5		0.14	± 0.2		0.13	± 0.3
	24-28 wk	1.77	± 0.4	^a	0.19	± 0.2	^b	0.33	± 0.4
	34-38 wk	1.75	± 0.4	^a	0.22	± 0.2	^b	0.11	± 0.4
	6-10 wk pp	0.58	± 0.4		0.09	± 0.2		0.00	± 0.4
Combined 'No'-Fats	16-20 wk	0.00	± 0.1		0.05	± 0.0		0.02	± 0.1
	24-28 wk	0.00	± 0.1		0.08	± 0.0		0.02	± 0.1
	34-38 wk	0.00	± 0.1		0.07	± 0.0		0.00	± 0.1
	6-10 wk pp	0.00	± 0.1		0.05	± 0.0		0.02	± 0.1
Soda	16-20 wk	0.90	± 1.8		2.43	± 0.6		3.16	± 1.2
	24-28 wk	2.70	± 1.5		2.67	± 0.6		2.56	± 1.5
	34-38 wk	0.09	± 1.7		2.10	± 0.6		2.39	± 1.5
	6-10 wk pp	3.23	± 1.5		3.30	± 0.7		3.44	± 1.5
Diet Soda	16-20 wk	-0.02	± 0.5		0.13	± 0.2		0.27	± 0.4
	24-28 wk	0.03	± 0.4		0.24	± 0.2		0.68	± 0.4
	34-38 wk	0.12	± 0.5		0.80	± 0.2		0.19	± 0.5
	6-10 wk pp	0.10	± 0.5		0.44	± 0.2		-0.01	± 0.5
Coffee/Tea	16-20 wk	2.04	± 1.7		2.42	± 0.6		3.06	± 1.2
	24-28 wk	1.93	± 1.4		2.69	± 0.6		4.32	± 1.4
	34-38 wk	2.36	± 1.6		2.19	± 0.6		5.17	± 1.5
	6-10 wk pp	4.52	± 1.5		5.44	± 0.7		4.62	± 1.5
Decaf Coffee/Tea	16-20 wk	1.53	± 0.9		0.40	± 0.3		0.37	± 0.6
	24-28 wk	0.79	± 0.7		1.00	± 0.3		0.59	± 0.7
	34-38 wk	1.03	± 0.8		0.85	± 0.3		-0.01	± 0.7
	6-10 wk pp	0.21	± 0.7		0.36	± 0.3		-0.08	± 0.8
Iced Tea	16-20 wk	0.20	± 1.3		1.04	± 0.4		1.17	± 0.9
	24-28 wk	0.13	± 1.1		2.00	± 0.4		0.93	± 1.1
	34-38 wk	0.02	± 1.2		1.16	± 0.5		0.31	± 1.1
	6-10 wk pp	0.11	± 1.1		1.33	± 0.5		2.08	± 1.1
Sweet Coffee Drinks	16-20 wk	-0.01	± 0.3	^{a,b}	0.09	± 0.1	^a	0.62	± 0.2
	24-28 wk	0.08	± 0.2		0.23	± 0.1		0.36	± 0.2
	34-38 wk	0.01	± 0.3		0.10	± 0.1		0.28	± 0.2
	6-10 wk pp	0.01	± 0.2	^a	0.11	± 0.1	^a	1.86	± 0.2

Appendix B: Ballots and Questionnaires

Taste Ballot

Subject #: _____

Instructions: Rinse your mouth thoroughly with water before you begin. Please taste the

sample and place a single mark on the line scale corresponding to the question.

1. How strong is the SWEETNESS of this sample?

None _____ Very Strong

2. How CREAMY is this sample?

None _____ Very Strong

3. How strong is the FLAVOR of this sample?

None _____ Very Strong

4. How much do you LIKE the SWEETNESS of this sample?

Dislike Extremely _____ Like Extremely

5. How much do you LIKE the CREAMINESS of this sample?

Dislike Extremely _____ Like Extremely

6. How much do you LIKE the FLAVOR of this sample?

Dislike Extremely _____ Like Extremely

FOOD FREQUENCY QUESTIONNAIRE

SUBJ #: _____

DATE: _____

Please consider each of the foods listed in this questionnaire and indicate how often you eat the food by noting the number of servings you consume either per DAY or WEEK.

Please use the **servicing sizes** noted for each food in answering.

For example, if you eat 2 slices of White Bread every day, you will enter “2” under the “DAY” column for White Bread. Or if you eat 2 Cups (C) of White Rice every week you will enter “4” under the WEEK column. Please note that (C) represents 1 cup and (T) represents 1 Tablespoon.

If you eat something once or twice per month, you may write that in as follows “2/mo”.

Servings per:

DAY <u>or</u>	WEEK	FOOD	SERVING SIZE (Consider this amt as 1 Serving)
_____	_____	White Bread (including Italian or white rolls, biscuits)	1 Slice
_____	_____	Whole grain bread (whole wheat, rye, oat etc.)	1 Slice
_____	_____	Crackers (regular fat & sodium)	4 Crackers
_____	_____	Crackers (low-fat, no- fat or low sodium)	4 Crackers
_____	_____	Tortillas (corn, flour)	1 6” Diam. Tortilla
_____	_____	Muffins (corn, blueberry, bran etc.)	1 3” Diam. Muffin
_____	_____	English muffins	1 muffin
_____	_____	Bagels	1 3.5” Diam. Bagel
_____	_____	Pita Bread	1 6” Diam. Pita

_____	_____	Pancakes, waffles, toaster pastry	2 4" Diam. Pancakes 2 4" Diam. Waffles 1 Toaster Pastry
_____	_____	Hot cereal	1C Cooked Cereal
_____	_____	Cold cereal	1C cereal
_____	_____	Granola, granola bars	1C Granola, 1 4" long bar
_____	_____	White rice	½ Cup Cooked Rice
_____	_____	Brown rice, wild rice	½ Cup Cooked Rice
_____	_____	Pasta (spaghetti, noodles, macaroni)	½ Cup Cooked Pasta
_____	_____	Fruit - fresh	½ Cup Fruit
_____	_____	Fruit – canned, frozen	½ Cup Fruit
_____	_____	Fruit – Tropical (mango, pineapple etc.)	½ Cup Fruit
_____	_____	Plantain	½ Cup Fruit
_____	_____	Fruit juice (OJ, cranberry, pineapple, apple etc.)	1C Juice
_____	_____	Fruit Drink (HI-C, Kool- aid etc.)	1 Cup
_____	_____	Vegetable Juice (tomato, V8)	1 Cup
_____	_____	Vegetable oil (corn, safflower, canola etc.)	1 Tablespoon (T)
_____	_____	Olive oil	1 T
_____	_____	Vegetable shortening	1 T
_____	_____	Lard	1 T
_____	_____	Margarine (regular)	1 T
_____	_____	Margarine (light version)	1 T
_____	_____	Butter	1 T
_____	_____	Mayonnaise (regular)	1 T
_____	_____	Mayonnaise (no cholesterol)	1 T
_____	_____	Mayonnaise (no fat)	1 T

_____	_____	Sour cream (regular)	1 T
_____	_____	Sour cream (reduced fat)	1 T
_____	_____	Salad dressing (regular)	1 T
_____	_____	Salad dressing (reduced calories)	1 T
_____	_____	Salad dressing (no fat)	1 T
_____	_____	Creamed cheese (regular)	1 T
_____	_____	Creamed cheese (reduced fat/calorie)	1 T
_____	_____	Creamed cheese (no fat)	1 T
_____	_____	Cream – heavy or light, whipping, coffee	1 T
_____	_____	Half & half	1 T
_____	_____	Non-dairy creamer (coffee whitener)	1 T
_____	_____	Milk - whole	1 C
_____	_____	Milk – reduced fat (1%, 2%)	1 C
_____	_____	Milk – skim	1 C
_____	_____	Flavored milk (chocolate, strawberry, etc)	1 C
_____	_____	Yogurt – regular fat	1 C (8 oz.)
_____	_____	Yogurt – low fat	1 C
_____	_____	Yogurt – no fat	1 C
_____	_____	Lettuce	1 Cup shredded
_____	_____	Dark green and leafy vegetables (broccoli, spinach, kale etc.)	½ C chopped
_____	_____	Cabbage	½ C cut
_____	_____	Carrots, celery	½ C cut
_____	_____	Tomatoes	½ C sliced
_____	_____	Green Beans	½ C
_____	_____	Corn	½ C kernels

_____	_____	Peas	½ C
_____	_____	Beans (Black, pinto, white)	½ C
_____	_____	Refried Beans	½ C
_____	_____	White potatoes (boiled, mashed, baked)	½ C
_____	_____	French fried potatoes, home-fried potatoes	½ C
_____	_____	Sweet potatoes, yams	½ C
_____	_____	Other vegetables (zucchini, squash, or anything not listed above)	½ C
_____	_____	Soda – regular	1 12 oz can
_____	_____	Soda – diet	1 12 oz can
_____	_____	Regular coffee, tea	1 C
_____	_____	Decaffeinated coffee, tea	1 C
_____	_____	Iced Tea (Snapple)	1 C
_____	_____	Sweet coffee drinks (Café Mocha, Cappuccino, Café Latte, Café con leche)	1 C
_____	_____	Hot chocolate	1 C
_____	_____	Beer – regular	1 8 oz. Bottle
_____	_____	Beer – light versions	1 8 oz. Bottle
_____	_____	Wine	1 6oz. glass
_____	_____	Liquor (rum, whiskey, scotch, vodka, etc.)	1 shot (2 oz)
_____	_____	Trail Mix (mixture of nuts, raisins and other dried fruit)	½ C
_____	_____	Peanut butter, nut butters	1 T
_____	_____	Tofu	½ C cubes
_____	_____	Veggie burger	¼ lb

_____	_____	Bacon	2 slices
_____	_____	Beef (roast, steak ground meat etc.)	3 oz
_____	_____	Poultry (chicken, turkey, duck etc.)	3 oz (1/3 pound)
_____	_____	Pork	3 oz (1/3 pound)
_____	_____	Veal	3 oz (1/3 pound)
_____	_____	Lamb, mutton	3 oz (1/3 pound)
_____	_____	Chicken and Rice	1 Cup
_____	_____	Canned fish – packed in oil (tuna, shrimp, salmon, etc.)	3 oz
_____	_____	Canned fish – packed in water	3 oz
_____	_____	Fresh fish (including fish sticks)	3 oz
_____	_____	Eggs	1 Medium or Large
_____	_____	Egg Substitute	¼ C
_____	_____	Cheese – regular fat	2 oz (1/4 pound)
_____	_____	Cheese – reduced fat	2 oz (1/4 pound)
_____	_____	Cheese – no fat	2 oz (1/4 pound)
_____	_____	Cottage cheese – regular fat	½ C
_____	_____	Cottage cheese – reduced fat	½ C
_____	_____	Lunch meats – regular fat	2 oz (1/4 pound)
_____	_____	Lunch meats – reduced fat	2 oz (1/4 pound)
_____	_____	Hot dogs, beef or pork, regular fat	1 hot dog
_____	_____	Hamburgers	¼ pound
_____	_____	Sausage – breakfast	2 links
_____	_____	Sausage - Italian	1 1” diam., 3” long link
_____	_____	Cookies – regular	2 3” diam. cookies
_____	_____	Cookies – reduced fat/calories	2 3” diam. cookies

_____	_____	Brownies	1 2" square
_____	_____	Doughnuts	1 3" diam.
_____	_____	Pastry – regular	1 4" diam.
_____	_____	Pastry – reduced fat/calories	1 4" diam.
_____	_____	Cake, no icing, regular	1 2" slice (2 oz)
_____	_____	Cake, with icing, regular	1 2" slice (2oz)
_____	_____	Fat free cakes and pastries	2 oz piece
_____	_____	Pie (cherry, apple, pumpkin, custard etc.)	1 2" slice
_____	_____	Jello – regular	½ C
_____	_____	Jello – no sugar	½ C
_____	_____	Pudding – regular	½ C
_____	_____	Pudding – reduced calorie	½ C
_____	_____	Pudding – fat free	½ C
_____	_____	Ice cream – regular (Sealtest, Breyers etc.)	½ C
_____	_____	Ice cream – super rich (Haagen Daaz, Ben & Jerry's)	½ C
_____	_____	Ice cream – reduced fat (ice milk etc.)	½ C
_____	_____	Ice cream – fat free	½ C
_____	_____	Frozen yogurt – regular fat	½ C
_____	_____	Frozen yogurt – reduced fat	½ C
_____	_____	Frozen yogurt – fat free	½ C
_____	_____	Other no fat frozen desserts	½ C
_____	_____	Candy – chocolate, chocolate bars	1.5 oz
_____	_____	Candy – hard, mints, lifesavers, lollipops etc.	1 piece
_____	_____	Candy – reduced sugar, calories	1 piece

_____	_____	Milk shakes	1 C
_____	_____	Pizza	1 4" slice
_____	_____	Popcorn – regular	1 C
_____	_____	Popcorn – reduced fat	1 C
_____	_____	Potato chips, corn chips, Doritos – regular	½ C
_____	_____	Chips – reduced fat/calories	½ C
_____	_____	Ketchup, catsup, mustard, relish	1 T
_____	_____	Tomato sauce, spaghetti sauce	½ C
_____	_____	Pickles	1 ½" diam., 3" long
_____	_____	Chewing gum – regular	1 stick
_____	_____	Chewing gum – sugarless	1 stick
_____	_____	Gravy	¼ Cup
_____	_____	Soup, clear based	1 C
_____	_____	Soup, cream based	1 C
_____	_____	Sugar	1 T
_____	_____	Artificial sweetener	1 packet

The Cravings Questionnaire*

Subj# _____ Estimated Height: ___ ft. ___ inches
 Age _____ Estimated Weight: _____
 Male _____ Female _____
 Are you currently on a diet? Yes No
 Please answer the following questions to the best of your ability

1. Have you ever experienced food cravings (i.e., an intense desire to eat a specific food)? Yes No
2. If you have experienced food cravings, we would like to know what it is that you crave. List below foods which you crave, starting with your strongest craving. Beside each, estimate how often you experienced that craving.

	CRAVED FOOD	FREQUENCY
Strongest craving	_____	_____ times/week
Next strongest craving	_____	_____ times/week
Etc.	_____	_____ times/week

3. The following questions refer only to the food which you indicated as your strongest craving (the food at the top of your craving list in Question 2)
 - a. Describe in as much detail as you can the food that you crave the most.
 - b. When you are experiencing a craving for the food you crave the most, is there any other food which would satisfy that craving?
 - c. When you are experiencing a craving for the food you crave the most, how often do you follow through and eat that food?
 _____ % of the time
 - d. How do you feel when you've eaten the food you crave the most?
 - e. (For women only) Do you feel that your cravings are related to your menstrual cycle?

No
 Yes If yes, then how?

Is there anything about your cravings you would like to tell us that we forgot to ask?

*Weingarten, HP and Elston, D. 1991. Food Cravings in a College Population. *Appetite* 17: 167-175.

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CURRICULUM VITA

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EDUCATION

- 2008 Ph.D. in Nutritional Sciences, Rutgers, The State University of New Jersey, New Brunswick, NJ
- 2001 Dietetic Internship, Saint Joseph College, West Hartford, CT
- 2000 Bachelor of Science in Nutritional Sciences, Rutgers, The State University of New Jersey, New Brunswick, NJ

PROFESSIONAL AND TEACHING EXPERIENCE

- 2003 – 2007 Graduate Research Assistant, Department of Nutritional Sciences, Rutgers, The State University of New Jersey, New Brunswick, NJ
- 2005 – 2007 Guest Lecturer: *Nutrition for the Developing Child*, Rutgers, The State University of New Jersey, New Brunswick, NJ
- 2005 Teaching Assistant, Department of Food Science, Rutgers, The State University of New Jersey, New Brunswick, NJ
- 2003 – 2005 Clinical Consultant Dietitian: Circle of Friends Adult Center, Springfield, NJ
- 2002 – 2003 Teaching Assistant, Department of Nutritional Sciences, Rutgers, The State University of New Jersey, New Brunswick, NJ
- 2001 – 2002 Clinical Dietitian: Rahway Hospital, Rahway, NJ

ABSTRACTS AND PUBLICATIONS

Belzer, L. and Tepper, B.J. 2005. Changes in Sweet Taste in Women with Gestational Diabetes Mellitus. 2005 Annual Meeting Abstracts [AChemS].

Belzer, L. and Tepper, B.J. 2006. Leptin, Insulin and Sweet Taste in Gestational Diabetes Mellitus. 2006 Annual Meeting Abstracts [AChemS].

Belzer, L., Tepper, B.J., Smulian, J. and Lu, Shou-En. 2007. Changes in Insulin and Leptin Across Pregnancy in Women with Diet-treated Gestational Diabetes Mellitus. 2007 Annual Meeting Abstracts [EB].

Belzer, L. (Projected Publication Date: 2/08). "Fat Taste." In Encyclopedia of Obesity. Published by SAGE publications. Thousand Oaks, CA. Lead editors: Kathleen Keller and Geoffrey J. Golson.