For my course development project, I choose to improve the NDFS 435 Nutritional Biochemistry course that I teach. The following information describe NDFS 435 and highlight the ways that I improved the course based on information that I was taught during the spring seminar.

**Course Background**

NDFS 435 “Nutritional Biochemistry” is the capstone course for students majoring in the Nutritional Science and Dietetics programs. It is generally taken the last semester of our student’s college career. NDFS 435 has Chem 481 or Chem 285 (Biochemistry or Bio-organic chemistry, respectively) as prerequisites. The purpose of this course is to study the pathways by which macro and micronutrients are used in the human body, with an emphasis on the biochemical processes that allow the use of these nutrients.

This course has been offered during Fall and Winter semesters each year, and I am the only professor teaching this course. Historically, the course has 10-15 students during fall semester, and 40-60 students during winter semester. During fall semester, the majority of the students are Nutritional Science majors, and during winter semester roughly 85% of the students are dietetics majors. This divergence in students causes a potential problem with the course, due to the varied level of training in chemistry.

**Learning Outcomes**

The learning outcomes for this course are as follows:

1. **Scientific Research and Writing**—Demonstrate ability to search, interpret, and summarize original scientific information in an efficiently written scientific paper. As scientists, our students should be able to determine what is “good science” and what is “bad science”. To do this, they must be able to read, interpret and judge scientific information. This course learning outcome supports the department learning outcome of Research: Search, interpret, and summarize peer-reviewed scientific literature. Evaluate nutritional claims for accuracy. Design research for nutritional science that includes ethical considerations of using human and animal subjects.

2. **Nutrient Chemical Structure and Metabolism**—Demonstrate knowledge of nutrient chemical structures, food sources, digestion, absorption, transport, metabolism and functions, and the metabolic consequences of nutrient deficiencies, interactions, imbalances and toxicities. As students majoring in nutritional science and dietetics, they will need to have a deep understanding of
how fuel is used in human biology. This outcome will help them understand the consequences of under and overnutrition, as well as understanding the biochemical methods by which fuel are utilized. This course learning outcome supports the department learning outcome of Nutritional Science Fundamentals: Identify the chemical structure of nutrients, their food sources, functions, and the scientific basis for nutrition requirements and dietary recommendations. Describe the processes of digestion, absorption, transport, and metabolism and the metabolic consequences of nutrient deficiencies, interactions, imbalances and toxicities.

3. **Experimental Design**—Demonstrate knowledge of appropriate design for scientific experiments. Our students need to understand how to properly design scientific experiments for future employment. In addition, this skill will allow them to properly evaluate scientific data, by determining if the experiments were done appropriately. This course learning outcome supports the department learning outcome of Research: Search, interpret, and summarize peer-reviewed scientific literature. Evaluate nutritional claims for accuracy. Design research for nutritional science that includes ethical considerations of using human and animal subjects.

4. **Biochemical Functions and Pathways**—Understand the biochemical functions and pathways of nutrients in the body. This outcome will ensure that students have the breadth and depth of knowledge that will help them in future careers. This course learning outcome supports the department learning outcome of Nutritional Science Fundamentals: Identify the chemical structure of nutrients, their food sources, functions, and the scientific basis for nutrition requirements and dietary recommendations. Describe the processes of digestion, absorption, transport, and metabolism and the metabolic consequences of nutrient deficiencies, interactions, imbalances and toxicities.

5. **Nutrient Metabolism and Body Homeostasis**—Identify physiological, biochemical and metabolic changes associated with deficient nutrient intake, excessive nutrient intake and selected metabolic diseases. This learning outcome will ensure that students can apply the biochemical pathways used in nutrient metabolism to understanding defects in metabolism frequently observed in human populations. This course-learning outcome supports two department learning outcomes. First it supports the Nutritional Science Fundamentals: Identify the chemical structure of nutrients, their food sources, functions, and the scientific basis for nutrition requirements and dietary recommendations. Describe the processes of digestion, absorption, transport, and metabolism and the metabolic consequences of nutrient deficiencies, interactions, imbalances and toxicities. Second, it supports the department-learning outcome for Diet in Health and Disease: Explain the role of dietary choices and interventions for optimizing health and preventing or ameliorating chronic disease and malnutrition.
**Course Activities**

In order to meet the course learning outcomes, I have employed the following activities in my course.

1. **Chapter study guides**: Prior to the first lecture on a chapter, and to help the students to complete the assigned reading, we have produced study guides that are to be completed and turned in prior to class. We choose five of the study guides that are turned in each time to be graded. Students are encouraged to work together in groups. The study guides direct the students to the important parts of the chapter so that in class discussion can be more in depth and useful. This activity allows us to have better in class discussions. It aligns with outcomes 2, 4, and 5.

2. **Pop Quizzes**: We have 12 pop quizzes during the course. The purpose, again, is to help the students’ study what is in the reading and to synthesize the information gone over in class. This is also an assessment tool used in the course. This activity aligns with outcomes 2, 4 and 5.

3. **Group work**: Students are assigned to groups of 4-5 on the first day of class. The students stay in these groups for the duration of the course. During lectures, we frequently stop and the students are asked to explain the concepts to each other, or to work out case studies together. Groups are then given the opportunity to explain their discussion and to be given feedback. This serves the purpose of allowing me to determine if the students truly understand the concepts. This aligns with learning outcomes 1-5.

4. **Primary literature discussion**: Every Monday we discuss an article from the current literature. This allows the students to read the most up to date information in the field. We discuss the figures in small groups and as a class. This aligns with outcomes 1 and 3.

5. **Writing Prompts**: Based on the weekly paper discussion, students are given a writing prompt to help them better understand the paper. They are encouraged to talk to me, the TA’s and other students in order to complete the writing prompt. The purpose is to help them synthesize the information presented in the paper, and to properly analyze the presented data. This aligns with outcomes 1 and 3.

6. **Case study**: Students are given the task to find a gene frequently found defective in a biochemical pathway involved with metabolism. The students study out the topic and build a written case study. Five case studies are turned in a week, and the best is given the opportunity to present in class for a small amount of extra credit. This aligns with outcome 5.

7. **Research Paper**: The students are required to complete a 5-page research paper on a topic in nutritional biochemistry. The assignment has
multiple benchmarks to assist the students to complete the paper in a timely manner. This project induces them to analyze the primary data, find a question that is yet unanswered, and propose and design a study to answer the question. This aligns with outcomes 1 and 3.

8. **Exams:** The course has 5 exams, made of short answer and multiple-choice questions. These questions come from the textbook reading, primary research articles discussed in class, the in class case study presentations and our discussion during lecture. Each exam is worth 100 points. The questions are such as to align with outcomes 1-5.

**Assessments of Student Learning**
To determine if students have met the learning outcomes, the following assessments are used:

1. **Chapter study guides:** Prior to the first lecture on a chapter, and to help the students to complete the assigned reading, we have produced study guides that are to be completed and turned in prior to class. We choose five of the study guides that are turned in each time to be graded. Students are encouraged to work together in groups. The study guides direct the students to the important parts of the chapter so that in class discussion can be more in depth and useful. This activity allows us to have better in class discussions. It aligns with outcomes 2, 4, and 5.

2. **Pop Quizzes:** We have 12 pop quizzes during the course. The purpose, again, is to help the student’s study what is in the reading and to synthesize the information gone over in class. This is also an assessment tool used in the course. This activity aligns with outcomes 2, 4 and 5.

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**Student Achievement of Learning Outcomes**

The following section provides quantitative and qualitative evidence that the course learning outcomes were met through the Fall 2014 iteration of this course. The evidence is provided for each learning objective:

1. **Scientific Research and Writing**-This outcome were met through four assessments: case study, writing prompts, research paper and exams. Each of these assessments provided the students with opportunities to read the primary literature, interpret the data, and convey information through scientific writing. Based on the outcomes of these four sets of assessments, it is my interpretation that this outcome was met. The evidence for this is defined below:

   a. **Case Study**-The case study was comprised of a two-page paper and a 10-minute oral presentation synthesized from primary literature on a disorder focusing on nutrient metabolism. The students were required to determine a topic, search out information about the topic, write a report on the topic, and teach the class about the topic. The class average for this assessment was 90.6%. The students did an excellent job in their written and in class presentation, as evidenced by their grades and by the case study sample contained in the appendix.

   b. **Writing prompts**-Each week we would discuss a paper from the primary literature in class. Based on the reading, and data presented in the paper, the students had a writing prompt to complete by the end of the week. Each prompt gave the students opportunities to analyze the presented data, and to design experiments that could be used to further the knowledge in the field. There were a total of 14 primary literature papers read during the course and 14 writing prompts. The papers supplemented the lecture topics for the week. The class average for all of the writing prompts was 92.4% for Fall 2014. The average improved after the first three weeks as the students became more familiar with reading and synthesizing information in the primary literature. Included in the appendix is an example of a writing prompt. From the end of year evaluations, the following was said regarding the primary literature and writing prompts: “I appreciate the challenge of reading and writing about the weekly scientific papers. It is a valuable learning experience.”, “I loved the
papers!! Don't take them away! It is a good thing for science students! " Based on the student comments, their papers and their overall score I feel that this was beneficial in meeting this outcome.

c. **Research Paper**-During the semester the students were required to research a topic in nutritional biochemistry. They were required to write a 4-5-page paper that presented a review of the scientific literature and proposed a set of experiments to address a hole that they have found in the literature. The assignment had multiple benchmarks to verify that students were moving along well with the assignment, including a library assignment, abstract and reference check, rough draft, peer review, TA review and a final grade for the paper. The class average in Fall 2014 was 91.4% for the library assignment, 100% for the abstract and reference check, 97.9% for the rough draft, 93% for the peer review, 100% for the TA review, and 90.6% for the final paper grade. An example of a well-done research paper is included in the appendix. Based on the students’ grades, I feel that this assessment was worthwhile in meeting this outcome.

d. **Exams**-The course had five 100-point exams, with the lowest exam grade dropped. Each exam was comprised of 50 points from multiple-choice questions, and 50 points from short answer questions. The students were given 6 questions based on concepts covered during the course lecture, PowerPoint slides and the textbook. The students were required to answer 3 of these questions. The students also received 4 questions that came directly from the paper discussions, of which they were required to answer 2. These questions presented data from the paper, which they were required to interpret. Furthermore, they were asked to design experiments that would answer questions proposed from the research article. Upon completion of the exam, the students were allowed to correct their incorrect short answer questions for 50% of the points that they lost. The average for all of the exams was 89%. An example of an exam question is included in the appendix (all exam were returned to the students, so no example of a students response is available). The students seemed to appreciate the way the exams were laid out. From the end of year student evaluations: "Dr. Tessem provides SO many opportunities for us to succeed - (not just three tests and five homework assignments like so many other science classes, where if you miss a single point you don't get an A. Thumbs down for those classes). I really liked that he had so many assignments and so many tests - this made it so much more possible to get a good grade. It also
helped make the tests really doable, by breaking them down into somewhat smaller units. “

2. **Nutrient Chemical Structure and Metabolism**-This outcome were met through three assessments: the pop quizzes, chapter study guides and the exams. Each of these assessments provided the students with opportunities to demonstrate their knowledge of nutrient chemical structure and metabolism. Based on feedback from the students, I feel that the chapter study guides need to be revamped. The assessments seemed to be beneficial and to help students meet this learning outcome, as evidenced by the data presented below.

   a. **Pop Quizzes**-We had 12 pop quizzes throughout the year, of which the lowest two were dropped. These quizzes were on the chapter that we were discussing. Generally we took 2-3 days per chapter. The quizzes gave the students the opportunity to demonstrate their knowledge of nutrient chemical structure and metabolism. The class average for all of the quizzes was 75.6%. While the quiz average was lower than the exam averages, it did serve the purpose of helping the students understand where to study so that they could be better prepared for the exams. An example of one of the quizzes used in the course is found in the appendix.

   b. **Chapter Study Guides**-Study guides were prepared for each of the 12 chapters. The students were required to complete the study guide prior to the first lecture where we discussed the chapter. This allowed us to have a good in class discussion and focus on areas that the students did not understand. These study guides had the students drawing out metabolic pathways and structures of nutrients. The students did not feel these were a good use of their time or worth the points that they earned. From the end of year student evaluations (which were echoed by my own evaluations and an evaluation by a SCOT representative): ‘By giving us the opportunity to talk in groups and being open to answer questions, I was able to understand the biochemistry presented. The only downside was the study guides due before each chapter was discussed. I think I would have learned more if the study guides were due or organized with each days readings and not the entire chapter. This would have expanded the time I had to read the chapter and retain the information. But other than that, Dr. Tessem was an excellent professor.”, “The course study guides are ridiculously long and not worthy enough points for how much work and time they required. Plus they didn't help prepare you for tests. It felt so pointless to have to waste time complete long the study guides and made me resent new material. The class as a whole already took tons of time, so I think the reading guides should be
an optional thing.”, “Dr. Tessem is very knowledgeable and his lectures are very interesting. I appreciate the challenge of reading and writing about the weekly scientific papers. It is a valuable learning experience. I believe the study guides would be more effective as a tool for students to use on their own without receiving a grade. I feel they are more just busy work (just something to get done) because they require so much detail and time to fill out. The study guides keep me from having time to read and study the chapters. I feel I would learn so much more if I had time to just read the chapters. Nutritional Biochem is a fascinating topic!” , “I absolutely loved this class. It was awesome and Dr. Tessem taught effectively and also made the class interesting. I loved everything in this class and thought it all contributed to my learning, except for the chapter study guides. They were far too long and I didn't feel like I got much out of them. I do think it was good to go over the chapter before we talked about it class, but I think there is a better way to do it. Otherwise, this class was the bomb! I loved it! My favorite class this semester!” Based on these feedback, I have shortened the study guides. They will be used during Winter 2015, and their usefulness will be reassessed. The class average for the study guides was 81.84%.

c. **Exams** - The course had five 100-point exams, with the lowest exam grade dropped. Each exam was comprised of 50 points from multiple-choice questions, and 50 points from short answer questions. The students were given 6 questions based on concepts covered during the course lecture, PowerPoint slides and the textbook. The students were required to answer 3 of these questions. The students also received 4 questions that came directly from the paper discussions, of which they were required to answer 2. These questions asked the student to use metabolic pathways, draw structures, and demonstrate a command of the area of nutrient metabolism that they were being tested on. Upon completion of the exam, the students were allowed to correct their incorrect short answer questions for 50% of the points that they lost. The average for all of the exams was 89%. An example of an exam question is included in the appendix (all exam were returned to the students, so no example of a students response is available). The students seemed to appreciate the way the exams were laid out. From the end of year student evaluations: “Dr. Tessem provides SO many opportunities for us to succeed - (not just three tests and five homework assignments like so many other science classes, where if you miss a single point you don't get an A. Thumbs down for those classes). I really liked that he had so many assignments and so many tests - this made it so much more
possible to get a good grade. It also helped make the tests really doable, by breaking them down into somewhat smaller units.

3. **Experimental Design** - This outcome was met through three assessments: the writing prompts, research paper and exams. Each of these assessments provided the students with opportunities to learn about experimental design, demonstrate experimental design, and interpret experimental design. Based on feedback from the students, I feel that this is one area where the class excelled. The students loved learning about how the data was acquired. The assessments were beneficial. From the student comments: “Dr. Tessem is very knowledgeable and his lectures are very interesting. I appreciate the challenge of reading and writing about the weekly scientific papers. It is a valuable learning experience.”

a. **Writing prompts** - Each week we would discuss a paper from the primary literature in class. Based on the reading, and data presented in the paper, the students had a writing prompt to complete by the end of the week. Each prompt gave the students opportunities to analyze the presented data, and to design experiments that could be used to further the knowledge in the field. There were a total of 14 primary literature papers read during the course and 14 writing prompts. The papers supplemented the lecture topics for the week. The class average for all of the writing prompts was 92.4% for Fall 2014. The average improved after the first three weeks as the students became more familiar with reading and synthesizing information in the primary literature. Included in the appendix is an example of a writing prompt. From the end of year evaluations, the following was said regarding the primary literature and writing prompts: “I appreciate the challenge of reading and writing about the weekly scientific papers. It is a valuable learning experience.”, “I loved the papers!! Don't take them away! It is a good thing for science students!” . Based on the student comments, their papers and their overall score I feel that this was beneficial in meeting this outcome.

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4. Biochemical Functions and Pathways - This outcome was met through three assessments: the pop quizzes, chapter study guides and the exams. Each of these assessments provided the students with opportunities to demonstrate their knowledge of biochemical functions and pathways. Again, the feedback from the students was that the chapter study guides need to be revamped. The assessments seemed to be beneficial and to help students meet this learning outcome, as evidenced by the data presented below.

a. Pop Quizzes - We had 12 pop quizzes throughout the year, of which the lowest two were dropped. These quizzes were on the chapter that we were discussing. Generally we took 2-3 days per chapter. The quizzes gave the students the opportunity to demonstrate their knowledge of nutrient chemical structure and metabolism. The class average for all of the quizzes was 75.6%.
While the quiz average was lower than the exam averages, it did serve the purpose of helping the students understand where to study so that they could be better prepared for the exams. An example of one of the quizzes used in the course is found in the appendix.

b. **Chapter Study Guides**—Study guides were prepared for each of the 12 chapters. The students were required to complete the study guide prior to the first lecture where we discussed the chapter. This allowed us to have a good in class discussion and focus on areas that the students did not understand. These study guides had the students drawing out metabolic pathways and structures of nutrients. The students did not feel these were a good use of their time or worth the points that they earned. From the end of year student evaluations (which were echoed by my own evaluations and an evaluation by a SCOT representative): "By giving us the opportunity to talk in groups and being open to answer questions, I was able to understand the biochemistry presented. The only downside were the study guides due before each chapter was discussed. I think I would have learned more if the study guides were due or organized with each days readings and not the entire chapter. This would have expanded the time I had to read the chapter and retain the information. But other than that, Dr. Tessem was an excellent professor.", "The course study guides are ridiculously long and not worthy enough points for how much work and time they required. Plus they didn't help prepare you for tests. It felt so pointless to have to waste time complete long the study guides and made me resent new material. The class as a whole already took tons of time, so I think the reading guides should be an optional thing.", "Dr. Tessem is very knowledgeable and his lectures are very interesting. I appreciate the challenge of reading and writing about the weekly scientific papers. It is a valuable learning experience. I believe the study guides would be more effective as a tool for students to use on their own without receiving a grade. I feel they are more just busy work (just something to get done) because they require so much detail and time to fill out. The study guides keep me from having time to read and study the chapters. I feel I would learn so much more if I had time to just read the chapters. Nutritional Biochem is a fascinating topic!", "I absolutely loved this class. It was awesome and Dr. Tessem taught effectively and also made the class interesting. I loved everything in this class and thought it all contributed to my learning, except for the chapter study guides. They were far too long and I didn't feel like I got much out of them. I do think it was good to go over the chapter before we talked about it class, but I
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5. **Nutrient Metabolism and Body Homeostasis**- This outcome was met through four assessments: the case study, pop quizzes, chapter study guides and the exams. Each of these assessments provided the students with opportunities to demonstrate their knowledge of nutrient metabolism and body homeostasis, in particular the deregulation of body homeostasis. Again, the feedback from the students was that the chapter study guides need to be revamped. The assessments seemed to be beneficial and to help students meet this learning outcome, as evidenced by the data presented below.

a. **Case Study**- The case study was comprised of a two-page paper and a 10-minute oral presentation synthesized from primary literature on a disorder focusing on nutrient metabolism. The students were required to determine a topic, search out information about the topic, write a report on the topic, and teach
the class about the topic. The class average for this assessment was 90.6%. The students did an excellent job in their written and in class presentation, as evidenced by their grades and by the case study sample contained in the appendix.

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**Steps Planned or Taken to Improve Teaching and Student Learning**

My goal as a professor is to improve my teaching skills in order to improve student learning. I have assessed my teaching through 1) Grades, 2) End of
year Evaluations, 3) BYU SCOT evaluation. I asked for peer evaluation, but it was not available for the Fall 2014 semester. Peer evaluations are currently ongoing, and I will use this information to improve my teaching. I will present this section by the changes that I am or will be making to improve NDFS 435.

1. **Splitting NDFS 435 into two courses**-NDFS 435 has historically been taught to the Nutritional Science students and the Dietetics students. These students take two different sets of chemistry courses. This has left us with difficulty as the NS students have taken Biochem 481 and the Dietetics students have only taken Chem 285. Based on feedback that I received in Winter 2014 semester from students and grades, this class is being split into two courses, NDFS 435 which the NS students will take in the Summer or Fall and NDFS 434, which the Dietetics students will take in the Winter. This will allow us to teach to the students training level. This will also improve student comprehension. A problem that we had was that the NS students felt the course was too easy, while the Dietetics students felt it was too hard. I will be teaching NDFS 434 with Dr. Kenealey.

2. **Change NDFS 435 from Fall/Winter to Summer/Fall**-A second observation that I made is that the NS students frequently would give up in the class. Most of these students, by the time they took the class in the winter semester, had already been admitted into graduate, dental or medical school. Therefore, the course did not have an impact on their future schooling. Many students told me that, they just didn’t care about the course because they had already been admitted into their professional schooling. Based on these observations, and discussion with my department, we have moved this course to be taught in the summer term and fall semester. I will teach the first iteration of this course Summer 2015.

3. **Classroom participation**-Both the end of year student evaluations and SCOT evaluation demonstrated that students appreciated taking time in class to discuss and teach their peers the difficult concepts. From the end of year evaluation “By giving us the opportunity to talk in groups and being open to answer questions, I was able to understand the biochemistry presented.” I have integrated more of this time into my class. I have students set up in groups of 4-5, and we frequently stop the lecture and have the groups describe a pathway, or draw the pathway on the board, or answer an application question. The students feel this helps them understand the questions better.

4. **Streamline Study Guides**-The greatest negative comment I received was in regards to the study guides. Of the student comments, 37% stated that they felt the study guides were not helpful and took too much time from their text reading. I have attempted to streamline the study guides for the Winter 2015 semester. I am currently evaluating their
effectiveness. The purpose is to help the students read the chapter and go through difficult topics before coming to class. Based on the evaluation during Winter 2015 I may remove these from the course.

5. **Paper Discussions** - The students loved the primary literature paper discussions. Both the end of year evaluations and the SCOT evaluation stated that this was their favorite part. I continue to choose new papers for each semester that are new hot topics in the areas of study. From the student evaluations “I appreciate the challenge of reading and writing about the weekly scientific papers. It is a valuable learning experience” and “I loved the papers!! Don’t take them away! It is a good thing for science students!”

6. **Changing PowerPoint slides** - One of the comments that came in the student evaluations mentioned the following: “One thing that is sort of a blessing and a curse: during lecture you basically read off the slides a lot. I liked that because I could just listen, enjoy and engage myself during lecture instead of worrying about taking really diligent notes but at the same time, I was a lot more susceptible to getting distracted and zoning out. I’m not sure if there is a happy solution or balance for this, but just something to think about. Maybe ask more questions to get us thinking before you go over the slides?” I have taken this information and am starting to revise the slides. I have made very thorough slides, but at times they have too much information. I am trying to improve that.
Appendix-Examples of Learning Assessment Tools

1. Case Study-Page 18
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Case Study Example
Brooke Bons

Cataract Formation from Galactokinase Deficiency

Jill decided to deliver her baby Sarah at home. Her midwife suggested she book an appointment at the family practitioner to get her newborn screening in the few days after she was born. A month after the heel prick, the doctor’s office phoned Jill to tell her that they hadn’t collected enough blood to do one of the last tests and asked her to return (and pay) for a second reprick. Frustrated, Jill decided that Sarah seemed perfectly healthy and decided to forgo the expensive reprick. As time went on, she noticed that Sarah had a hard time following objects with her eyes. She had difficulty reaching out and grabbing things held out to her. She was not smiling very much either in response to interactions. At first, she thought Sarah was just a shy, less-interactive baby. After a while, she began to notice a subtle cloudiness in her eyes. She took her to the doctor and after blood and urine sample tests, the doctor diagnosed her with a rare form of galactosemia: galactokinase deficiency.

Galactokinase is an enzyme involved in galactose metabolism. Galactose in primarily ingested into the body in the form of lactose, a disaccharide made up of two monosaccharides, glucose and galactose, joined together by a β (1-4) glycosidic bond. Lactase hydrolyzes that bond and releases galactose as a monosaccharide to be absorbed by the enterocytes in the small intestine. Galactose is sent through the bloodstream to the liver, where it is converted to glucose 1-phosphate through a series of enzymes. Once in its glucose 1-phosphate form, it can be easily changed to glucose 6-phosphate and used for glycolysis.

Figure 1. Mechanism of galactokinase activity on galactose. A conserved aspartic acid residue catalyzes the phosphorylation at C1.

The first step in converting galactose to glucose 6-phosphate involves phosphorylation at C1 on galactose by an enzyme called galactokinase. Individuals with galactokinase deficiency have
a deletion mutation at the \textit{GALK1} gene. This mutation leads to lowered expression of the gene and/or decreased functionality of the enzyme. Lowered galactokinase activity stalls galactose phosphorylation, resulting in galactose accumulation in the cells. These high monosaccharide cellular concentrations activate aldose reductase, an NADPH-dependent oxidoreductase, which reduces the sugar to its alcohol derivative, galactitol. Galactitol is not easily metabolized, nor can it be converted to glucose.

Even without galactokinase deficiency, the lens epithelial cells in the eye are favorable sites for galactose accumulation simply due to site-specific slower rates of phosphorylation. Lens galactose concentrations are even higher for galactokinase deficient subjects, leading to large build-ups of galactitol in the eye. As galactitol accumulates, osmotic pressure forces water into the lens. The lens fibers swell until they ultimately rupture. The vacuole cleans the damage and leaves behind large clefts filled with precipitated proteins. These proteins create opaqueness on the lens and thus, a cataract forms.

So what was happening with Sarah? Due to genetic inheritance, she had 1 of over 20 mutations possible at the \textit{GALK1} gene, which affected gene expression and/or functionality of galactokinase. The inability to convert galactose to galactose 1-phosphate led to high lens concentrations of galactose, which was converted to galactitol. Galactitol build-up caused lens epithelial cell swelling and rupture and protein congregation in ruptured areas led to cataract formation. The cataracts impaired her vision and delayed development of some physical and social skills. However, with proper diet and aid of drugs, cataracts can disappear—making galactokinase deficiency one of the less severe forms of galactosemia.

Questions:
1. What are some dietary instructions the doctor would give for Sarah?
2. What are possible targets for drugs that prevent cataract formation?
3. Diabetes can produce eye problems such as cataracts as well. What is a probable mechanism for cataract formation in diabetes?
4. Even when galactose is withheld from the diet, problems still are seen sometimes. Why?

References:
The function of PEPCK is to synthesize phosphoenolpyruvate (PEP) from oxaloacetate for gluconeogenesis. PEPCK-C and PEPCK-M are two isoforms of PEPCK that both are used for this reaction. PEPCK-C is different from PEPCK-M because PEPCK-C is found in the cytosol of the cell and PEPCK-M is found in the mitochondria of the cell.

The primary question asked by this paper was what the effects on gluconeogenesis would be if PEPCK-M was silenced, as no assessments of this had been performed before. Experimental tools used to address this question included treating rats with antisense oligonucleotides to silence the PEPCK-M gene, allowing researchers to see the effects of the absence of PEPCK-M. They then measured hormones and metabolites of the mice after feeding and fasting stages. They also did experiments in hepatocytes and used silencing RNA to prevent translation of the PEPCK-M gene.

The key finding of the paper was that when PEPCK-M was silenced, gluconeogenesis was impaired. This was shown by decreased glucose formation from lactate, reduced liver glycogen, and increased glycerol turnover, as well as lowered plasma levels of glucose, insulin, and triglycerides. This finding changes the interpreted role of PEPCK-C vs. PEPCK-M because PEPCK-M was previously thought to be irrelevant in gluconeogenesis, as it is located in the mitochondria and gluconeogenesis happens in the cytosol, hence why PEPCK-C was thought to be the primary PEPCK involved in
gluconeogenesis. However, this paper shows that the previous assumptions were incorrect, and that PEPCK-M is responsible for approximately one-third of gluconeogenesis.
Research Paper Example

Possible Mechanisms of High Fructose Diet-Induced Visceral Fat Deposition.

Rachel Fisher
NDFS 435
Sep. 24, 2014
Dr. Tessem

Abstract:

Studies have shown that high fructose diets increase visceral fat, thus inducing obesity and other pieces of metabolic syndrome that plague the US. One could argue that this link can be explained by the excess caloric intake of subjects consuming high fructose diets. However, Kavanagh et al. demonstrated that a high fructose diet induced ectopic fat deposition in a calorically controlled setting. Bursac et al. evaluated the role of glucocorticoids in fat deposition. They found that fructose consumption led to glucocorticoid signaling that caused leptin resistance and adipogenesis, particularly in visceral tissue. Another study found that rats fed a high fructose diet had increased lipid deposition due to increased mitochondria and lipogenic enzymes in hepatic tissue. Most studies administer pure fructose; however, common sweeteners are about half glucose and half fructose. A study is proposed to investigate visceral adipose tissue accumulation from the consumption of sucrose or high fructose corn syrup at different levels. Each of these proposed mechanisms may play a role in the increased lipid deposition that accompanies high fructose diets. Determining the complete mechanism of high fructose consumption and visceral adipose tissue accumulation will help to prevent obesity and other metabolic syndrome related diseases.

Key Words:

Fat deposition, fructose, glucocorticoids, lipogenesis
Interestingly, the rise in the percentage of obese individuals in the US coincides with the introduction of high fructose corn syrup in many foods (1). This link between high fructose consumption and obesity prompted numerous studies on the effect of high fructose consumption on increased visceral adipose tissue and other pieces of metabolic syndrome.

Fructose is absorbed and metabolized differently than glucose (2). Metabolism of glucose through glycolysis is regulated by phosphofructokinase (PFK) which reacts to ATP, insulin and citrate levels to slow down glycolysis when needed; however fructose enters the glycolytic pathway after the PFK step so fructose metabolism is virtually unregulated. Also, unlike glucose, almost all of the fructose absorbed in the small intestine is taken up by the liver and shunted right into glycolysis. This results in a buildup of triose-phosphates when fructose consumption is high. These excess triose-phosphates stimulate lactate production which leads to gluconeogenesis and hepatic de novo lipogenesis.

Researchers have hypothesized many factors that may affect fructose-induced lipogenesis including health status, BMI, dietary protein, enzyme activity, hormones and the combination of foods/sugars consumed with fructose (2-3). It is important to consider all of these factors in order to truly understand the mechanism behind fructose-induced fat deposition.

Bursac et al. (4) hypothesized that high fructose diets contribute to visceral specific adipose accumulation through interactions of glucocorticoids. In this study, two groups of rats were fed ad libitum but one group got normal water and the other group received a 60% fructose solution. After nine weeks on this diet, researchers measured the
rats’ corticosterone, insulin, leptin, and adipose tissue. The amounts of adipose transcription factors, peroxisome-proliferator-activated receptor γ (PPARγ) and sterol regulatory element-binding protein-1 (SREBP-1) were measured. Fructose fed rats had significantly greater visceral adipose tissue as well as signs of leptin resistance and increased PPARγ and SREBP-1 which likely lead to larger adipocytes (4). This study found no change in total body mass of fructose fed rats, however the rats exhibited an increased proportion of visceral fat mass to total body mass. The results point to the conclusion that fructose is less satiating and more lipogenic than other sugars, and thus contributes more significantly toward obesity.

Crescenzo et al. (1) focused on a different factor in the fructose-induced fat deposition puzzle. To evaluate the effect of mitochondrial energetics on de novo lipogenesis from a high fructose diet, researchers fed rats a high fructose or control diet for 8 weeks and then measured the body composition, degree of mitochondrial coupling, de novo lipogenesis, mitochondrial mass, and other relevant parameters. The fructose fed rats had significantly increased rates of de novo lipogenesis and lipogenic enzyme activity including increase fatty acid synthase activity. The fructose rats also had greater mitochondrial protein mass and degree of coupling than the control rats. This means that with a high fructose diet, less substrate is needed to produce ATP. These results lead to the conclusion that fructose increases mitochondrial efficiency and thus leads to increased visceral adipose and whole body fat deposition.

In animal models, ectopic lipid deposition in the liver is seen shortly after the initiation of a high fructose diet, and later on, there is increased hepatic de novo lipogenesis, decreased whole body fat oxidation, and decreased energy expenditure;
however, there is little evidence of these effects in humans when fructose is consumed at moderate levels (2-3). Kavanagh et al. (5) showed that high fructose consumption increased liver fat deposition even without overall weight gain, but they did not observe any effects on visceral adipose tissue. In non-human primates, high fructose corn syrup diets induce visceral favored obesity more than isocaloric glucose diets. Many studies administered pure fructose rather than the glucose/fructose mixtures found in commonly added sugars (3). While this allows researchers to isolate the effects of fructose, these results may not be applicable since normal diets contain both fructose and glucose. The increased lipogenesis seen in many studies may be due to the excess total calories consumed and the overall weight gained as a result rather than the effect of fructose consumption alone (2).

Since fructose absorption is affected greatly by the simultaneous digestion and absorption of glucose, it could be hypothesized that the consumption of glucose with fructose could have synergistic effects on the metabolism of fructose, thus decreasing visceral adipose tissue accumulation (3). To test this hypothesis, a study should be conducted in healthy-weight and obese, non-diabetic patients who do not take prescription drugs or weight loss products. Subjects would be fed a diet containing varying amounts of high fructose corn syrup (HFCS) or sucrose within their beverages at daily caloric intakes calculated to maintain total initial weight. This will eliminate the confounding effect of whole body weight gain. In addition to a control group, three groups would consume sucrose at 10%, 25%, and 40% of their total daily caloric intake and three groups would consume HFCS at 10%, 25%, and 40% of their total daily caloric intake. The total body weight would be measured weekly to ensure maintenance of
original body weight. Visceral adipose tissue would be measured by calculating waist to hip ratio as well as by fat selective magnetic resonance imaging. Measurements should be collected monthly for 1 year in order to test the short term and long term effects of these diets.

The most commonly consumed sweeteners, HFCS and sucrose are roughly a 50/50 mixture of fructose and glucose. So, if the hypothesis for the proposed study were true, most of the research findings about deleterious effects of high fructose consumption would no longer be relevant to humans. If the hypothesis were false, this study would still help to define what levels of fructose consumption cause increased visceral fat deposition.

In conclusion, there is strong evidence which claims that high intakes of pure fructose cause lead to the development of many symptoms of metabolic syndrome including visceral, liver, and ectopic fat deposition. Evidence is very limited as to whether commonly consumed sweeteners, HFCS, and sucrose, have these same effects of pure fructose. Much more research is needed to analyze the effect of fructose consumption on healthy versus obese individuals, the effect of various fructose/glucose mixtures, the effect of diets consisting of different percentages of total calories, and to generate the full picture of the effects of fructose.
References


Response to Reviewers

Reviewer 1: End sentence here and begin new one with “Each…”

The revised abstract split the sentences as suggested.

Reviewer 1: Is this the study you are proposing? If so consider making it more explicit.

I already stated at the beginning of that sentence, “a study is proposed…” the only way to make that more explicit would be to say “My proposed study…” but the paper instructions say not to use first person.

Reviewer 1: Place a colon here or reword the sentence because as it is it doesn’t make sense.

This has been reworded. “potential mechanisms” was deleted.

Reviewer 1: need commas between these

Comma was added

Reviewer 1: This covers a lot consider making it 2 sentences to make it easier for the reader to follow.

This has been fixed

Reviewer 1: The period should go on the other side of the parenthesis.

This has been fixed.

Reviewer 1: Administered?

The verb was changed to past tense.

Reviewer 1: You might want to add some of the data to show how significant their finding were.

This sentence explains that their studies did not have a significant effect, so I find it unimportant to include p-values to demonstrate that it wasn’t significant.

Reviewer 1: unnecessary comma
The comma was deleted.

**Reviewer 1:** insert comma

A comma was inserted.

**Reviewer 1:** pluralize

The plural form was used.

**Reviewer 1:** starting weight?

This was fixed.

**Reviewer 1:** This sentence could use some rewording or different punctuation to make it more clear.

The sentence was reworded.

**Reviewer 1:** What is this supposed to say?

This was fixed.

**Reviewer 1:** Some of the conjunctions in this sentence don’t make sense.

This was fixed.

**Reviewer 1:** Of?

This was changed.

**Reviewer 1:** There are a few grammatical and clarity issues but a simple proof-reading should fix those.

Errors were fixed.

**Reviewer 1:** It might be nice to include some of the data on your studies to show how significant their findings are.

The page limits for this assignment do not allow for the space required to explain data in a way that would make sense to the reader. Thus data was presented only as significant or not for simplicity purposes.

**Reviewer 1:** The last paragraph on page 3 doesn’t explain the study being discussed very well.

The study was discussed in the previous paragraph. I combined the paragraphs to help the reader know that both paragraphs were referring to the same study.

**Reviewer 1:** In the study you are proposing, are your obese individuals’ diabetic?

The wording was switched to clarify.

**Reviewer 1:** I would like a little more explanation as to what you are hoping to discover from your study.

The first sentence of the first paragraph talking about the proposed study clearly states that the study is looking to see if consuming both fructose and glucose together has synergistic effects. This would make other studies which only study pure fructose consumption inapplicable to humans who don’t consume pure fructose.

**Reviewer 1:** Be more explicit in why the findings of your studies wouldn’t be applicable to humans if your hypothesis proved true.

I said that the findings of previous studies wouldn’t be applicable any longer, not that my study findings would not be applicable.

**Reviewer 2:** To keep consistant may want to list the name of the researchers as you had done with the previous two studies.

I believe it is unnecessary to “stay consistent” with the way I reference each study in the abstract. Only the text of the paper is supposed to be cited in a consistent way, and I think that it helps to have some variety in the abstract so it isn’t too repetitive.

**Reviewer 2:** Portions
The change is unnecessary. Both words mean the same thing.

**Reviewer 2: add S to stimulate**
This is a grammatically incorrect suggestion. Since triose-phosphates is plural, the verb should be stimulate with no S at the end.

**TA Review: Don’t forget your name.**
My name was added.

**TA Review: In-text citation should be inside the punctuation.**
Punctuation order was corrected throughout the paper.

**TA Review: Use a different word.**
An alternate word was used.

**TA Review: Did the paper talk about many studies that used pure fructose or did paper 3 use pure fructose?**
Paper 3 is a review paper and so it does reference many studies that use pure fructose in their experiments.

**TA Review: This seems like a good introduction to your study. It shows why you would use HFCS and sucrose.**
The paper was rearranged to put this section right before the introduction to my proposed study.

**TA Review: This seems to go with the first part of the paragraph**
The paragraph was rearranged accordingly.
Exam #1, Fall 2014

BRIGHAM YOUNG UNIVERSITY
Department of Nutrition, Dietetics and Food Science

NDFS 435
Part II- Essay Section
Dr. Jeffery Tessem

52 Points
Name___________________________

Answer any 3 questions from 1-7

1. (10) Draw and label a parietal cell. Include all cell surface transporters and enzymes necessary for parietal cell function. Define the two primary factors secreted by the parietal cell and the mechanism for secretion of one of these factors. Define the hormones that influence parietal cell function, where these hormones are produced, and what controls their secretion.
2. (10) Glucose metabolism is not the same in all tissues. Muscle cells and hepatocytes both take up glucose under fed conditions. Explain how muscle and hepatocytes are different in terms of enzymes, transport proteins, relative Km and the process of glycogenesis.
3. (10) Describe the process of glycogenolysis. Define the enzymes involved in glycogenolysis and their functions. Explain how liver and muscle are different in terms of this process. Finally, define the primary enzyme that is regulated in this pathway and explain how it is regulated in terms of hormones, post-translational modifications and allosteric interactions.
4. (10) The enzymes involved in glycolysis and gluconeogenesis are essentially the same. Explain why gluconeogenesis uses four enzymes that glycolysis does not. Name the four enzymes; define how hormones, signaling molecules and substrate cycling, regulate these enzymes.
5. (10) What is the purpose of the pentose phosphate pathway? What are the three potential outcomes of the pathway? Which tissues express this pathway, and why is it expressed there? What is the key regulated gene in this pathway, and how is it regulated?
6. (10) Contrast glucose and fructose metabolism through glycolysis. How are these two fuel sources treated differently? How does this affect the eventual metabolism and downstream consequences of these fuel sources?
7. (10) What is the purpose of the TCA cycle? What enzymes are required for pyruvate to enter the TCA cycle? What nutrients are involved in this process? Explain how reducing equivalents are transported into the mitochondrial matrix for use in the ETC. Explain how the tissue type determines the use of these reducing equivalents, how the energy from these reducing equivalents are placed in a usable form, and how and why this determines the amount of energy that can be harvested from the ETC. Define the ETC enzymes that allow this to occur.
Answer any 2 questions from 8-11
8. (10) In the paper Reprogramming of the Circadian Clock by Nutritional Challenge, it was shown that circadian rhythms of various genes and metabolites are profoundly changed due to high fat feeding. Please describe the findings presented below. Explain what experiments are used to acquire this data, what this data means, and the effect of these changes on gene expression. Finally, what would the overall findings of this paper be in the animals were but on a high carbohydrate or high fat/carbohydrate diet?
9. (10) In the paper *Postprandial Metabolite Profiles Reveal Differential Nutrient Handling after Bariatric Surgery Compared With Matched Caloric Restriction*, the overall conclusion was that “a greater disposal of glucose and AAs and more complete β-oxidation of fatty acids are early, and positive changes in metabolic flexibility [found] post-RYGB surgery”. Which pieces of information from the following data support their conclusion? Using your knowledge about the digestion system, give a rational for why this may be.

| TABLE 1. Plasma Metabolites in Overnight Fasted Subjects and 2 Hours After a MMT Test |
|-----------------------------------------------|---------------|----------------|---------------|----------------|---------------|----------------|
|                                | Fasting (% Change) | CR | RYGB Surgery | \(^1P\) | CR | RYGB Surgery | \(^2P\) |
| Cys AAs                         | -10.65 ± 5.07* | -18.93 ± 5.48* | 0.012 | 1.09 ± 8.62* | -23.94 ± 7.15* | 0.040 |
| Glutamine                       | 5.27 ± 6.57* | 22.71 ± 7.06* | 0.059 | -1.77 ± 6.47* | -21.20 ± 6.48* | 0.054 |
| Leucine                         | 1.80 ± 5.66* | -1.72 ± 5.99* | 0.863 | 3.31 ± 7.46* | -19.93 ± 6.85* | 0.016 |
| Methionine                      | -3.66 ± 4.97* | -1.75 ± 2.97* | 0.027 | -4.32 ± 2.37* | -7.27 ± 4.91* | 0.011 |
| Valine                          | -2.07 ± 6.98* | -1.50 ± 5.24* | 0.143 | -1.63 ± 4.73* | -12.74 ± 6.99* | 0.013 |
| Proline                         | 1.52 ± 3.06* | -8.75 ± 3.87* | 0.039 | -2.71 ± 3.73* | -21.20 ± 6.79* | 0.045 |
| Phosphate                       | 1.03 ± 3.01* | -16.62 ± 2.56* | 0.001 | 2.59 ± 4.02* | -21.82 ± 2.38* | 0.001 |
| Taurine                         | 1.37 ± 7.57* | -9.12 ± 6.96* | 0.262 | 3.48 ± 5.85* | -16.64 ± 3.77* | 0.112 |
| Aspartame             | 5.10 ± 6.61* | 10.59 ± 8.35* | 0.048 | 17.69 ± 6.47* | 33.46 ± 6.17* | 0.219 |
| Glutamine-glutamine           | -4.79 ± 3.96* | 9.55 ± 4.96* | 0.105 | 1.05 ± 6.74* | 9.20 ± 12.32* | 0.385 |
| Arginine                       | -0.14 ± 4.81* | -1.04 ± 5.86* | 0.080 | 2.45 ± 5.90* | -17.67 ± 5.07* | 0.022 |
| BCAAs                           | -0.36 ± 4.16* | -12.67 ± 4.17* | 0.039 | -1.85 ± 4.83* | -19.65 ± 6.27* | 0.027 |
| Aromatic AAs                 | 0.30 ± 4.50* | -13.52 ± 3.42* | 0.029 | 2.34 ± 4.81* | -21.30 ± 2.30* | 0.001 |
| Total AAs                        | -3.36 ± 3.79* | -6.75 ± 2.70* | 0.401 | 1.39 ± 4.67* | -13.83 ± 2.22* | 1.017 |
| Aminoacids                        | C2            | 49.79 ± 14.55* | 75.39 ± 17.15* | 0.220 | 25.39 ± 10.16* | -50.92 ± 18.02* | 0.273 |
| C2 + C3                        | 71.04 ± 5.37* | 20.26 ± 7.13* | 0.010 | -7.69 ± 5.14* | -16.47 ± 9.30* | 0.762 |
| Medium-chain                  | 45.10 ± 10.31* | 7.21 ± 8.61* | 0.031 | 36.14 ± 14.11* | 9.15 ± 7.66* | 0.073 |
| Long-chain                    | 31.73 ± 4.11* | 50.01 ± 6.35* | 0.059 | 29.13 ± 5.47* | 33.17 ± 14.63* | 0.768 |
| Total aminoacids              | 41.70 ± 8.29* | 58.58 ± 13.06* | 0.290 | 22.48 ± 7.69* | 38.52 ± 14.14* | 0.356 |
| Urea cycle products           | Ornithine      | -4.06 ± 7.11* | -7.36 ± 5.12* | 0.794 | -7.32 ± 4.44* | -13.90 ± 5.38* | 0.178 |
|                          | Gln            | 8.93 ± 9.57* | -1.87 ± 9.93* | 0.446 | -0.67 ± 9.81* | -14.91 ± 10.16* | 0.809 |
| Free fatty acid and products   | NEFA           | 49.50 ± 10.63* | 27.9 ± 11.06* | 0.271 | 35.2 ± 18.11* | 29.0 ± 22.51* | 0.083 |
|                          | Total ketones   | 32.19 ± 79.5* | 56.4 ± 22.91* | 0.279 | 40.6 ± 53.8* | 142.4 ± 62.93* | 0.229 |
|                          | 3β-hydroxybutyrate | 319.3 ± 288.9* | 220.9 ± 288.4* | 0.239 | 34.2 ± 48.6* | 144.6 ± 102.4* | 0.180 |
|                          | Glucose        | -15.1 ± 5.4* | -9.4 ± 11.2* | 0.956 | -9.1 ± 7.6* | -24.1 ± 8.3* | 0.191 |

Data are expressed as percentage change postprandial relative to preprandial for the CR or RYGB surgery group. Data are presented as mean ± standard error. \(^1P\) and \(^2P\) are values for comparison between changes occurring as a result of RYGB surgery vs CR, adjusted for differences in body weight.

\(^\ast\) p < 0.05 for differences between post CR or pre and post RYGB surgery.

Total AAs indicates the sum of all amino acids measured; BCAAs, sum of branched chain amino acids (Val + Leu + Ile); Aromatic AAs, sum of the aromatic amino acids (Phe + Tyr). The \(*\) indicates statistical significance.
10. In the paper *A Role for Mitochondrial Phosphoenolpyruvate Carboxykinase (PEPCK-M) in the Regulation of Hepatic Gluconeogenesis*, they clearly demonstrate that PEPCK-M knock down results in changed endogenous glucose production during the fed state. Using the following data, explain what the increased Glycerol and decreased Lactate levels indicate regarding gluconeogenesis in the knock down animals.
The paper *Artificial sweeteners induce glucose intolerance by altering the gut microbiota* is a recent “hot topic”. Using the data presented below, explain the findings of the paper. Using what you have learned regarding fiber, bacteria and energy metabolism, propose a model for what may be happening in the body.
Quiz

NDFS 435

Name_____________________________

Quiz #6: Protein Part 3

1. (1) Ketogenic amino acids are catabolized to:
   A. Acetyl CoA
   B. Acetoacetate
   C. Pyruvate
   D. Succinyl CoA
   E. Both A and B
   F. All of the above

2. (1) Match the nitrogen-containing non-protein compound with its function.
   ___ Glutathione   A. High energy bonds. Can make ATP.
   ___ Carnitine     B. Methyl Donor
   ___ Creatine      C. Major antioxidant
   ___ Choline       D. Fatty acid transporter

3. (.5) Which vitamin is required for transamination reactions?
   A. Vitamin C (ascorbic acid)
   B. Folate
   C. Vitamin B12 (cobalamin)
   D. Vitamin B6 (PLP)

4. (1) What is the purpose of the urea cycle?

(.5) True / False: Several amino acids including glutamine and glycine are involved in purine synthesis.

6. (1) Name two hormones involved in protein metabolism: ONE that stimulates anabolism and ONE that stimulates catabolism.

   Anabolic hormone:

   Catabolic hormone:
**Chapter Study Guides**

Chapter 6 Study Guide

**SETHRINA DUNLAP**

A. Amino Acid Classification

1) Structure
   i. What is the basic structure of an amino acid, what makes them unique
   
   \[
   \text{H}_2\text{N-CH-COOH} \text{ with R group attached to CH}
   \]
   
   Central carbon at least one **amino group** (\(\text{NH}_2\)) at least one **carboxy group (acid) group** (\(\text{COOH}\)) and a side chain (\(\text{R}\))

2) Net Electrical Charge
   i. Define **zwitterion**
      
      Dipolar ion with no carboxy or amino groups in their side chain to add additional charge to the molecule.
   
   ii. Which amino acids are negatively charged? Which are positively charged? Why?
      
      Due to the presence of an additional carboxy group in the side chain the dicarboxylic amino acids are **negative**: aspartic (aspartate) and glutamic (glutamate).
      
      Due to presence of an additional amino group in the side chain produce **positive** amino acids: lysine, arginine, histidine.
      
      Both neg and positive have these qualities at a pH of 7.0.

3) Polarity
   i. Which amino acids are polar, and which are nonpolar. How does this effect the location and function of the amino acids?
      
      **NonPolar**: Alanine, glycine, isoleucine, leucine, methionine, proline, valine within central region or core portion of protein b/c hydrophobic.
      
      **Polar**: serine, threonine, cysteine, asparagine and glutamine.
      
      Interact with aqueous environment and form salt bridges and interact with electrolytes and minerals. Surface of proteins or facing inward at proteins binding site. Interact with hydrogen bonds in water.

4) Essentiality
   i. Which amino acids are essential? Which are conditionally indispensable? What does conditionally indispensable mean?
      
      **Essential**: Body cannot make the nutrient that must be supplied by the diet.
      
      Phenylalnine, Valine, Threonine, Methionine, Tryptophan, histidine, Isoleucine, Leucine, Lysine.
      
      **Conditionally Indispensable**: organ failure or improper function.
      
      Tyrosine, cysteine, proline, arginine, glutamine

B. Sources of amino acids
1) What are the two primary sources of proteins presented to the digestive tract?
   - Animal products
   - Plant products

C. Digestion
   1) Stomach
      i. What is the purpose of HCl in terms of protein digestion
         - The low pH of HCl allows for denaturing of proteins in the stomach.
      ii. How does pepsin function?
         - Breaks the peptide bonds in the protein

2) Small Intestine
   i. Which hormones and regulatory peptides are secreted? What are their functions?
      - Secretin and CCK stimulate the pancreas to secrete juices.
      - What are zymogens? Which are secreted from the pancreas?
      - How are they activated? What are their functions?
      - Inactive enzymes that help in digestion, will need to be activated to do protein hydrolysis.
      - Trypsinogen: carboxy end of basic aa
      - Chymotrypsinogen: carboxy end of aromatic aa
      - Procarboxypeptidases A&B: exopeptidases
      - All are activated by trypsin except trypsinogen which is activated by enteropeptidase.
   ii. Which peptidases are produced by enterocytes? Where are these peptidases located?
      - Location: Ileum
      - Aminopeptidases
      - Dipeptidylaminopeptidases
      - Tripeptidases

D. Absorption
   1) Intestinal Brush Border Membrane Absorption
      i. Where are most amino acids absorbed?
      - Deuodenum and upper jejunum
      ii. How are amino acids absorbed?
      - Using carriers, a little paracellular
      iii. What mechanisms do amino acid transporters use?
      - Passive: uniporters, exchangers.
      - Active: ion gradient
      - Most: sodium dependant transporters
      iv. Describe sodium dependent amino acid transport
      - Sodium first binds to carrier which increases affinity for aa.
      - (For some aa this process is reversed). This produces a conformational change and results in delivery of sodium, and aa into cytosol. Na is pumped out by Na/K ATPase.
v. What affects the speed of amino acid absorption? Describe the mechanism of peptide absorption.

Affinity by hydrocarbon mass of aa side chain and net electr. Charge of aa. As mass increases affinity increases. Branched aa are generally absorbed faster than smaller aa. Neutral highest absorbance.

2) Intestinal Basolateral Membrane Transport

i. Describe how amino acids leave the enterocyte

Sodium independent transporters same as transporters found on membrane of nonepithelial cells.

3) Amino Acid Absorption into Extraintestinal Tissues

i. Describe how the γ-glutamyl cycle transports amino acids into cells (synthesis (include locations, metabolites, enzymes, etc.)

Gluthathine acts as carriers of neural amino acids into cells. Glutathione reacts with γ-glutamyl transpeptidates located in cell membranes forming γ-glutamyl enzyme complex, which binds a neutral amino acid at the cell surface and transports it into the cytosol for use.

E. Amino Acid Metabolism

1) Amino Acid Catabolism Overview

i. Where does most amino acid catabolism occur?

Liver.

ii. Define transamination. Transfer or removal of an amino acids amino group.

What are the necessary substrates for transamination? Amino acid carbon skeleton and amino acid group that become alpha keto acid.

What are the products? What enzyme class is involved?

Enzymes: aminotransferases and transaminases

Products: alpha keto acid and amino acid 2

What coenzymes are needed?

Vitamin B6 pyridoxal phosphate

What are the two most common aminotransferases?

Tyrosine and aminotransferase (ALT)

iii. Define deamination. Only removal of amine group from another aa with NO transfer of an aa group to another compound.

What are the necessary substrates for deamination?

Amino group and lyases

What are the products?

Alpha keto acid and ammonia or ammonium ion

What enzyme class is involved?

Lyases: dehydrateses or dehydrogenases

iv. What are the sources of ammonia in the body?

The body’s pH and ammonia converted into ammonium ion due to deamination of aa. Generation by deamidation of amide
groups from glutamine and asparagine. Ingestion of food (cheese, processed meats). Generation from bacterial lysis and aa of GI tracts

v. Describe how glutamate and glutamine synthesis remove free ammonia from the body.
Glutamate dehydrogenases use ammonia or ammonia ion and alpha ketoglutarate to make aa glutamate.
Glutamine synthesis uses ammonia or ammonia ion for the amidation of glutamate’s gamma carboxy group to form glutamine in an ATP dependent reaction that also requires magnesium.

vi. Starting with ammonia in the liver, describe how urea is produced and how the urea cycle interacts with the TCA cycle (include locations, metabolites, enzymes, etc.)
- \( \text{NH}_3 + \text{CO}_2 \) form carbomoyl phosphate catalyzed by CPSI using two moles of ATP and Mg2+ and NAG (made in liver).
- Then carbamoyl phosphate reacts with ornithine in mitochondria, using OTC to form citrulline. Citrulline inhibits OTC activity.
- Apsartate reacts with citrulline once it has made it to the cytosol. This is rate limiting step catalyzed by arginine succinate synthetase. ATP and Mg2+ are required for the reaction and argininosuccinate is formed. Argininosuccinate and arginine and AMP+ PPi inhibit enzyme.
- Argininosuccinate is cleaved by argininosuccinase in cytosol to form fumarate and arginine. Both fumarate and argigine inhibit argininosuccinate activity.
- Urea is formed and ornithine is reformed for the cleavage of arginine by arginase. May be rate limiting because needs manganese for manganes requiring hepatic enzyme.

vii. What can the carbon skeletons be used for?
Energy, glucose, ketone bodies, cholesterol, fatty acids

2) Hepatic Catabolism and Uses of Aromatic Amino Acids
i. **Phenylalanine and Tyrosine:** What cofactors are necessary for phenylalanine conversion to tyrosine? Phenylalanine hydroxylase, iron, vitamin C and tetrahydrobiopeterin
What process is necessary for tyrosine degradation?
Transamination by vit B6 dependant tyrosine amino transgerase
What carbon skeleton is needed? \( P\text{-hydroxyphenylpyruvate} \)
What are the metabolic products from this pathway? Fumarate and acetoacetate
Is this process glucogenic, ketogenic or partially ketogenic and glucogenic? Why?
Partially glucogenic b/c degraded to fumarate and partially ketogenic because phenylalanine and tyrosine are catabolized to acetoacetate.

ii. **Tryptophan:**
What cofactors are necessary for degradation?
Tryptophan dioxygenase heme iron induced by glucagon as well as cortisol.
What are the metabolic products from this pathway?
B vitamin niacin, NAD and NADP
Is this process glucogenic, ketogenic or partially ketogenic and glucogenic? Why?
Partially glucogenic b/c catabolized to form pyruvate also partially ketogenic b/c forms acetyl CoA

3) Hepatic Catabolism and Uses of Sulfur Containing Amino Acids

i. **Methionine:**
What cofactors are necessary for degradation?
SAM by methionine adensyl transferase and ATP
What are the metabolic products from this pathway?
Cystine, alpha ketobutyrate
Is this process glucogenic, ketogenic or partially ketogenic and glucogenic? Why?
Glucogenic b/c catabolized form succinyl CoA

ii. **Cysteine:**
What cofactors are necessary for degradation?
Taruine B amino sulfonic acid
What are the metabolic products from this pathway?
Pyruvate and sulfite
Is this process glucogenic, ketogenic or partially ketogenic and glucogenic? Why?
Glucogenic b/c produces pyruvate and sulfite

4) Hepatic Catabolism and Uses of Basic Amino-Acids

i. **Lysine:**
What cofactors are necessary for degradation?
SAM, alpha ketoglutarate
What are the metabolic products from this pathway?
Alpha ketoadipase
Is this process glucogenic, ketogenic or partially ketogenic and glucogenic? Why?
Ketogenic b/c makes acetyl CoA

ii. **Arginine:**
What cofactors are necessary for degradation?
Alpha ketoglutarate
What are the metabolic products from this pathway? Urea, ornithine (decarboxylated to form putrescine, spermine and spermidine) or proline via transamination
Is this process glucogenic, ketogenic or partially ketogenic and glucogenic? Why?
Glutogenic b/c generates TCA cycle intermediates

iii. Histidine:
What cofactors are necessary for degradation?
Beta alanine, bit B6
What are the metabolic products from this pathway?
Alpha ketoglutarate
Is this process glucogenic, ketogenic or partially ketogenic and glucogenic? Why?
Glucogenic b/c produces alpha ketoglutarate

5) Hepatic Catabolism and Uses of Other Amino Acids
i. Threonine:
What cofactors are necessary for degradation?
Cytosolic threonine dehydratase
What are the metabolic products from this pathway?
Alpha ketobutyrate, propionyl coa the d methymalonyl coa and l meth-ylmalonyl coa and then succinyl COA
Is this process glucogenic, ketogenic or partially ketogenic and glucogenic? Why?
Glucogenic and ketogenic b/c metabolized by three different pathways.

ii. Glycine and Serine:
What cofactors are necessary for degradation?
Folate (THF)
What are the metabolic products from this pathway?
glycine and serine, glutathione, creatine, porphyrrins bile salt glycocholate, ethanolamine, choline for phospholipids
Is this process glucogenic, ketogenic or partially ketogenic and glucogenic? Why?
Unclear

F. Functional Roles of Proteins and Nitrogen-containing Nonprotein Compounds
1) Nitrogen-Containing Nonprotein Compounds
   i. Glutathione: What are its components, what is needed for its synthesis (enzymes, cofactors, etc.), what are its functions.
      -Components: glycine, cysteine, glutamate tripeptide
      -Synthesis: must have available cysteine for GSH synthesis, contains sulfur
      -Function: antioxidant, transporter of aa’s in gamma-glutamyl cycle, part of synthesis of leukotriines, part of prostaglandin conversion
ii. **Carnitine**: What are its components, what is needed for its synthesis (enzymes, cofactors, etc.), what are its functions.
   - **Components**: methylated & hydroxylated lysine
   - **Synthesis**: cofactors are vit B₆, vit C, niacin
   - **Function**: stored in muscle (though not synthesized there), needed for fatty acid transport across inner mitochondrial membrane, needed for ketone catabolism for energy

iii. **Creatine**: What are its components, what is needed for its synthesis (enzymes, cofactors, etc.), what are its functions.
   - **Components**: arginine + glycine form guanidinoacetate
   - **Synthesis**: occurs in kidneys. Amidinotransferase catalyzes it. Needs SAM as methyl donor to guanidinoacetate
   - **Function**: mostly in muscles as phosphocreatine. A storehouse for high-energy phosphate – replenishes ATP in a muscle that is rapidly contracting.

iv. **Carnosine**: What are its components, what is needed for its synthesis (enzymes, cofactors, etc.), what are its functions.
   - **Components**: histidine + β-alanine
   - **Synthesis**: energy-dependent rxn by carnosine synthetase
   - **Function**: mostly in skeletal & cardiac muscle. May act as a buffer & antioxidant in cells. May reduce calcium for muscle contractility.

v. **Choline**: What are its components, what is needed for its synthesis (enzymes, cofactors, etc.), what are its functions.
   - **Components**: methylated serine. Found in foods usually as lecithin (a phospholipid)
   - **Synthesis**: SAM as methyl donor to serine
   - **Function**: methyl donor, helps form platelet aggregating factor, helps secretion of VLDL from liver. Part of sphingomyelin, phosphatidyl choline (lecithin), & neurotransmitter acetylcholine

vi. **Pyrimidine bases**: What are its components, what is needed for its synthesis (enzymes, cofactors, etc.), what are its functions?
   - **Components**: uracil, cytosine, thymidine. Six-membered rings containing nitrogens at 1 & 3.
   - **Synthesis**: building blocks for DNA & RNA
   - **Function**: building blocks for DNA & RNA

vii. **Purine bases**: What are its components, what is needed for its synthesis (enzymes, cofactors, etc.), what are its functions?
   - **Components**: Adenine, guanine. Two fused rings w/ N's at 1, 3, 7, 9.
   - **Synthesis**: in liver. Requires 2 ATP, glutamine, aspartate
   - **Function**: building blocks for DNA & RNA

**G. Interorgan “flow” of Amino Acids and Organ-Specific Metabolism**

1) **Intestinal Cell Amino Acid Metabolism**

i. **Glutamine:** What is it used for in the enterocyte?

ii. **Glutamate:** Where does it come from, and what is it used for in the enterocyte?
   - Either comes from diet or from glutamine metabolism
   - Can be transaminated with pyruvate to form α-KG & alanine, or can be used to make glutathione or can go to proline (which goes to circulation), or can make ornithine when combined with arginine
   - Very little glutamate actually goes into circulation as glutamate

iii. **Aspartate:** What happens to aspartate in the enterocyte?
   - Transaminated to oxaloacetate, leaving the amino group to form ornithine (very little asp goes into circulation as asp)

iv. **Arginine:** How is the arginine used by the enterocytes? What happens to its products?
   - Oxidized to citruline & urea. The NH₃ can be used for carbamoyl phosphate synthesis. Citruline is absorbed, then goes to kidneys for arginine synthesis.

v. **Methionine:** What is methionine used for in the enterocytes?
   - Can form cysteine, which can then form glutathione. Can be metabolized to taurine, or to pyruvate + sulfite.

2) **Amino acids in the plasma**
   i. How large is the plasma amino acid pool? Which types of amino acids make up most of the pool?
   - ~150 g. Mostly made up of nonessential amino acids (alanine, glutamate, aspartate, glutamine), & some essential (lysine, threonine)

3) **Glutamine and the Muscle, Intestine, Liver and Kidneys**
   i. What is the primary purpose of glutamine in muscle, lungs, heart, etc?
   - Ammonia transport. (Glutamine synthetase catalyzes the utilization of ammonia w/ glutamate to form glutamine)

   ii. Describe the process of ammonia transfer and release from the muscle.
   - Glutamate generated in muscle when BCAAs are transaminated w/ α-KG
   - Some glutamate deaminated to α-KG + ammonia
   - Ammonia formed from AMP deamination (AMP generated in muscle from ATP degradation, especially during exercise)
Glutamine synthesis catalyzes the formation of glutamine from ammonia & glutamate

iii. How is glutamine used in the liver? How is it used in the kidneys?
- In the absorptive state (or during alkalosis), liver glutaminase activity increase, yielding NH4 for urea cycle
- In the acidotic state, glutamine for urea diminishes, & the liver releases glutamine into the blood for transport to & uptake by the kidneys for acid-base balance.

4) Alanine and the Liver and Muscle
i. Describe the alanine-glucose cycle during periods of low circulating glucose levels.
- In between meals, with excessive glucose need, during illness, or during low glycogen (eg: fasting) states, glutamate transfers its amino group to pyruvate forming a-KG+ alanine.
- This alanine is then released from the muscle to go to liver, where it is transaminated back to pyruvate, which can be used to remake glucose. (That also produces a glutamate, which can be deaminated to provide NH4 for urea synthesis)

5) Skeletal Muscle
i. Describe Branch Chain Amino Acid catabolism in the muscle. What cofactors are needed by BCKAD? Which BCAA is glucogenic and which is ketogenic?
- Catabolism of BCAAs: branched-chain aminotransferases transaminate the amino acids, leaving a-keto acids (which can be further oxidized in the muscle or go into circulation)
- BCKAD (branched chain a-keto acid dehydrogenase): 3 subunits; needs thiamin, niacin, Mg, & pantothenic acid (CoA)
- Valine is glucogenic (goes to succinyl-CoA). Isoleucine is glucogenic (succinyl-CoA) & ketogenic (acetyl-CoA). Leucine is ketogenic (acetyl-CoA & acetoacetate).

ii. What are the indicators used to measure muscle mass and muscle degradation?
- Creatinine (muscle mass) & 3-methylhistidine (degradation)

6) Kidneys
i. What are the kidneys’ roles in amino acid metabolism?
- Glutamine & glycine catabolism for acid-base balance
- Serine synthesis from glycine
- Arginine & glycine use to form guanidinoacetate for creatine
- Glutathione catabolism
- Arginine synthesis from citruline
- Tyrosine synthesis from phenylalanine
- Histidine generation from carnosine degradation
- Synthesis of arg, his, ser

7) Brain and Accessory Tissues
i. How does the brain acquire amino acids?
   - Active transport for neutral, basic, & acidic aa’s (most of them are fully saturated at normal plasma concentrations)

ii. Which amino acids function as neurotransmitters? What are their functions?
   - Glycine: in spinal cord as inhibitory neurotransmitter
   - Taurine: probably an inhibitory neurotransmitter
   - Aspartate: (from glutamate thru asp aminotransferase in neural tissue), excitatory neurotransmitter in the CNS
   - Glutamate: in brain & spinal cord, excitatory
     * Can also be decarboxylated to GABA, inhibitory

iii. What biogenic amines are produced in the brain? What are their starting points?
   - Tryptophan: used to synthesize serotonin, excitatory neurotransmitter (biogenic amine) in the CNS, & a vasoconstrictor in circulation, & stimulator of smooth muscle contraction.
   - Tyrosine: used in sympathetic neurons to make catecholamines (dopamine-behaviors, movement, norepinephrine-alertness, sleep, epinephrine-low in brain, but hormone/nutrient metabolism effects in circulation)
   - Histidine: decarboxylated to histamine, which is involved in attention, alertness, & many more roles.

iv. What are the differences between neuropeptides and neurotransmitters? Where are they expressed and how are they secreted?
   - Neuropeptides have more diverse effects. Derived from aa’s but not necessarily biogenic amines. Can be hormone-releasing factors, have endocrine effects, or modulatory action on transmitter functions, mood, or behavior.

v. How does the brain use Glutamate? How does the brain use Leucine?
   - Glutamate: helps get rid of ammonia in the brain (glucose in brain metabolized to a-KG, which can go to glutamate, then if there’s excess NH4, glutamine can be interconverted with glutamate to get rid of it)
   - Leucine: provide nitrogen to the brain. Leucine is taken up & aminotransferases remove its amino group for synthesis of glutamate & glutamine.

H. Catabolism of Tissue Proteins
1) Describe lysosomal degradation, how does it occur and what is being degraded?
   - Proteins from all over are endocytosed into the cell & stored in endosomes, which fuse w/ lysosomes. Lysosomes have digestive enzymes that are active w/ a low pH, so they have a proton pump to achieve acidity. Once active, no energy is needed for lysosomal
digestion. The aa's released afterward can be reused for protein synthesis or degraded, depending on cell needs

2) Describe Proteasomal degradation, how does it occur and what is being degraded?

-Proteasomes (protease systems/complexes) have large cavities for protein degradation. Proteins are marked for degradation by ligation to ubiquitin, a polypeptide. ATP required to unfold proteins, then ubiquitin added, then proteins degraded.

I. Perspective: The impact of starvation, metabolic stress, and low-grade inflammation on body proteins and protein metabolism.

1) What occurs during starvation? What signals are produced, what signals are shut off, what energy is used?

- Protein synthesis decreases (due to decreased mRNA needed for protein translation & decreased peptide bond formation).
- Protein degradation also decreases to avoid nitrogen losses.
- Hormone concentrations change: insulin drops (& muscles/fat become resistant to it, to prevent nutrient storage), glucagon & epinephrine increase, cortisol increases (promotes fatty acid mobilization, ketone production & proteolysis)

- First few days: glycogen depleted in liver. Muscle proteolysis (releases alanine a lot, which stimulates (thru glucagon) & is a substrate for gluconeogenesis). Glucose made from lactate & pyruvate (Cori cycle). Glutamine from muscles circulates for uptake & metabolism (mainly by gastrointestinal tract & kidneys)

- Continued fasting: continues use of fatty acids & glucose, but also ketone body production in liver from fatty acid oxidation. This allows protein catabolism & gluconeogenesis to decrease. This creates acidosis, which directs glutamine to kidneys (& away from other organs that would use it as fuel) for acid-base balance (amino group produces NH4, carbon skeleton produces glucose in kidney).

2) What occurs with metabolic stress? What signals are produced, what signals are shut off, what energy is used?

- Metabolic stress = hypermetabolic, catabolic state in response to sepsis, injury, trauma, or some diseases.
- Leads to degradation of body tissues: adipose undergoes lipolysis (but no ketones, b/c ketogenesis inhibited by insulin). So, body proteins degraded to use amino acids for glucose production.

- Hormones: glucocorticoids (cortisol), epinephrine, insulin, & glucagon release increase...but tissues become resistant to insulin & hyperglycemia is present despite insulin presence. Cortisol levels in blood are elevated, promoting proteolysis & hyperglycemia.
Syllabus

NDFS 435 - Nutritional Biochemistry
Fall 2014
Section 001: 1002 JKB on M W from 9:00 am - 10:50 am

Instructor/TA Info
Instructor Information
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Wed 11:00am-1:00pm
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Course Information
Description
In NDFS 435 Nutritional Biochemistry, we will learn about the macronutrients, vitamins and minerals that are essential for life. We will learn how the body digests, transports, stores and excretes these nutrients. We will focus on the metabolic pathways where these nutrients function, and the biochemical manner by which they are essential for life. We will apply these biochemical and metabolic functions to disease states that correlate with over-nutrition, under-nutrition and genetic deficiencies.

Prerequisites
NDFS 200, PDBio 305, Chem 481 or equivalent.

Materials

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Learning Outcomes
**Scientific Research and Writing**
Demonstrate ability to search, interpret, and summarize original scientific information in an efficiently written scientific paper.

**Nutrient Chemical Structure and Metabolism**
Demonstrate knowledge of nutrient chemical structures, food sources, digestion, absorption, transport, metabolism and functions, and the metabolic consequences of nutrient deficiencies, interactions, imbalances and toxicities.

**Experimental Design**
Demonstrate knowledge of appropriate design for scientific experiments.

**Biochemical Functions and Pathways**
Understand the biochemical functions and pathways of nutrients in the body.

**Nutrient Metabolism and Body Homeostasis**
Identify physiological, biochemical and metabolic changes associated with deficient nutrient intake, excessive nutrient intake and selected metabolic diseases.

**Grading Scale**

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**Grading Policy**
Grades for chapter study guides, quizzes, literature writing prompts, case-study, research paper, midterms and final examination will contribute to a total of 1000 points. More information on each of the individual assignments can be found below. Make-ups will be allowed ONLY for those having a justifiable excuse (my discretion) or who made previous arrangements with me AND have unavoidable circumstances. The only extra credit for this course is completion of the end of course evaluation, which is worth 10 points.

**Chapter Reading Guides**—There are 13 chapter reading guides, each worth 5 points. These will all be due the first day that we discuss a chapter. Prior to class you should read the chapter and answer the questions on the reading guide. These are due prior to 9am submitted on learning suite. Any assignment submitted after 9am will lose 10% from the total pre-graded score for each 24 hour period (i.e. if you turn in the assignment at 9:30 am, you have lost 10%, if you turn the assignment in at 9:30 am the next day then you have lost 20%). Three to four chapter reading guides will be graded at random from the class for each assignment—if yours is not graded you will receive full points for having it completed and turned in on time.
Quizzes-There will be 12 quizzes given at random throughout the semester, each worth 5 points. These quizzes will be on the days assigned chapter. You must be in attendance to take the quiz. If you are not in class when the quiz is given you will not be able to take the quiz. Your lowest 2 quizzes will be dropped.

Class participation-You will earn 50 points through the year from class participation. These points will be earned from my evaluation of your participation and preparedness and your group members assessment. You can lose points by not being in attendance, by leaving class early, by not helping with the discussions, etc.

Literature writing prompt-We will discuss on each Wednesday a recent peer-reviewed primary literature article that is relevant to our current topic. At the end of the in class discussion, you will be given a writing prompt. You should use the article and any other relevant data that you can find from peer-reviewed articles (PubMed) to complete the one page, 12 point font, double spaced prompt (worth 5 points per prompt). These assignments fulfill three purposes-1) you will become comfortable reading the primary biochemical and metabolic literature, 2) it will prepare you for the exam where you will see some of the same figures, and 3) you will improve your ability to write as a scientist.

Case study-You will be required to develop and present a case study to the class during the semester, worth 50 points. The presentation will take no more than 10 minutes. Based around a principle of metabolism for the nutrient you choose, you will develop a story around a scenario. Detailed instructions and examples are provided in a separate document.

Research Paper-You will complete a 4-5 page, 12 point font, double spaced, research paper. This research paper will focus on the biochemistry of nutrients. You must use a minimum of four peer-reviewed primary literature articles to define the question and then propose a study to address the question. No text books, meta-analysis, or reviews can be used as references. Your topic must first be approved by me. The following benchmarks will give you different levels of points: complete a library assignment with Dr. Nelson (10 points), abstract and reference review (25 points), rough draft for peer review (25 points), peer review (30 points), TA review (15 points), final paper (100 points). Detailed instruction and examples are provided in a separate document.

Exams-There will be four mid-term exams, each worth 100 points. Each will be half multiple choice and half short answer. There will be one extra credit question on each of the four mid-term exams. Exam 1 will cover chapters 1-4, Exam 2 will cover chapters 5-7, Exam 3 will cover chapters 9-10, and Exam 4 will cover chapters 11-14. All midterm exams will be given in the testing center. A comprehensive final examination will be given in class on December 17th, from 11:00am-2:00pm. If you are satisfied with your grade going into the final, you do not need to take the final. You will need to let me know your decision by Tuesday December 16th at noon. If your final exam grade is greater than the average of the four midterms, I will set this grade as your exam average grade.

Extra credit-There will be only one form of extra credit. Completion of the end of year evaluation will give you 10 points.

Participation Policy
Participation is expected. We will frequently be working in groups during the semester. Your group will be fixed from the beginning of the semester. Fifty points of your grade will come from class participation. Those points will come from my assessment of your participation and your peers assessment.

**Attendance Policy**

Attendance is expected. Much of what will be on the examinations will be discussed in class. Group work will occur in class. You will receive points for in class participation. It is in your best interest to be in class. Students that do not attend class more than 75% of the time usually do not earn a grade greater than a C. To help you attend class, twelve quizzes will be given throughout the semester. Your lowest two quizzes will be dropped at the end of the semester, thus allowing you to miss two quizzes without it affecting your grade. If you are absent on a day of a quiz and you have not coordinated with me prior to the beginning of class, you will not be allowed to take the quiz. Quizzes will be given at any point during the two hour lecture period.

**Classroom Procedures**

In class schedule

Our classroom procedures will be primarily based around questions that you as students bring to class regarding the reading. We will discuss the key points, and you will be given the opportunity to teach each other. We will be working in groups, and as such you will need to stay with your group for the duration of the semester. In addition, each Monday we will end the lecture/discussion at 10:40 am for a 10 minute case study presentation. There will be exam questions that come from the case studies. On Wednesday, the lecture/discussion on the text will end at 9:50 am. After a 10 minute break, we will begin a discussion of the assigned primary literature paper. This will be the basis of the week’s writing prompt due by Saturday night (turned in on Learning Suite, with the exception of the final prompt, due the last day of classes). Furthermore, questions from these current literature will be present on all exams. Quizzes will be given throughout the semester, at any time during the two hour lecture period.

Questions

If you have questions about grades, points, assignments, due dates, etc. it is in your best interest to contact the TA first. The TA will be able to respond more rapidly, and if it is something that the TA can not deal with, then it will be passed on to me. I will deal with all policy issues. Everything else should go to the TA first.

Consultation

My office hours are Tuesday, Thursday, and Friday from 9:30-10:30am. Please come and visit with me. If you have questions, these are the best times to find me. I will be available to discuss class lecture material, other aspects of the class, or to talk about life! It is better to come early to get clarification or help.

**Study Habits**

We will cover much information this semester. Here are some things that will help you in your study:

1. **Read and take notes on the text before coming to class**- The purpose of the Chapter reading guide is to try to help you find some of the key points from the reading. It will not cover everything. The best way that I have found to study from
this text is to outline the chapter. Focus on the key points—what pathways is the nutrient involved in, where does the nutrient come from, how is the nutrient digested, how is it transported, where is it stored, how is it excreted, why is it necessary, what chemical reactions is it involved in, what is its global function.

2. **Come to class with questions**—It is your responsibility to come with questions so our lecture can focus on things that you do not yet understand. If you do not have questions, that means you completely understand the topic and their is not need for the lecture.

3. **Pay attention to the areas focused on in lecture**—In addition to clarifying concepts where you have questions, lecture periods also let us revisit the most salient points. These will be things that are important for the exam.

4. **Study with a group**—You will be assigned to a group for discussions your first day. You are encouraged to find time to study together out side of class. The best way to learn a concept is to teach it. I encourage you to meet together, and teach each other (don’t just read back notes) the concepts that are emphasized in lecture. The students who do the best in this course study as a group one or two times a week.

5. **Put in enough time**—This is a four credit hour class. The rule of thumb is that for every hour spent in class you should spend three hours out of class preparing for the course. Based on that arithmetic, you should spend 12 hours a week preparing for this class. Reading the chapter once will not be sufficient. The most effective methods to learn include out of class preparation (reading), participation (hearing, speaking, writing, drawing, applying), practice (speaking, writing, drawing, applying), feedback, analysis, iteration and cooperation.

**Teaching Philosophy**

When I was a student a professor was seen as a "Sage on the stage". I believe that the professor plays a greater role as a "Guide on the side". My philosophy is that I am training scientists. As you graduate with a degree from BYU in the basic sciences, you should be the best example of a scientist. My goal is to help you think critically about all data. Memorization does not make a great scientist. The ability to apply concepts to solve new problems does. I believe that when goals are set high, individuals will stretch to reach those goals. As a graduate student, post-doc and faculty member at other Universities, I observed how in demand BYU graduates were. These graduates were always in the top of the class. They learned to stretch and to do hard things. I believe being a great scientist is within each of your grasps. My goal is for each of you to become that.

**Assignments**

**Assignment Descriptions**

**Pop Quiz 1**
Due: Wednesday, Sep 03 at 11:59 pm

**Chapter 1**
Due: Friday, Sep 05 at 9:05 am

**Writing Prompt 1-HF diet paper**
Due: Saturday, Sep 06 at 11:59 pm
HF diet and circadian rhythm paper

**Chapter 2**
Due: Monday, Sep 08 at 9:05 am

**Pop Quiz 2**
Due: Wednesday, Sep 10 at 11:59 pm

**Writing Prompt 2-Postprandial Metabolite Profiles**
Due: Saturday, Sep 13 at 11:59 pm

**Chapter 3**
Due: Monday, Sep 15 at 9:05 am

**Pop Quiz 3**
Due: Monday, Sep 15 at 11:59 pm

**Library Assignment**
Due: Wednesday, Sep 17 at 11:59 pm

**Library Assignment**
**Writing Prompt 3-A Role for Mitochondrial Phosphoenolpyruvate Carboxykinase**
Due: Saturday, Sep 20 at 11:59 pm

**Chapter 4**
Due: Wednesday, Sep 24 at 9:00 am

**Abstract and References due**
Due: Wednesday, Sep 24 at 11:59 pm

**Abstract and References due**

**Pop Quiz 4**
Due: Wednesday, Sep 24 at 11:59 pm

**Writing prompt 4-Artificial sweeteners induce glucose intolerance**
Due: Saturday, Sep 27 at 11:59 pm

**Exam 1-Chapters 1-4 and associated papers 9/25, 26, 27, 29 in testing center**
Due: Monday, Sep 29 at 12:59 am

Exam 1-Test opens 9/25 in the testing center. Closes 9/29 in the testing center. You should give your self about 2 hours.

**Chapter 5**
Due: Monday, Sep 29 at 9:00 am

**Writing Prompt 5-Cardiomyocyte specific loss of diacylglycerol acyl transferase 1**
Due: Saturday, Oct 04 at 11:59 pm

**Rough draft for peer review**
Due: Monday, Oct 06 at 11:59 pm
Turn in completed rough draft (1 abstract page, 2 data summary pages, 1 future experiment page, references on page 5). Paper can be between 4 and 5 pages without references.

**Pop Quiz 5**
Due: Monday, Oct 06 at 11:59 pm

**Chapter 6**
Due: Wednesday, Oct 08 at 9:00 am

**Writing Prompt 6**-Metabolic profiling of PPARα−/− mice reveals defects in carnitine
Due: Saturday, Oct 11 at 11:59 pm

**Peer Review of Research Paper**
Due: Monday, Oct 13 at 11:59 pm
Submit the reviews of your fellow students' Paper 1 drafts to the TA email.

**Pop Quiz 6**
Due: Monday, Oct 13 at 11:59 pm

**Chapter 7**
Due: Wednesday, Oct 15 at 9:00 am

**Writing Prompt 7**-A Branched-chain amino acid-related metabolic signature that differentiates
Due: Saturday, Oct 18 at 11:59 pm

**Chapter 9**
Due: Monday, Oct 20 at 9:00 am

**Exam 2**-Chapters 5,6,7. 10/16,17,18,20 in testing center.
Due: Monday, Oct 20 at 12:59 pm
Exam 2-Test opens 10/16 in the testing center. Closes 10/20 in the testing center. You should give your self about 2 hours.

**Pop Quiz 7**
Due: Monday, Oct 20 at 11:59 pm

**Writing Prompt 8**-Folate deficiency-induced oxidative stress
Due: Saturday, Oct 25 at 11:59 pm

**Chapter 10**
Due: Wednesday, Oct 29 at 9:00 am

**TA review of Research Paper**
Due: Wednesday, Oct 29 at 11:59 pm
TA review

**Writing Prompt 9**-Gla-Rich Protein is a Potential New Vitamin K Target
Due: Saturday, Nov 01 at 11:59 pm

**Pop Quiz 8**
Writing prompt 10-Treatment of Tumors with Vitamin E Suppresses Myeloid Derived suppressor Cells and

Due: Saturday, Nov 08 at 11:59 pm

Chapter 11

Due: Monday, Nov 10 at 9:00 am

Exam 3-Covers Chapters 9 and 10, 11/6,7,8,10 in Testing Center

Due: Monday, Nov 10 at 10:59 pm

Exam 3-Test opens 11/6 in the testing center. Closes 11/10 in the testing center. You should give your self about 2 hours.

Pop Quiz 9

Due: Monday, Nov 10 at 11:59 pm

Writing prompt 11-Remodeling of Channel-Forming ORAI proteins

Due: Saturday, Nov 15 at 11:59 pm

Chapter 12

Due: Monday, Nov 17 at 9:00 am

Pop Quiz 10

Due: Wednesday, Nov 19 at 11:59 pm

Final Research Paper Due

Due: Wednesday, Nov 19 at 11:59 pm

Research Paper is due-Must have the following parts: 1st page-abstract (1 page), Introduction, discussion of previous data, proposed experiment and experimental design (3-4 pages), references-up to 1 page (only primary literature. No reviews, not meta-analysis, no text books, no websites. Only peer reviewed primary literature). Also include 1-2 pages describing what your peer and TA review suggested you change, and what you did-point by point.

Writing prompt 12-Metformin Treatment of Diabetes Mellitus Increases the Risk

Due: Saturday, Nov 22 at 11:59 pm

Chapter 13

Due: Monday, Nov 24 at 9:00 am

Pop Quiz 11

Due: Monday, Dec 01 at 11:59 pm

Writing prompt 13-Distinct patterns of hepcidin and iron regulation

Due: Saturday, Dec 06 at 11:59 pm

Case Study

Due: Monday, Dec 08 at 12:00 am

Case Study

Chapter 14
Due: Monday, Dec 08 at 9:00 am

Exam 4-Chapters 11, 12, 13. 12/4, 5, 6, 8 in Testing Center.

Due: Monday, Dec 08 at 10:59 pm

Exam 4-Test opens 12/4 in the testing center. Closes 12/8 in the testing center. You should give your self about 2 hours.

Pop Quiz 12

Due: Monday, Dec 08 at 11:59 pm

End of year class evaluation

Due: Thursday, Dec 11 at 11:59 pm

Class participation

Due: Thursday, Dec 11 at 11:59 pm

Writing Prompt 14-Effects of vanadium (III,IV,V)-chlorodipidolinate

Due: Thursday, Dec 11 at 11:59 pm

Exam 5-Comprehensive Exam-11:00am-2:00pm

Due: Wednesday, Dec 17 at 2:00 pm

Comprehensive Final Exam

University Policies

I reserve the right to change this syllabus at any point as I see fit.

At times things change during the semester. I am a scientist, and I will change my methods based on the results. I reserve the right to change the class as I see fit throughout the semester. Please note that my goal is for you to succeed in this course. If I change the course, it is for that goal to be met.

Honor Code

In keeping with the principles of the BYU Honor Code, students are expected to be honest in all of their academic work. Academic honesty means, most fundamentally, that any work you present as your own must in fact be your own work and not that of another. Violations of this principle may result in a failing grade in the course and additional disciplinary action by the university. Students are also expected to adhere to the Dress and Grooming Standards. Adherence demonstrates respect for yourself and others and ensures an effective learning and working environment. It is the university’s expectation, and every instructor’s expectation in class, that each student will abide by all Honor Code standards. Please call the Honor Code Office at 422-2847 if you have questions about those standards.

Sexual Harassment

Title IX of the Education Amendments of 1972 prohibits sex discrimination against any participant in an educational program or activity that receives federal funds. The act is intended to eliminate sex discrimination in education and pertains to admissions, academic and athletic programs, and university-sponsored activities. Title IX also prohibits sexual harassment of students by university employees, other students, and visitors to campus. If you encounter sexual harassment or gender-based discrimination, please talk to your professor or contact one of the following: the Title IX Coordinator at 801-422-2130; the Honor Code Office at 801-422-2847;
the Equal Employment Office at 801-422-5895; or Ethics Point at http://www.ethicspoint.com, or 1-888-238-1062 (24-hours).

**Student Disability**

Brigham Young University is committed to providing a working and learning atmosphere that reasonably accommodates qualified persons with disabilities. If you have any disability which may impair your ability to complete this course successfully, please contact the University Accessibility Center (UAC), 2170 WSC or 422-2767. Reasonable academic accommodations are reviewed for all students who have qualified, documented disabilities. The UAC can also assess students for learning, attention, and emotional concerns. Services are coordinated with the student and instructor by the UAC. If you need assistance or if you feel you have been unlawfully discriminated against on the basis of disability, you may seek resolution through established grievance policy and procedures by contacting the Equal Employment Office at 422-5895, D-285 ASB.

**Academic Honesty**

The first injunction of the Honor Code is the call to "be honest." Students come to the university not only to improve their minds, gain knowledge, and develop skills that will assist them in their life’s work, but also to build character. "President David O. McKay taught that character is the highest aim of education" (The Aims of a BYU Education, p.6). It is the purpose of the BYU Academic Honesty Policy to assist in fulfilling that aim. BYU students should seek to be totally honest in their dealings with others. They should complete their own work and be evaluated based upon that work. They should avoid academic dishonesty and misconduct in all its forms, including but not limited to plagiarism, fabrication or falsification, cheating, and other academic misconduct.

**Deliberation Guidelines**

To facilitate productive and open discussions about sensitive topics about which there are differing opinions, members of the BYU community should: (1) Remember that we are each responsible for enabling a productive, respectful dialogue. (2) To enable time for everyone to speak, strive to be concise with your thoughts. (3) Respect all speakers by listening actively. (4) Treat others with the respect that you would like them to treat you with, regardless of your differences. (5) Do not interrupt others. (6) Always try to understand what is being said before you respond. (7) Ask for clarification instead of making assumptions. (8) When countering an idea, or making one initially, demonstrate that you are listening to what is being said by others. Try to validate other positions as you assert your own, which aids in dialogue, versus attack. (9) Under no circumstances should an argument continue out of the classroom when someone does not want it to. Extending these conversations beyond class can be productive, but we must agree to do so respectfully, ethically, and with attention to individuals' requests for confidentiality and discretion. (10) Remember that exposing yourself to different perspectives helps you to evaluate your own beliefs more clearly and learn new information. (11) Remember that just because you do not agree with a person’s statements, it does not mean that you cannot get along with that person. (12) Speak with your professor privately if you feel that the classroom environment has become hostile, biased, or intimidating. Adapted from the Deliberation Guidelines
Devotional Attendance

Brigham Young University’s devotional and forum assemblies are an important part of your BYU experience. President Cecil O. Samuelson said, "We have special and enlightening series of devotional and forum assemblies...that will complement, supplement, and enrich what will also be a very productive period in your classrooms, laboratories, and libraries. We look forward to being with you each Tuesday...and hope that you will regularly attend and bring your friends and associates with you...A large part of what constitutes the unique 'BYU experience' is found in these gatherings where the Spirit has been invited and where we have the opportunity to discuss and consider things of ultimate worth and importance that are not afforded to the academic community on almost any other campus" (from the address "The Legacy of Learning", 30 August, 2005). Your attendance at each forum and devotional is strongly encouraged.

Inappropriate Use Of Course Materials

All course materials (e.g., outlines, handouts, syllabi, exams, quizzes, PowerPoint presentations, lectures, audio and video recordings, etc.) are proprietary. Students are prohibited from posting or selling any such course materials without the express written permission of the professor teaching this course. To do so is a violation of the Brigham Young University Honor Code.

Plagiarism

Intentional plagiarism is a form of intellectual theft that violates widely recognized principles of academic integrity as well as the Honor Code. Such plagiarism may subject the student to appropriate disciplinary action administered through the university Honor Code Office, in addition to academic sanctions that may be applied by an instructor. Inadvertent plagiarism, which may not be a violation of the Honor Code, is nevertheless a form of intellectual carelessness that is unacceptable in the academic community. Plagiarism of any kind is completely contrary to the established practices of higher education where all members of the university are expected to acknowledge the original intellectual work of others that is included in their own work. In some cases, plagiarism may also involve violations of copyright law. Intentional Plagiarism-Intentional plagiarism is the deliberate act of representing the words, ideas, or data of another as one's own without providing proper attribution to the author through quotation, reference, or footnote. Inadvertent Plagiarism-Inadvertent plagiarism involves the inappropriate, but non-deliberate, use of another's words, ideas, or data without proper attribution. Inadvertent plagiarism usually results from an ignorant failure to follow established rules for documenting sources or from simply not being sufficiently careful in research and writing. Although not a violation of the Honor Code, inadvertent plagiarism is a form of academic misconduct for which an instructor can impose appropriate academic sanctions. Students who are in doubt as to whether they are providing proper attribution have the responsibility to consult with their instructor and obtain guidance. Examples of plagiarism include: Direct Plagiarism-The verbatim copying of an original source without acknowledging the source.
Paraphrased Plagiarism-The paraphrasing, without acknowledgement, of ideas from another that the reader might mistake for the author's own. Plagiarism Mosaic-The borrowing of words, ideas, or data from an original source and blending this original material with one's own without acknowledging the source. Insufficient Acknowledgement-The partial or incomplete attribution of words, ideas, or data from an original source. Plagiarism may occur with respect to unpublished as well as published material. Copying another student's work and submitting it as one's own individual work without proper attribution is a serious form of plagiarism.

**Respectful Environment**

"Sadly, from time to time, we do hear reports of those who are at best insensitive and at worst insulting in their comments to and about others... We hear derogatory and sometimes even defamatory comments about those with different political, athletic, or ethnic views or experiences. Such behavior is completely out of place at BYU, and I enlist the aid of all to monitor carefully and, if necessary, correct any such that might occur here, however inadvertent or unintentional." I worry particularly about demeaning comments made about the career or major choices of women or men either directly or about members of the BYU community generally. We must remember that personal agency is a fundamental principle and that none of us has the right or option to criticize the lawful choices of another." President Cecil O. Samuelson, Annual University Conference, August 24, 2010  

"Occasionally, we ... hear reports that our female faculty feel disrespected, especially by students, for choosing to work at BYU, even though each one has been approved by the BYU Board of Trustees. Brothers and sisters, these things ought not to be. Not here. Not at a university that shares a constitution with the School of the Prophets." Vice President John S. Tanner, Annual University Conference, August 24, 2010

**Schedule**

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<td>M Nov 17</td>
<td>Monday</td>
<td>Water and Electrolytes</td>
<td>Ch 12 (pgs. 455-480)</td>
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<td>W Nov 19</td>
<td>Wednesday</td>
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<td>Ch 12 (pgs. 455-480)</td>
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<td>F Nov 21</td>
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<td>Sa Nov 22</td>
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<td>Writing prompt 12- Metformin Treatment of Diabetes Mellitus Increases the Risk</td>
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<td>M Nov 24</td>
<td>Monday</td>
<td>Essential Trace and Ultratrace Minerals</td>
<td>Ch 13 (pgs. 481-500)</td>
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<td>T Nov 25</td>
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<td>Ch 13 (pgs. 500-519)</td>
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<td>Ch 13 (pgs. 519-546)</td>
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<tr>
<td>Sa Dec 06</td>
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<td>Writing prompt 13-Distinct patterns of hepcidin and iron regulation</td>
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<tr>
<td>M Dec 08</td>
<td>Monday</td>
<td>Nonessential Trace and Ultratrace Minerals Ch 14 (pgs 547-561)</td>
<td>Exam 4-Chapters 11, 12, 13. 12/4, 5, 6, 8 in Testing Center. Chapter 14 Pop Quiz 12</td>
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<td>W Dec 10</td>
<td>Wednesday</td>
<td>Nonessential Trace and Ultratrace Minerals Ch 14 (pgs 547-561)</td>
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<tr>
<td>Th Dec 11</td>
<td>Thursday</td>
<td>Writing Prompt 14-Effects of vanadium (III,IV,V)-chlorodipidolinate</td>
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<td>M Dec 15</td>
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<td>W Dec 17</td>
<td>Wednesday</td>
<td>Exam 5-Comprehensive Exam-11:00am-2:00pm</td>
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Scholarship Strategies Proposal-Final Report

As a graduate student I studied pancreatitis induced diabetes, and the protective role of macrophages in this disorder under the mentorship of Dr. James DeGregori at the University of Colorado Health Sciences Center. I completed a postdoctoral fellowship at Duke University’s Sarah W. Stedman Center Nutrition and Metabolism Center with Dr. Christopher Newgard. There I studied beta-cell biology and control of beta-cell growth as a potential therapeutic for diabetes. I have published in the Proceedings of the National Academy of Sciences and Diabetes. Since August 2013 my group here at BYU has been continuing these studies with many interesting findings that we hope will soon lead to publications and external grant funding. My lab currently has 19 undergraduates who all work a minimum of 10 hours a week on various research projects. My interactions with these students involved daily mentoring and guiding of research projects, as well as teaching laboratory techniques. Nine of these students applied for an ORCA grant, and 5 received it. For 2015.

Below are the goals that I set for myself during the 2014 Spring Seminar. These goals were to be completed by February 2015. I felt that I made good progress on these goals. I felt that these goals have helped me grow as a scientist and has allowed me to have success in my research program. I have addressed each of the goals and strategies, with an assessment in italics of how I did with these goals.

Scholarly Goals to be completed by February 2015

1. Submit a proposal as a primary investigator to the National Institutes of Health, American Diabetes Association and BYU for the Mentored Environment Grant. *I was successful in completing this grant. I applied for 7 external grants in 2014 (JDRF, NIH, AHA-local, AHA-National, DRWF, DARF, PhRMA Foundation). I was unable to apply for the ADA grant as the granting agency cancelled their two granting cycles during 2014. I also applied for 3 internal grants from BYU, the Widtsoe Grant, MEG and Andersen Diabetes Grant. My grants were not funded by the NIH, AHA-local, AHA-national, DRWF and PhRMA foundation. I have not yet heard from the JDRF or the DARF. I did not receive the Widtose award, but I did receive the Andersen Grant and the MEG.*

2. Submit 1-2 manuscripts this year for publication. *My lab has submitted one manuscript to the Journal Islets. It has been through one round of review and is now in review after revisions. We are confident that it will be published shortly. We have a second manuscript that we will submit by the end of February.*

3. Develop a lab website to attract quality graduate students-My lab has developed a website. I have two students that frequently up date the website. We have received communication from students about possible graduate training.
4. Submit one abstract for presentation at the Experimental Biology conference in April 2015. My lab submitted three abstracts to the EB conference. Two students will accompany me to the meeting.

Below are the strategies that I used to complete the scholarly goals. These were all very helpful. The explanation for each strategy is listed below:

**Strategies of Scholarly Productivity**

To meet the 4 goals listed above, I will use the following strategies to help me be more efficient and productive in my scholarly activity:

1. Set aside daily blocks of 30 minutes a day write (grants, manuscripts)- This was extremely helpful. I found having a dedicated writing time very helpful. This is a strategy that I will continue to use in my career.

2. Write in the library so that distraction from colleagues, students and phone are diminished-I have a room in the library that I use. This is a wonderful opportunity. It allows me to get quite a bit of writing done, and allows me to focus on my task.

3. Meet with Merrill Christensen (department mentor) bi-weekly to discuss early drafts and to receive critical feedback. -Our bi-weekly meetings have been very important. Dr. Merrill has given feedback on manuscripts; grant reviews and help regarding the teaching of my course and lab management. This has been extremely helpful for me.

4. Meet with collaborators every two weeks to discuss projects and to maintain accountability. -Dr. Hancock and I have been collaborating on a paper and a grant. We have met frequently to discuss experiments and to discuss results. This has been a helpful experience.
Citizenship Project Proposal-Final Report

As a member of the NDSF department, Life Sciences College and BYU I recognize the responsibility to serve the organization as a whole. I recognize that citizenship in this community as a right, privilege and duty. I am anxious to serve in any capacity that I am needed. I recognize that it is through this infrastructure that BYU can continue to improve in its goals of educating our wonderful students. As a member of the NDFS department, I have had the opportunity to serve on the MEG review committee, as a member of the department undergraduate education committee and on the seminar committee. I have been able to participate in two faculty search committees. I have enjoyed the opportunity to mentor students in my laboratory research and in the classroom. I will eagerly accept any new committee assignments given to me by my department chair.

Specific Goals:

The following are the goals that I set for myself to complete by February 2015 as my citizenship project. I feel that I was successful in implementing these goals. Listed after each goal is an explanation of how I completed these goals in italicized text.

Between now and February 2015, I will work on the following citizenship based goals:

1. **Observe colleagues’ teaching and invite colleagues to observe yours** - I will invite Chad Hancock, Jason Kenealey and Merrill Christensen to attend my course (NDFS 435-Nutritional Biochemistry). I will solicit feedback from these bench scientists on better teaching in my nutritional biochemistry course. I will also attend courses taught by Dr. Hancock and Dr. Christensen to learn their techniques. Dr. Kenealey and Dr. Christensen have observed my teaching. I have asked them for feedback with the techniques that I use. I have attended Dr. Kenealey’s course to watch his teaching style. I was not able to attend Dr. Hancock’s course, but I did attend one of Dr. Christensen’s lectures. All together, I feel the feedback that I received from colleague observation of my teaching and from watching their teaching styles has been helpful. I have picked up techniques and have implemented their usage in my teaching.

2. **Collaborate on a research project with a colleague** - I will begin working with Dr. Hancock on a project looking at mitochondrial activity in beta cells. We will present our findings at the 2015 EB meeting and anticipate submitting a manuscript by the end of the year. Dr. Hancock and I have worked together on a grant and a research project. We did not feel we had sufficient data to present this project at the 2015 EB meeting, however we are on target to complete this project to be submitted by the end of 2015. Our grant received critical feedback. We are currently revising the grant to be submitted at the end of February (NIH R15 grant). In addition, I have
begun collaborations with other professors in my department (Dr. Kenealey) and out of my department (Dr. Bikman).

3. **Develop a new course and team-teach with a colleague**—Dr. Jason Kenealey and I will develop NDFS 434—Nutritional Bioorganic chemistry for dietetics students. We will begin teaching the first iteration of this course in Winter 2015. This will allow me to apply some of the techniques that I learned in the spring semester, as well as increase collaborations with Dr. Kenealey. —Dr. Kenealey and I are currently teaching the pilot course of NDFS 434 (listed this semester as NDFS 603R) During this iteration we are teaching five students, and are working together to define the concepts that will be taught in the course. We meet frequently to discuss the course, assignments, exams and how the course will be graded. I feel that this process is being very helpful in preparation for the teaching of NDFS 434, which will begin in Winter 2016.

I feel that these goals have been very helpful in directing me to find opportunities for collaboration within my department. I feel, based on these experiences, that I have potential collaborations with three colleagues in my department. Furthermore, I feel that my teaching has improved by watching the other Professors in my department. This process has been beneficial, and I will use these concepts throughout my career.