<u>**Portfolio**</u>

Personal Plan:

1. *Although I looked at my personal plan halfway through the semester, I am reading it now with the knowledge that I have reached my goal. I understand the impact socioeconomic status, stress, and the impact healthcare access has on individuals. Through this course I am also better able to analyze studies through category identification, biases, and confounders. I have explored the health effects from high fructose corn syrup and the obesity crisis from many angles, which helped me to understand epidemiology principles.*

<u>**Directions in which I want to develop:**</u>

Although I have strong knowledge on disease transmission and what an infectious disease can do to an individual, I would like to develop a better understanding of the impact social factors have on health and to broaden my views beyond the patient in the bed at a hospital.

2. *I chose this glossary term because I was able to transfer the knowledge I gained in class to my job. Understanding biases helped me to understand how misinformation can occur, and reminds me that a study flaw can have a wide effect. In the future, when I read about or conduct a study, I will be able to better evaluate for biases.*

<u>**Glossary: Informational Bias**</u>

"Informational bias occurs when the means for obtaining the information about the subjects in the study are inadequate so that as a result, some of the information gathered regarding exposures and/or disease outcomes is incorrect." (Gordis, p264)

Clostridium difficile is a contagious and a potentially deadly infectious disease. There are two examples of informational bias with this condition.

We once believed it was caused by antibiotics, which effected the normal flora in the bowel and allowed the Cdiff (which is part of the normal flora) to proliferate.

Later, I learned it is transmitted via the environment, and it is the antibiotics which makes you susceptible to the long living spores that you are exposed to. The spores only were in the environment if left there by a previous

actively-diseased Cdiff person.

Now, the research is showing that even colonized individuals, those carrying the disease but not actively ill with it, are also able to transmit the spores into the environment. These people were never considered to be able to transmit the disease. Many people in the community are colonized, and develop the disease after antibiotic exposure.

The biases are #1: the disease is caused by antibiotics, and #2 the disease is only spread by actively infected people. The disease transmission and who can transmit the disease were not looked at appropriately, revealing flawed methodology.

Gordis, L. (2014). *Epidemiology* (5th ed). Philadelphia, PA: Elsevier.

3) *I chose this glossary term because I was able to map out a disease which I see at the hospital, and I could share with my classmates the process of an infectious disease (although a picture may have been more effective in displaying this). The chain describes each step which must be followed in order for disease transmission to occur.*

Glossary Term: Chain of Infection

"Chain of infection" must be followed for an infection to occur. I will explain using Lyme disease as an example:

- A pathogen or causative agent
    - Bacteria: *Borrelia burgdorferi*
- A reservoir (human, animal, or environmental source of the pathogen)
    - Deer tick
- A portal of exit from the reservoir
    - Body fluids from the tick
- Mode of transmission
    - Tick bite (lasting for at least 24 hours)
- A portal of entry into a susceptible host
    - Into the bloodstream
- A susceptible host
    - Human being (without bug spray!)

Breaking one link in the chain will stop the disease transmission.

CDC: http://www.cdc.gov/OralHealth/infectioncontrol/guidelines/slides/008.htm

http://www.ems-ceu.com/courses/375/index_ems.html

4) *I chose this annotation because it is when I first considered socioeconomic status (SES) as a classification which is determined by a standard method. I always assumed income level was the only way classify this, but I discovered many other methods could be used and that some of them may not appropriately identify true SES.*

Davey Smith, G. (2000). Learning to live with complexity: ethnicity, socioeconomic position, and health in Britain and the United States. American Journal of Public Health. 90(11). 1694-1698.

This article reviews the flaws in socioeconomic indicators in that they do not explain the whole story of health differences. Indicators can vary among ethnic groups, can be impacted by culture, and can change over time and across regional areas. Standardizing the evaluation of socioeconomic indicators throughout regions can leave knowledge gaps in understanding population health.

Davey Smith describes the limitations to such thinking, and gives examples of how factors such as mixed ethnicity, income differences within ethnic groups, and the differences in meaning for certain socioeconomic indicators in different ethnic groups. Also, the current indicators assume the current indictors reflect current social circumstances, and may not be transferable across ethnic groups. He also discusses the early life SES affects health through adulthood.

This article is an example of how the categorization can be faulty, despite being generally accepted. It is an example of the importance of how classification occurs as an important part of analyzing data.

5) *I not only chose this annotation because the article was eye opening for me as to the access to healthcare in Canada, and I was also able to tie a few different week's reading into it. Differences in healthcare across countries was not a topic I had explored before, and with Canada being a close neighbor to us with universal healthcare, I was intrigued. Other articles we read mentioned how universal healthcare was not based on successful evidence, and with our country heading in this direction I am somewhat fearful of having these statistics in the US. My hospital does not have the equipment to perform coronary angiography, but they typically have it performed within 24 hours (usually within hours).*

Alter, D. A., C. D. Naylor, et al. (1999). "Effects of socioeconomic status on access to invasive cardiac

procedures and on mortality after acute myocardial infarction." New England Journal of Medicine 341: 1359-1367.

Canada instituted universal healthcare to improve the health status of all of their citizens, with a focus on need, not income. Over a 3 year period, this study evaluated acute myocardial infarction (MI) admits in hospitals in the Ontario area and compared certain outcomes with the socioeconomic status (SES): use and wait time for coronary angiography and revascularization procedures after admission, and the death rate after one year. The SES status was stratified by neighborhoods, rather than actual income. They concluded a higher SES resulted in a 23% increase in angiography, 45% decrease in wait time, and an inverse relationship between the income and death rate: each $10,000 increase in median income resulted in a 10% reduction in death rate.

Angiography is not available in all hospitals, therefore the wait time varies according to the hospital: if the angiography was not onsite, wait times ranged from 34.5 days (low SES) to 23.3 days (high SES), or onsite services the rate was 6.9 days (low SES) to 4.6 days (high SES). As a nurse who works in a hospital without cardiac angiography services, I found these times overwhelming, since we will transfer patients immediately for treatment within hours.

This study ties into our reading topics from this week and last week. There is a relationship between SES and access to services and cardiac mortality. The authors stated in the article there were disproportionate number of patients presenting with MI were from the lower SES. I feel that this statement says so much, and implies there are other confounders at play here, including primary prevention strategies, including diet, smoking, or unhealthy workplace environment. Also, diabetes mellitus is a prevalent disease which can impact microvasculature and would need to be managed before, and especially after, an MI.

There were other factors which are not discussed in the study which can also have an impact on outcomes, including the administration of aspirin and wait time to seek out heath care services. It would be interesting to explore the post-management of these patients as well, which medications were they prescribed (and if they took them), what was their access to cardiac rehab, and their health literacy regarding the importance of cardiac risk factors. SES is more than just income; is encompasses so much more than that.

6) *I chose this annotation because I found the topic extremely interesting. I knew about the Human Genome Project, but was unaware of the results of it or the numerous amount of studies which had resulted from it. This information was transferred to my later sketch on the genetics of obesity. I am curious as to where this information will take us, including newer treatments and interventions in the future. I am aware of how close we are to personalized medicine and how difficult it will be to sort through all of the evidence which is resulting.*

Khoury, M. J., J. Little, M. Gwinn and J. P. Ioannidis (2007). "On the synthesis and interpretation of consistent but weak gene-disease associations in the era of genome-wide association studies." International Journal of Epidemiology 36: 439-445.

The Human Genome Project was a catalyst for investigating genes and environmental interactions which cause disease, but this journey has not been a speedy one. Although the International Haplotype Map (HapMap) project has made it possible for less expensive and more efficient genomics technologies, and the Genome Wide Association (GWA) has provided ways to analyze associations with diseases using case controlled studies, there is some debate as to if the causative factors can truly be identified in these large scale "fishing expeditions".

Genome-wide association studies brings to light the challenge of differentiating associations from confounders and biases. Although only a few associations have been able to be replicated, true causative factors can be diluted in large studies, and it may be the seemingly weaker associations which are the most important.

Khoury proposed a need to have systematic approaches to identify what associations are actually causative, and grading the evidence should be based on its relevance to health practices; even true genetic associations may not explain the outcomes due to unmeasured variables involved. Before we use the results on population medicine, the methods need to be more sophisticated, since weak associations may be the basis to identify the relationships of genetic factors and disease.

7) Sketch 3 was chosen because it was when I first took a deep dive into the HFCS debate and I was able to identify some trends with the polarizing sides. On the one side there are more scientists and biologists, and on the other side there are more people tied to the corn refiners and some nutrition professionals which may provide some biases. Also, I saw how categories and data can be skewed to fit the intension of the researcher.

<u>**Sketch 3: Categories HFCS Typical Consumption Patterns**</u>

The debate on the health effects of high fructose corn syrup (HFCS) is highly dependent on its consumption amounts: if there is no increase in use, then the increase in poor health outcomes, such as obesity, cannot be blamed on this substance. Therefore, the category of normal/typical use needs to be explored, both historical and currently, to evaluate if there has been an increase. There are two distinctly opposite opinions on this: there has been a dramatic increase versus no substantive increase.

White (2013) proposes the intake of fructose has not had a profound per capita increase since 1920 and has been steadily decreasing since 1999, without any correlation with obesity. He reports the historical data is incorrectly represented, with the omission of fats and oils as other trending food items, and 'fructose increase from caloric
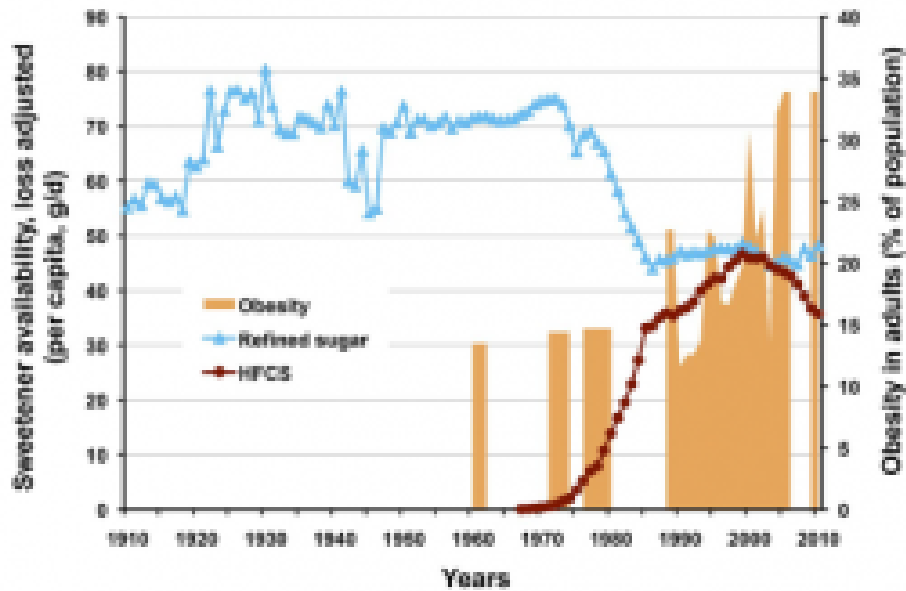
sweeteners over the past 90 years has increased very little averaging 39 plus/minus 4g/day/person" (p249). His data source was the USDA Economic Research Service per capita consumption data (although adjusted for loss) and the World Health Organization Global Database for BMI. In opposition, Bray (2004) evaluated the data from the US Department of Agriculture, reported HFCS has increased by >1000% between 1970 and 1990, and HFCS daily average of 132kcal for all American's >/= 2 years old, with the highest consumers reaching 316kcal/day. US Department of Agriculture food consumption tables and the National Center for Health Statistics were utilized as a data sources. Although the consumption data for both were obtained from similar sources, their categorization must have been varied.

Data can be displayed in graphs aid the reader in comprehending data, and both authors used them to show their results. Data displays can be somewhat deceiving since it can be manipulated to have the visual effects an individual desires. For example, White goes back further in time to look at fructose use, which appears to no be a substantial increase by graphing back to 1910 and categorizes the data scale for grams per day from 0-120g, whereas Bray has a shorter timeline, from 1961 to 2000, and tighter scale from 0-100g.
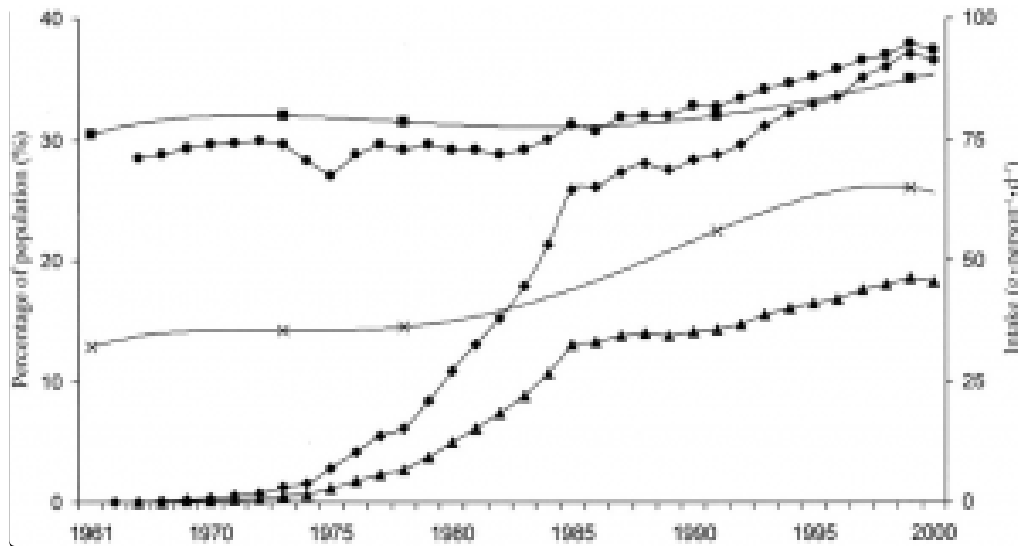
Estimated intakes of total fructose (•), free fructose (▴), and high-fructose corn syrup (HFCS, ♦) in relation to trends in the prevalence of overweight (▪) and obesity (x) in the United States. Data from references 7 and 35. (Bray, 2004)

Historical trends in sucrose and high-fructose corn syrup (HFCS) consumption (availability) versus rates of obesity in adults. After significant gain in market share at the expense of sucrose, HFCS consumption has been decreasing since 1999 and there is no correlation with obesity. From USDA Economic Research Service per capita consumption data, adjusted for loss and WHO Global Database on BMI. (White, 2013)

This is a good example of how categorizing data into tables and graphs can change perceptions of the information, and of bias when interpreting data. Anecdotally, it appears most of the opponents to HFCS are biologists, and look at the health effects from a cellular perspective, whereas the proponents tend to be involved with agriculture, such as the Corn Refiners Association, like John White, who believes there is "bias in the biochemical outcomes" in the opposing studies (p250).

"Dr. White worked in food industry research and management for 13 years, developing a specialization in nutritive (caloric) sweeteners. He is one of the foremost experts in fructose and high fructose corn syrup (HFCS), the result of 32 years of research on the production, functionality, applications, consumption and metabolism of these sweeteners. He enjoys present and past affiliations with the American Chemical Society (Washington, DC), the Institute of Food Technologists (Chicago, IL), the American Council on Science and Health (Washington, DC), the International Life Sciences Institute (Washington, DC), the Calorie Control Council (Atlanta, GA) and the Corn Refiners Association (Washington, DC)." (from LinkedIn).

Dr. Bray is the Chief of the Division of Clinical Obesity and Metabolism and Professor Emeritus at the Pennington

Biomedical Research Center. He "is an internationally recognized researcher whose major research interests have been in obesity and diabetes at both the experimental and clinical level. His grant funding is for the Diabetes Prevention Program, a multi-center NIH funded trial that is studying strategies for reducing the conversion to diabetes of people at high risk for diabetes; for the Look AHEAD multi-center trial, also funded by NIH, asking whether reducing body weight will impact overall health risk in diabetics; and for an experimental study comparing two strains of animals, one which become obese eating a high fat diet, and one which does not." (from Pennington Biomedical Research Center).

Bray, G. (2004). Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *The American Journal of Clinical Nutrition*. (79). 537-543.

White, J. (2013). Challenging the fructose hypothesis: new perspectives on fructose consumption and metabolism. *Advances in Nutrition*. (4). 246-256.

8) *Sketch 4 was chosen because I learned a lot on the history of nutritional decisions created in the United States. Low-fat diets were recommended in the 1940's and are now being revised in 2015 in response to the obesity crisis. It brought to light that not all policies are created on evidence, and by not circling back to check on the success of these interventions, policies not based on facts can perpetuate for decades. When identifying an association, it is important to circle back to see if the predictions hold up after an intervention to assert the intervention is affecting the suspected cause.*

Sketch 4: Associations, predictions, causes, interventions

LaBerge provides a historical account of the low fat diet phenomenon which has occurred in the United States over the past decades. "Scientific studies dating from the late 1940's showed a correlation between high-fat diets and high-cholesterol levels, suggesting that a low-fat diet might prevent heart disease in high-risk patients" (p139). Many studies at this time, including the Framingham study, investigated the causes of CHD since it was the leading cause of death.

In the 1950's medical researchers began to recommend the low fat diet, as a factor of risk for coronary heart disease (CHD). At this time, the population generally believed a low fat was appropriate for weight reduction, although not necessarily for every person. Years later, a low fat diet was recommended to all Americans for preventing overweight bodies and decreasing cardiac risk. "After 1980, the low-fat approach became an overarching ideology, promoted by physicians, the federal government, the food industry, and the popular health media. Many Americans subscribed to the ideology of low fat, even though there was no clear evidence that it prevented heart disease or promoted weight loss" (p139).

Why didn't this intervention impact the outcome? Perhaps the cause was not correctly identified. There could be many other associations between CHD and diet which were not investigated in these studies; by missing an association, the intervention can fail. For example, the high fat consumption individuals may be more likely to be sedentary, be concurrently ingesting high amounts of carbohydrates, or be impacted by socioeconomic status. A meta-analysis of dietary studies was conducted, and revealed people were substituting simple, refined carbohydrates for fat; now the American Heart Association (AHA) no longer recommends a 10% fat diet, and is now increasing it to 25-35% (O'Riordon, 2015).

Because the prediction of decreasing fat in the diet to decrease CHD and obesity was incorrect, the outcome gives us some insight to a cause for obesity. By substituting carbohydrates for the lost fats, there is now an association being made with sugars and fructose and their negative health impact. Although it is not feasible for a randomized control study with fructose alone, one way to evaluate the causation is to continue to monitor weight trends after these new dietary recommendations from the AHA.

LaBerge, A. (2008). How the ideology of low fat conquered America. *Journal of the History of Medicine and Allied Sciences*. 63(2).139-177.

O'Riordon, M. (Feb 12, 2015). No evidence to support dietary fat recommendations, meta-analysis finds. *Medscape.* Online only. Retrieved on Feb 16, 2015 from http:www.medscape.com/viewarticle/ 839708.

9) *I had to completely redo the next sketch because I misunderstood what heterogeneity really was! I chose this sketch because I was able to explore the differences in obesity in men and women, and learned there are biological differences in their metabolism of sugars. Although many population differences studies are based on ethnicity, I enjoyed looking at the heterogeneity between the sexes.*

Sketch: Heterogeneity in obesity: men and women are not alike

Obesity is a worldwide pandemic. Globally, obesity rapidly increased between 1980 and 2008, although there is a higher incidence among females than males. Across 68 countries, obesity in women is much greater than in men, with 3 obese women for every 2 obese men, which cannot be accounted for by any distribution variability of obesity (Wells, et al, 2012).

There have been some hypotheses which suggest social determinants vary by gender. Wells, et al (2012) reports the prevalence of obesity increases with economic development; there is an inverse association between per capita gross domestic product and female obesity. Throughout almost all populations, obesity in females is more prevalent than in males. Another socioeconomic influence may be under-nutrition in early life, which is associated with female

obesity later in life (Wells, et al, 2012).

The availability of processed food may impact the higher obesity rates in females. Many developing countries have had a significant infiltration of processed foods, including refined carbohydrates and added sweeteners, which correlates with rapid increases in obesity (Kanter & Caballero, 2012). The accessibility to these food impact women more than men because "…there are gender differences in carbohydrate metabolism that cause a greater increase in triglyceride levels in women. Therefore, the increased refined carbohydrate intake in developing countries may affect excess weight gain in women more than men" (Kanter & Caballero, p495). As opposed to men, women seem to prefer these foods high in added sugars, including high calorie processed foods (including cookies, chocolate, and ice cream). Women consume more dairy foods than men (Kanter & Caballero, 2012) which includes some higher sugar foods.

In the United States, there has been an increase in prevalence of obesity and type 2 DM, which coincides with an increase in soft drink consumption. Schultze, et al (2004) conducted a prospective study from the Nurses' Health Study II which evaluated the relationships between sugar-sweetened beverages, weight gain, and type 2 diabetes mellitus (DM) in women. Their result found that women who increased their consumption of sugar sweetened soft drinks from 1 or fewer drinks per week to 1 or more per day had the highest amount of weight gain and increased BMI. The lowest weight gain and BMI occurred in women who decreased their intake of sugar sweetened soft drinks. These were adjusted for physical activity and known risk factors. Women who drank 1 or more sugar sweetened soft drinks a day had a progressively higher risk of type 2 DM, after adjusting for confounders. Other poor health behaviors associated with higher sugar-sweetened soft drinks including less physically active, smoke more, higher intake of total calories.

Women have a higher incidence of obesity worldwide, despite living in the same communities and homes as men. Inherent food preferences which may play a part, with partiality for sweet food. We also need to look closely at the biological differences in women in the metabolism of foods, especially with sugars and sugar sweetened beverages.

Kanter, R. & Caballero, B. (2012). Global gender disparities in obesity: a review. *American Society for Nutrition. 3:* 491-498.

Schulze, M., Manson, J., Ludwig, D., Colditz, G., Stampfer, M., Willett, W. & Hu, F. (2004). Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in you and middle-aged women. *Journal of the American Medical Association. 292(*8)**: 927-34.

Wells, J., Marphatia, A., Cole, T. & McCoy, D. (2012). Associations of economic and gender inequality with global obesity prevalence: understanding the female excess. *Social Science & Medicine. 75:* 482-490.