FROM FARM TO FAT KIDS: THE INTERSECTION OF
AGRICULTURAL AND HEALTH POLICY

A Dissertation
Presented to
The Graduate Faculty of The University of Akron

In Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Lisaann S. Gittner
December, 2009
ABSTRACT

A descriptive retrospective study was performed to display the intersection of U.S. Agricultural Policies (which created the U.S. food supply composition) and Health Policies (which focused on disease treatment) on an individual level disease, obesity (described as BMI units). The intersection in the 20th century between U.S. agricultural and health policy shaped the nutrient composition of the food supply and school children's growth. However, here is a disproportionate increase in obesity occurring among US children as compared to children in 13 other developed countries and adults possibly because selection has been occurring for phenotypes that have ‘thrifty’ metabolic settings expressed as obesity. Obesity is a disease of mal-nutrition caused by unhealthy diet quality and quantity with the lifespan cost of obesity increasing if obesity is developed in childhood because of the longer duration of co-morbid conditions (CSDH, 2008; Deckelbaum & Williams, 2001). Policies to resolve childhood obesity need to reflect the changing nature of agricultural production, provide a food supply that provides affordable appropriate nutrition to the entire population, and create a healthy food environment to foster appropriate body weights.

Federal manipulation of agricultural production, distribution, and price accelerated after WWII. Unintended consequence of agricultural policies
changed the food supply creating a niche for cheaper alternative processed and refined foods. Increases in nutritional programs and exponential increases in crop subsidy programs began in the 1960s creating a food supply that was very different in both which crops were produced and price from the early part of the century. The U.S. Diet changed over the century from one that supplied a diet low in fat and high in unprocessed grains fruits and vegetables to a diet high in fat, sugar, and processed foods and low in vegetables and fruit. Consumption patterns also changed during of the century; as consumption of processed foods and protein increased, a sharp rise in children’s BMIs was also seen. The linkage between the changing food supply and children’s increases in BMI suggest that the food supply affected the health of the nation’s children.
DEDICATION

To Gregory, who said any good writing always starts “Once upon a time,
in a land far far away. . .” Thank you for your support encouragement and the
belief that I could do this even though there were many times that I didn’t believe,
you always did.

And to Noah, I know you also ‘got’ the PhD with me because you were
there every step of the way giving me exactly what I needed when I needed it.
Hopefully, you will now get your Mom back.

And finally to Kathy, who pushed me so hard to go back to school before
she died, I wish you could be here to see this. . .
ACKNOWLEDGEMENTS

To my mentor, Susie Ludington for without her, I would have never followed the path I did to study children.

To my Chair, Raymond W. Cox III, who always pointed me in the correct direction and wouldn’t let me stay in my comfort zone of medical gobbledygook and pushed me to be policy relevant.

To my son, Kevin, who followed Mom to graduate school because I was ‘his inspiration’ now he will know that there were many times in the last few years where he was my inspiration because he shamed me into studying because he was and I would have felt guilty if I didn’t.

To my friend, Denise Radovick, who without her organizational skills I would have never been able to write the first word let alone over 70,000 words, I would still be drowning in paper.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>LIST OF FIGURES</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>x</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LIST OF GRAPHS</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>xi</td>
</tr>
</tbody>
</table>

## CHAPTER

### I. INTRODUCTION

- Purpose and Objectives ................................................................. 1
- Problem Statement ........................................................................... 2
- Methods and Objectives ................................................................... 25
- Results and Discussion .................................................................... 28

### II. LITERATURE REVIEW

- Background ...................................................................................... 33
- Childhood Obesity Intersection with Policy Streams ....................... 34
- Obesigenic Food Environment ........................................................... 49
- Childhood Obesity Definition, Scope, Causal Factors ....................... 55
- Consequences of Pediatric Obesity .................................................... 81
- Conceptual Frameworks ...................................................................... 83

### III. METHODOLOGY

- Purpose ............................................................................................ 99
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1</td>
<td>Life course Model of Health</td>
<td>87</td>
</tr>
<tr>
<td>2.2</td>
<td>Multiple Streams Framework of Policy Formulation</td>
<td>90</td>
</tr>
<tr>
<td>2.3</td>
<td>Framework From Government Policy to Individual Disease</td>
<td>97</td>
</tr>
</tbody>
</table>
## LIST OF GRAPHS

<table>
<thead>
<tr>
<th>Graph</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.1</td>
<td>Children’s BMI Trend During the 20th Century (1900-2000)</td>
<td>127</td>
</tr>
<tr>
<td>4.2</td>
<td>6-9 year old Males BMI Trend During the 20th Century (1900-2000)</td>
<td>131</td>
</tr>
<tr>
<td>4.3</td>
<td>9-12 year old Males BMI Trend During the 20th Century (1900-2000)</td>
<td>132</td>
</tr>
<tr>
<td>4.4</td>
<td>6-9 year old Females BMI Trend During the 20th Century (1900-2000)</td>
<td>133</td>
</tr>
<tr>
<td>4.5</td>
<td>9-12 year old Females BMI Trend During the 20th Century (1900-2000)</td>
<td>134</td>
</tr>
<tr>
<td>4.6</td>
<td>Males Age 6-9 BMI Trend vs. Per Capita Consumption of Red Meat</td>
<td>137</td>
</tr>
<tr>
<td>4.7</td>
<td>Males Age 9-12 BMI Trend vs. Per Capita Consumption of Red Meat</td>
<td>138</td>
</tr>
<tr>
<td>4.8</td>
<td>Females Age 6-9 BMI Trend vs. Per Capita Consumption of Red Meat</td>
<td>138</td>
</tr>
<tr>
<td>4.9</td>
<td>Females Age 9-12 BMI Trend vs. Per Capita Consumption of Red Meat</td>
<td>138</td>
</tr>
<tr>
<td>4.10</td>
<td>Males Age 6-9 BMI Trend vs. Per Capita Consumption of Poultry</td>
<td>139</td>
</tr>
<tr>
<td>4.11</td>
<td>Males Age 9-12 BMI Trend vs. Per Capita Consumption of Poultry</td>
<td>139</td>
</tr>
<tr>
<td>4.12</td>
<td>Females Age 6-9 BMI Trend vs. Per Capita Consumption of Poultry</td>
<td>139</td>
</tr>
<tr>
<td>4.13</td>
<td>Females Age 9-12 BMI Trend vs. Per Capita Consumption of Poultry</td>
<td>140</td>
</tr>
<tr>
<td>4.14</td>
<td>Males Age 6-9 BMI Trend vs. Protein Prices (FY1967 dollars)</td>
<td>140</td>
</tr>
</tbody>
</table>
4.15 Males Age 9-12 BMI Trend vs. Protein Prices (FY1967 dollars) .......... 140
4.16 Females Age 6-9 BMI Trend vs. Protein Prices (FY1967 dollars) ........ 141
4.17 Females Age 9-12 BMI Trend vs. Protein Prices (FY1967 dollars) ...... 141
4.18 Males Age 6-9 BMI Trend vs. Per Capita Consumption of Eggs .......... 142
4.19 Males Age 9-12 BMI Trend vs. Per Capita Consumption of Eggs ........ 143
4.20 Females Age 6-9 BMI Trend vs. Per Capita Consumption of Eggs ...... 143
4.21 Females Age 9-12 BMI Trend vs. Per Capita Consumption of Eggs ..... 143
4.22 Males Age 6-9 BMI Trend vs. Egg Prices (FY1967 dollars) ............... 144
4.23 Males Age 9-12 BMI Trend vs. Egg Prices (FY1967 dollars) ............. 144
4.24 Females Age 6-9 BMI Trend vs. Egg Prices (FY1967 dollars) .......... 144
4.25 Females Age 9-12 BMI Trend vs. Egg Prices (FY1967 dollars) .......... 145
4.26 Males Age 6-9 BMI Trend vs. Per Capita Consumption of Milk ......... 148
4.27 Males Age 9-12 BMI Trend vs. Per Capita Consumption of Milk ........ 148
4.28 Females Age 6-9 BMI Trend vs. Per Capita Consumption of Milk ...... 149
4.29 Females Age 9-12 BMI Trend vs. Per Capita Consumption of Milk ..... 149
4.30 Males Age 6-9 BMI Trend vs. Milk Prices (FY1967 dollars) ............. 150
4.31 Males Age 9-12 BMI Trend vs. Milk Prices (FY1967 dollars) .......... 151
4.32 Females Age 6-9 BMI Trend vs. Milk Prices (FY1967 dollars) .......... 151
4.33 Females Age 9-12 BMI Trend vs. Milk Prices (FY1967 dollars) .......... 151
4.34 Males Age 6-9 BMI Trend vs. Per Capita Consumption of Wheat/Grains ................................................................. 152
4.35 Males Age 9-12 BMI Trend vs. Per Capita Consumption of Wheat/Grains .................................................................. 153
4.36 Females Age 6-9 BMI Trend vs. Per Capita Consumption of Wheat/Grains .......................................................................................................................... 153

4.37 Females Age 9-12 BMI Trend vs. Per Capita Consumption of Wheat/Grains .......................................................................................................................... 154

4.38 Males Age 6-9 BMI Trend vs. Flour / Bread Prices (FY1967 dollars) .... 154

4.39 Males Age 9-12 BMI Trend vs. Flour / Bread Prices (FY1967 dollars) ................................................................................................. 154

4.40 Females Age 6-9 BMI Trend vs. Flour / Bread Prices (FY1967 dollars) .......................................................................................................................... 155

4.41 Females Age 9-12 BMI Trend vs. Flour / Bread Prices (FY1967 dollars) .......................................................................................................................... 155

4.42 Males Age 6-9 BMI Trend vs. Per Capita Consumption of Corn Products ................................................................................................. 156

4.43 Males Age 9-12 BMI Trend vs. Per Capita Consumption of Corn Products ................................................................................................. 157

4.44 Females Age 6-9 BMI Trend vs. Per Capita Consumption of Corn Products ................................................................................................. 157

4.45 Females Age 9-12 BMI Trend vs. Per Capita Consumption of Corn Products ................................................................................................. 158

4.46 Males Age 6-9 BMI Trend vs. Sugar Prices (FY1967 dollars).............. 159

4.47 Males Age 9-12 BMI Trend vs. Sugar Prices (FY1967 dollars).............. 159

4.48 Females Age 6-9 BMI Trend vs. Sugar Prices (FY1967 dollars) .......... 159

4.49 Females Age 9-12 BMI Trend vs. Sugar Prices (FY1967 dollars) ....... 160

4.50 Males Age 6-9 BMI Trend vs. Per Capita Consumption of Sweeteners ................................................................................................. 161

4.51 Males Age 6-9 BMI Trend vs. Per Capita Consumption of Sugar and High Fructose Corn Syrup Sweeteners .................................................................................. 161

4.52 Males Age 9-12 BMI Trend vs. Per Capita Consumption of Sweeteners ................................................................................................. 162
4.72 Females Age 6-9 BMI Trend vs. Total Fat Consumption ......................... 173
4.73 Females Age 9-12 BMI Trend vs. Total Fat Consumption ...................... 174
4.74 Males Age 6-9 BMI Trend vs. Major Fats Consumption ...................... 175
4.75 Males Age 9-12 BMI Trend vs. Major Fats Consumption ...................... 175
4.76 Females Age 6-9 BMI Trend vs. Major Fats Consumption ...................... 175
4.77 Females Age 9-12 BMI Trend vs. Major Fats Consumption ...................... 176
4.78 Males Age 6-9 BMI Trend vs. Per Capita Consumption of Cheese ....... 177
4.79 Males Age 9-12 BMI Trend vs. Per Capita Consumption of Cheese ..... 177
4.80 Females Age 6-9 BMI Trend vs. Per Capita Consumption of Cheese ... 177
4.81 Females Age 9-12 BMI Trend vs. Per Capita Consumption of Cheese ................................................................. 178
4.82 Males Age 6-9 BMI Trend vs. Butter and Margarine Prices (FY1967 dollars) .............................................................................................................. 179
4.83 Males Age 6-9 BMI Trend vs. Butter and Margarine Consumption ...... 179
4.84 Males Age 9-12 BMI Trend vs. Butter and Margarine Prices (FY1967 dollars) .............................................................................................................. 180
4.85 Males Age 9-12 BMI Trend vs. Butter and Margarine Consumption ...... 180
4.86 Females Age 6-9 BMI Trend vs. Butter and Margarine Prices (FY1967 dollars) .............................................................................................................. 180
4.87 Females Age 6-9 BMI Trend vs. Butter and Margarine Consumption .... 181
4.88 Females Age 9-12 BMI Trend vs. Butter and Margarine Prices (FY1967 dollars) .............................................................................................................. 181
4.89 Females Age 9-12 BMI Trend vs. Butter and Margarine Consumption ......................................................................................................................... 182
4.90 Males Aged 6-9 BMI Trend vs. Federal USDA Spending ...................... 184
4.91 Males Aged 9-12 BMI Trend vs. Federal USDA Spending ...................... 185
4.92 Females Aged 6-9 BMI Trend vs. Federal USDA Spending ............... 185
4.93 Females Aged 9-12 BMI Trend vs. Federal USDA Spending ............ 186
4.94 Males Age 6-9 BMI Trend vs. Crop Subsidy (FY1967 dollars) ........... 187
4.95 Males Age 9-12 BMI Trend vs. Crop Subsidy (FY1967 dollars) .......... 187
4.96 Females Age 6-9 BMI Trend vs. Crop Subsidy (FY1967 dollars) ......... 187
4.97 Females Age 9-12 BMI Trend vs. Crop Subsidy (FY1967 dollars) ........ 188
4.98 Males Age 6-9 BMI Trend vs. % Household Income Spent on Food .... 189
4.99 Males Age 9-12 BMI Trend vs. % Household Income Spent on Food ... 189
4.100 Females Age 6-9 BMI Trend vs. % Household Income Spent on Food .......................................................... 189
4.101 Females Age 9-12 BMI Trend vs. % Household Income Spent on Food .......................................................... 190
4.102 Generational Pattern Mother’s BMI trends (b. 1888-1911) vs. Son’s BMI Trends (b.1912- 1935) ................................................................. 193
4.103 Generational Pattern Mother’s BMI trends (b. 1888-1911) vs. Daughter’s BMI Trends (b.1912- 1935) ..................................................... 194
4.104 Generational Pattern Mother’s BMI trends (b. 1903-1917) vs. Son’s BMI Trends (b.1922- 1939) ................................................................. 194
4.105 Generational Pattern Mother’s BMI trends (b. 1903-1917) vs. Daughter’s BMI Trends (b.1922- 1939) ..................................................... 195
4.106 Generational Pattern Mother’s BMI trends (b. 1907-1927) vs. Son’s BMI Trends (b.1933- 1947) ................................................................. 195
4.107 Generational Pattern Mother’s BMI trends (b. 1907-1927) vs. Daughter’s BMI Trends (b.1933- 1947) ..................................................... 196
4.108 Generational Pattern Mother’s BMI trends (b. 1921-1936) vs. Son’s BMI Trends (b.1941- 1957) ................................................................. 196
4.109 Generational Pattern Mother’s BMI trends (b. 1921-1936) vs. Daughter’s BMI Trends (b.1941- 1957) ..................................................... 197
4.110 Generational Pattern Mother’s BMI trends (b. 1930-1946) vs. Son’s BMI Trends (b.1957- 1973)........................................................................................................... 197

4.111 Generational Pattern Mother’s BMI trends (b. 1930-1946) vs. Daughter’s BMI Trends (b. 1957- 1973)...................................................................................................... 198

4.112 Generational Pattern Mother’s BMI trends (b. 1941-1957) vs. Son’s BMI Trends (b. 1963- 1985)........................................................................................................... 198

4.113 Generational Pattern Mother’s BMI trends (b. 1941-1957) vs. Daughter’s BMI Trends (b. 1963- 1985)...................................................................................................... 199

4.114 Generational Pattern Mother’s BMI trends (b. 1951-1969) vs. Son’s BMI Trends (b. 1979- 1994)........................................................................................................... 199


4.116 Three Generational Overlay early to mid century Grandmothers (b. 1898-1918), Mothers (b. 1922-1942), and Daughters (b. 1951 - 1971)........................................................................................................... 202

4.117 Three Generational Overlay mid to late century Grandmothers (b. 1922 -1942), Mothers (b. 1951-1969), and Daughters (b. 1989-1994)...................................................................................................... 203
CHAPTER I
INTRODUCTION

“As I see it, every day you do one of two things: build health or produce disease in yourself.”  Adelle Davis

Purpose and Objectives

The purpose of this study was to describe a 100-year time span for the growth of U.S. school children during the 20th century overlaid upon changes in federal agricultural policies that directly affected the composition of the U.S. food environment. The objectives of the study were as follows:

1) Describe trends in children’s weights following adoption of different federal agricultural policies as measured by commodity component’s availability and price in the U.S. food supply.

2) Describe generational trends in children’s weight status overlaid with historical context such as adoption of significant U.S. health policies and medical practices (immunizations and widespread antibiotic usage) which contribute to a healthy childhood environment, food shortages during various periods, and food
supply changes due to adoption of significant U.S. agricultural policies (school lunches, crop subsidies and various commodity production programs).

Problem Statement

An increase in the prevalence of obesity is occurring among people of all ages throughout the world (Speiser et al., 2005), however, the disproportionate increase in obesity among U.S. children is even more unsettling than the analogous increase in adults (Riley, Bass, Rosenthal, & Merriman, 2005). The lifespan cost of obesity increases dramatically if obesity is developed in childhood (CSDH, 2008; Deckelbaum & Williams, 2001). Co-morbid diseases developed because of obesity are not only exhibited in adulthood but are also exhibited in childhood, extending the duration of disease and increasing the impact of the associated disabilities throughout the life span (Deckelbaum & Williams, 2001), thus obesity policies need to address multifactoral long-term life course issues, with the acknowledgment that there are no short term simple fixes. Overweight and obese children are exhibiting diseases that used to be considered 'adult diseases' such as hypercholesterolemia and type 2 diabetes (Deckelbaum & Williams, 2001). Because pediatric obesity is correlated with a number of serious medical complications and increased mortality in both children and adults (Power, Lake, & Cole, 1997a, 1997b), prevention of early development of obesity without affecting normal growth is critical. Evidence
indicates that children are able to regulate energy intake to maintain normal
growth as long as there is no outside influence or pressure about when and what
to eat (Birch & Fisher, 1997; Satter, 1990). However, the obesigenic nature of
the U.S. food supply created by U.S. Farm Policies may be contributing to
disregulation of eating behaviors uncoupling caloric consumption from nutritional
needs (Putnam, Allshouse, & Scott Kantor, 2002).

Public policies proposed to combat obesity are fragmented and originate
in more than one policy stream and no linkage between the divergent policy
streams of agriculture and health is occurring. Consequently, obesity policy is
primarily formulated in the health policy stream but health policies ‘treat’ obesity
as a disease. In the health policy stream, there is a basic underlying limitation,
because the focus is on disease prevention and/ or treatment. Thus, a disease
has to manifest or present in the pre-stages before ‘treatment’ initiates; therefore
health policies are viewed as ‘a treatment’ rather than a long term life course
solutions (AAP, 2006; Whitaker, Sherman, Chamberlin, & Powers, 2004). By the
time disease related policies effect an individual, there already is either a
potential problem or a problem that has already manifested shifting the solution
paradigm from a public policy to an individual treatment. It is difficult to change
the treatment model that pervades health policies because of the established
pattern of medical care as a treatment. Thus, when facing a multifaceted life
course problem like obesity, the model of individual disease treatment fails when
applied to population policies (Deckelbaum & Williams, 2001). W. Dietz, CDC
Director testified in March 2009, before the U.S. House Committee on Agriculture
and stated “While medical treatment for disease management is essential, our nation needs a better balance between treating diseases and preventing them (Current Status and Activities to Decrease the Prevalence of Obesity Among U.S. Children and Adolescents. Statement of William H. Dietz, MD, PhD, Director Division of Nutrition, Physical Activity, and Obesity National Center for Chronic Disease Prevention and Health Promotion Centers for Disease Control and Prevention, 2009).” The best potion to deal with life course diseases such as obesity is to prevent occurrence altogether by creating effective public policy.

Historically, in the United States, food has always been an important public policy. However during most of the nation’s history, the problem has been insufficient food supplies creating malnutrition and hunger in the population rather than the current problem of a food supply creating an obesigenic environment causing obesity and over nutrition in the population. Rampant malnutrition found in military recruits during and post World War II prompted passage of the National School Lunch Program in 1946 specifically to address childhood under nutrition ("National School Lunch Act," 1946). The National School Lunch Act 1946 Public Law 79-396 was created to “safeguard the health and well-being of the nation’s children and to encourage the domestic consumption of nutritious agricultural commodities and other food" ("National School Lunch Act," 1946). Social welfare proponents lobbied that the School Lunch program should be part of a broad social safety net welfare agenda and that it did not make sense for the permanent program to administered by the
USDA because it was a health policy and it should administered by a federal department that understood the health needs of children (Levine, 2008). This did not occur because the School Lunch program was never designed as a health program *per se*, it was an agricultural program designed to stabilize crop prices by eliminating surpluses (Levine, 2008). Nutritional standards were developed and established on the basis what was known about the dietary needs of school children ("National School Lunch Act," 1946). The program was operated with USDA funds and donated food stocks, it had a dual purpose to feed children but also to remove agriculture commodity surpluses from the market ("National School Lunch Act," 1946). Further expansion of the National School Lunch Program was authorized in The Agricultural Act of 1949 which allowed the Commodity Credit Corporation to donate commodities acquired by it under its price support activities to various agencies, first and foremost was the School Lunch Program ("Agricultural Act of 1949," 1949; Gunderson, 1971). The main reason the 1949 Agricultural Act passed was because of fear that commodity over production surpluses (that did not exist during the war) would reoccur so the program was developed to ‘dispose’ of excess commodities so the retail market would remain stable (Levine, 2008). The legislation was touted as a way to expand demand for American produced food commodities by introducing children (through school lunch rooms) to new and different foods (United States. Congress. House. Committee on Education and Labor., 1961; United States. Congress. House. Committee on Education and Labor. Subcommittee on
General Education., 1960) and was purely an agricultural commodity program not a human nutrition (health) program.

However, in the 1960s, hunger emerged as a policy problem in the agricultural policy stream. The problems regarding food insufficiency and malnutrition that were widespread in the population resulted in new federal food assistance programs and expanded food assistance became a top policy priority. By 1966, school lunch programs to meet the nutritional needs of children were expanded, extended and strengthened "in recognition of the demonstrated relationship between food and good nutrition and the capacity of children to develop and learn . . . to encourage the domestic consumption of agricultural and other foods. . . to meet more effectively the nutritional needs of our children. . ." ("Child Nutrition Act ", 1966a). Additionally, the first significant changes in the National School Lunch Act of 1946 were adopted in 1966, because of rising concern regarding poverty and a desire to help schools lacking resources to provide low-cost meals to poor children (United States. Congress. House. Committee on Education and Labor., 1961; United States. Congress. House. Committee on Education and Labor. Subcommittee on General Education., 1960). By 1975, legislation was passed to extend and make food programs for women, infants and young children permanent ("Child Nutrition Act of 1966 Amended Permanent Authorization of WIC as National Health and Nutrition Program," 1975) and in 1977, the School Lunch and free milk programs were extended to Summer and Daycare programs ("Child Nutrition Act of 1966 Amended Permanent Authorization of WIC as National Health and Nutrition
Program," 1975). Since the inception of the first nutritional subsidy programs, the programs have continued to grow and expand to meet nutritional requirements for U.S. children, but inherent to the programs is the creation of more abundant food but not always food with the best nutritional content. For the previous 60 years, the agricultural policy stream was focused on providing food for the nation and supporting food production by creating short term policies for food production and distribution rather than long term policies to support life course nutrition. Thus, a paradigm shift in the agricultural policy stream will need to occur for policies to be effective in combating obesity which is considered a health related ‘disease’. Policies to reduce obesity need to span the human life course than responding to an immediate agricultural commodity supply problem.

From 1976 to 2004, the prevalent risk of a U.S. child being overweight or obese increased more than five-fold among 6-11 year old children and almost seven-fold among adolescents 12-17 years old between the two National Health and Nutrition Examination Surveys, NHANES (Ogden et al., 2006; Ogden, Flegal, Carroll, & Johnson, 2002; C.L. Ogden et al., 2002). Between the NHANES Surveys (1976/80 to 2003/4), the percent of children with BMIs above the 85th percentile, the diagnostic level for overweight, increased from 6.5% (+/- 0.6%) to 37.2% (+/- 1.9%) for 6-11 year olds and increased from 5.0% (+/- 0.5%) to 34.3% (+/- 2.6%) for 12-17 year olds (Ogden et al., 2006; Ogden, Flegal, Carroll, & Johnson, 2002). Obesity is traditionally not measured in pre-school children, however, in just 3 years (2000 to 2003), obesity in the cohort of 2-5 year old U.S. children increased over 10% from 3.6% to 13.9% +/- 1.6 (Ogden et al.,
Additionally, the United States has the highest prevalence of pediatric obesity as compared to 13 European countries and Israel (Cole, Bellizzi, Flegal, & Dietz, 2000). According to the Healthy People 2010 Midcourse Review performed in 2007, pediatric obesity increased and moved away from the target by 5% (United States Department of Health and Human Services, 2007). Rather than a reduction in obesity that was predicted by the implementation of HP2010 policies, obesity increased over and above the baseline threshold during the measurement period (2000-2005). Therefore the DHHS stated that “prevention rather than treatment may hold the highest potential in reversing the trend in the United States” (United States Department of Health and Human Services, 2007). Obesity is a complex manifestation of an individual’s gene-environment interaction. During human evolution, physical exercise and food procurement have been inseparably interwoven creating a link between eating and physical activity (Barness, Opitz, & Gilbert-Barness, 2007). The link sets and regulates body metabolism and weight preferentially selecting genotypes that are adapted to their nutrition and activity level of their environment (Barness et al., 2007). However, environmental changes that altered a grandparent or parent’s genes may through a fetally driven feedback loop create an obese phenotype in the ensuing children which was not displayed in the previous generations because of the changed nutritional environment (Prentice, 2005).

In the contemporary United States starvation is rarely an issue. Additionally, sedentary lifestyles have increased, thus the inherited link between eating and physical activity has been broken disconnecting the human genome
from human metabolism possibly leading to a large increase in obesity in the population (Barness et al., 2007). Therefore looking at obesity as purely an individual disease or a population disease is not adequate to address the complexity of development of obesity. There is research evidence that an association exists between early childhood overweight, the mother’s pre-pregnancy body size and both their nutritional statuses (Barker, 2007a; J. Eriksson, T. Forsen, J. Tuomilehto, C. Osmond, & D. J. Barker, 2001). Nevertheless, there has been little research looking at the influence of diet on development of obesity/overweight in children. The studies that have been conducted had limited sample sizes, did not include representative samples, were cross-sectional, and did not adjust for confounding factors (Hawkins & Law, 2006). Thus, this study addresses a gap in the evidence because it proposes to look at the intersection of childhood obesity trends and the nation’s diet which is proscribed by agricultural policies which create the food supply.

Federal agriculture policies regulating food production and pricing have created an U.S. food supply where food is more abundant, available, and cheaper than ever before compared to food supplies in any other nation during any other historical era. But, the composition of the United States food supply has gradually changed over the previous century to one high in processed high calorie foods (USDA, 2000). Highly processed energy dense foods such as sodas and snack foods are inherently obesigenic (J. E. Brown et al., 2005; R. Kleinman, 2004; Windham, Wyse, & Hansen, 1983). The caloric content of the food supply has increased by 23% in the previous 30 years (Buttet & Dolar,
and the composition of the United States food supply is directly related to both crop subsidies and the industrialization of farming. Until 500 generations ago, all humans consumed only unprocessed food foraged and hunted from the environment (O'Keefe & Cordain, 2004). Unprocessed food provided a diet high in lean protein, polyunsaturated fats, monounsaturated fats, fiber, vitamins, minerals, antioxidants, and was low in sugar and saturated fats (O'Keefe & Cordain, 2004). The described diet is the one that shaped human genotypes and is the opposite of the currently available food supply in the U.S. The current U.S. food supply provides a diet that is high in fat, sugar and processed grains and low in vegetables and fruit (i.e. average 3,900 calories per capita per day with 24% of the calories from fat, 17% from added sweeteners, 24 % from processed grains and only 13% from meat, 5% from vegetables, and 3% from fruit) (Hiza & Bente, 2007). This is a very different food environment than even 100 years ago.

Politics greatly influenced (and still influences) U.S. agricultural policies by determining which crops were / are supported which direct affects the retail market price of foods (Drewnowski & Barratt-Fornell, 2004; Pollan, 2003). The U.S. food environment has been shaped by political and economic forces, agribusiness, and food processing companies which control what commodities are produced, processed, and sold. Economics and politics direct food prices, amounts, availabilities, and choices (Tillotson, 2003a, 2003b). After World War II, industrialized farming in conjunction with the very politically developed crop subsidy program encouraged food overproduction and further lowered the retail price for food (Cochrane & Runge, 1992). The politicization of food production
(i.e. for profit over production) has created an obesigenic food supply in the U.S. (Tillotson, 2003a, 2003b).

To understand the development of the current obesigenic environment in the United States, multiple policies need to be factored into the equation: Agriculture Policy, Transportation Policy and Health Policy combined with increasing population affluence (Economic Policy) conjoined to create an obesigenic national food environment. Historically in the 20th century, U.S. public health policies related to childhood growth were targeted to address either immunizations or mal/under nutrition and did not even consider childhood obesity as a problem that would require a policy solution (Institute of Medicine. Committee on Prevention of Obesity in Children and Youth, Koplan, Liverman, & Kraak, 2004; Institute of Medicine. Committee on the Impact of Pregnancy Weight on Maternal and Child Health, 2007). However, there is limited evidence on any direct relationship between policies and obesity in children (Wang & Lobstein, 2006). Policies can influence the development or resolution of childhood obesity and its precursors through legislation, regulation, fiscal policy, or through direct recommendations targeted at children, parents, service organizations, and local communities (Wang & Lobstein, 2006).

The current study provides a 100-year time sequence of the growth of U.S. school children during the 20th century which will be matched to changes in federal agricultural policies that directly affected the composition of the U.S. food environment. Patterns in the 100-year graph in children’s growth, as measured by BMI that follow adoption of different federal agricultural policies, as measured
by commodity component’s availability and price in the food supply, was graphed to describe emergent patterns between children’s body mass and the food supply. For any research related to obesity, both the policies that created the obesigenic environment and the progression of obesity in individuals need to be reconciled into a coherent comprehensive framework. Recommendations and solutions can then be contextually studied at both the macro- and the micro-level. Thus, a framework is necessary to allow the integration of theories across the disparate domains of medicine and public administration so the effect of macro-level policy changes can be discerned regarding micro-level health in an individual. There are problems with the combination of theories from two distinct and disparate disciplines because words are defined and understood differently. Thusly, a discussion of the proposed combination framework and the literature review will follow in Chapter 2. Medical and public administration policy theories would otherwise be examined in isolation from each other; complexities of the macro-level policy process need to be combined with the micro-level medical models of health to create a new framework that bridges policies and health throughout the life course of individuals within the societal context. New sociological theories, such as the Ecological Theory and Social Determinants of Health Theory do try to span macro and micro level interactions to explain how societal influences contribute to individual outcomes. However both are limited in use when looking at a chronology of generational life courses for policy and individual effects to manifest. Theories causally link observed or modeled phenomena, providing interpretive structure. Conceptually, health theories are
concerned with the progression of disease states and do not address the construction of the environment that lead to a particular disease except in broad terms such as the environmental context as a confounder or causal factor. Health theories do not attempt to explain the environment or the factors that determined the construct of the environment. Thusly, the environment is described and used as either input or as a confounder in most health theories, if it even addresses them at all. In contrast, policy theories are concerned with the construct of social, political, organizational environments and do not address the progression of a disease in specific individuals except in the broadest sense. The disease is considered a measurable descriptor of the population rather than an outcome from policy decisions that created the environment. Limitations in the conceptualization of both medical and policy theories are that the bridge between them does not exist. Policy models stop with the environment as an outcome and health models start by using the environment as an input. To solve complex health policy issues, new conceptual frameworks are necessary. A combination of health and policy theories would be necessary to provide a more comprehensive approach so that individual health issues can be directly related to policy choices. An argument could be made that there are sociological and psychological theories already developed which bridge between medical and policy theory but these theories are limited. They use the individual as the center or starting point and then link to ‘outside the individual’ to explain human development. They cannot explain inherited traits that develop because of environmental interactions. Disciplines differ profoundly in the approach they use
to address the issues of health, illness, and health care. Therefore, each theory brings with it, the disciplinary ‘baggage’ of definition and context when trying to conceptually frame a study. Stone points out, paradoxes especially when disciplines need to cross boundaries to explain a phenomenon because each discipline “lives in their own worldview” and it is necessary to step out of a singular disciplinary perspective to understand and engage life course health problems but then the clarity of definition becomes critical so as to not miscommunicate (Stone, 2002).

Barker’s Theory of Developmental Origins of Adult Diseases uses the environment as an input factor, stating that adult diseases are related to both genetics and phenotypical expression both pre- and post-natally; the causal factor that determines phenotype expression is environment (Barker, 1995b). For obesity, the environment of the fetus creates an intrauterine ‘thrifty phenotype’ that translates into a propensity for obesity post-birth (Hales & Barker, 2001). However, the environment is described as a given and there is no explanatory power regarding how the environment itself became obesigenic. In contrast, Kingdon’s Multiple Policy Streams Framework approach to policy formation explains that policies are developed by policy entrepreneurs when forces create a policy window where multiple independent policy streams combine to produce an agenda that will affect the formulation of a responsive policy which in turn, after policy adoption, will change or create a social environment (Zahariadis, 2007). The Multiple Streams Theory clearly articulates the chasm between the agricultural policy stream and the health policy stream.
However, policy theory readily describes the development of the United States food supply environment was created but does not explain the individual level metabolic impact (obesity) from the environment (food supply) that the policies created. It is critical to align a conceptual framework that encompasses both policy formulation and disease progression so meaningful solutions can be developed to stop the expanding obesity epidemic in the United States. Combining Kingdon’s Multiple Policy Streams Framework with Barker’s Theory of Developmental Origins of Adult Diseases creates a conceptual model that begins with macro-level policy and ends with micro-level disease. Use of Kingdon’s Multiple Policy Streams Framework will provide an understanding of the profound influence politics has had on the agricultural policy stream that has shaped the U.S. food supply and the enormity (and longevity) of the agricultural policy stream when compared to the health policy stream. Politics shape the policy agenda and significantly favors the agribusiness portion of the agricultural policy stream, health and nutrition policy have a minor impact. Alignment of current U.S. agricultural policy to assure a food supply that had the appropriate dietary recommended nutrient intakes for normal growth and development is critical, but agriculture policies which are highly politicized and are a remnant of early American settlement policies do not intersect the health policy stream. The agricultural policy stream is a torrent that at this point in its evolution is difficult to change direction. Finding a policy entrepreneur that is willing to commit to policies that will take multiple life times before fruition and the difficulty in findings someone as a champion will be difficult in the agricultural policy stream. It is
easy to find policy entrepreneurs’ willing to champion either agribusiness or farming interests because the policies have a near term ‘pay off’. The Multiple Streams Framework does accurately describe the policy history that has created the U.S. food supply. Barker’s Theory of Developmental Origins of Adult Diseases provides a life course theory that spans multiple generations as well as the current individual. Complex gene environment interactions spanning multiple generations can be explained when using these two theories because the time span can expand to account for long term societal policy and family dynamics which change individuals.

Eating is a cultural and social event not just a necessity of life that balances energy intake with energy expenditures. A complex web of inter related factors determine preferred diet and eating behaviors; some of these factors begin before birth and span the life course (J. A. Mennella & Beauchamp, 1991, 1997; J. A. Mennella, Griffin, & Beauchamp, 2004; J. A. Mennella, Jagnow, & Beauchamp, 2001). New research indicates that certain genes either directly or indirectly contribute to eating preferences and food metabolism. Some of the alleles select for both diets and metabolisms that when superimposed on the modern food environment cause a tendency toward obesity (Corella, Arnett et al., 2007; Corella, Lai et al., 2007; Qi, Corella et al., 2004). The feeding relationship is complex between the parent and child; effective feeding provides the groundwork for growth patterns and healthy eating behaviors (Satter, 1990). Children are genetically programmed to prefer energy-dense foods over energy-dilute foods they learn to relate food flavors to the positive physiologic feedback
that occurs from eating energy-dense foods associated with satiety cues involved in nutrient digestion and absorption (Birch & Fisher, 1998; Julie A. Mennella, Pepino, & Reed, 2005). However, energy intake, from appropriate diet and not energy expenditure due to physical activity was the primary determinant of obesity status in one to two year old children (Stunkard, Berkowitz, Schoeller, Maislin, & Stallings, 2004). Two to three year old children of mothers who skipped breakfast and did not eat fruits and vegetables had the highest incidence of poor nutritional status (Lee, Hoerr, & Schiffman, 2005). Thirty year trends (1970 to 2000) in children’s food choices correspond with the national food supply and are influenced by taste, television, and cultural norms (R. E. Allen & Myers, 2006). The consumption of milk, vegetables, grains, and eggs has declined while the consumption of fruit juices, sweetened beverages, poultry, and cheese has increased (R. E. Allen & Myers, 2006). But, Swinburn (2009) found that increases in children’s energy intake, alone, from 1970 to 2000 were able to explain the entire weight increase for that time period (Swinburn, 2009).

Contrary to longstanding views that a decrease in physical activity as a result of higher TV viewing and video game playing coupled with an increase in consumption contributed to the increase in weight of American children from 1970 to 2000 (Andersen, Crespo, Bartlett, Cheskin, & Pratt, 1998; Coon & Tucker, 2002; Dietz & Gortmaker, 1985; Expert Committee Recommendations on the Assessment, Prevention, and Treatment of Child and Adolescent Overweight and Obesity 2007; Mendoza, Zimmerman, & Christakis, 2007). The Framingham Children’s Study reported that parental eating habits directly affected the nutrient
intakes of young children; children whose parents ate diets high in saturated fat also ate diets high in saturated fats (Oliveria et al., 1992). Parents role model food preferences therefore it stands to reason that children’s food preferences are positively correlated to their parent’s food preferences (Cathey & Gaylord, 2004). Children’s repeated exposure to food advertisements for particular types of foods and ease of access to these foods in the home may cultivate preferences for energy-dense, nutrient poor foods (Birch & Fisher, 1997, 1998; McGinnis, Gootman, & Kraak, 2005). If high calorie non-nutritious food (junk food) is the only food available in the home, the child will develop a preference for that type of food, establishing lifetime poor eating habits (R. E. Allen & Myers, 2006). Home environments where energy-dense foods are predominant may create an obesigenic feedback loop predisposing the child for future obesity (Birch & Fisher, 1998). Jointly, United States economic and agriculture policies influence the home food environment by constraining the availability of affordable food choices; many affordable foods contain significant amounts of non-nutritive calories at artificially lowered prices. Couple an inherited tendency toward obesity with a family food environment that selects for energy dense non-nutritive foods and a cultural pattern that focuses on eating, it is not surprising that the population level obesity is exponentially rising in children. The Survey of Food Intakes by Individuals (CSFII), in 1994-96 reveals that 67% of the population age 2 or older consumes less than one serving of whole grains a day and only 10% consume the recommended daily allowance of three or more servings (Putnam et al., 2002). The data reveal that individuals eat more refined grain, fat,
cholesterol, and sugar and eat less whole grains and fiber than recommended (Putnam et al., 2002). The food supply provided 5.2 servings of fruits and vegetables, including legumes, per capita per day in 2000, higher than the minimum recommendation shown in the Food Guide Pyramid but below recommended 7 daily servings (Putnam et al., 2002). The Survey of Food Intakes by Individuals (CSFII) 1994-96 data confirm that intake of fruits and vegetables increase with age, income, and education (Putnam et al., 2002). Additionally, half of all young children (> two years old) consumed less than one serving of fruit per day (Putnam et al., 2002). The survey revealed that iceberg lettuce, white potatoes, and canned tomato products accounted for 43% of the fruit and vegetables consumed in an average American’s diet; the nutritional content of these three fruits and vegetables is limited and does not provide adequate dietary intake of vitamin and minerals (Putnam et al., 2002).

Approximately 1.61 per capita daily servings of dairy products were produced in the United States in 2000; less than the recommended 2.2 servings per individual that are recommended in the USDA Food Pyramid Guidelines (Putnam et al., 2002). Cheese and whole milk, both very high in fat content, account for > 50% of the dairy products consumed in the United States (Putnam et al., 2002). Consumption of high fat milk and cheese products accounted for 22% of the saturated fat in the U.S. diet (Putnam et al., 2002). However, poultry consumption has doubled since the 1970s, but red meat accounts for 48% of the total meat servings consumed by Americans; the consumption of this quantity of red meat contributes to 20% of the saturated fat in the U.S. diet (Putnam et al.,
The inflation adjusted ‘real’ price of added sugar (high fructose corn sweetener), saturated fat, and non-enriched flour / grain has declined by 16% since 1977 (Buttet & Dolar, 2008). However, the inflation adjusted ‘real’ price for fruits, vegetables, non-subsidized lean meat, and non-subsidized low fat dairy products has risen by 25% in the same 30 year period (Buttet & Dolar, 2008). The market pricing of food might have produced the dramatic increase in saturated fat and sugar and the lowered consumption of fruit/vegetables in the average American's diet. Crop subsidies account for approximately $20 billion each year in USDA’s food expenditures (Cohen; Dimitri, Effland, & Conklin, 2005; Effland, 2000). Subsidies were created to protect farmers’ incomes and insure that low commodity prices or natural disasters (weather) that create widespread crop failures do not bankrupt the farmer (“USDA--NASS Quick Stats (Crops),” 2007).

U.S. agriculture policies have created an artificial low cost for meat, dairy products, and poultry because corn is the major component of animal feed and created a new market for corn based sweeteners which replaced beet and cane based sweeteners (Drewnowski & Barratt-Fornell, 2004; Pollan, 2003) and trans fats (Beach, Murray, Piggott, & Wohlgenant, 2002) which replaced beef tallow and lard in the food supply. Since the main components of the fast food industry are beef, chicken, soda (high fructose corn syrup is the primary ingredient), and vegetable shortening, federal crop subsidy policies have effectively subsidized that industry (Cochrane & Runge, 1992; Tillotson, 2003b). By the 1970s, agriculture policies created a situation that for farmers to maintain a constant
income, it was necessary to grow more corn which lead to even lower prices; and significantly drove down the cost of many foods products based on corn and created a niche market for high fructose corn syrup (Cochrane & Runge, 1992; Tillotson, 2003b). Whole milk products are actually subsidized more than once (during production and then again in food aid programs); cattle feed crops (corn and sorghum) and milk fat products are subsidized before and after; the combination of programs creates a consumer market where high fat dairy products are lower priced than low fat products (Buttet & Dolar, 2008).

Government crop subsidies in recent years have supported a system of relative stable crop prices as compared to increasing per capita income levels ("Farm Security and Rural Investment Act of 2002," 2002; Regional Economic Information System, Bureau of Economic Analysis, U.S. Department of Commerce," 2007). Creating a situation in which income levels rise and food costs decline as a percentage of real income. Food costs as a percentage of family income have decreased; food prices have risen below the annual inflation rate during 1913 to 1946, and 1972 to 2000 ("Consumer Price Index - All Urban Consumers [Data File]." 2008). However, the trend is slowing, since 2005, U.S. food prices have risen faster than at any time since 1990; prices for all food products has increased by 4.0% in 2007, higher than the 2.4% rise in 2006 (Clauson, 2008). Even with the recent food price increases the per capita ‘real’ cost of food is actually less than it was 20 years ago ("Consumer Price Index - All Urban Consumers [Data File]." 2008; Table 2.3.1. Percent Change From Preceding Period in Real Personal Consumption Expenditures by Major Type of
Average daily per capita caloric intake in the United States has increased by 12% since 1985 and physical activity rates have not increased at the same rate (Putnam et al., 2002). Food prices rose at an accelerated rate in 2007 as compared to the previous 30 years, however Americans overall still spent less than 10% of their disposable income on food; they spent 13.9% in 1977 as compared to 9.8% in 2005 (Clauson, 2008). This drop occurred because prices of other consumer goods outpaced the price of food, and incomes rose at a faster rate than food prices; disposable personal income increased 5.7% in 2007 and 5.9% in 2006 (Clauson, 2008). Of the total food purchased, 58% was for consumption at home and 42% was consumed away from home, predominately ‘fast food’ (Clauson, 2008). Consumption of fast food among children in the United States has an adverse effect on dietary quality in ways that plausibly increases the obesity risk (Bowman, Gortmaker, Ebbeling, Pereira, & Ludwig, 2004). However, socioeconomic status also affects food consumption, with lower SES households spending a higher level of their disposable income on food (Clauson, 2008). Lower SES households are also more impacted by USDA Farm policy and food subsidies than other income level American households (Clauson, 2008). Currently, low income households spend between 19 and 25% of their disposable income on food and also receive the bulk of federal food after market subsidies (Clauson, 2008); the subsidies, such as WIC (Women Infants and Children Federal Food Program), Food Stamps and School Lunch Programs.
strongly support purchases of high caloric foods with marginal nutritional values (Institute of Medicine. Committee to Review the WIC Food Packages, 2005; McGinnis et al., 2005). Income levels are inversely associated with poor nutrition, low SES individuals are less likely to meet the Dietary Reference Intakes, DRI (Otten, Hellwig, & Meyers, 2006), for fruits and vegetables, have higher fat and sweetened beverage intakes than more affluent individuals (Dietary Risk Assessment in the WIC Program, 2002; Krebs-Smith, Cleveland, Ballard-Barbash, Cook, & Kahle, 1997; Munoz, Krebs-Smith, Ballard-Barbash, & Cleveland, 1997; Welsh et al., 2005). Mixed-race neighborhoods, white high-poverty areas and all African American areas (regardless of income) were less likely than predominantly white higher-income communities to have access to foods that enable individuals to make healthy choices (Baker, Schootman, Barnidge, & Kelly, 2006). Because WIC program obesity rates are well over the national average rates of obesity for children in the U.S. population; the federally subsidized food program has a noteworthy effect on obesity in its enrollees (Health, United States, 2006 with Chartbook on Trends in the Health of Americans, 2006). Using the WIC Program as an example of federal food subsidy programs shows that obesity rates in WIC enrolled children are consistently higher than the general population consequently there must be something in the WIC food policies that create an obesigenic environment (Bartlett, Bobronnikov, & Pacheco, 2006). The obesity rate in WIC served children by the time they are 2 years old is higher (15.4% are overweight and 22.3% are obese) than the general population rate of 13.9% for both overweight
and obesity (Bartlett et al., 2006; *Health, United States, 2006 with Chartbook on Trends in the Health of Americans*, 2006). Obesity rates are even worse for minority children participating in WIC, in 2004, at 1 year of age obesity rates range from: 42.7% overweight, 34.9% obese in Native Americans; 37.3% overweight, 29.6% obese in Hispanics; 32.1% overweight, 25.2% obese in African Americans; to 31.3% overweight, 24.0% obese in Whites (*Survey of the Public Health Nutrition Workforce 1999-2000*, 2003). White children enrolled in WIC were 48% more likely to become overweight at age 5 than non WIC enrolled white children (Rose, Bodor, & Chilton, 2006). U.S. Population obesity is likely to worsen because over half the infants in the United States are living in households eligible for WIC (Bogen, Hanusa, & Whitaker, 2004). Additionally, the proportion of WIC enrolled children developing later life adult obesity is 300% higher compared to the general non-WIC population (Bogen et al., 2004). Since 2004, WIC has created a new programmatic policy to classify children as ‘at risk for’ overweight if their mothers were obese during pregnancy; but, there have not been any changes to the composition of the food packages for the group of children who are now labeled ‘at risk for’ overweight (Bogen et al., 2004). The Institute of Medicine (IOM) report “WIC Food Packages” (2005) called for major changes to the food packages coupled with intensive systematic nutritional educational programming to try to stem the rise in obesity (Institute of Medicine, 2005).
Methods and Objectives

Chapter 3 presents the methods used to describe the food supply and define the generational BMI trends that have occurred in the 20th century; it will discuss the data sets accessed and the graphical presentation used to present results. A descriptive retrospective study was performed that displayed how U.S. Agricultural Policy externalities (which directly create the U.S. food supply composition) lead to individual level diseases such as obesity and its co-morbidities. The study describes, using graphical displays, the changes in pediatric Body Mass Index (BMI) and overlays children’s weight trends with the U.S. food supply during the course of the 20th century. The study describes trends in children’s weights following adoption of different federal agricultural policies as measured by commodity component’s availability and price in the U.S. food supply. Commodities were chosen to represent the average U.S. diet and reflect the average daily calorie consumption. The food supply was described by commodities in four main categories: proteins, carbohydrates, fats, and vitamin and minerals. Average daily calorie consumption in the United States in 2000 was 12 percent (300 calories) above the 1985 consumption level (Putnam et al., 2002). By 2008, the average Americans’ daily caloric intake was 500 calories greater than 30 years ago equating to an average weight gain of 1 pound per person per week if activity levels do not increase proportionately (Buttet & Dolar, 2008). Commodity production for human consumption in the U.S. food supply was used to describe the relative availability of calories for each
type of nutritional category. However, nutritional needs vary based upon the growth and health status of the individual, thus in 1994, the National Academy of Science Institute of Medicine (U.S.) and Health Canada began an initiative to develop a broad set of dietary reference values and by 2005 and have now developed DRIs for a healthy diet (Otten et al., 2006). Generalizations of a healthy diet were determined by age classifications based upon growth and developmental stages (Otten et al., 2006). DRI groupings are based on developmental milestones for the following age groups: Toddlers (1 year through 3 years); Early Childhood (4 to 8 years); Puberty (9 to 13 years); and Adolescence (14 to 18 years) (Otten et al., 2006). Comparing the Dietary Recommended Intakes with the actual U.S. food supply produced reveals that there are not enough fruit and vegetables grown to meet the appropriate dietary needs of all Americans (McGinnis et al., 2005). A U.S. National Academy study in 2005 determined that every pediatric age group did not to eat enough vegetables to meet the DRIs (Hendricks, Briefel, Novak, & Ziegler, 2006).

Anthropometric data is available for school aged children, defined as 6-16 years old, however, the DRI age categories regarding caloric intake do not match the anthropometric data categories, and therefore the DRI for puberty will be used as the standard for children’s nutritional requirements. The age of the children in this study will be 6 to 12 years old (broken into 2 categories, 6 to 9 years old and 9 to 12 years old). Childhood obesity at 6-11 years is predictive of adolescent obesity especially in minorities (Saha, Eckert, Pratt, & Shankar, 2005), which is then predictive of adult obesity status (S. S. Guo, Wu, Chumlea, & Roche, 2002;

The graphs were produced using Excel for Vista. The x-axis was the time period from 1900 to 2000 in 5-year increments, the primary y-axis displays the school children’s BMI using WHO standards for weight cutoffs (WHO, 2003) and the secondary y-axis varies depending upon commodity. Major historical events from 1900 to 2000 that either affected the food supply or public health will be discussed. The x-axis remained constant for all graphs. Trend lines for the BMI variable as compared to the commodity, price, household income, and government spending variables were fitted using the Excel trend line function. The Excel trend line function uses the following formula functions to fit the trend lines: for the polynomial trend line, y = mx + m1x^2 + b for all males (age 6 to 9 and
9 to 12) and females (age 6 to 9) and for exponential trend line used for females (age 9 to 12) \( y = bm^x \), to the data set by minimizing the sum of the squares of the error (Kleinbaum, Kupper, Muller, & Nizam, 1998). Over-specifying the trend line was avoided by checking the Excel function LINEST to assure that the lowest order polynomial trend line was chosen as the best fit curve. LINEST identified if the coefficients were statistically significant and if they were not, a lower order polynomial was chosen. Confidence intervals were not displayed because the graphs were difficult to interpret because of the multiple variables with the numerous corresponding lines. For the Generational trends a line was fitted between at least two time points spanning a generation using the Excel trend line function with the following equation: \( y = mx + b \) to the data set through minimizing the sum of the squares of the error terms for data that is linear (Kleinbaum et al., 1998). Graphically, the \( R^2 \) measures how close the regression line is to all of the observations and will be presented on all the generational graphs.

Results and Discussion

Chapter 4 presents the graphical results to answer the research questions. RQ1) Did the aggregation of U.S. agricultural and nutrition policies throughout the past century create a selection process for that is biased towards obesity? RQ1 was answered by overlaying the anthropometric measurements of children over the past century with commodities consumption and price, that
were affected by agricultural policies either positively or negatively, which then affected the food supply. The anthropometric measurement pattern was then explained in terms of the ensuing agricultural policies in effect during the time period. RQ2) Was the large spike in obesity that has been seen in the last 30 years in the United States created by expression of the thrifty phenotype across the generations? RQ2 was answered by overlaying the anthropometric measurements of generational patterns (i.e. children generational patterns with their mother's generational pattern with their mother's mother (grandmother) generational pattern, respectively). Patterns between the generations emerged showing the thrifty phenotype; during periods of food scarcity the mother’s generation was smaller than their children’s generation and during food plenty the mother’s generation was either similar or bigger with their children.

Chapter 5 interprets the results with regard to current Federal obesity policy proposals. Two main policy streams affect childhood obesity, the health and the agricultural streams and two other streams, the financial and transportations stream also have minor effects; each stream flows independently and is driven by different forces. However, for pediatric obesity policy, the health stream appears to be the primary stream that attempts to set; the other streams do not even consider unintended effects the policies would have on obesity during their agenda setting (Wildavsky, 2002). Many policies and programs outside the health policy stream that directly affect the food supply and population physical activity are developed with blinders on in regard to unintended policy consequences linked to obesity. There has often been “no
policy response” to obesity, making the problem even worse because of lack of consensus among researchers, policy specialist sub groups, and policy makers about the policy solutions required (Exworthy, 2008), and accordingly created more disconnection in already disconnected policy streams.

Policy disconnects between multiple streams have created an obesity epidemic that cannot be solved by policy changes in only one stream. However, since obesity is a life course problem, there are no short term solutions; hence the policy cycle timeline would need to be generational rather than administrational in length. Thus enters the problem of sustainability and feasibility of policies that need to remain in effect and be monitored for generations. Limitations of the current study need to be considered. First, the descriptive nature of the study does not provide causal data or relational data. Thus, it is difficult to ascertain how or if there are relationships between the food supply and children’s body size; there appears to be trends that indicate a possible relationship but validation of the existence of or significance of the relationships cannot be determined from this study. It is always difficult to obtain data across such a prolonged time span but even with the incomplete data, trends were able to be described for the 20th century. However, the relationships between pediatric BMIs and the commodities were not possible with the data used for this study. Trends in body weight and trends in the food supply caused by federal agricultural policy were described; unfortunately it was not possible to posit relationships.
Body weight equals energy input (i.e. calories consumed) minus energy expended (i.e. physical activity); consequently, the major limitation of this study is that the energy expenditure side of the equation was not measured. The inability to measure any type of surrogate for energy expenditure was the main limitation in the data, because there was no good proxy measure that was consistent over the century for children’s physical activity. Television viewing was considered as a proxy measure for physical activity but, the rising trends in obesity began in the 1950s 20 to 30 years before television was identified as a factor that reduced children’s physical activity levels (Coon & Tucker, 2002; Mendoza et al., 2007). However a new analysis of the past 30 years of NHANES data, released in May 2009, indicates that physical activity may not be as important as previously thought as causing the appreciable weight gain in U.S. children during the time span (Swinburn, 2009). Swinburn (2009) found that increases in children’s energy intake, alone, from 1970 to 2000 were able to explain the entire weight increase for that time period (Swinburn, 2009). The latest findings from the Swinburn (2009) study lower expectations regarding what types of weight reduction can be achieved with exercise so public health policy will need to shift its focus toward eating less rather than physical activity(Swinburn, 2009).

Agriculture policy effects agriculture production, food prices and the availability of food. However, data that links the actual availability of food and eating behavior in the population is lacking. This study described the broad effects that diverse policy streams (agricultural, health) has on U.S. farm production; it shows the need for policies to be developed collaboratively across multiple policy streams
rather than politically so the policies: reflect the changing nature of agricultural production, provide a food supply that provides affordable appropriate nutrition to the entire population and creates a health food environments that fosters appropriate body weights.
CHAPTER II
LITERATURE REVIEW

“There are risks and costs to a program of action. But they are far less than the long-range risks and costs of comfortable inaction.” John F. Kennedy

Background

The current U.S. food environment has been shaped by economics, politically influenced policies, and by agribusiness which strongly influence the types and quantities of food commodities that are produced, processed and sold (Tillotson, 2003b). The politics and business of ‘food’ directs price, amount, availability, and choices and has created an obesigenic food supply in the U.S. (Tillotson, 2003a, 2003b). Evidence indicates that there is a mismatch between the modern diet and lifestyle and the inherited human Paleolithic genome, which has remained principally unchanged since the agricultural revolution 10,000 years ago to assure survival in during periods of food shortages (Barness et al., 2007; O'Keefe & Cordain, 2004). The “thrifty” phenotypes, designed to store excess calories as adipose tissue and slow metabolism, inherited from the
Paleolithic genome as postulated as the cause of the increases in obesity, hypertension, diabetes, and cardiovascular disease seen in modern humans (Barness et al., 2007; O'Keefe & Cordain, 2004). The United States has the highest prevalence of obesity in children when compared to other industrialized nations (Cole et al., 2000). According to the Healthy People 2010 Midcourse Review performed in 2007, pediatric obesity increased and moved away from the target by 5% (United States Department of Health and Human Services, 2007). Rather than a reduction in obesity that was predicted by the implementation of HP2010 policies, obesity increased over and above the baseline threshold during the measurement period, 2000-2005 (United States Department of Health and Human Services, 2007) and in 30 years, 1976-2004, the prevalent risk of a U.S. child being overweight or obese increased more than five-fold (5.7x) among 6-11 year old children and six-fold (6.9x) among adolescents 12-17 years olds (Ogden et al., 2006; C. L. Ogden et al., 2002; C.L. Ogden et al., 2002).

Childhood Obesity Intersection with Policy Streams

While the cause of the pediatric obesity is multi-faceted, the food environment is viewed as one of the main contributors. U.S. agricultural policy facilitated programs to alleviate hunger and provide food assistance, direct commodity production and processing which influenced the amounts and types
of food produced and shaped the nation’s food environment without regards as to the health consequences of the food supply. The absence of diversity in the nation’s diet because of the food supply has lead to poorer population health and the burgeoning obesity epidemic (Luna, 2004). The nation’s health and agriculture policies are two distinct and separate policy streams that have not historically intersected. U.S. health policy has developed through collaboration with the major medical and health professional organizations and the pharmaceutical industry utilizing a policy process that was effectively expert driven because of the scientific complexity of the issues being debated. Whereas, U.S. agricultural policy developed through a politically influenced land grant system that provided free land to anyone willing to settle and farm it, creating a farm system that focused on production supports to assure continued commodity production. The national problem that is pediatric obesity straddles both the health and agricultural policy streams creating an intransigent conundrum because traditionally these streams have not worked together. Formulation of effective policies to reduce obesity means that policies will have to be formulated across policy streams that have never worked together before and have historically developed very different processes for agenda setting and policy formulation.

Post World War II social norms in the United States called for increasing children’s weights (i.e. equating to better health) when utilizing the currently available research which again was focused on the deleterious effects of under nutrition (Joint Committee on Health Problems in Education., 1957; Michelman,
1976; United States Congress Senate. Select Committee on Nutrition and Human Needs, 1972; United States. Dept. of Agriculture. et al., 1971). Policies to address pediatric obesity did not impact the U.S. policy agenda until the 1990s. Prior to the 1990’s nutrition and food policies were designed to address food insufficiency, food insecurity, and under/ mal nutrition. The first mention of pediatric obesity as a national policy was in an addenda to a 1995 Institute of Medicine Report “Weighing the Options: Criteria for Evaluating Weight-Management Programs” which was focused on adult weight loss programs (Institute of Medicine. Committee to Develop Criteria for Evaluating the Outcomes of Approaches to Prevent and Treat Obesity, 1995). The appendix on childhood obesity was written by Beatrice S. Kanders, one of the committee members, and discussed the state of the science regarding pediatric weight trends and the alarming increase in childhood obesity rate that was seen between NHANES I (1971-75) and NHANES III (preliminary data 1993) with an increase of almost 30% in the 20 year time span (Institute of Medicine. Committee to Develop Criteria for Evaluating the Outcomes of Approaches to Prevent and Treat Obesity, 1995; National Health and Nutrition Examination Survey Data. NHANES I," 1975; National Health and Nutrition Examination Survey Data. NHANES III," 1997). During the final formulation of Healthy People 2000 in 1991 (United States Department of Health and Human Services, 1991), a national goal was set for reduction of adolescent obesity by 5% of the population base level of 11%; however, the Healthy People 2000 priority public health goals did not address childhood obesity prior to adolescence. In 1999, both the
Department of Health and Human Services and the Institute of Medicine called for a national research agenda to build the evidence bases regarding the etiology, progression, definition and prevention of childhood obesity. There was a paucity of evidence, because prior to the late 1990, there were only 3 evidence based studies on obesity rates in children and only 1 study from 1971 that was longitudinal (Institute of Medicine. Committee to Develop Criteria for Evaluating the Outcomes of Approaches to Prevent and Treat Obesity, 1995). The state of the science regarding pediatric obesity was dismal with little scientific evidence but the population trends showed a marked increase in obesity rates in the population (including in children) leading both the NIH and HRSA (MCHB) to determine to set national research agendas to rectify the lack of obesity research data in 2000. However, it took until 2004 for the NIH to release its Strategic Plan for Obesity Research that included a research priority area “to increase the knowledge base for obesity in children” (NIH, 2004), which was closely followed by the IOM report on Preventing Childhood Obesity: Health in the Balance (Institute of Medicine. Committee on Prevention of Obesity in Children and Youth et al., 2004) and the USDA Team Nutrition: Getting it Started and Keep it Going program (United States Department of Agriculture, 2004). With the final release of Healthy People 2010 (HP2010) in 1998, a national priority public health goal of a 5% reduction in pediatric obesity in all childhood age groups, from 5-19 years old was set (United States Department of Health and Human Services, 1991, 1998). HP2010 also called “childhood obesity a public health emergency” and called pediatric obesity an epidemic (United States Department of Health and
Human Services, 1998). The government also reached out to the medical community through the AAP to educate pediatricians and the ADA (American Dietetic Association, i.e. Clinical Nutritionist’s Professional Society) regarding healthy eating habits and appropriate growth tracking of children (2001). The voluntary nature of educating healthcare providers that treat children is not working to stem population obesity; by 2007, when the HP2010 midcourse review data was released, trends indicated that the current policies that were to reduce pediatric obesity by 5% were not working (United States Department of Health and Human Services, 2007). In fact, pediatric obesity rates moved away from trend by an average of 5% for all age groups; what this means is there was an overall increase in obesity rates of > 5% (HP2010 midcourse review, 2007). For some groups such as ethnic and minority children, especially females the increase was greater than 10%; and in younger children (preschool age) the increase was growing exponentially larger each year (HP2010 midcourse review, 2007). Thusly, the focus of U.S. health policy has changed during the course of the 20th century and in the previous decade has begun to focus on the control of childhood obesity; recognition is occurring that the food environment needs to be included in any health policy that impact obesity.

Prior to the 1990’s agricultural policies dealing with nutrition fostered food policies designed to address food insufficiency, food insecurity, and under/ malnutrition (Garvue, Flanagan, & Castine, 1971; Gould, 1972; Gunderson, 1971; Joint Committee on Health Problems in Education., 1957, 1962; National School Lunch Act," 1946; Smith et al., 1971; United States Congress Senate. Select
Committee on Nutrition and Human Needs, 1972; Young, 1971; Young & Nokkeo, 1970) and health policies were designed to reduce disease transmission and severity. The Public Health Service Act of 1944 consolidated and revised all existing legislation relating to the Public Health ("Public Health Service Act," 1944). In the 20th century the main health policy focus for children was the prevention and control of diseases ("Public Health Service Act," 1944); therefore disease diagnosis, epidemiology and immunization were the primary policy programs. The high incidence of malnutrition that was found in military recruits during and post World War II prompted passage of the National School Lunch Program in 1946 specifically to address childhood under nutrition ("National School Lunch Act," 1946) which was expanded in the 1950’s and 60’s to assure adequate milk (calcium) and calorie consumption ("Agricultural Act of 1954," 1954; Agricultural Act of 1956," 1956; Child Nutrition Act ", 1966a; Food and Agricultural Act of 1962," 1962; Food and Agricultural Act of 1965," 1965) and continues to be re-authorized, again stressing caloric consumption rather than diet quality ("An Act to Amend the Child Nutrition Act of 1956 and the National School Lunch Act to Revise and Extend Certain Authorities Contained in Such Acts and for Other Purposes," 1989; An Act to Amend the Child Nutrition Act of 1966 and the National School Lunch Act to Promote Healthy Eating Habits for Children and to Extend Certain Authorities Contained in Such Acts through Fiscal Year 1998, and for Other Purposes," 1994; An Act to Amend the National school lunch act and the Child nutrition act of 1966 in order to revise and extend the summer food program, to revise the special milk program," 1977; An Act to
The School Lunch Program was designed under agricultural policies based on food cost with the predominance of subsidized commodity [cheap] foods being used in the program; calories were increased but nutritional deficiencies were not addressed (Food Marketing to Children and Youth Threat or Opportunity?, 2006). In a 2008 review of school children’s diets, the IOM found that for all children ages 5–18 years, the mean intakes of total vegetables (45%), fruit (80%), whole grains (24%), total meat and beans (70%), and milk (80%) was of the DRI recommendations (Institute of Medicine. Food and Nutrition Board, 2008). Additionally, school children consumed larger than recommended amounts of trans fats (> 400 calories) and added sugars (19-29 teaspoons) each day (Institute of Medicine. Food and Nutrition Board, 2008). The food programs did not limit the types of food that could be purchased and were more focused on increasing calories in children’s diets rather than the nutritional quality of the diet (Institute of Medicine. Committee to Review the WIC Food Packages, 2005). Interestingly, most scientific studies by the IOM from the 1970’s to the 1990s looked at diet and growth in adolescents once they entered the military service; and recommendations on childhood nutrition were geared to produce ‘fit’ military recruits (studies for females looked at milk consumption and calcium intake sufficient to produce health bones and for males looked at nutritional requirements necessary for muscle development and strengthening (Institute of Medicine. Committee on Body Composition Nutrition and Health of Military Women, 1998; Institute of Medicine. Committee on Military Nutrition Research, Carlson-Newberry, & Costello, 1997; Institute of Medicine. Committee
In the previous 60 years (1949 to 1999), agricultural policies were focused on assuring that children were appropriately fed and had enough calories for growth. John Dobbing's pioneering brain maturation studies in the early 1960s created a body of evidence showing consumption of fat was necessary for children to develop normal brain function thereby affecting intelligence (Dobbing, 1964, 1965). Dobbing's studies affect medical and nutrition clinical practice to this day and perpetuate the myth that fat intake should never be restricted in children, even if the child is ‘at risk for’ or obese (Butte, 2006; Dobbing, 1972, 1974a, 1974b; Dobbing & Hopewell, 1971; Embleton, Pang, & Cooke, 2001; Hamilton, Schiller, & Boyne, 1994). The 1960s saw an exponential increase in the amounts of hydrogenated vegetable oil use and was one of the significant changes that occurred in the U.S. diet. Hydrogenated vegetable shortening is primarily produced from soybeans and cotton which are commodity crops that receive large annually production subsidies. Because of many changes to both the soybean and cotton subsidy programs, prices for hydrogenated vegetable shortening are lower than what they would be without federal intervention creating a niche for this alternative inexpensive food product (Beach et al., 2002). Hydrogenated vegetable oil has been produced since 1910 but did not significantly enter the food supply until the 1960s due to the commodity programs that amassed huge stocks of crop commodities (Beach et al., 2002) which because of their low cost were processed into hydrogenated oils to replace the
more costly fats (butter and lard). Hydrogenation creates a more dense fat has a consistency like butter and is much cheaper. The problem with hydrogenation is that the essential fatty acids are no longer active interfering with the satiety response and causing overeating and it also creates trans fats which have been causally linked to high cholesterol levels and coronary heart disease (Institute of Medicine. Committee on the Scientific Evaluation of Dietary Reference Intakes, 2005).

Nor did agricultural policies reflect the large increase in sweetener consumption during the century in the U.S. Nearly all sugars add empty calories to the human diet, but High Fructose Corn Syrup was not even established in the food supply until the early 1970s (Gross, Li, Ford, & Liu, 2004; Major Trends in U.S. Food Supply, 1909-99, 2000). Since its introduction in the late 1960s, in less than 40 years, HFCS has become the predominate sweetener in the American diet; 80 pounds per person per year consumed in 1909 versus 160 pounds per person per year in 1999 (USDA, 2000), some of the rise in HFCS consumption is due to the sugar commodity programs. U.S. agricultural policies for sugar production and import have increased the price of sugar making the low price for HFCS (subsidized by the corn crop program) more attractive for use in food products (Jurenas, 2007). All sugars/ sweeteners add empty calories to the diet but HFCS has thirteen percent more calories per tablespoon than beet or cane sugar (J. E. Brown et al., 2005). The consequence of a diet containing large amounts of HFCS is metabolic; HFCS does not trigger the satiety response like cane or beet sugar, that were previously in the American diet, leading to over
consumption of calories (Bray, Nielsen, & Popkin, 2004; Butte et al., 2007; Montague et al., 1997). HFCS is made from corn but the nutritional value is very different than an ear of corn (J. E. Brown et al., 2005). HFCS is composed of 45% glucose and 55% fructose. Glucose is the form of sugar that is transported in the blood and is used for energy and is regulated by insulin metabolism, but fructose does not stimulate insulin secretion or require insulin to be transported into cells, thus short circuits the insulin-leptin satiety response and does not signal the brain to stop eating (Gross et al., 2004). An associated problem with HFCS digestion is that it requires a different metabolic pathway (Acetyl CoA) than glucose digestion further stimulating fat deposition (Bray et al., 2004; Gross et al., 2004; Sunehag et al., 2002). Fructose is the primary sugar found in fruits and only becomes a problem when high levels are consumed such as in processed foods that do not have the associated nutrients and fiber content of fruits (Bray et al., 2004; Gross et al., 2004; Moshfegh, Friday, Goldman, & Ahuja, 1999; Park & Yetley, 1993; Sunehag et al., 2002). High intakes of HFCS can lead to higher caloric intake, increased bodyweight, miscued hunger signals, thereby decreasing the intake of nutrient-dense foods and increasing insulin resistance and adipose tissue formation (Raben, Vasilaras, Moller, & Astrup, 2002). The subsidized low corn prices created a niche market for highly sweetened cheap foods that contain a high proportion of HFCS (Tillotson, 2003a). A small fast food soda has a caloric content of 150 calories with all the calories from HFCS equaling 41 grams of sugar; there is no other nutritional value in the product, there are no nutrients, vitamins or minerals, supersizing the
soda increase the calories to 410 calories equaling 113 grams of sugar which is almost one third of the daily recommended calorie intake for a child (J. E. Brown et al., 2005).

The historical policy agenda regarding appropriate childhood nutrition for growth never accounted for the changing U.S. food supply; from 1969 to 2000 there was an increase of 500 calories per person per day in the U.S. food supply (Buttet & Dolar, 2008). The policies did not account for the increase in affluence that decreased the amount of income necessary for food purchases; from 1909 to 1990 there was a decrease from >20% to less than 12% of the household budget spent on food (Beydoun, Powell, & Wang, 2008; Boutelle, Fulkerson, Neumark-Sztainer, Story, & French, 2007; Bowman et al., 2004; Burdette & Whitaker, 2004; Demory-Luce, 2005; Hawkins & Law, 2006; Jekanowski, 1999; Table 2.3.1. Percent Change From Preceding Period in Real Personal Consumption Expenditures by Major Type of Product [Data File], 2008; Table 2.3.3. Real Personal Consumption Expenditures by Major Type of Product, Quantity Indexes [Data File], 2008; Trends in intake of energy and macronutrients--United States, 1971-2000, 2004; What we eat in America, 1996). As population affluence increased more meals were eaten outside the home, increasing the consumption of higher fat and non-nutritive food stuffs, such as sweets and convenience foods (Beydoun et al., 2008; Boutelle et al., 2007; Bowman et al., 2004; Burdette & Whitaker, 2004; Demory-Luce, 2005; Hawkins & Law, 2006; Jekanowski, 1999; Table 2.3.1. Percent Change From Preceding Period in Real Personal Consumption Expenditures by Major Type of
Product [Data File]," 2008; Table 2.3.3. Real Personal Consumption Expenditures by Major Type of Product, Quantity Indexes [Data File]," 2008; Trends in intake of energy and macronutrients--United States, 1971-2000," 2004; What we eat in America," 1996). The changing food environment created a situation where children's weights increased approximately 30% in a 30 year period, 1971 to 2000 (Advanced Data, Mean Body Weight, Height, and Body Mass Index, United States1960-2002 2004; Buttet & Dolar, 2008; United States Congress Senate. Select Committee on Nutrition and Human Needs, 1972; United States. Congress. Senate. Committee on Appropriations. Subcommittee on Agriculture Rural Development and Related Agencies., 2001; USDA, 2000). Arcan et al., 2007 suggests that parents have the most influence in their children's consumption of fruits, vegetables and dairy establishing an eating pattern that is the foundation of their adult diet; thusly appropriate access and availability of healthy food choices in the home strongly affect the next generations' diet and food choices (Arcan et al., 2007). Post birth, poor maternal diet quality (with resulting poor maternal nutritional status (Demmelmair, von Rosen, & Koletzko, 2006; Ong, Emmett, Noble, Ness, & Dunger, 2006)) predicted poor diet quality of the child (Lee et al., 2005).

The feeding relationship is complex between the parent and child; effective feeding provides the groundwork for healthy growth and later life healthy eating behaviors (Satter, 1990). For example, parental role modeling directly influences the child's preferences for fruits and vegetables (Bisset, Gauvin, Potvin, & Paradis, 2007). A preference for energy-dense foods is influenced by
past tasting experience and perceived satiety but can be overridden by parental child-feeding styles (Bisset et al., 2007; Oliveria et al., 1992; Orrell-Valente et al., 2007; Satter, 1990). Obesity status in infants is primarily influenced by diet (consumption of appropriate caloric intake) rather than by physical activity (Stunkard et al., 2004). Stunkard, et al also found that growth pattern and size of a 2 year old can be predicted by calories consumed at 3, 6, 9 months; this indicates a correlation between future body size and current metabolism rate measured by energy intake (Stunkard et al., 2004). A common belief (Grummer-Strawn & Mei, 2004) is that solid food supplementation in the first 6 months of life is related to childhood obesity (Grummer-Strawn & Mei, 2004; Kramer, 1981). Two-to-three year old children of mothers who skipped breakfast and did not eat fruits and vegetables had the highest incidence of poor nutritional status (Lee et al., 2005). Additionally, children’s meal frequency was inversely associated with their BMI, thus, children who had less formal meals had higher BMIs (Beyerlein, Fahrmeir, Mansmann, & Toschke, 2008).

Research indicates that fruit and vegetable consumption in children is developed by modeling parental eating habits (Bisset et al., 2007). Evidence also indicates that prenatal and early postnatal exposure to a flavors produced by vegetables and spices enhanced the chance that the infant would eat and prefer that flavor in solid foods after weaning; very early flavor experiences may provide the foundation for cultural and ethnic differences in food preferences (J. A. Mennella et al., 2001). College educated mothers were more likely to provide adequate nutrition for their children; but all children routinely do not to eat the
daily recommended intake of vegetables (J. E. Brown et al., 2005; Hendricks et al., 2006; Otten et al., 2006). Parents, mothers in particular, directly control nutritional status [and eating habits] in children: in higher SES families, restrictive feeding practices occurred when a school-aged child exhibited a tendency toward overweight, but these practices appeared to elicit the opposite response and produce additional child weight gain (Faith et al., 2004).

Evidence indicates that children's eating behavior has familial roots, and particular family behavior patterns are associated with the development of obesity (Bisset et al., 2007). But there is also a genetic component to eating preferences, the TAS2R38 gene has been linked to preferences for sweet taste and research indicates that in children the gene significantly affected eating behaviors (Mennella et al., 2005). Flavor is the primary way that young children determine food acceptance so a genetic basis for food preference will directly affect eating behavior; children who were heterozygous for the TAS2R38 gene added more sweeteners to their food (Mennella et al., 2005). However, by adulthood, there was no correspondence between the RAS2R38 gene and sweet taste preferences, indicating that cultural forces, learned behaviors and experience eventually overrode the genotype (Mennella et al., 2005). Much of the genetic research focuses on explaining how the brain affects the variety of patterns of how human eat. “It should be kept in mind that eating is 100% behavior, and this activity links the internal world of molecules and physiological processes with the external world of physical and cultural systems” (Blundell, 2006), consequently it is hard to distinguish whether human eating is a function
of physiology or environment. Research eventually needs to reconcile the effects of the physiological mechanism responsible for eating control in the obese with the actual displayed eating patterns (i.e. eating phenotypes) and with the cultural environmental pressures that affect eating behaviors (Blundell, 2006).

Obesigenic Food Environment

During human evolution, physical exercise and food procurement have been inseparably interwoven creating a link between eating and physical activity setting and regulating the body metabolism and weight (Barness et al., 2007). However, in the modern environment starvation is rarely an issue and sedentary lifestyles have increased. Consequently the inherited link between eating and physical activity has broken, thus disconnecting human genetics from metabolism leading to a large increase in obesity in the population (Barness et al., 2007). Until 500 generations ago, all humans consumed only unprocessed food foraged and hunted from the environment; the unprocessed food provided a diet high in lean protein, polyunsaturated fats, monounsaturated fats, complex carbohydrates (unrefined), fiber, vitamins, minerals, antioxidants, and other beneficial phyto-chemicals and low in sugars and saturated fats (O’Keefe & Cordain, 2004). This unprocessed food low sugar and fat diet that shaped human genotypes is the opposite of the current diet in the U.S. Agricultural policies established in the 1929-1954 New Deal Period have proven harmful
because the crop support “programs became preferential, profligate, and perennial”, and supported food commodities in a partisan political fashion without consideration of potential harmful nutritional impact that these foods would have on a significant portion of the population (P. Allen, 2004; Luna, 2004). More processed and refined foods were entering the food supply during the course of the century adding more calories with less nutritional value. Comparing the DRI with the food products actually currently produced in the U.S. reveals that there are not enough fruit and vegetables grown to meet the appropriate dietary needs of all Americans (Food Marketing to Children and Youth Threat or Opportunity?, 2006). In 2000 the food supply provided 5.2 servings of fruits and vegetables, including legumes, per capita per day, far below the DRI recommended 7 daily servings (Putnam et al., 2002). Grains, fruits, vegetables, and sugars and sweeteners are the primary source of carbohydrates in the food supply but the types of carbohydrates have shifted from unprocessed to highly processed during the course of the century (Hiza & Bente, 2007). In 1909-19, the major types of carbohydrates consumed were: 55% whole grains, followed by 23% refined and natural sugars / sweeteners, and 15% fruits and vegetables collectively (Hiza & Bente, 2007). However by 2004 the types of carbohydrate consumption had changed to: 40% processed grains, 37% sugars / processed sweeteners, and 15% fruits and vegetables (Hiza & Bente, 2007). Finally, current U.S. food production of dairy was approximately 1.61 daily servings of dairy products, which is again is less than the DRI recommended 2.2 servings (Putnam et al., 2002). Cheese and whole milk account for > 50% of the dairy
products consumed in the U.S. and both are very high in fat, accounting for 22% of the saturated fat in the U.S. diet (Putnam et al., 2002). Federal farm policies related to the U.S. food supply and agricultural production have created a system with the lowest priced foods being those with the lowest nutritional value; commodity prices dictate retail prices with those foods that are highly subsidized having the lowest retail price. A nutritional analysis of the U.S. food supply revealed that the food supply currently did not contain enough of the USDA DRI recommended nutrients to feed the population (Putnam et al., 2002).

In the previous, 50 years, a considerable shift has occurred in the adolescent diet (Cavadini, Siega-Riz, & Popkin, 2000). Over 97% of children under 8 years old had adequate intakes of vitamins and minerals but by age 14 more than 50% of the adolescents had inadequate intakes of nutrients such as Vitamin E, Vitamin A, and Magnesium and greater than 20% had inadequate intakes of Zinc, Phosphorus, Iron, Thiamin, Folate, Vitamin B₁₂, Vitamin B₆ and Vitamin C (Institute of Medicine. Food and Nutrition Board, 2008). Research indicates that psychosocial interactions are also linked to eating patterns and maladaptive eating styles (ones that are not associated with hunger and satiety) in early adolescences (Bisset et al., 2007). From childhood (age 9) to early adolescence (age 14) there is an overall decrease in the consumption of fruit and vegetables and an increase in low quality snacking (Bisset et al., 2007). U.S. adolescents, in general, have a diet high in fat and sodium, and low in vegetables, fruits, whole grains and dairy foods that is not tightly associated with parental dietary intakes (Arcan et al., 2007). But, there was significant
association between frequency of serving vegetables at family dinners and adolescent intakes of vegetables (Arcan et al., 2007). Home food availability of non-nutritious foods (such as chips and soda) was associated with more frequent purchases of fast food by both adolescents and parents (Boutelle et al., 2007). Fast food as the primary component of family meals was associated with an unhealthy food environment in the home, and was also associated with higher consumption of salty snack foods, and decreased intake of vegetable servings (Boutelle et al., 2007). Adult eating behaviors are also developed during late childhood to early adolescence; some of the development of eating patterns is associated with psychosocial tendencies such as dietary restraint (Bisset et al., 2007). A study in Ireland found that children’s intake of micronutrients decreased as the frequency of consumption of foods outside the home increased because a much wider range of more nutritious food is normally found in the home environment (Burke et al., 2007). It appears that during adolescence, diet is more influenced by external factors with adolescents developing eating habits different from their parents (Arcan et al., 2007). Interestingly, as adolescents age over a 5-year period, their parent’s dietary intakes predicted their dietary intakes as young adults; the young adults modeled their parent’s feeding styles and eating practices and ‘reverted’ back to the eating patterns of their family (Arcan et al., 2007).

Adolescent’s diets changed from consumption of lower calorie unrefined foods to one that had large consumption increases in high fat processed potato products (french fries, chips) and high calorie and fat mixed dishes such as pizza
and macaroni and cheese, etc. . . (Cavadini et al., 2000). Also, low fat milk consumption increased with the corresponding decline in higher-fat milk consumption, but total milk consumption decreased by 36% (Cavadini et al., 2000). The decline in milk consumption was accompanied by an increase in the consumption of soft drinks and highly sweetened non-citrus juices (Cavadini et al., 2000). The increase in high fat potato consumption did increase vegetable intake, but the number of servings for fruits and vegetables is still lower than the recommended 5 per day (Cavadini et al., 2000). However, increased consumption of french fries and potato chips is not a good indicator of a healthy nutritious diet. The Survey of Food Intakes by Individuals (CSFII) reveals that 67% of the population age 2 or older consumes less than one serving of whole grains a day and only 10% consume the recommended daily allowance of three or more servings (Putnam et al., 2002). CSFII data also show that more refined grain, fat, cholesterol, and sugar servings are consumed and less whole grains and fiber are consumed than recommended in the DRIs (Putnam et al., 2002). Half of all individuals above age 2, consumed less than 1 serving of fruit per day (Putnam et al., 2002). The types of fruit and vegetables consumed were not those with the highest nutritional content, rather were iceberg lettuce, white potatoes, and canned tomato products which accounted for 43% of fruit and vegetables consumed in the U.S. (Putnam et al., 2002).

Since beef, chicken, diary, and HFCS are central components of the fast food industry, it is plausible to suggest a link between subsidies, poor diet, and obesity (Tillotson, 2003a). The proportion of household food expenditures on
fast food increased from 29% to 34% between 1982 and 1997 (Jekanowski, 1999). Between 1999 and 2003, the global number of food service restaurants grew at a rate of 4% each year to an estimated 11.2 million establishments (Burke et al., 2007). The Happy meal was introduced in 1979, a high calorie, cheap meal with a toy that was marketed directly to children (Linn & Novosat, 2008). The branding of the high calorie low nutrient children’s meals cause problems for parents because many high profile children’s action hero and beloved cartoon characters are paired with these meals (Linn & Novosat, 2008). The fast food industry supplied meals that appealed to parents because of convenience and price but the typical child’s meal including: cheeseburger, small fries and chocolate milkshake from McDonalds has 740 calories, and cheeseburger, small fries and small coke from Burger King has 690 calories (Linn & Novosat, 2008). The calorie content of these meals is almost half of the daily recommended intakes for children 4 to 8 years old and significantly adds to the already high fat content of children’s diets (J. E. Brown et al., 2005). A McDonald’s cheeseburger contributes 22% of the daily recommended calories from fat in a child’s diet, the french fries another 15%, and the milkshake contributes 12%, making the daily fat content of a Happy Meal 59% of the daily calories; the DRI recommendation is to limit calories from fat to below 10% of a daily diet (J. E. Brown et al., 2005).
Childhood Obesity Definition, Scope, Causal Factors

There was no clear consensus on a definition of overweight or obesity in preschool aged (1–5 years) and school aged (6–9 years) children (Institute of Medicine. Committee to Develop Criteria for Evaluating the Outcomes of Approaches to Prevent and Treat Obesity, 1995). In the 1995, the IOM called for further research to provide standard guidelines for screening and identification of children ‘at risk’ for and already overweight because of the lack of clear clinical and research evidence that there was even an obesity problem occurring in children (Institute of Medicine. Committee to Develop Criteria for Evaluating the Outcomes of Approaches to Prevent and Treat Obesity, 1995). In Healthy People 2000, which was written in 1990, the federal government proposed a goal to reduce obesity prevalence to less than 15% percent in adolescents (12 to 19 years) but there were no recommendations for younger children (United States Department of Health and Human Services, 1991). By 1998, the federal government, when writing Healthy People 2010, proposed to reduce obesity prevalence to less than 5% for all children (6 – 19 years) acknowledging that there was little data that addressed “the etiology, treatment, and prevention of childhood obesity” (United States Department of Health and Human Services, 1998). In the late 1990s, the federal government called for a knowledge base regarding pediatric obesity to be developed and for research to determine the definition, etiology, treatment, eating behaviors, weight maintenance requirements, and prevention protocols for obesity in children (NIH, 2004; United
States Department of Health and Human Services, 1998). There was no coordinated federal effort before the mid 2000s to build any type of knowledge base that could be used to formulate policies to respond to the increasing obesity in American children. Between the NHANES Surveys (1976/80 to 2003/4), the percent of children with BMIs above the 85th percentile increased from 6.5% +/- 0.6% to 37.2% +/- 1.9% for 6-11 year olds and 5.0% +/- 0.5% to 34.3% +/- 2.6% for 12-17 year olds (Ogden et al., 2006; C.L. Ogden et al., 2002). African American, Hispanic, and Native American children are at a higher risk for obesity than whites (Ogden et al., 2006; C.L. Ogden et al., 2002; Rosner, Prineas, Loggie, & Daniels, 1998; Salsberry & Reagan, 2005). In just 3 years (2000 to 2003), obesity in American children 2-5 years old increased 3.6% to 13.9% +/- 1.6 (Ogden et al., 2006). Further, the majority of obese American pre-school aged children have not been clinically diagnosed representing an underreporting of pediatric obesity (Riley et al., 2005). However, the appropriate time in the life-course to begin obesity prevention is not yet known. Abrantes, et al (2003) states that “There is an urgent necessity to establish consensual criteria for obesity definition in childhood that would predict morbidity and mortality in adulthood” (Abrantes, Lamounier, & Colosimo, 2003). The difficulty in defining pediatric obesity causes both clinical and policy problems because some individuals can be misclassified as ‘normal or obese’ and then receive interventions inappropriate to their actual nutritional status (Abrantes et al., 2003). Obesity status can change by age; individuals who are normal at one time depending upon their growth pattern may or may not remain in the same. It
is difficult to define obesity limits in children, adult measures of BMI take into account morbidity and mortality data coupled with weight, height and age to determine classification points for obesity; however, using adult standards and applying them to children is more difficult (Abrantes et al., 2003; Cole et al., 2000). Measures used to 'diagnose' overweight children with a very low chance of giving false-positive results are not yet agreed upon; it is particularly important in young, rapidly growing children to have clear definitions of the cut off point for overweight to avoid detrimental to growth food restrictions.

Many parents do not recognized that their child is overweight; research indicates that less than 20% of the parents realized that their pre-school child was overweight and in some studies of toddlers only 2% of the parents recognized that their child was overweight but only a small percentage were concerned about the ramifications of the child’s weight status (Crawford, Timperio, Telford, & Salmon, 2006). Seventy-one percent of parents of overweight 5 to 6-year old children were not at all concerned about their child’s current weight, but 57% of parents of 10 to12-year-old overweight children were concerned about their overweight child (Crawford et al., 2006). Evidence shows that mothers decide if their child is overweight not by using height and weight measures, but by whether or not their child is being teased about their weight or if they have developed limitations in physical activity (Crawford et al., 2006). Most children (43%), their parents (40%), and their pediatricians (33%) all underestimated the child’s degree of overweight when using either a word scale or a picture scale (Chaimovitz, Issenman, Moffat, & Persad, 2008). Clinicians
are reluctant to intervene even if the child is clearly overweight using standard height weight charts as long as the child is ‘eating well’ and doesn’t exhibit any significant disease related co-morbidities (Demory-Luce, 2005; Satter, 1990). Research has demonstrated the need to educate pediatricians to recognize obesity and overweight so that they can provide appropriate counseling (Chaimovitz et al., 2008). Other research suggests that parents do not consider their children as overweight if they were active and had a good appetite (Crawford et al., 2006). Obesity status can change by age; individuals who are normal at one time depending upon their growth pattern may or may not remain in the same category as they age and grow.

Obesity is defined as a body composition with an excess of adipose mass as compared to lean body mass (Maynard et al., 2001). The gold standard method to determine body composition and determine obesity status is to directly measure body fat using dual-energy X-ray absorptiometry (DEXA) but DEXA is costly to perform and not feasible as a diagnostic indicator (Komiya, Masubuchi, Mori, & Tajima, 2008; Maynard et al., 2001). Therefore, adipose mass can be estimated by Body Mass Index, BMI (Maynard et al., 2001). Age and gender affect BMI in children but BMI is not a direct measure of adiposity, sharp upward trends in BMI that occur without a corresponding upward trend in stature do indicate disproportionate increase in adipose tissue which are linked to later life obesity (Barker, 2007a; Eriksson, Forsen, Osmond, & Barker, 2003; Maynard et al., 2001). Though BMI has not been considered accurate in children under age 2 because weight changes are considerable during the first two years of life
(O'Brien, Holubkov, & Reis, 2004), it is important to understand that BMI considers the height of the child as well as weight, and thus, may be less variable than weight change alone. The Rohrer Index has also been used to determine obesity (calculated as the weight in kilograms divided by the height in meters cubed) and has been shown to be less age and height dependent than BMI but there is little research on the correlation of the Rohrer Index with other measurements of body composition (lean muscle or percent body fat), and most importantly, there is no widely available reference population or established cutoff points for the Rohrer index (Komiya et al., 2008). Multiple skinfold anthropometry, bioelectrical impedance analysis are useful as clinically non-invasive, low-cost and transportable methods to measure body composition, but there is little evidence regarding the feasibility of using them longitudinally to monitor obesity status in children (Campanozzi, Dabbas, Ruiz, Ricour, & Goulet, 2008; Komiya et al., 2008). An additional problem occurs because there is a significant lack of agreement between the methods (multiple skinfold anthropometry, bioelectrical impedance analysis and DEXA) so they cannot be used interchangeably to determine body composition (Campanozzi et al., 2008). The discrepancies between methods increase as obesity increases making standardization almost impossible (Campanozzi et al., 2008). Out of all the methods to determine obesity in children, BMI appears to have the highest specificity (>99%) but has low sensitivity (0.60) allowing for clinical underestimation of obesity (Komiya et al., 2008) and children whose BMI is in the upper third of the BMI percentile range had more accurate prediction of actual
adipose-tissue-mass than those who had lower BMIs (Maynard et al., 2001). Racial minority (black and Hispanic) females (5-12 years) had greater BMIs than white females at the same ages; the difference in BMI values between races needs to be further explored to assure that BMI is measuring actual adipose tissue mass (Rosner et al., 1998). Hence, a limitation of using standardized BMIs to determine appropriate weight/height ratios for all children between 2 to 6 years may be related to variation of different ethnic (and gender) groups (Rosner et al., 1998).

Controversy exists regarding the use of BMI in children less than two years old, deOnis, et al (1997) stated that the growth standard for all children irrespective of race or national origin should be based on BMI as a growth reference for breastfeed infants living in healthy environments (de Onis, Graza, & J-P., 1997). The American Academy of Pediatrics in 2006 recommended that pediatricians use change in BMI to identify rate of excessive weight gain relative to linear growth to determine childhood growth patterns, especially obesity risk ("Active Healthy Living: Prevention of Childhood Obesity Through Increased Physical Activity, Pediatrics. 2006;1834-1842," 2006). Agreement on the method to accurately measure body composition and estimate adipose tissue in children is one issue that still needs consensus but once there is a measurement it becomes necessary to have standardized age and gender specific growth measures that clearly define cutoff values for obesity status. Currently, there are two different standards, the WHO 2006 or the CDC 2002 Reference Growth Charts, used to determine the cut off values for obesity in children. Both define
obesity status in children according to percentiles, i.e. weight/ height/ BMI, on age and gender specific growth charts (de Onis, 2004; de Onis, Graza, & Habicht, 1997; C. L. Ogden et al., 2002). Both also define that if the BMI that exceeds the 95th percentile on age and gender specific growth charts (WHO, or CDC) the child is considered obese, BMI between the 85th and 94th percentiles is considered “at risk” for overweight, BMI between the 25th – 84th percentiles is normal and BMI below the 25th percentile is under- or mal-nourished (C. L. Ogden et al., 2002; WHO, 2003). Age specific obesity status can be determined by determining the percentile where the individuals’ BMI, weight and height plot on Reference Growth Charts, but the two growth charts, WHO and CDC, have the cut off percentiles and appearance of the normal curves at different BMI values which is causing significant confusion regarding the clinical identification of childhood obesity.

During 2003, the WHO collected growth data from 8500 children in 6 countries (Brazil, Ghana, India, Norway, Oman and the U.S.) and developed a new set of standardized child Reference Growth Charts (WHO, 1999, 2003). The WHO Reference Growth Charts 2006 purpose was to develop a “scientifically reliable yardstick of children’s growth achieved under desirable health and nutritional conditions” and are based on a prospective, international sample of infants selected to represent an optimum growth pattern as established by using the breastfed infant as the gold standard for growth (de Onis & Blossner, 2000, 2003; de Onis, Garza, Onyango, & Borghi, 2007; WHO, 1999). The breastfed infant was chosen as the gold standard for early life
course growth and is the normative model for human growth irrespective of race or national origin because it is based on a reference population that would reach their maximum genetic growth potential with the effect of early feeding synched to match 10,000 years evolutionary feeding in the human species (de Onis, Graza, & J-P., 1997; Toselli, Argnani, & Gualdi-Russo, 2005). All alternative infant feeding methods can then be evaluated for effect on later life growth, health and development and then compared to the standardized growth chart of the breastfed infant (de Onis, Graza, & J-P., 1997; Toselli et al., 2005). Adoption of the WHO growth standard in the U.S. is controversial; debate ranges from ‘international standards do not accurately reflect population norms’ (de Onis et al., 2007) to ‘U.S. children would not track appropriately on international charts because of differences in nutritional status because of feeding differences during infancy’ (de Onis, 2004). However, U.S. national guidelines (Healthy People 2010) recommend breastfeeding as the optimal method of feeding for infants (United States Department of Health and Human Services, 2007) so growth charts such as the WHO which are based on the breastfed infant as the norm should provide an optimal growth pattern measurement tool for U.S. children. Additionally, the U.S. children’s data included in the WHO sample did have a growth pattern that was on the median of the WHO Reference Growth Charts; however the U.S. children included in the sample were breastfed for > 6 months, which is different than most U.S. infants (de Onis et al., 2007; WHO, 2003). The conflict arises because the CDC also developed new Reference Growth Charts in the same time period; but the CDC charts only include BMI for children over 2
years of age ("Centers for Disease Control and Prevention, National Center for Health Statistics.  CDC growth charts: United States.," 2000b; de Onis et al., 2007; C.L. Ogden et al., 2002). The CDC Reference Growth Charts are based on U.S. only data sets; the mathematical modeling that was used to smooth the growth patterns because of the small sample size (<100 infants for some time points) might have influenced the patterns to reflect heavier weights as the norm in the United States ("Centers for Disease Control and Prevention, National Center for Health Statistics.  CDC growth charts: United States.," 2000a; de Onis et al., 2007). Variability in height, supine versus standing heights, in some of the data sources also caused a break at 2 years between the growth charts of infants and children; the break leads to an underestimation of overweight and an overestimation of under nourished infants on the CDC charts (de Onis et al., 2007). Healthy breastfed infants plot on the mean (50th percentile) on the WHO charts, but plot well below the 50th percentile on the CDC charts from 2 months onwards (de Onis et al., 2007). This creates a conundrum in using the CDC charts to measure breastfed infant growth because their growth pattern is different than the CDC derived growth pattern (de Onis et al., 2007). The differences between the CDC (C.L. Ogden et al., 2002) and the WHO (WHO, 2003) charts are largest from birth to 2 years because of methodological differences (smaller sample size for Birth to 2 months in the CDC sample), different feeding methods (more formula-feeding, in the U.S. versus the breastfed international sample), and more variability in the height measurement in the CDC sample (Kuczmarski et al., 2000). The shape of the curve of the CDC growth pattern reflects shorter and
heavier growth as the norm compared to the WHO charts (de Onis et al., 2007). If the CDC percentiles were transposed onto WHO growth charts, the results would reflect lower rates of under nutrition and higher rates of overall obesity; meaning that the CDC charts under-report obesity and over-report under nutrition (de Onis et al., 2007). An over estimation of under nutrition in infants may cause U.S. pediatricians to recommend early feeding supplementation leading to less exclusive breastfeeding, faster growth, and increased deposition of adipose tissue in early life (Bogen et al., 2004; Kramer, 1981; Schanler, O'Connor, & Lawrence, 1999). The first wide scale field testing of the World Health Organization 2006 growth standards in the U.S. and Canada has been completed; the study quantified differences between the WHO 2006 and 2000 CDC growth charts for children younger than 2 years (Nash, Secker, Corey, Dunn, & O'Connor, 2008). The WHO growth charts identified more children younger than 2 years as ‘at risk’ for overweight or obesity (21.0%) compared to the CDC charts (16.6%) and fewer children as under nourished (18.6% WHO vs. 23.0% CDC) (Nash et al., 2008). The data describes for the first time the difference in the number of children screened as under nourished (4.4% decrease) or over nourished (4.4% increase) when using the WHO growth charts instead of the CDC charts in a tertiary care setting in North America (Nash et al., 2008). An effort needs to be made to obtain a general agreement on the definition of obesity in children, but even without a standard definition research is continuing to elucidate the factors that lead to obesity.
Many parental factors have been causally linked to child obesity status and can be classified as genetic and environmental/behavioral influences, respectively. The genetic factors are: maternal weight gain during pregnancy (Ravelli, van Der Meulen, Osmond, Barker, & Bleker, 1999), parental obesity status-BMI (Robl et al., 2008; Whitaker, 2004) and parental race (Lombard, Forster-Cox, Smeal, & O'Neill, 2006; C. L. Ogden et al., 2002). The environmental/behavioral factors are: maternal nutritional status during pregnancy (Ravelli et al., 1999), breastfeeding intention and duration (Arenz, Ruckerl, Koletzko, & von Kries, 2004; Bogen et al., 2004; Thomas Harder, Bergmann, Kallischnigg, & Plagemann, 2005; Oddy et al., 2006), family diet quality (Demmelmair et al., 2006; Ong et al., 2006), family socio economic status (Burdette & Whitaker, 2004), maternal educational level (Grummer-Strawn & Mei, 2004; Hawkins & Law, 2006; Oddy et al., 2006; Scariati, Grummer-Strawn, & Fein, 1997), and maternal smoking (Ong, Preece, Emmett, Ahmed, & Dunger, 2002). Individual factors also have been causally linked to childhood obesity status, and again can be classified as genetic and environmental/behavioral factors. They are: birth weight (de Onis et al., 2007; C. L. Ogden et al., 2002; Saha et al., 2005; Salsberry & Reagan, 2005), early life course programming (Barker, 1995b), gender and inherited genotypes that become “thrifty” phenotypes (Barness et al., 2007; Ordovas, 2007; Qi, Shen et al., 2004), birth weight (Parsons, Power, & Manor, 2005), liquid diet weaning age/introduction of solid foods (Michels et al., 2007; Owen, Martin, Whincup, Smith, & Cook, 2005), poor nutrition (Kranz, Lin, & Wagstaff, 2007; Kranz, Smiciklas-Wright, Siega-Riz,

The prenatal period, infancy, and early childhood are the life stages which are particular vulnerability for development of obesity because they are unique periods of rapid cellular differentiation and development (Barness et al., 2007). The relationship between early growth (body size and growth rate) and adult diseases are postulated to be the consequences of 'programming', where a stimulus or insult at a critical, sensitive period in early life has permanent effects on structure, physiology and metabolism (Godfrey & Barker, 2001). The plasticity and programming of the fetus results from fetal adaptation in utero because the maternal-placental nutrient supply fails to match the fetal nutrient demand (Godfrey & Barker, 2001). Genetic factors related to obesity are just beginning to emerge postulating that the increasing prevalence of population obesity might be the interaction of a Paleolithic genome that creates a “thrifty” phenotype with the modern environment that has changed the maternal in utero environment (Barness et al., 2007). The expression of genetic tendencies
toward obesity that were advantageous for survival in a Paleolithic environment may not be in the modern environment (Barness et al., 2007). The Paleolithic genome which assured survival in during periods of food shortages by storing fat and increasing tissue resistance to insulin now appears to be a common underlying trigger of both obesity and diabetes (Barker, 2006; Barker, Osmond, Forsen, Kajantie, & Eriksson, 2005; Barness et al., 2007; O'Keefe & Cordain, 2004). Thusly, the “thrifty” phenotype turned on in utero during plasticity in fetal development may be an important explanation of an inherited tendency toward obesity (Barness et al., 2007). A significant body of research has revealed the strong relationship between the impact of intrauterine conditions on offspring health, intelligence, emotional development, and even SES during later life (Kajantie et al., 2005).

One of the major determinants of fetal growth and the in utero environment is maternal nutritional status during pregnancy (Barker et al., 2005; Godfrey, Redman, Barker, & Osmond, 1991; Godfrey et al., 1996). Additionally, there are other factors which also affect the maternal in utero environment including the mother's body composition, endocrine profile, diet, and physical activity around the time of conception (Inskip et al., 2006). A mother's ability to support a fetus is established during her own fetal life and by her nutrition in childhood and adolescence which determines her body size, body composition and metabolism (Hales & Barker, 2001). Consequently, the previous generations’ body composition greatly affects the next generations’ composition (Hales & Barker, 2001). Fetuses in chronically malnourished populations show a
pattern of reduced growth probably due to not only to poor pregnancy nutrition but also due to the body composition of the mother (Law et al., 2001). Body composition of the mother before conception not just during pregnancy appears to also affect fetal development (Phillips et al., 2000) and the maternal diet during pregnancy can alter characteristics of the offspring (Godfrey & Barker, 2001; Hales & Barker, 2001). In humans, a diet high in protein, low in carbohydrates during pregnancy produced higher systolic blood pressure in the offspring (Shiell et al., 2001). There is sparse longitudinal evidence regarding the relationship between weight gain during pregnancy/ maternal nutritional status during pregnancy and offspring obesity status (Raiten, Kalhan, & Hay, 2007; Ravelli et al., 1999).

Fetal growth has direct effects upon later life growth and metabolism, including the development of diseases such as obesity (Barker, 1995b). Adaptations to under or over nutrition that occur during fetal development can permanently alter the structure and function of the body (Hales & Barker, 2001). However, the adaptations that enable the fetus to continue to grow may nevertheless have adverse consequences for health in later life (Roseboom et al., 2001). Therefore, diseases like coronary heart disease could be viewed as the price paid for successful adaptations to an adverse intra-uterine environment (Roseboom et al., 2001).

Plasticity during development enables the production of phenotypes that are better matched to the environment than would be possible if the same phenotype was produced in all environments (Barker, 2006). However, the fetal
environment may not reflect the environment the individual will experience as an adult and could cause phenotypes that would not be adapted to their adult environment. Additionally, heritable homozygous genotypes with obesity alleles predispose the offspring to obesity with and without environmental triggers. But “thrifty” phenotypes which are expressed as high BMIs and high percent body fat compositions are heterogeneous phenotypes which results from the combined effects of genes, environmental factors, and their interactions (Borecki, Rice, Bouchard, & Rao, 1991). It appears that there is prominent heterogeneity between generations suggesting that there may be significant developmental (genetic or environmental) effects during development to adult pattern phenotypes, particularly for the complex indicators of body composition (Borecki et al., 1991). The most current research evidence (2005) has shown that there are 176 human obesity single-gene mutations (alleles) in 11 different genes and 50 allele loci related to human obesity related Mendelian syndromes with causal genes or strong candidates identified for most of the syndromes (Rankinen et al., 2006). Genetic mutations in leptin (LEP) and its receptor (LEPR) have been shown to result in a severe obesity phenotype (Clement et al., 1998; Montague et al., 1997; Rolland et al., 1998). Leptin deficiency because of the mutation in the LEP is associated with both early-onset morbid childhood obesity and stunted pubertal development indicating that the endocrine system is involved when obesity geno- and phenol- types are selected (Clement et al., 1998; Rolland et al., 1998). Additionally, adipose (fat) cells secrete hormones and cytokines that if mutated, or if the adipose tissues’ receptors have mutations, fat and glucose
metabolism and storage are affected which can lead to obesity phenotypes (Chang, Lee, Chang, Lee, & Chuang, 2008; Fasshauer & Paschke, 2003). Fetuses in early human development may have to adapt to a limited supply of nutrients, and by this adaptation, permanently change their physiology and metabolism originating a number of adult diseases including coronary heart disease, stroke, diabetes, obesity, and hypertension (J. G. Eriksson, T. Forsen, J. Tuomilehto, C. Osmond, & D. J. Barker, 2001). Mechanisms of developmental plasticity and compensatory growth in early life have been directly linked to higher risks of metabolic diseases such as coronary heart disease and type 2 diabetes in later life (Barker, 2002). The recently discovered, FTO gene on chromosome 16, is strongly associated with control of body mass index; 16% of adults who are homozygous for the allele weighed significantly more and had a 1.67-fold increased risk of obesity when compared with those without the allele (Barness et al., 2007). The association in people with and without the FTO allele was observed beginning at 7-years of age and demonstrated specifically an increase in fat mass (Barness et al., 2007). The PLIN gene which modulates adipocyte lipid metabolism has been linked to a statistically significant association in women with obesity risk (Qi, Corella et al., 2004). Women with the recessive allele had lower obesity risk, lower plasma glucose and lower weights than women with the wild type genotype but the lowered risks were not found in males, thus, statistically significant gene-gender interactions were determined (Qi, Corella et al., 2004). Gene mutations that affect the efficiency of muscle metabolism and the composition of skeletal muscle also affect BMI by altering
the lean muscle to adipose tissue ratios and the relative energy expenditures during physical activity; the Quebec Family study found that certain alleles of the gene, UCP3, expressed primarily in skeletal muscle by producing heat instead of ATP during exertion were directly related to the phenotypic expression of BMI, percent body fat and skin-fold thickness (Lanouette et al., 2001). In another study, lower levels of expression of calpain 3 protein (coded for by the CAPN3 gene) in skeletal muscle was associated with reduced carbohydrate oxidation, elevated circulating glucose, and also with increased body fat (Walder et al., 2002). It appears that alleles of the CAPN3 gene particularly control the amount of abdominal fatness which is linked to obesity related co-morbidities (Walder et al., 2002). In twins studies, genetic analyses showed that BMI (~60%) and waist circumference were significantly affected by genetics but there was also a 40% independent genetic effect for both (Wardle, Carnell, Haworth, & Plomin, 2008). Interestingly, the environmental effect for BMI status in twins was very small, <1%, (Wardle et al., 2008). Li et al postulates that because of the genetic link to obesity that reductions in the incidence of childhood obesity in the current population may reduce obesity in their children, i.e. future generations (L. Li, Law, Lo Conte, & Power, 2009). In summary, widespread evidence shows that inherited genetic factors predispose an individual to obesity (O'Rahilly & Farooqi, 2006). It has been assumed that these inherited factors would influence either metabolic rate or the selective conversion of excess calories into fat; but as evidence accumulates “... it is notable that, thus far, all monogenic defects causing human obesity actually disrupt hypothalamic pathways and have a
profound effect on satiety and food intake” rather than on metabolism and “it seems that from an aetiological/genetic standpoint, human obesity appears less a metabolic than a neuro-behavioural disease” (O’Rahilly & Farooqi, 2006).

When fetal growth is differentially changed, organ systems through the entire body develop differently from the norm. Disproportionate fetal growth is associated with the redistribution of blood flow to support the brain, heart and adrenal glands instead of other organs including the liver and kidney (Barker, 2004). Small at birth persons are vulnerable to later disease through three kinds of process: less functional capacity in key organs, such as the kidney (hypertension is initiated by the reduced number of glomeruli); setting of hormones and metabolism (i.e. undernourished fetus establishes a “thrifty” way of handling food maintaining high blood glucose concentrations for the benefit of the brain); and are more vulnerable to adverse environmental influences in later life (Barker, 2006). "Thrifty" metabolic settings include resistance of tissues to the effects of insulin predisposing the individual to either early onset diabetes or metabolic syndrome (Barker et al., 2005; Prentice, 2005). New research indicates that genetic variations of the human adiponectin gene are associated with these “thrifty” metabolic phenotypes such as: obesity, insulin sensitivity, and diabetes (Loos et al., 2007; W. S. Yang et al., 2007). Babies who are thin or short at birth lack muscle which persists into childhood because there is little cell replication in muscle after birth; suggesting that rapid weight gains in the postnatal period lead to disproportionately high fat mass versus muscle mass (Barker et al., 2005). Under nutrition during early development when followed by
improved nutrition causes compensatory growth which has biologic costs such as shortened life span due to a higher rate of cell division causing rapid shortening of the protective ends of the chromosomes, hastening cell death and organ degradation (Barker, 2002). Reduced growth during gestation and during infancy is also associated with an increase in age related diseases (Hales et al., 1991). Poor fetal growth, including low early weights, thinness, and shortness at birth, are related to raised plasma fibrinogen (Barker et al., 1992), raised serum cholesterol concentrations (Fall et al., 1992), impaired glucose tolerance (Hales et al., 1991), and reduced arterial compliance (Martyn et al., 1995). Low weight gain in infancy coupled with rapid childhood weight gain are factors in adult development of cardiovascular disease (Ravelli et al., 1999). Risk for disease fell with increasing birth weight and rose with increasing BMI from 3 to 11 years of age (Barker, 2002). Birth weight alone is not sufficient to represent the entire fetal development experience (Barker, 2003). Both early size and growth rates had effects on the propensity for developing later life co-morbidities; the combination of smaller birth size with high early growth rate raised the likelihood of developing adult co-morbidities especially obesity (Barker, 2002).

Evidence now shows that people who were small at birth remain biologically different to people who were larger (Barker, 2003). Infants who have low birth weight, are thin at birth, and lack muscle will have a disproportionately high ratio of adipose-to-lean mass if they become overweight (J. Eriksson, T. Forsen, J. Tuomilehto, C. Osmond, & D. Barker, 2001). Infants with low birth weight are especially vulnerable to excess fatness, disproportionate fat to lean
tissue mass in adulthood at any/all BMIs (Barness et al., 2007). Infants who were small at birth lack muscle, a deficiency that persists into childhood (there is little cell replication in muscle after birth), suggesting that rapid weight gain in the post natal period leads to disproportionately high fat mass rather than muscle mass (Barker et al., 2005). The higher percentages of fat-tissue-mass increases the risk of coronary heart disease and associated co-morbidities (J. Eriksson, T. Forsen, J. Tuomilehto, C. Osmond, & D. Barker, 2001). Conversely, high BMIs associated with high birth weights and heights may reflect increased lean-tissue-mass which does not affect coronary heart disease risk (J. Eriksson, T. Forsen, J. Tuomilehto, C. Osmond, & D. Barker, 2001). BMI of both genders increases as the child ages and is similar for all races; however there is more variation in females which is probably due to females having a higher percentage of adipose-to-lean mass than males (Rosner et al., 1998).

Maternal pre-pregnancy BMI is a strong predictor of infant adipose tissue mass at birth (R. Li, Jewell, & Grummer-Strawn, 2003). Higher percentage body fat in infants has been related to both gestational age and maternal pre-pregnancy overweight or obesity status (Influence of Pregnancy Weight on Maternal and Child Health. Workshop Report., 2007; Salsberry & Reagan, 2005). Evidence has shown that maternal obesity before and during pregnancy doubles the risk of obesity in the children at 2-4 years of age (Whitaker, 2004). Most of the time the association between maternal BMI and offspring BMI does not appear until the child is an adult (Influence of Pregnancy Weight on Maternal and Child Health. Workshop Report., 2007). In children born in the previous 30
years, evidence indicates that genetics was the main influence on both BMI and abdominal adiposity; confirming that BMI is a highly heritable geno- and phenotype (Wardle et al., 2008). Excessive BMIs in both parents during childhood and adulthood were associated with a higher BMI and risk of obesity in their offspring (L. Li et al., 2009). In the previous 30 years, childhood BMI increased on average by 0.25-1.10 between the parent and child generations and depending on the sex and age group overweight / obesity increased from 10% to 16% between the successive generations (L. Li et al., 2009). Both parents' BMI in childhood and adulthood independently influenced their offspring BMI, but no significant difference in the strength of influence was observed between the parents (L. Li et al., 2009). For example, the adjusted increase in BMI for offspring (4-8 years old) was equivalent to 0.37 and 0.23 for a 1-SD increase in maternal BMI at 7 and 33 years old, respectively; a similar increase in offspring BMI was observed for risk of overweight/obesity when compared to paternal BMI (L. Li et al., 2009). Other studies have shown that both maternal BMI and post birth infant weight gain was significantly associated with a child’s body composition, lean to fat tissue mass ratio (Beyerlein et al., 2008).

Breast feeding, currently, is the only early life identified factor with a considerable body of established evidence that shows a slight association with a reduction for later life obesity (Arenz et al., 2004; Bogen et al., 2004; Cope & Allison, 2008; Demmelmaier et al., 2006; Gartner et al., 2005; Thomas Harder et al., 2005; T. Harder, Schellong, & Plagemann, 2006; Mayer-Davis et al., 2006, 2007; Oddy et al., 2006; Owen et al., 2005; Rose et al., 2006). Maternal pre-
pregnancy obesity is associated with poor lactation, regardless of pregnancy weight gain, obese mothers were less likely to initiate and sustain breastfeeding than those with a normal pre-pregnancy BMI, so offspring from non-obese mothers were more likely to breastfeed (R. Li et al., 2003; Oddy et al., 2006). This further confuses the relationship between breast feeding's protective effect against obesity since mothers who were not obese were also more likely not to have genetic factors that influenced obesity and were more likely to have a healthier lifestyle (R. Li et al., 2003; Oddy et al., 2006). Since causality has not been categorically determined between early life breast feeding and a protective effect against later life obesity, further research is indicated (Cope & Allison, 2008). A protective effect of breast feeding has been shown in some studies such that breast fed infants develop into normally sized school aged children (Kramer, 1981; Liese et al., 2001). Higher rates of obesity developed in 4 year old children who had either never breast fed or breastfed less than 7 weeks; lower rates of obesity were found in children who had breast fed for greater than 7 weeks without concurrent formula supplementation (Bogen et al., 2004). But the critical duration for breast feeding to have a significant effect on decreasing childhood obesity is still not uncontroversially determined, in some studies it was 6 months with concurrent formula usage and in others 16 weeks without formula usage (Bogen et al., 2004). However, breast feeding duration did show a dose-response, protective relationship with the risk of overweight among white children but the dose response relationship was not observed in other races (Grummer-Strawn & Mei, 2004). Breast feeding duration with and without concurrent
formula supplementation correlated with decreasing obesity among White, but not Black, children (Bogen et al., 2004). Again though, there were differences between the races not only in breast feeding but in socioeconomic status and education which are known to change life style habits which lead to better nutrition and healthier environment (Bogen et al., 2004). Predictors of successful breastfeeding duration again link to overall healthier lifestyles and are higher socioeconomic status (Ahluwalia, Morrow, Hsia, & Grummer-Strawn, 2003) and a college education (Ryan, Zhou, & Gaston, 2004). Even matched for comparable SES, Blacks have consistently lower breastfeeding rates, 14% lower than Whites which could be one of the reasons for the higher pediatric obesity rate in blacks in the United States (R. Li, Fridinger, & Grummer-Strawn, 2004). The National Immunization Survey (2001) found that 71.4% of all U.S. children had been breastfed for some duration (i.e. one breast feeding session in the hospital is included in the duration percentage), however exclusive breast feeding rates are very low in the United States (at 42.5% at 3 months- and 13.3% at 6 months-post birth) (R. Li, Darling, Maurice, Barker, & Grummer-Strawn, 2005).

The race of both parents impacts the child’s obesity status (C. L. Ogden et al., 2002; Salsberry & Reagan, 2005), again a link to either a heritable genetic component or a significant difference in behavior and environment between the races. Recent evidence indicates that the specific level of co-morbid health risks associated with a given level of obesity may be different depending on gender, race and socioeconomic condition (Paeratakul, Lovejoy, Ryan, & Bray, 2002). There is a paucity of evidence regarding when in the life course obesity
develops, however, the time to overweight during childhood and adolescence has been determined to vary by race, with obesity developing 25% earlier in blacks than whites (Saha et al., 2005). According to the Healthy People 2010 (HP2010) midcourse review, obesity rates for black children (6-19 years) increased from 14% to 21% in the past 5 years as compared to whites (10% to 14%) in the same time period (United States Department of Health and Human Services, 2007). Obesity rates have been consistently higher in minority populations (blacks 30 - 39%) as compared to whites (22 - 30%).and the HP2010 midcourse review showed that obesity rates have increased overall 9% in blacks and 8% in whites (United States Department of Health and Human Services, 2007). These statistics need to be interpreted with care since race is also related to food insecurity (Williams, 2002). Minorities, especially urban blacks, have less access to and availability of healthy foods (Williams, 2002). Rather than a genetic predisposition to obesity, the differential between the races maybe an unhealthily food environment as the cause of obesity in minorities (Williams, 2002). Living in a rural area and being black was a significant predictor for higher intakes of total fat, saturated fat, cholesterol, and sodium (Johnson, Guthrie, Smiciklas-Wright, & Wang, 1994).

Data conflicts regarding the effect of SES on childhood obesity and feeding practices. (Burdette & Whitaker, 2004) Some studies have determined that high SES increases the risk of family obesity (Wang & Lobstein, 2006) while others have found relationships between lower family SES and higher BMI in children (Ness et al., 2006). In lower SES families there is a perception that if a
child is “chubby” that means the child is healthy (Crawford et al., 2006). Lower SES families consume more sweetened drinks than higher SES families and there has been a link between consumption of sweetened drinks and obesity (Hawkins & Law, 2006; Welsh et al., 2005). Children's familial predisposition to obesity may differentially affect their beverage consumption patterns, especially since sweetened beverages are cheaper than milk or 100% fruit juice (Kral et al., 2008). Systematic reviews have indicated that excessive fruit juice consumption relates to frequency of overweight in children (Hawkins & Law, 2006; Welsh et al., 2005). Consequently, the link between eating behavior, growth pattern and SES needs to be further explored. Children with a high risk for obesity consumed a greater percentage of daily calories from beverages at 3 years of age, more fruit juice at ages 3 and 4, more soft drinks (including fruit juice) at ages 3-5, and more soda at age 6 compared to children not at risk (Kral et al., 2008). Longitudinal analyses showed that a greater 3-year increase in soda intake was associated with an increased change in waist circumference, whereas a greater increase in milk intake was associated with a reduced change in waist circumference (Kral et al., 2008).

The effect of maternal smoking pre- and during pregnancy on offspring obesity status is not fully understood. (Salsberry & Reagan, 2005) Smoking during pregnancy may be a significant risk factor for development of overweight during childhood (Salsberry & Reagan, 2005). Infants of mothers who smoked during pregnancy were generally small at birth, but exhibited rapid catch-up growth during the first year post birth (Ong et al., 2002) and could predispose the
child to later life obesity (Barker et al., 2005). Children whose parents smoked more than a half-a pack of cigarettes per day had higher saturated fat and cholesterol intakes and lower fiber intakes in comparison to children of non-smokers (Johnson, Wang, Smith, & Connolly, 1996).

Pediatric obesity is a critical precursor of adult obesity because the early onset of obesity is the forerunner of lifespan obesity (S. S. Guo et al., 2002; Must et al., 1999) with obesity status appearing to persist over time, a healthy weight 2 year old child is less likely to become overweight at a later age (Salsberry & Reagan, 2005). Twenty-six percent of children obese at 6 months to 5 years of age were also obese as adults; the risk ratio is 1.77 for an obese infant becoming an obese adult as compared to a non-obese infant (Garn, 1985). Obesity trends from the first year of life remain consistent corresponding to obesity during adulthood (Garn, 1985; Garn & LaVelle, 1985; S. S. Guo et al., 2002; Vogels et al., 2006). Modeling of obesity risk factors and early growth have shown that a growth pattern established at a very young age (< 2-4 years) persists throughout childhood and can be used to identify children who are at risk for obesity (Salsberry & Reagan, 2005). The relative risk for becoming an overweight child among overweight infants (versus non-overweight infants) at 1 and 2 years old was 4.3 times and 3.5 times the normal weight infants' respective risk (Mei, Grummer-Strawn, & Scanlon, 2003). A thirty year study in girls showed that obesity status in adolescence predicted obesity status as an adult; the highest risk factor for adult obesity was low educational attainment and obesity as an adolescent (Iughetti et al., 2008). Low birth weight children have the highest risk
ratio to remain overweight once they become overweight when compared to normal- and high- birth weight children (Mei et al., 2003). Growth rates in infants correlate to obesity rates in school-aged children (age 5) and adults (Baird et al., 2005). A majority of overweight 3 year-olds (62.5%) were still overweight a year later and very few non-overweight 3-year-olds (4.1%) became overweight a year later (Mei et al., 2003). One year BMI has been related to 12 year BMI (Nader et al., 2006; Vogels et al., 2006). Three-, six- and nine-month caloric consumption predicts body size, growth pattern and metabolic rate at two years (Stunkard et al., 2004); and six-month BMI predicts adult obesity just as well as it predicts adolescent BMI (Eriksson et al., 2003).

Consequences of Pediatric Obesity

The lifespan costs associated with obesity related morbidity and mortality increases dramatically if obesity is developed in childhood (Deckelbaum & Williams, 2001). Children who are overweight have a tendency towards hypertension, a shortened life expectancy, increased diabetes risk, increased osteoporosis risk, increased destruction of the hips, premature puberty, and hepatic steatosis (i.e. fatty liver) (Barker, 2007a; Barness et al., 2007; Eriksson et al., 2003; Erlanson-Albertsson & Zetterstrom, 2005; Law et al., 1993). Children with high levels of cardiovascular risk tend to have high level as adults and the converse is also true (Barker, 1999, 2007a; Eriksson et al., 2003; J. G. Eriksson
Disease risk persists from childhood to adulthood with obesity linked to numerous co-morbidities (Must et al., 1999). Overweight and obesity during childhood are risk factors for increased co-morbidity; obesity co-morbidities not only are exhibited in adulthood, but, are also exhibited in childhood. The extension of the duration of expression of co-morbid conditions increases the extent and impact of the associated disabilities from the co-morbidities (Deckelbaum & Williams, 2001). The increase in co-morbid disease because of obesity also relates to worker productivity and has a direct costs to society; there is a distinct inverse relationship between obesity and productivity with obesity associated with both increased disability and early workforce retirement (Popkin, Kim, Rusev, Du, & Zizza, 2006). Additionally, the onset of obesity in early life has created a cadre of employees who are more frequently sick and have limitations on work as compared to normal weight individuals; leading to approximately a 33% decrease in overall U.S. productivity (Popkin et al., 2006). Predictive models show that by 2025, the indirect costs of lost productivity (increased healthcare costs, more sick time and reduced work capacity) due to obesity could be almost 10% of total GNP (Popkin et al., 2006). Further research shows that (controlling for education and work experience) overweight and obese employed adults are more likely to earn lower wages in the labor market as compared to normal weight individuals (Z. Yang & Hall, 2008) because the obese are more likely to suffer chronic diseases and/or related acute medical events that lead to more expensive medical bills; thusly
employers shift the higher medical care cost to the heavier employees by offering lower wages (Z. Yang & Hall, 2008). Health care expense for medical care for the obese are between 12 (males) and 17 (females) percent higher than for normal weight individuals (Z. Yang & Hall, 2008). The Bogalusa Heart Study showed that over 60% of the overweight 5-10 year olds had cardiovascular risk factors such as hypertension, hyperlipidemia or elevated insulin levels (Freedman, Dietz, Srinivasan, & Berenson, 1999). Many of these same children (~20%) had 2 or more cardiovascular risk factors (Freedman et al., 1999). Overweight and obese children are exhibiting diseases that used to be considered ‘adult diseases’ such as hypercholesterolemia and type 2 diabetes (Deckelbaum & Williams, 2001). Because pediatric obesity is correlated with a number of serious medical complications and increased mortality in both children and adults (Power et al., 1997a, 1997b), prevention of early development of obesity without affecting the normal growth pattern is important.

Conceptual Frameworks

For a policy issue such as pediatric obesity, it becomes necessary to cross disciplinary realms and combine medical and public administration theories so that the usual conceptual limitations are minimized when translating from macro-level national health policy to micro-level individual health. According to Brown and Summerbell, “There is a need for research to view behaviour change
within the context of an obesigenic environment (T. Brown & Summerbell, 2008)

accordingly obesity policies that affect behavior are not enough on their own but
must be coupled with policies that affect both the food supply and the build
environment. Normative medical theories to explain health and health behaviors
begin with a basic assumption regarding a disease or disease process.

Individual interaction with a disease is the basis of the micro-level medical
theories. Personal individual factors are interlaced with psychosocial and
behavioral factors to conceptualize individual level disease. Social cognitive
theories like the Health Belief Model (Rosenstock, 1982; Rosenstock, Strecher, &
Becker, 1988), Stages of Change Theory (J. M. Prochaska, Prochaska, &
DiClemente, 1992; J. O. Prochaska & Velicer, 1997) and the Theory of
Reasoned Action (Ajzen & Fishbein, 1980; Fishbein & Ajzen, 1975) explore the
relationships between disease status and health behaviors where individual
cognition, beliefs, perceptions affect health decisions; the construct of the
physical environment is only used tangentially in these types of medical model
health theories. Theories of health create a context of meaning within which the
clinician diagnosis and patients comprehend individual body experiences. Both
medical professional and patient decisions regarding health behaviors are used
as factors to affect individual level disease status. The individual is seen as the
crucible where all factors, including environment created by collective choice
policies, individual behavior, social attitudes and beliefs, combine to create a
specific disease state. All medical theories have the same paradigmic fault when
dealing with public health policy issues; the theory, model, or framework all deal with a ‘disease’ and obesity is not per se a ‘disease’. When dealing with a ‘disease’ the practitioner then lapses into a mode of prevent, manage and/ or treat the disease, once this occurs, societal policy is no longer relevant, the relevant issue is now a disease in an individual and society is now tangential as a vector pool that either caused the contamination of the individual or that is at risk from contamination by the individual.

For example, The Health Belief Model (HBM) (Rosenstock et al., 1988) The Stages of Change Theory - Transtheoretical Model (J. O. Prochaska, 1991, 1995; J. O. Prochaska & Velicer, 1997), and The Theory of Reasoned Action (Ajzen & Fishbein, 1980), all have limited explanatory potential when applied to government policy related to a life course population problem such as obesity. However, The Theory of Developmental Origins of Adult Diseases (Life-course Model of Health) proposed by Baker in the 1980s states that characteristics and health of the infant and influences from the environment during infancy, toddlerhood, and early childhood are associated with the development of adult acute and chronic disease, disability and death (Barker, 1995b). Barker’s theory has also come to be known as the Life-course Model of Health (Forrest & Riley, 2004; Halfon & Hochstein, 2002; Hertzman, 1999).

Normative public administration theories on the other hand, elucidate policy development in a given political system and begin with a basic assumption that final collective choice policies will change the bio-physical environmental context thusly impacting the populations’ behaviors. Public administration
theories such as the *Institutional Analysis and Development Framework* (Ostrom, 2007), *Advocacy Coalition Framework* (Sabatier, 2007; Sabatier & Jenkins-Smith, 1993), and the *Punctuated Equilibrium Model* (Baumgartner & Jones, 2005; Zahariadis, 2007) explore the translation of societal problems into governmental policies aimed to ameliorate the cause and illustrate the explanatory gap that occurs when trying to predict individual level health behavior change when the environmental context is modified by public policy change. Public Administration theories account for individuals as decision makers and stakeholders; the public agenda; and context of policy development but do not predict policy impacts at an individual level. Public Administration theories are predicated on the assumption that as policies change the environment, the environmental change will impact the individual. Individuals are defined differently in public administration theories as compared to medical model health theories; individuals are either decisional agents who affect policy change or stakeholders in the implementation of the ultimate policy. Individuals are not considered as cognitive performers such that their individual behaviors' interaction with a policy creates a change in personal status (i.e. disease state). For example, The Institutional Analysis and Development (IAD) framework (Ostrom, 2007), The Advocacy Coalition Framework (Sabatier, 2007), and The Punctuated Equilibrium Model (Baumgartner & Jones, 2005), all consider individuals as actors in the policy realm who either represent themselves or institutions; personal / individual status is accounted for as a belief system that
causes an actor to advocate from a certain policy relevant perspective rather than as an individually representative condition per se.

A conceptual solution to the limitations of medical/health and public administration theories will be discussed to link a population policy theory with an individual health theory such that Kingdon’s Multiple Streams Theory of Policy Formulation (Kingdon, 2003; Zahariadis, 2007) would be linked to Barker’s Theory of Developmental Origins of Adult Diseases (Barker, 1995b). The linkage of two disparate disciplinary theories combines and generates a macro to micro level conceptual framework for the study of pediatric obesity. The discussion of the type of normative framework necessary to link medical and public administration theories will be explored positing that a combination of both disciplinary theories would then create a continuum, from macro level policy formulation to micro level individual disease status.

Accordingly, infancy and toddlerhood are critical periods for health status across the life-span (Barker, 2001). Effects of slow fetal growth, low birth weight,
and effects of early postnatal development, are determined by genetic and environmental influences and developmental pathways that precede and follow (Barker, 2006). Programming, the term used to describe lifelong changes in function that follows a particular event in an earlier period of the life span, is defined as the incident where metabolic change leads to altered metabolism and then to disease later in life (J. G. Eriksson et al., 2001). Organisms are often malleable or ‘plastic’ during their early development and can be molded by the growth environment (J. G. Eriksson et al., 2001). Developmental plasticity is defined as the phenomenon by which a single genotype can give rise to a range of different physiologic or morphologic states in response to different environmental conditions during development (Barker, 2003, 2006). There is a critical period, normally in utero, when the biological systems are ‘plastic’ and sensitive to the environment, followed by loss of plasticity and development of fixed functional capacity; the determinants of the fixed capacity will also be the determinants of adult disease states (Barker, 2006). The Life-course Model of Health theory proposes that fetal undernutrition causes an adverse event of ‘small birth size’ which is associated with increased mortality and morbidity from chronic disease in later life (Barker, 1995a; Barker, Osmond, Golding, Kuh, & Wadsworth, 1989; Barker, Osmond, & Law, 1989).

Gene/environment interactions, both nutritional and non-nutritional, during gestation are critical factors which determine the origin of chronic adult diseases (Law et al., 2001). Criticism of the Life-course Model of Health centers on the fact that most research using this theory looks at only the individual and takes the
environment that the individual lives in as a given. However, individuals are not isolated entities making autonomous decisions, but rather are embedded in a social system; therefore, the major limitation is the absence of the concept of ‘linked-lives’ (Institute of Medicine. Committee on the Health and Safety Needs of Older Workers. Board on Behavioral Cognitive and Sensory Sciences, 2004).

Individuals need to be treated as products of social interaction and shared decision making within families, personal networks and governments, so environments that create later life individual disease is linked to society at large (Institute of Medicine. Committee on the Health and Safety Needs of Older Workers. Board on Behavioral Cognitive and Sensory Sciences, 2004).

Dannefer and Uhlenberg (1999, p309) identify what they consider three significant problems regarding the life course theory: there is a tendency to equate the significance of social forces with actual environmental changes, intra-cohort variability is not addressed, and there is undue emphasis placed on behavior choices to mitigate outcomes in the life course (Dannefer & Uhlenberg, 1999). The life course theory has been seen as only a micro-level theoretical framework with limited value for explaining macro-level influences of public policy and social institutions on health and disease free aging (Institute of Medicine. Committee on the Health and Safety Needs of Older Workers. Board on Behavioral Cognitive and Sensory Sciences, 2004).
The Multiple Streams Framework provides a better understanding of the structure of policy development in governmental agenda setting (Kingdon, 2003). The Multiple Streams Framework defines policy formulation as three process streams which are instrumental in the creation of, or framing of policy windows (Kingdon, 2003). The Multiple Streams approach presents an illustration of “a policy primeval soup” where policy alternatives, problems and solutions float about “within a community of specialists” be they, politicians, academicians, advocacy groups, etcetera waiting for the moment of convergence thereby developing an adopted policy (Burgess, 2002). The first of the process streams defined is the problem stream involving problem identification and recognition which may or may not be based on a ‘focusing’ event (Zahariadis, 2007). The next is the policy stream which is composed of disparate policy alternatives and proposals (Zahariadis, 2007). The final process stream is the politics stream composed of public opinion; governmental administrations; elected officials and
bureaucrats; and special interest groups which determine popular receptivity and political feasibility (Zahariadis, 2007). Each stream flows independently, is driven by different forces, and are coupled by a policy entrepreneur when a policy window opens at critical points in time thusly influencing agenda setting and creating policies (Burgess, 2002). When a policy window opens in either the political stream or the problem stream, it provides the opportunity for action on a policy (Kingdon, 2003; Zahariadis, 2007).

How a problem becomes a policy is the central explanatory function of the Multiple Streams Framework. Kingdon cites focusing events and feedback as the most important factors influence the attention given to a problem (Kingdon, 2003). Quantifiable systemic indicators are used to illustrate that a problem exists but they can also be used to argue that a problem conversely does not exist and is what is occurring regarding the pediatric obesity epidemic; conflicting opinions abound creating a lack of focus and consensus. Focusing events are more powerful than indicators because they immediately raise awareness of a problem, both on a policy level and in the public realm (Burgess, 2002; Kingdon, 2003) but there really have not been any events that truly focused a laser beam on the issue of growing obesity rates in U.S. children. The health and agricultural policy sub-governments are fragmented; the policy community of specialists is not uniform across the policy streams, thusly a policy entrepreneur from the academic or research community will probably not be able to impact both streams. The health specialists do have a major role in the proposal of health policy alternatives (i.e. the IOM, AMA, and NAS do effectively impact national
health policy); conversely, the agricultural specialists do not factor highly in the proposal of farm policy alternatives (i.e. the ADA and DRI researchers at the USDA have not effectively coupled research with obesity policy agendas). A confounding issue is that for any food-based policies to be effective, they need to be based on the prevailing patterns of the country for which they are being developed, and take into account the social and cultural elements of that society (Burke et al., 2007).

Problematically for both health and agricultural policy streams, many of the policy alternatives do not emerge from specific specialist communities, but rather from within the legislature itself; there is a politicization of policy rather than policy fostered by research in both health and farm policy similar to what is happening in fine art policy (Burgess, 2002). The onus for any specific policy community is to generate proposals acceptable to a majority of legislators; the policy must be politically feasible for it to have any chance of reaching the agenda. For example, many times a turnover needs to occur in elected officials before a policy window opens and the policy become politically feasible for passage (Greathouse, Hahn, Okoli, Warnick, & Riker, 2005). As Kingdon notes, “Part of a group’s stock . . . in affecting all phases of policy making… is its ability to convince governmental officials that it speaks with one voice and truly represents the preferences of its members” (Kingdon, 2003); health policy advocates, such as the AMA, IOM and NAS are very good at this and have high profile government bureaucracies (i.e. NIH) to assert considerable technical ‘muscle’ to advocate their policy alternatives. However, agricultural policy
specialists are a diverse and divisive community and do not speak with one voice; there are many sub groups in the fragmented agricultural policy community, such as Soil and Crop Scientists, Environmentalists, Farmers, and the ADA (i.e. Nutritionists); the USDA is a disjointed bureaucracy that administers diverse programs ranging from school lunches to soil conservation programs and thusly does not present a strong united policy voice ("Farm Security and Rural Investment Act of 2002," 2002). Strategic behavior of scientific experts has been shown to not only open policy windows but to advance policy stream coupling with the political stream (Hart & Victor, 1993). Select elite scientists were able to parlay their research agendas onto the political agenda to influence politicians to support research policy that would change research across multiple disciplinary domains (Hart & Victor, 1993). These 'elite' scientists were able to mediate between science and politics to secure support for research by linking unconnected policy streams becoming policy entrepreneurs who capitalized on open policy windows to secure funding that benefited not only themselves but other scientists who were not as adept (Hart & Victor, 1993). The example suggests that a high level scientist from one policy domain maybe strategically positioned as to couple disparate policies streams of health and agriculture when a political opportunity opens a policy window (Hart & Victor, 1993).

Finally, for a policy alternative to endure it must be technical feasible, cost appropriate, and attuned to the values of both politicians and the general public (Burgess, 2002; Kingdon, 2003). In effect, advocates for a proposal must anticipate details and technicalities by “gradually eliminating inconsistencies,
attending to the feasibility of implementation, and specifying the actual mechanisms by which an idea would be brought into practical use” (Kingdon, 2003). The impact of focusing events to bring a problem to the forefront of policy development can be seen in many diverse policy arenas. Exworthy and Powell (2004) argue that there are numerous sub policy streams in each domain that can be linked across domains to form a major cross disciplinary policy (Exworthy & Powell, 2004). A skilled policy entrepreneur can focus a policy window that is open in one domain to expand into another domain thereby creating cross-disciplinary policies; taking a small policy window open in one domain and creating a large policy window that can accommodate many disparate institutions and specialists (Exworthy & Powell, 2004). Disparate policy ‘silos’ can be linked to formulate cross cutting policies that are implementable across hierarchical bureaucracies; Exworthy and Powell’s expansion of Kingdon’s Multiple Streams approach makes the framework amenable for explanation of policies that address life-course health issues (Barker, 2007b; Exworthy & Powell, 2004; Kingdon, 2003). The Multiple Streams Framework can be further extended to explain coalition building by scientific leadership in governmental agencies to raise a cross-cutting scientific issue to the policy agenda (Felix, 2007). This type of creative policy agenda setting needs to be pursued at both the Department of Health and Human Services (DHHS) and at the USDA if effective solutions to the pediatric obesity problem are to be forged.

Merger of health and public policy theory however, does bring its own set of problems. Stone argues that disciplines have their own linguistics and
communication pattern which leads to miscommunication when disciplines work together on multidisciplinary problems (Stone, 2002), the need for a common framework to solve critical individual problems with government policy becomes compelling. Two theories, one a medical health theory and the other a public administration policy theory, that may lend themselves to combination in a conceptual framework to model a complete process flow from macro policy to individual disease to solve problems related to the social determinants of health were discussed. Social determinants of health are multi-faceted problem phenomena with multiple causations, such as obesity, do not lend themselves neatly to a conceptual framework (Exworthy, 2008). The life-course perspective on health (Barker, 1995a) delivers a conundrum to policy makers because policy time scales are not measured over a life span (50-70 years) but rather are measured in terms of legislative or executive office election cycles (4 -8 years) (Exworthy, 2008). In order to sustain a life course policy to fruition where its affects become perceptible and significant change is realizable, either a policy entrepreneur or policy community will need to continue to support continued policy action. Committed support over such a long period of time may be extremely difficult or impossible to muster (Exworthy, 2008). Life course policies require policy action across different organizations and policy domains, however, most health policy responses are disease specific. Formulating long-term partnerships between divergent organizations are a critical component for successful cross-cutting policy formulation and implementation, but partnerships are difficult to create and many times are unsustainable in hierarchical
governmental organizations because of differing accountabilities, values and performance criteria ("ADA supports USDA School Meals Initiative for Healthy Children but recommends more improvements for child nutrition," 1994; AMA, 2007; Exworthy, 2008; Institute of Medicine. Committee on Prevention of Obesity in Children and Youth et al., 2004; Institute of Medicine. Committee on Progress in Preventing Childhood Obesity, 2006; NIH, 2004; Reaffirmation of AAP Policy Statement: Prevention of Pediatric Overweight and Obesity.," 2007). An additional problem for policy that proposed to effect long-term change are the political realities which reinforce short time spans to show policy results which has become necessary because of the reduced attention span of the public to supporting policy change.

An effective conceptual solution is the combination of Kingdon’s Multiple Streams Framework (Kingdon, 2003) with Barker’s Life-course Theory of Health (Barker, 1995b) into one theoretical framework that could have explanatory power across the public policy and medical disciplines to recognize, define, and determine policies which impact the bio-physical environment creating social determinants which are the causal factors of obesity. In this study both individuals and policy makers interaction with the framework begins in the middle with Common Pool Environment/ Resources which are created by public policies and which then determine the amount of resources available to the individual. Common Pool Environment/ Resources are operationalized for as the amount and availability of food commodities produced for human consumption.

Resources, Components and Factors Available to the Individual are
operationalized as the price of a commodity when adjusted for inflation and household income which drives would drive individual access to the resources. *Growth and Development* will be operationalized as pediatric anthropometric measurements of BMI which affect *Development of Disease*. Problems for the policy maker would begin as the population outcomes of collective individual health which would set the stage for policy intervention.

![Diagram](image)

**Figure 2.3 Framework From Government Policy to Individual Disease**

The merger of the problem and solution streams occurs in two disparate policy streams, agriculture and health; however the policy window that is open for pediatric obesity policy is beginning to be linked by Congress (*Chairwoman McCarthy Statement Subcommittee Hearing On "Improving Child Nutrition Programs to Reduce Childhood Obesity",* 2009; *Current Status and Activities to Decrease the Prevalence of Obesity Among U.S. Children and Adolescents*.}
Statement of William H. Dietz, MD, PhD, Director Division of Nutrition, Physical Activity, and Obesity National Center for Chronic Disease Prevention and Health Promotion Centers for Disease Control and Prevention, 2009; Hearing to examine how improvements to child nutrition programs can help fight the nation's childhood obesity crisis. Statement of Michelle Patterson, First Lady of New York State, 2009). Many policy makers deem that because obesity cannot be defined as a single problem, policy solutions will be ineffective either because of social forces or because the problem is personal to an individual rendering it invisible to the society (Exworthy, 2008). Therefore, there has often been “no policy response”, which has frequently made the problem definition even worse by the lack of consensus among researchers, policy specialist sub groups, and policy makers about the policy solutions required (Exworthy, 2008). Targeting the family may be vital for obesity prevention in the earliest years, but longer-term weight control will require a combination of individual engagement and society-wide policies to modify the food and built environments, especially for children at high genetic risk for obesity (Wardle et al., 2008).
CHAPTER III

METHODOLOGY

“What’s right about America is that although we have a mess of problems, we have great capacity - intellect and resources - to do some thing about them.” Henry Ford II

This chapter outlines the methods and procedures used in the study and consist of the following sections: 1) Purpose, 2) Method and Data Sources, and 3) Limitations.

Purpose

The purpose of this study was to provide a 100-year time sequence of the growth of U.S. school children during the 20th century which was overlaid upon changes in federal agricultural policies that directly affected the composition of the U.S. food environment. The objectives of the study were as follows:

1) Describe trends in children’s weights following adoption of different federal agricultural policies as measured by commodity component’s availability and price in the U.S. food supply.
2) Describe generational trends in children’s weight status overlaid with historical context such as adoption of significant U.S. health policies and medical practices (such as immunizations and widespread antibiotic usage) which contribute to a healthy childhood environment, food shortages during various periods, and food supply changes due to adoption of significant U.S. agricultural policies (such as school lunches, crop subsidies and various commodity production programs).

Method and Data Sources

A descriptive retrospective study is proposed to depict the intersection between U.S. agricultural policy externalities which shape the nutrient composition of the U.S. food supply and anthropometric measurements regarding body typology related to obesity in American children during the 20th century (1900 to 2000). The study will seek to describe if children’ weights/ body sizes change following adoption of different federal agricultural policies which affect the national food supply by changing availability, amount or access to varying commodities. Additionally, trends in children’s weight status will be overlaid with adoption of significant U.S. health policies (federal supplemental food programs) and medical practices (such as vaccinations programs and widespread antibiotic usage) which account for better health and contribute to healthy childhood growth.
The study describes, using graphical displays, trends over the 20th century in pediatric Body Mass Index (BMI) and will overlay the children’s growth with the composition of the U.S. food supply. Pediatric BMI trends was overlaid with the availability of selected nutrient components of the U.S. food supply and graphed versus a timeline of significant historical events which might also affect children’s growth patterns. The U.S. food environment was described in terms of ‘food availability’ defined as both the per capita retail food consumption (i.e. in pounds or gallons, etc. . .) and Price (i.e. cost per unit of food which is adjusted for inflation); these measures provide a picture of overall amounts of food available for human consumption each year and the actual accessibility of the food to the average person. To complete the trend picture the percent of household income spent annually on food will also be displayed.

Descriptive research attempts to accurately describe a particular state of affairs by identifying the variables that exist and explicating the relationships existing between or among these variables. The time series graphs will display the pattern and trend in U.S. school children’s weights and display the relationship to both historical events and food commodity availability and prices. Children’s weight measurements (criterion variable) trends over the 20th century will be compared according to the classification variables of food availability and price. The current time series study will attempt to describe the increase in overweight and obesity in America’s children from 1900 to 2000. These series of related graphs will display trends in children’s obesity and additionally overlay these trends with historical events that possibly effected population health during
the twentieth century which could also affect obesity rates or the obesigenic food environment. The research questions are:

RQ1) Did the aggregation of U.S. agricultural and nutrition policies throughout the past century create a selection process that is biased towards obesity?

RQ1 will be measured by overlaying the BMI of children over the past century with commodity consumption and price.

RQ2) Was the large spike in obesity that has been seen in the last 30 years in the United States created by expression of the thrifty phenotype across the generations?

RQ2 will be measured by the anthropometric measurements (i.e. BMI) of children overlaid with their mother’s generation and then the mother’s generation overlaid with their mother’s generation, respectively. If there is thrifty phenotype expression then during periods of food scarcity the mother’s generation will be smaller than their children’s generation and during periods of food plenty the mother's generation should be either similar or bigger than their children.

Graph Production

The data will be graphed using Excel for Vista. Trend lines for the BMI variable as compared to the commodity, price, household income, and government spending variables will be fitted using the Excel trend line function.
The Excel trend line function uses the following formula functions to fit the trend lines: for the polynomial trend line, \( y = mx + m_1x^2 + b \) for all males (6 to 9 yo and 9 to 12 yo) and females (6 to 9 yo) and for exponential trend line used for females (9 to 12 yo) \( y=bm^x \), to the data set by minimizing the sum of the squares of the error (Kleinbaum et al., 1998). Over-specifying the trend line was avoided by checking the Excel function LINEST to assure that the lowest order polynomial trend line was chosen as the best fit curve. LINEST identified if the coefficients were statistically significant and if they were not, a lower order polynomial was chosen. Confidence intervals were not graphed because the graphs would have created graphs that would have been difficult to interpret because of the multiple variables with the numerous corresponding lines.

For the Generational trends a line was fitted between at least two time points spanning a generation using the Excel trend line function with the following equation: \( y = mx + b \) to the data set through minimizing the sum of the squares of the error terms for data that is linear (Kleinbaum et al., 1998). The \( R^2 \) is calculated by the formula

\[
\frac{\text{Sum of Squares}}{\text{Sum of Squares} + \text{Sum of Squares residual}}
\]

and measures how well the regression fits the observed data ranging from 0 to 1; the closer to 1 the better the fit (Kleinbaum et al., 1998). Graphically, the \( R^2 \) measures how close the regression line is to all of the observations and will be presented on all the generational graphs.
The x-Axis

x-Axis will be the time period from 1900 to 2000 in 5-year increments. Major historical events from 1900 to 2000 that either affected the food supply or public health will be discussed. The x-axis will remain constant for all graphs. Historical events from 1900 - 2000 that may have affected the population health or nutritional status, and agricultural policies that may have affected food supply availability or price will be noted when the patterns are described (See Appendix 1 for a description of the pertinent historical events).

Primary y-Axis

BMI data was calculated from weights and heights obtained from various Public
Health Service Data sources throughout the century. Data was obtained from
Public Health Service published articles and data sets explained for each subset
as follows: in 1900 data was used from the published data from the Byers Study
(Byer, 1901) obtained from Cambridge MA schools for males 10 to 12 and
females 10 to 12 of aggregate data for height and weight, and in 1900 from the
published data set from Bowditch (Bowditch, 1900) obtained in Boston MA for
schools for children classified as American, Irish, German or English on
aggregate height and weight, in 1915 from individual data from the Turner Study
(J. Turner, 1915) of Philadelphia, PA school Children for 6 to 12 yo males and 6
to 12 yo females; in 1919, 1921, and 1922 from aggregate data from The
Newton, MA School Education Experiment (C. Turner, 1920, 1928) for school
children classified as American, Jewish and Italian for 6 to 12 yo males and 6 to
12 yo females; in 1921 from aggregate data from the NY Welfare Department
Improving Social Conditions of the Poor Study (Dublin & Gebhart, 1923) in the
Mulberry District New York City in Italian children 6 to 10 yo; in 1923 from
aggregate data in Chicago and New York (Palmer, 1924) for 9 yo males in 1927,
1928, 1934, 1935, 1941, and 1942 from aggregate data from The Division of
Medical Services of the Board of Public Education Philadelphia Pennsylvania
records (Hundley, Mickelsen, Mantel, Weaver, & Taber, 1955) in 6 to 12 yo male
and female white children in Philadelphia schools in 1928 from aggregate data
Definition of the primary y-axis variables follows:

1) Gender: Reported value of either male or female.

2) School Age: Ages from 6 to 12 years old defined as 'school age'; the age range was chosen because of it is a relatively stable flat growth period during the child's life cycle, coming after the adiposity
rebound growth stage (Hawkins & Law, 2006; Wang & Lobstein, 2006), and before the puberty growth spurt (de Onis & Blossner, 2003; Mei, Yip, Grummer-Strawn, & Trowbridge, 1998; WHO, 2003). Additionally, children are predominately institutionalized in primary school for a significant portion of the calendar year with over 50% eligible for Federal School Lunch Programs (Dahdah, 2004; Duniform & Kowaleski-Jones, 2002, 2004).

3) Weight: Reported value of weight in pounds. Weight as an anthropometric method has well documented variation. Due to the retrospective nature of the study, weight measurements are assumed to be accurate and reliable to within 1-2% as has been found in clinical practice (Corkins, Lewis, Cruse, Gupta, & Fitzgerald, 2002) and will be used to calculate the BMI value.

4) Height: Reported value of height in pounds. Height as an anthropometric method has well documented variation. Due to the retrospective nature of the study, height measurements are assumed to be accurate and reliable to within 1-2% as has been found in clinical practice (Corkins et al., 2002) and will be used to calculate the BMI value.
5) BMI: Calculated value derived from the height and weight using the standard formula:

\[
BMI = \frac{\text{weight (lbs) x 703}}{[\text{height (inches)}]^2}
\]

BMI percentile ranges that distinguish the levels of growth (normal 20-84\textsuperscript{th} percentile, overweight 85-94\textsuperscript{th} percentile, and obese >95\textsuperscript{th} percentile) were developed by the “WHO Multi-Centere Growth Study” and will be used to demarcate the normal growth range on the graphs (WHO, 2003). The WHO growth standards were chosen because of the small variability in the WHO growth sample due to the highly standardized method of data collection across the various country samples and that the children in the sample were predominantly breastfed which is considered the gold standard for healthy early life course growth (WHO, 2003).

Secondary y-Axis

The secondary y-axis will display the classification variables chosen as surrogate markers for dietary intakes of Protein, Carbohydrates, Fat, and Vitamins and Minerals which are essential components of a healthy pediatric diet. Additionally, household income, and consumption expenditures will be used to display the amount of income needed for food purchases and if there is a trend...
when compared to pediatric weight trends. The data will be obtained from the Department of Commerce Bureau of Economic Analysis National Income and Product Account tables (1929-1947 and 1947-2008).

The food commodities will be displayed in various ways:

1) Per capita level of the type of food available,

2) Inflation-adjusted retail price to demonstrate the actual availability on a household level of the food commodity and/ or

3) Commodity farm market price to demonstrate the actual produced supply.

Data sets will be obtained from the USDA ERS Data base: food availability for human consumption ("Food Availability: Spreadsheets," 2009); from The Statistical Abstract of the United States, published since 1878 which has the retail prices of food available, composition, and production levels of food from 1878 to 2004 (U.S. Census Bureau. The Statistical Abstracts of the United States, 1900- 2004); and the Recommended Dietary Intake Levels will be obtained from Dietary DRI Reference Intakes The Essential Guide to Nutrient Requirements compiled by the National Academy of Science (Otten et al., 2006). Commodity price and price support data will also be obtained from the archived USDA Electronic Research Service databases (1908- 1992)("United States Department of Agriculture NASS Quick Stats (Crops)," 2007) and The Statistical
Protein quality is considered when assessing healthy diets with > 95% of the protein easily digestible and containing the essential amino acids, thusly foods such as meat, poultry, and egg protein can be used to gauge the health quality and availability of the food environment (R. Kleinman, 2004). Dietary protein is necessary to replace losses from the body when energy intake is balanced (i.e. maintenance requirement), however for children protein is required for growth; dietary protein requirements are approximately 65% of dietary intake at birth and decrease to 5% of intake by 5 years of age (R. Kleinman, 2004). By the time the child is school age, dietary intakes of protein should be approximately 14% of their total caloric intake to maintain a healthy body weight (J. E. Brown et al., 2005). Growth requires approximately 0.23 g of dietary protein for every gram of new tissue deposited, thusly, the amount and availability of protein the food environment should be an indicator of a food environment conducive to healthy pediatric growth (Otten et al., 2006). The effects of too much dietary protein have not been extensively studied but evidence does show a correlation (but no causal link has yet been discovered) between excessive protein intake and arthrosclerosis and hypercalciuria (interferes with bone mineralization) (R. Kleinman, 2004).

Carbohydrates provide 50-60% of the calories consumed in the average American diet even though relatively little carbohydrate is actually necessary for a healthy diet (R. Kleinman, 2004; Otten et al., 2006). Carbohydrates are
primarily obtained from fruit, vegetables and milk consumption as either starches or sugars. In the infant, carbohydrates are primarily obtained from milk lactose; however, as the child ages, lactose is replaced by a variety of different sugars and starches. According to USDA DRI s school aged children should be consuming approximately 55% of their total caloric intake as carbohydrates (J. E. Brown et al., 2005). Additionally, fruits and vegetables are the primary source of dietary fiber which is necessary for appropriate regulation of the digestive tract; the fiber regulates intestinal transit of food. Diets high in fiber appear to have a protective effect against colorectal carcinomas, atherosclerosis, and obesity; but the causal link to obesity is more so that populations with a high fiber diet also have low caloric intakes (R. Kleinman, 2004). Therefore, commodities such as milk, wheat (and bread), corn (including high fructose corn syrup) and cane and beet sugar will be used to gauge the health quality and availability of carbohydrates in the food supply. There is a deficiency in information regarding the composition of the U.S. food supply for the availability of fresh fruits and vegetables before the 1960s. Thus, even though fruit and vegetable commodities are a significant part of the food environment and are thought to correlate to obesity status, the paucity of data during the timeframe forces the study to exclude them as a variable.

Fat is required for healthy growth and body maintenance (i.e. to maintain optimal fatty acid composition in the tissues), in a healthy non growing human, approximately 5% of the total dietary intake should be fat (R. Kleinman, 2004; Otten et al., 2006). However, in young children higher fat intakes are required for
appropriate nervous system and brain development (Dobbing, 1970, 1971, 1973). By the time a child is school aged (over 4 years old), brain cell differentiation has slowed and the need for a high dietary fat intake is reduced (Dobbing, 1972, 1973, 1974a, 1974b; Dobbing, Hopewell, & Lynch, 1971; Dobbing & Smart, 1974). Commodities that can be used as surrogate markers for dietary fat availability in the food supply will be: butter, oleomargarine, vegetable shortening, high fat cheese and eggs.

Vitamins and Minerals are essential for healthy growth and development in children; there are thirteen vitamins and fifteen minerals in this category and are normally found in vegetables, fruit, grain, and milk products. Iron, Calcium and Vitamin D will be used as the primary markers for appropriate dietary consumption (J. E. Brown et al., 2005), thus, red meat and milk will be used as the indicators for appropriate Vitamins and Minerals in the diet.

Representative Food Commodities Graphed on the Secondary y-axis

**Meat** will be used as a measure of available Protein and Vitamins and Minerals (i.e. iron).

1) Per capita adjusted annual amounts available for consumption of red meat, beef and fish per year (pounds per capita per year) from the USDA/Economic Research Service, U.S. Department of
Commerce/National Marine Fisheries Service for fishery products database,

2) Inflation adjusted retail prices for red meat and beef (cost per pound) from Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce.

**Poultry/ Eggs** will be used as a surrogate measure of available Protein and Fat (i.e. cholesterol in eggs).

1) Per capita adjusted annual amounts available for consumption of poultry and chicken (broilers) per year (pounds per capita per year) from the USDA/Economic Research Service, U.S. Department of Commerce/National Marine Fisheries Service for fishery products database,

2) Inflation adjusted retail prices for poultry from the Bureau of Labor Statistics data base for consumer price indices,

3) Per capita adjusted annual amounts of total (processed and in shell) eggs (number per capita per year) available for consumption from the USDA/Economic Research Service database,
4) Inflation adjusted retail prices for eggs. Averages are for fresh eggs for all years. For 1942, prices are for the highest grade sold in volume in each store; for 1943-1944, U.S. extras or Grade A; for 1945-1952, the highest grade and size sold in volume in each store; since 1953, large Grade A eggs in most cities, although some ungraded eggs included in some small cities from the Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce.

**Dairy** will be used as a surrogate measure of available Protein, Fat, and Vitamin and Minerals (i.e. Vitamin D).

1) Per capita adjusted annual production levels of whole milk from the Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce,

2) Inflation adjusted retail prices for gallon of whole milk. Until 1935, prices are for fresh fluid milk, raw or pasteurized, no grade designation, in quart bottle or in bulk, delivered to homes; for raw or pasteurized milk of the dominant grade in each city in quart bottles or cartons; for 1947-1949, same grades, but sizes included 1-quart, and 4-quart containers; for 1950-1956, pasteurized milk, homogenized or non-homogenized, without Vitamin D, of the volume-selling in quart or half-gallon cartons or bottles; for 1957-
September 1966, pasteurized, homogenized milk with Vitamin D added, 3.25% or over butterfat content in quart or half-gallon cartons or bottles; beginning in October 1966, prices are for half-gallon containers; since May 1970, prices are for fresh whole milk, pasteurized, homogenized, Vitamin D added.

3) Natural equivalent of cheese and cheese products in pounds per capita per year. Excludes full-skim American and cottage, pot, and baker’s cheese. American cheese includes Cheddar, Colby, washed curd, stirred curd, and Monterey jack. Other cheese includes Romano, Parmesan, mozzarella, Ricotta, other Italian cheeses, Swiss, brick, Muenster, cream and Neufchatel, blue, gorgonzola, Edam, Gouda, imports of Gruyere and Emmenthaler, and other miscellaneous cheeses. Calculated from unrounded data from the USDA/Economic Research Service database.

Corn will be used as a measure of consumption of Carbohydrates.

1) Number of bushels produced from Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce.
2) Price in dollars per bushel from Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce.

Cane and Beet Sugar, High Fructose Corn Syrup (HFCS), Total

Sweeteners and Chocolate (i.e. Cocoa and Hershey Bars) will be used as a measure of Carbohydrates.

1) Pounds per capita availability since introduction in the U.S. food supply in 1966; data from USDA/Economic Research Service database.

2) Tons of sugar sweeteners produced annually from beets and cane from Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce.

3) Prices per pound are for white granulated cane or beet sugar but the size package has varied. For 1890-1916, prices for the volume-selling quantity were quoted; for 1917-1928, 1 pound; for 1929-1942, 10 pounds; and for 1943-1970, 5 pounds. For a short period during World War the 2-pound unit was the only one available. Data obtained from Retail Prices of Selected Foods in U. S. Cities 1890 to 1970.
4) Retail price of Hershey Candy Bar per ounce (1908 to 2003) from The Food Timeline.

Wheat/Grains and Bread will be used as a measure of Carbohydrates.

1) Number of pounds produced per capita from Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce.

2) Price per loaf of bread from Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce.

3) Price per pound of flour from Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce.

4) Price per pound from Retail Prices of Selected Foods in U.S. Cities 1890 to 1970 Prices are for white bread, pan style, excluding all specialty type bread. For 1913-1936, prices were obtained from bakeries for 16 or 18 ounces in the dough and converted to 16 ounces baked weight. Both wrapped and unwrapped breads were included. Beginning in 1937, prices were obtained primarily from grocery stores for the volume-selling size loaf of wrapped bread.
The baked weight on the wrapper or reported by the store was converted to 16 ounces. Data obtained from Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce.

Butter, Oleomargarine and Vegetable Shortening will be used as a measure of Fat.

1) Retail prices for uncolored oleomargarine, animal and vegetable, in 1 pound cartons for 1919-1948. For 1949 and 1950, uncolored vegetable margarine in 1-pound cartons was quoted. For 1951-1970, averages are for colored vegetable margarine in 1-pound cartons. Data is from the Statistical Abstracts of the United States published annually by the Bureau of Statistics Department of Commerce,

2) Pounds produced annually from the data from USDA/Economic Research Service database.

Limitations and Assumptions

Retrospective studies allow analysis of large amounts of data over long periods of time but are limited in scope because of the reliance on variables that
were collected which might not be optimum to answer the research questions. Accordingly, research questions must be designed to and fit within the limitations of the data; for this study only generalized descriptive trends can be reported and causality cannot be determined. Retrospective research is limited to the data that is available; thusly there is error in the variables for both the primary and secondary y-axis. The researcher does not have control of the type of data collected, the quality and frequency of the data collection or the definition / specification of the variables. Analysis is limited by the data available.

The population data from the beginning of the century is so generalized as not to be able to be distinguished by race, region, or culture. As the century progresses the data becomes more discrete and is able to be broken out into many different categories, however definitions of race changed significantly during the century and it is impossible to discern comparable racial categories from the early data (pre-1920) to the more recent data. Evidence indicates that there are racial differences in rates of pediatric obesity (X. Guo, Warden, Paeratakul, & Bray, 2004; Johnson et al., 1994; Jolliffe, 2004; C. L. Ogden et al., 2002; Paeratakul et al., 2002); however because of the varying definitions of race, the data was combined to produce a generalized trend picture for the average child by gender and race was not used as a distinguishing factor.

The definition of ‘school aged’ children also varies and is reported differently in some of the data, therefore for the purpose of this study, data from ages 6 to 12 was combined to display a composite school aged child. The rationale for this was the leveling off of growth during the period between the
rapid growth phases during the pre-school/ toddler period and puberty (Kuczmarski et al., 2000) combined with the 1994, the National Academy of Science Institute of Medicine (U.S.) and Health Canada guidelines for nutrition which were based upon developmental life stages (Otten et al., 2006) of Early Childhood ages 4 to 8 years; and Puberty ages 9 to 13 years. The normalized picture displayed by a school aged child is an extreme generalization because of the wide age span that was combined to present the anthropometric trends. The population data varied between local, state and national level data depending upon the years when the data was collected; collection methods for the anthropometric measures varied over the century and changed from health care worker collection, to self report by parents, to researcher collected which added further variability to the data. Thus the usefulness of the anthropometric data is limited because of the mixture of individual and aggregate level data; data was obtained from many sources; the sources include not only Federal statistical data warehouses and other organizations such as State Health Departments and Local School Districts that collect statistics and academic researcher’s data sets.

The data for the commodity production levels also has significant collection variability during the course of the century. Definitions for commodities have changed, as the century progressed and reporting methods have also changed as to what is considered as part of the food supply for human consumption. For example at the turn of the century poultry was defined as both hens and broilers in a generic category of poultry but by mid century the definition of poultry changed from a generic term that included laying hens and
broilers to broilers only. Also at the turn of the century, there is discrepancy in the data about what exactly constitutes poultry, just chickens or a combination of chickens and turkeys, it is difficult to discern what is being reported. The food supply is also calculated using the disappearance method of calculation which relegates to the food supply all residual uses of food stocks for which data are not available. This means that miscellaneous nonfood uses, stock changes at retail and consumer levels, and sampling and measurement errors are included in the estimation of the human food supply. An example would be that an increasing proportion of turkey (especially backs, necks and giblets) is made into pet food, but this portion is still currently included in the human food disappearance data. The data probably over states human consumption and is actually the upper limit on the amount of food available for human consumption. Finally, data using food disappearance estimates overstates actual human consumption because it includes spoilage and waste in the marketing system and in the home.

The methodology for data collection has changed for food prices during the century from retail prices in selected U.S. cities to national average prices (U.S. Census Bureau. The Statistical Abstracts of the United States, 1900-2004). In addition, the methods used to adjust for inflation have also changed during the course of the century and in some of the oldest data (pre 1930) it is not clear if the prices have been inflation adjusted. However, all price data was chained and presented in 1967 dollars so a standard dollar value could be visualized across the century in all the graphs.
“Never discourage anyone...who continually makes progress, no matter how slow.”  Plato

Chapter 4 describes the analyses of the data collected to address the research questions. The chapter is organized into four sections: 1) explanation of BMI trends, 2) commodity comparisons to BMIs, 3) comparison of BMI and federal program budgets relating to agriculture and food, and 4) BMI trends over subsequent generations.

Purpose

The purpose of the research was to describe trends in children’s weights following adoption of different federal agricultural policies as measured by commodity component’s availability in the food supply (defined by per capita consumption and retail price) and during different historical events that would
affect the food supply (such as wars, economic depression, etc. . .). Trends in children’s BMI are discussed in the context of significant U.S. health policies, historical events, and nutritional legislation which might also contribute to childhood growth. Finally, BMI generational patterns are overlaid to describe the presence or absence of generational phenotypic changes. When generational phenotypic changes are suspected, U.S. historical events and policy was used to possibly describe the existence of a pattern.

Graphical presentation of children’s BMI, per capita consumption and food prices will depict patterns between the variables. Children’s BMIs and the Food Supply (amount and prices) will be graphed and discussed in terms of Government Policies, and Historical Events.

The graphs also describe trends and conditions which lead to patterns of change in the children’s BMI. School children’s BMI has risen over the century; however the rise in BMI is more pronounced in females and older males (9-12 year old) than at the younger ages. The graphical data is presented separately by gender and age grouped into 4 subsets (Males aged 6-9; Males aged 9 -12; Females aged 6-9 and Females aged 9-12). The BMI rise is rather gradual until mid century with BMI trends in the early part of the century 1900 to 1920 either stable or slightly higher than the 1930s. BMI then decreases to its nadir in the 1930’s and begins a gradual rise through the 1940s and 1950s but by the mid 1960s children’s BMIs begin to exponentially increase.

The graphs also describe the effect of U.S. agricultural and health policies that affect the nutrition of the population and indirectly affect BMI trends. For RQ
1: the aggregation of our agricultural and nutrition policies throughout the past century has created a selection process for phenotypes that are ‘thrifty’, the graphical data will be presented for the four gender/age sub sets of the children, Males aged 6-9; Males aged 9-12; Females aged 6-9 and Females aged 9-12.

For RQ 2: the expression of the 'thrifty' phenotype in the current food environment has created the large spike in obesity that has been seen in the last 30 years in the United States, the graphical presentation will be presented in separate generational graphs for using females and their male and female children.

Overview of the Graphs

The graphs are presented in the following fashion: the x-axis always displays years ascending in increments of 5-years from 1900 to 2000; the primary y-axis always displays on the left side of the graph with BMI units ascending in increments of 0.5 BMI from 15 to 21; and the secondary y-axis varies according to which commodity/ commodity program is being presented. The secondary y-axis units varies with commodity but will either present a variation of consumption in pounds per capita or chained dollars (representing either price or program costs).

Each variable is graphically displayed as points and the best fit line is graphed. Linear trend lines are used where ever possible, however lower order
polynomials better fit some of the commodity trends. Polynomial trend lines are used when the data oscillated to describe the numerous fluctuations. When polynomials are necessary to describe the trend lines, extrapolation beyond the time span of the data set are not valid and were not used to describe or make any prediction regarding future trends (Cowden, 1963). Trends that are polynomial in nature are grounded in the economic, social and historical time span for the data set so the historical events become critical in describing the fluctuations (Cowden, 1963).

Presentation of the Graphs

The graphs are presented in the following order: BMI (Graphs 4.1 – 4.5). Then graphs will be displayed as BMI vs. per capita consumption of Protein (Graphs 4.6 - 4.13; 4.18 – 4.21) included are Red Meat, Poultry, Eggs (which could be included also in the Fat category); as BMI vs. Protein prices in chained 1967 dollars (Graphs 4.14 - 4.17; 4.22 - 4.25) included are Beef, Chicken, and Eggs (which could be included also in the Fat category); BMI vs. per capita consumption of Carbohydrates (Graphs 4.26 - 4.49; 4.34 – 4.37; 4.42 – 4.45; 4.50 – 4.57; 4.62 – 4.65) included are Milk (which could be included also in the Protein category), Wheat Products, Corn Products, Sweeteners (Sugar, HFCS), and Cocoa; and BMI vs. Carbohydrate prices in chained 1967 dollars (Graphs 4.30 – 4.33; 4.38 – 4.41; 4.46 – 4.49; 4.58 – 4.61; 4.66 - 4.69) included are Milk.
(which could be included also in the Protein category), Flour/ Bread, Corn (crop), Sugar, and Hershey Bar prices; and BMI vs. per capita consumption of Fats (Graphs 4.70 - 4.81; 4.83; 4.85; 4.87; 4.89) included are Total Fats, High Fat Cheese, Selected Fats (Vegetable Shortening, Lard, Butter, Oleomargarine; and BMI vs. Fat prices in chained 1967 dollars (Graphs 4.82; 4.84; 4.86; 4.88) included are Butter and Oleomargarine. Then BMI vs. USDA Program Budgets (Graphs 4.90 - 4.93), and vs. Commodity Price Supports are presented separately (Graphs 4.94 - 4.97), and vs. Percent Household Income Spent on Food (Graphs 4.98 - 4.101). Finally, Generational Graphs of BMIs are presented (Graphs 4.102 - 4.117).

Children’s BMIs

RQ1) Did the aggregation of U.S. agricultural and nutrition policies throughout the past century create a selection process for that is biased towards obesity?

RQ1 was answered by overlaying the anthropometric measurements of children over the past century with commodities consumption and price, that were affected by agricultural policies either positively or negatively, which then affected the food supply. The anthropometric measurement pattern was then explained in terms of the ensuing agricultural policies in effect during the time period. Graphs 4.1 to 4.101 display the descriptors for RQ#1.
Graph 4.1: Children's BMI Trend During the 20th Century (1900-2000)

BMI data was calculated from weights and heights obtained from various Public Health Service Data sources throughout the century. Data was obtained from Public Health Service published articles and data sets explained for each subset as follows: in 1900 data was used from the published data from the Byers Study (Byer, 1901) obtained from Cambridge MA schools for males 10 to 12 and females 10 to 12 of aggregate data for height and weight (n=156 for 10 yo males, n=436 for 11 yo males, n=454, for 12 yo males, n=159 for 10 yo females, n=434 for 11 yo females n=465, for 12 yo females); and in 1900 from the published data set from Bowditch (Bowditch, 1900) obtained in Boston MA for schools for children classified as American, Irish, German or English on aggregate height and weight (n=910 for 6 yo males, n=1008 for 7 yo males, n=1061, for 8 yo males, n=1504 for 9 yo males, n=987 for 10 yo males, n=953, for 11 yo males, n=923 for 12 yo males, n=674 for 6 yo females n=825, for 7 yo females, n=883
for 8 yo females, n=787, for 9 yo females, n=664 for 10 yo females, n=666 for 11 yo females, n=644 for 12 yo females); in 1915 from individual data (n=29 for each age and gender) from the Turner Study (J. Turner, 1915) of Philadelphia, PA school Children for 6 to 12 yo males and 6 to 12 yo females; in 1919, 1921, and 1922 from aggregate data (n= 273 for each year) from The Newton, MA School Education Experiment (C. Turner, 1920, 1928) for school children classified as American, Jewish and Italian for 6 to 12 yo males and 6 to 12 yo females; in 1921 from aggregate data ( n= 1878 males and n= 2169 females) from the NY Welfare Department Improving Social Conditions of the Poor Study (Dublin & Gebhart, 1923) in the Mulberry District New York City in Italian children 6 to 10 yo; in 1923 from aggregate data in Chicago and New York (Palmer, 1924) for 9 yo males (only mean height and weight data was reported); in 1927, 1928, 1934, 1935, 1941, and 1942 from aggregate data from The Division of Medical Services of the Board of Public Education Philadelphia Pennsylvania records (Hundley et al., 1955) in 6 to 12 yo male and female white children in Philadelphia schools(mean height and weight data was reported for each year by age and gender); in 1928 from aggregate data from The School Health Program in Iowa (Stuart & Meredith, 1946) for white 7 and 11 yo males and 7 and 11 yo females; in 1934 and 1935 from aggregate data from Boston MA school children (Wilson, 1935) for 6 to 12 yo females (n=275); in 1945 from aggregate data (n=208) from The Iowa School Children Study (Eppright & Roderuck, 1955) in white 11 and 12 yo males; in 1948 and 1953 from aggregate data from The Iowa School Children Study (Eppright & Roderuck, 1955) for white 6 to 12 yo males
(n=485) and 6 to 12 yo females (n= 488); in 1963 from individual data from the NHES II ("National Health Examination Survey, Cycle II (NHES II)," 1965) for white 6 to 11 yo children (n=489 for 6 yo males, n= 551 for 7 yo males, n= 537 for 8 yo males, n= 524 for 9 yo males, n= 509 for 10 yo males, n= 512 for 11 yo males, n= 461 for 6 yo females n= 512 for 7 yo females, n= 498 for 8 yo females, n= 494 for 9 yo females, n= 505 for 10 yo females, n= 445 for 11 yo females), in 1965 from individual data from the NHES III ("National Health Examination Survey, Cycle III (NHES III)," 1970) for white 12 yo children (n= 30 for 12 yo males, n= 32 for 12 yo females), in 1976, 1977, 1978, 1979 from individual data from the NHANES II ("National Health and Nutrition Examination Survey Data. NHANES II," 1980) for white 6 to 12 yo children (n=114 for 6 yo males, n= 118 for 7 yo males, n= 123 for 8 yo males, n= 121 for 9 yo males, n= 123 for 10 yo males, n= 126 for 11 yo males, n= 120 for 12 yo males, n= 100 for 6 yo females, n= 119 for 7 yo females, n= 103 for 8 yo females, n= 123 for 9 yo females, n= 114 for 10 yo females, n= 113 for 11 yo females, n= 118 for 12 yo females); in 1990 from individual data from the NHANES III ("National Health and Nutrition Examination Survey Data. NHANES III," 1997) for white 6 to 12 yo children (n=128 for 6 yo males, n= 121 for 7 yo males, n= 115 for 8 yo males, n= 124 for 9 yo males, n= 114 for 10 yo males, n= 126 for 11 yo males, n= 46 for 12 yo males, n= 114 for 6 yo females, n= 104 for 7 yo females, n= 133 for 8 yo females, n= 129 for 9 yo females, n= 110 for 10 yo females, n= 119 for 11 yo females, n= 43 for 12 yo females); in 2000 from individual data from the NHANES 1999-2000 ("National Health and Nutrition Examination Survey Data.
NHANES 1999 -2000," 1999) for white 6 to 12 yo children (n=26 for 6 yo males, n= 24 for 7 yo males, n= 25 for 8 yo males, n= 14 for 9 yo males, n= 17 for 10 yo males, n= 22 for 11 yo males, n= 37 for 12 yo males, n= 23 for 6 yo females, n= 21 for 7 yo females, n= 19 for 8 yo females, n= 20 for 9 yo females, n= 20 for 10 yo females, n= 21 for 11 yo females, n= 35 for 12 yo females). The data is limited because of the mixture of individual and aggregate level data; data was obtained from many sources; the sources include not only Federal statistical data warehouses and other organizations such as State Health Departments and Local School Districts that collect statistics and academic researcher’s data sets.

Consequently, the data vary considerably as to time periods, definitions of race and the number and frequency of time periods for which data were available. The statistics presented were obtained and tabulated by SPSS to determine means and frequencies and by Excel for graphical display. Some of the year’s BMI statistics are based on complete enumerations of a data set while others are based on aggregate samples. Therefore, the estimation procedures used to determine annual BMIs by age and gender vary from highly sophisticated scientific techniques, to crude “informed best guesses.”

Males age 6-9 show a very slight curvilinear increase in BMI over the century. The BMI change for 6-9 year old males during the course of the century is very slight, less than< 0.4 BMI units beginning at 16.4 BMI units in 1900 and ending at 16.9 BMI units in 2000. Interestingly, the BMI throughout the entire century is above the WHO classification for normal range but does not cross into the diagnostic overweight range until the 1980s. However, looking at the trend,
the BMI was relatively flat with the major increase in BMI beginning for this group in the 1970s.

Conversely, males' age 9 to 12 BMI shows a marked curvilinear increase over the century but their weights did not cross out of the WHO normal weight range until the 1950s. Beginning in the 1940s through the end of the century their BMI increases much more sharply than previously in the century. The BMI change for 9 to 12 year old males during the course of the century is fairly large, 2.1 BMI units during the century, beginning at 17.1 BMI units in 1900 and ending at 19.2 BMI units in 2000.

Graph 4.2: 6-9 year old Males BMI Trend During the 20th Century (1900-2000)
Graph 4.3: 9-12 year old Males BMI Trend During the 20th Century (1900-2000)

Females age 6 to 9 show a curvilinear increase in BMI over the century. Their BMI throughout the entire century is slightly above the WHO classification for normal range but never crosses into the diagnostic overweight range. The BMI in 6 to 9 yo females is fairly variable in the century with a large range at each time point. However, beginning in the late 1960s, their BMI begins to sharply increase. The BMI change again is fairly large for 6 to 9 yo females, 1.75 BMI units during the century from 15.8 BMI units in 1900, to 17.55 BMI units in 2000.
Graph 4.4: 6-9 year old Females BMI Trend During the 20th Century (1900-2000)

Females age 9-12 show an exponential BMI increase over the century. Their BMI in the early half of the century is in the WHO classification for normal weight range but in the 1950s crosses above the normal range and by 1975 crosses into the diagnostic overweight range. The BMI change for 9-12 year old females is very large, 3.05 BMI units during the century from 16.25 BMI units in 1900 and ending at 19.3 BMI units in 2000.
Graph 4.5: 9-12 year old Females BMI Trend During the 20th Century (1900-2000)

Food Consumption and Price Trend Graphs to Describe Children’s BMIs

Protein Types Consumption and Retail Prices, and Federal Policies that regulated protein in the U.S. food supply will be displayed in the next series of Graphs (4.6 – 4.25). Protein quality needs to be considered when assessing healthy pediatric diets with > 95% of the protein easily digestible and containing the essential amino acids, thusly foods such as meat, poultry, and egg protein can be used to gauge the health quality and availability of the food environment (R. Kleinman, 2004). Since the 1930s with the establishment of federally sponsored commodity support programs ("Agricultural Adjustment Act," 1938; Agricultural Adjustment Act of 1933," 1933; Commodity Credit Corporation Charter Act," 1948) which supported farm production, especially feed grain
production, U.S. agriculture policies have created an artificial low cost for meat (dairy products: Milk discussed in the Carbohydrate section and Cheese discussed in the Fat section), and poultry. Because corn is the major component of animal feed, the agricultural policies created a new market for cheap animal based protein (Drewnowski & Barratt-Fornell, 2004; Pollan, 2003). Since the fast food industry is based on inexpensive products of which beef and chicken are central, subsidy policies effectively created and subsidized the fast food industry (Cochrane & Runge, 1992; Tillotson, 2003b). By the 1970s, U.S. agriculture policies created a situation that for farmers to maintain a constant income, it was necessary to grow more corn which lead to even lower prices which significantly drove down the cost of many foods products based on corn and created a niche market for fast foods (Cochrane & Runge, 1992; Tillotson, 2003b). Thus, it was expected that as animal production increased, prices would fall, consumption would increase and so would BMIs increase. Looking at the BMI vs. per capita consumption of Protein (Graphs 4.6 - 4.13; 4.18 – 4.21) for Red Meat, Poultry, and Eggs and BMI vs. Protein prices in chained 1967 dollars (Graphs 4.14 – 4.27; 4.22 – 4.25) for Beef, Chicken and Eggs was expected to show that BMI increased along with rising consumption and falling prices. Red meat, defined as beef, veal, pork, lamb, and mutton by the USDA Economic Research Service ("Food Availability: Spreadsheets," 2009), consumption shows curvilinear trends with consumption decreasing during the depression and WWII then rising continuously post WWII reaching its peak in the 1970s then beginning to decrease towards the end of the century. Red meat consumption was at its nadir
in 1935 with of 76.9 lbs per capita and reached its peak in 1971 with 136.1 lbs per capita. Beef has lower overall consumption than red meat ranging from a high of 88.8 lbs per capita in 1977 to a low of 32.1 lbs per capita in 1937 (Beef Graphs not presented). Fish consumption has remained relatively flat during the century with < 15 lbs per capita consumed in any year [Fish Graphs not presented]. Overall protein consumption has risen since WWII and poultry consumption has also steadily increased since the 1930s. Poultry consumption has increased from < 12 lbs per capita before 1940 to 67.9 lbs per capita in 2000. The increase in protein consumption begins just after grain feed production subsidies were implemented in the Agricultural Adjustment Act of 1937 ("Agricultural Adjustment Act," 1938) and further augmented in the amended Agricultural Acts of 1947, ‘48 and ‘54 ("Agricultural Act of 1948," 1948; Agricultural Act of 1949," 1949; Agricultural Act of 1954," 1954), however the increase in BMIs are not predominant until the 1970s which leads one to postulate that food price might be inversely related to BMI.

Prices for protein have fallen during the century, with beef prices on the decline since the mid 1970s, and chicken prices on a steady decline beginning in the 1950s. The price of beef (chained dollars) rose to its highest point from the 1950s to 1970s, then prices declined and toward prices at the end of the century were comparable to prices in the early part of the century (1990’s vs. 1910’s). However, chicken prices were more variable throughout the century; prices by the 1980s were lower than prices at any other point in the century. The drop in red meat and chicken prices coupled with the large increase in consumption that
began post WWII and accelerated in the 1970s appears to have similar timing as when children’s BMIs began to increase.

Consumption of large amounts of protein could cause rapid growth trajectories and could explain the timing of the large increased in BMI seen in children. Another confounder that needs to be addressed is the advent of antibiotics and vaccinations of school children in the 1960s which would lead to better health and a corresponding increase in growth. Finally, beginning in the 1970s, antibiotics and growth hormones were routinely added to animal diets in the U.S. causing rapid growth (i.e. weight gain) and shorter times to market (meaning lower prices) in cattle and chicken (D. L. Kleinman, Kinchy, & Handelsman, 2005) which has been previously postulated to affect human growth.

Graph 4.6: Males Age 6-9 BMI Trend vs. Per Capita Consumption of Red Meat
Graph 4.7: Males Age 9-12 BMI Trend vs. Per Capita Consumption of Red Meat

Graph 4.8: Females Age 6-9 BMI Trend vs. Per Capita Consumption of Red Meat

Graph 4.9: Females Age 9-12 BMI Trend vs. Per Capita Consumption of Red Meat
Graph 4.10: Males Age 6-9 BMI Trend vs. Per Capita Consumption of Poultry

Graph 4.11: Males Age 9-12 BMI Trend vs. Per Capita Consumption of Poultry

Graph 4.12: Females Age 6-9 BMI Trend vs. Per Capita Consumption of Poultry
Graph 4.13: Females Age 9-12 BMI Trend vs. Per Capita Consumption of Poultry

Graph 4.14: Males Age 6-9 BMI Trend vs. Protein Prices (FY1967 dollars)

Graph 4.15: Males Age 9-12 BMI Trend vs. Protein Prices (FY1967 dollars)
It was expected that egg consumption would decline towards the end of century because of the aggressive health promotion campaign begun by the American Heart Association("Cholesterol," 2009) in the 1970s that linked eggs to cholesterol which ‘caused’ heart attacks. An Egg contains approximately 200mg cholesterol (about 2/3rd of the daily recommended intake), 5g of fat (2g is saturated fat) and 6 g protein; the yolk contains all the fat, vitamins (A,D,E) and half the protein (J. E. Brown et al., 2005). At the beginning of the century, eggs were thought of as a good source of protein that would contribute to healthy
nutrition but by the end of the century, eggs were perceived as an unhealthy source of ‘bad fat’ (i.e. cholesterol) (Ariouat & Barker, 1993; What we eat in America,” 1996).

Through the century, both egg consumption and prices were expected to decline and be inversely related to the rise in children’s BMIs. However, eventually egg consumption might be affected by the declining price which could level out consumption patterns when the price becomes so low that the use of egg products in low cost snack foods (such as cookies, cakes, pastries) would increase, this could affect BMIs in the opposite direction. Egg consumption during the century follows a u-shaped curve with peak consumption occurring in the 1940s and 50s with a nadir in 1990 and consumption beginning to rise toward the end of the century. Egg prices did fall throughout the century as predicted. However, the BMI trend does not appear to be similar to either egg consumption or prices, BMI rose as egg consumption and prices fell.

Graph 4.18: Males Age 6-9 BMI Trend vs. Per Capita Consumption of Eggs
Graph 4.19: Males Age 9-12 BMI Trend vs. Per Capita Consumption of Eggs

Graph 4.20: Females Age 6-9 BMI Trend vs. Per Capita Consumption of Eggs

Graph 4.21: Females Age 9-12 BMI Trend vs. Per Capita Consumption of Eggs
Graph 4.22: Males Age 6-9 BMI Trend vs. Egg Prices (FY1967 dollars)

Graph 4.23: Males Age 9-12 BMI Trend vs. Egg Prices (FY1967 dollars)

Graph 4.24: Females Age 6-9 BMI Trend vs. Egg Prices (FY1967 dollars)
According to USDA DRIs school aged children should be consuming approximately 55% of their total caloric intake as Carbohydrates (J. E. Brown et al., 2005). Therefore, commodities such as milk, wheat (and bread), corn (including high fructose corn syrup, HFCS), raw sugar (i.e. beet and cane) and candy (chocolate) will be used to gauge the health quality and availability of carbohydrates in the food supply. Looking at the BMI vs. per capita consumption of Carbohydrates (Graphs 4.26 – 4.29; 4.34 – 4.37; 4.42 – 4.53; 4.62 – 4.65) for Milk, Wheat Products, Corn Products, Sweeteners (Beet and Cane Sugar, HFCS), and Cocoa was expected to show that BMI increased as the type of carbohydrate consumed changed. Looking at the BMI vs. Carbohydrate prices in chained 1967 dollars (Graphs 4.30 – 4.33; 4.38 – 4.41; 4.54 – 4.61; 4.66 – 4.69) for Milk, Flour/ Bread, Corn (crop), Sugar, and Hershey Bar prices was expected to show that that BMI increased along with rising consumption of non-nutritive...
carbohydrates (HFCS and candy) and falling prices for the less nutritious carbohydrates (i.e. HFCS and candy).

Milk is defined by the USDA as the total flavored and unflavored whole milk plus low fat and fat free milk including 2% reduced fat milk, low fat milk (1%, 0.5%, and buttermilk), and skim milk /fat-free ("Food Availability: Spreadsheets," 2009). The DRI for milk recommends that children get 30-50% of their daily carbohydrate intake from milk; which is three 8 ounce glasses per day (J. E. Brown et al., 2005). It was expected that the consumption of milk would greatly increase from the mid to end of the century as federal subsidy programs were implemented to provide free and low cost milk to children. Thus, beginning in 1954 with the enacting of The Special Milk Program which was further augmented by The Child Nutrition Act of 1966 which permanently authorized WIC as a National Health and Nutrition Program, the expectation was that milk consumption would increase dramatically because of the federal policies and that retail milk prices would also increase because of the large demand. However, this is not what occurred, milk consumption shows a curvilinear trend with consumption slightly decreasing during the depression, then reaching its peak in 1945 and gradually decreasing through the rest of the century. Milk consumption was at its peak in 1945 with of 44.6 gallons per capita and has declined ever since even though legislation was enacted in 1954 to increase children’s milk consumption ("The Special Milk Program 1954 ", 1954) and further legislation was enacted throughout the 1960s, 70s and 80s to increase milk consumption ("An Act to Amend the Child Nutrition Act of 1956 and the National School Lunch
Act to Revise and Extend Certain Authorities Contained in Such Acts and for
Other Purposes," 1989; An Act to Amend the National School Lunch Act ", 1968;
An Act to Amend the National school lunch act and the Child nutrition act of 1966
in order to revise and extend the summer food program, to revise the special milk
An Act to Extend and Amend the special supplemental food program and the
child care food program, and for other purposes," 1978; Child Nutrition Act ",
1966b; Child Nutrition Act of 1966 Amended Permanent Authorization of WIC as
National Health and Nutrition Program," 1975; Food Security Improvements Act
found that dairy and calcium intakes are inadequate in 4 to 18 year-old children
and only 2 to 3year-olds met the USDA DRI recommendations; plus more than
half of the milk and milk products consumed was either whole milk, or the highest
fat varieties of cheese, yogurt, and ice cream (Kranz et al., 2007). Even with the
USDA focusing nutrition efforts on increasing the intake of the low fat dairy
products, and increasing calcium intake in school age children and adolescents,
milk consumption continues to fall (Kranz et al., 2007); this could be due to the
myriad of other low cost beverage choices that were introduced beginning in the
1970s. By 2000, only children who did not consume sweetened beverages (i.e.
soda, fruit juices) had calcium intakes that met the USDA DRI (Frary, Johnson, &
Wang, 2004). At the end of the century, lower than USDA DRI intakes for milk
and calcium was the worst in the lowest socioeconomic children who qualify for
the federal programs with 44% reporting lower than recommended intakes
(Nitzan Kaluski, Basch, Zybert, Deckelbaum, & Shea, 2001); therefore looking at milk consumption patterns shows that federal milk programs have not performed to mandate.

Graph 4.26: Males Age 6-9 BMI Trend vs. Per Capita Consumption of Milk

Graph 4.27: Males Age 9-12 BMI Trend vs. Per Capita Consumption of Milk
It was expected that milk prices would either remain steady or slightly increase because of the federal distribution policies, and thus not have an effect on children’s BMIs. Milk prices plateaued (with small peaks and valleys during the beginning to mid part of the century) and plummeted during the final part of the century (after 1965). As milk consumption fell at the end of the century so did milk prices. Currently, in 2009, dairy farmers are sending their cattle to the slaughterhouse because they are worth more as beef rather than as milking
cows (Cone, 2009); the retail price for milk is approximately half what it costs ($0.80/ gal) to produce the milk ($1.65/ gal); retail prices are continuing to falling with dairy farmers predicting a massive herd cull and requesting the federal government to implement policies to reverse the trend. The 2009 milk crisis is occurring because feed prices have risen because of the demand for feed grains to produce ethanol and the global recession has lowered the demand for butter and cheese U.S. exports (Cone, 2009). The falling prices of milk did not affect consumption but the lower prices might have had an effect of making high fat, high calorie milk products more readily available. Cheaper prices for product containing higher calories might have an inverse affect on children’s BMIs, therefore milk consumption would not need to increase to provide more calories contributing to an increase in calories consumed with the resulting increase in obesity.

Graph 4.30: Males Age 6-9 BMI Trend vs. Milk Prices (FY1967 dollars)
Graph 4.31: Males Age 9-12 BMI Trend vs. Milk Prices (FY1967 dollars)

Graph 4.32: Females Age 6-9 BMI Trend vs. Milk Prices (FY1967 dollars)

Graph 4.33: Females Age 9-12 BMI Trend vs. Milk Prices (FY1967 dollars)
It was expected that wheat subsidies beginning in the 1940s with the Steagall Amendment and Agricultural Adjustment Act and continuing into the early 1960s with the Feed Grain Adjustment Act ("Agricultural Act of 1948," 1948; Feed Grain Adjustment Act of 1961", 1961; Steagall Amendment of 1941 (Steagall Commodity Credit Act)," 1941), which was enacted to level out the price, production and supply of wheat, would then contribute to the creation of a niche market for inexpensive, refined non-nutritive ‘junk’ food, which in turn contributes to an increase in calories consumption with a resulting increase in obesity. This is not what happened with wheat consumption; both flour and grain consumption has decreased during the century with approximately 300 lbs per capita of wheat/grain products consumed in the early part of the century which dropped to approximately 200 lbs per capita by late century. But in the 1970s the decline did reverse which might indicate that subsidized refined wheat products were contributing to the increase in obesity in the past 30 years.

Graph 4.34: Males Age 6-9 BMI Trend vs. Per Capita Consumption of Wheat/Grains
Graph 4.35: Males Age 9-12 BMI Trend vs. Per Capita Consumption of Wheat/Grains

Graph 4.36: Females Age 6-9 BMI Trend vs. Per Capita Consumption of Wheat/Grains
Graph 4.37: Females Age 9-12 BMI Trend vs. Per Capita Consumption of Wheat/Grains

Graph 4.38: Males Age 6-9 BMI Trend vs. Flour / Bread Prices (FY1967 dollars)

Graph 4.39: Males Age 9-12 BMI Trend vs. Flour / Bread Prices (FY1967 dollars)
HFCS production has risen from 525,000 tons in 1975 to over 6.1 million tons in 1990 (Rendleman, 1991). However, corn consumption does exhibit a pattern consistent with the expectation that inexpensive corn [products] would contribute to the creation of a niche market for low-cost, refined non-nutritive ‘junk’ food (i.e. refined corn products and high fructose corn syrup), which sequentially contributes to an increase in calories consumed with the ensuing increase in obesity. Corn consumption showed relatively stable consumption (~
60 lbs per capita annually) during most of the century until the 1970s, when consumption exponentially expanded reaching a high of 196.2 lbs per capita in 1999. The burgeoning expansion of total corn consumption can be explained by the increase in HFCS use. By 1990, almost 50% of total caloric sweeteners consumed in the United States was HFCS (Rendleman, 1991); sweeteners are broadly defined and include cane and beet sugar, HFCS, honey, maple syrup, and artificial sweeteners. Thus as HFCS consumption has grown, the demand for corn to produce it has also significantly increased between the 1970s and 1990s leading to more crop subsidies and more consumption of non-nutritive food products and the increase in obesity in children.

Graph 4.42: Males Age 6-9 BMI Trend vs. Per Capita Consumption of Corn Products
Graph 4.43: Males Age 9-12 BMI Trend vs. Per Capita Consumption of Corn Products

Graph 4.44: Females Age 6-9 BMI Trend vs. Per Capita Consumption of Corn Products
However, there is a complicated relationship between corn production and consumption used for HFCS and sugar price subsidies; the Commodity Credit Corporation lends money to sugar cane processors to refine sugar and to pay growers ("Commodity Credit Corporation Charter Act," 1948; Rendleman, 1991). If the market price of sugar falls below the price when the loan originated, sugar processors can forfeit the sugar in lieu of repayment. Therefore, the government, to assure that they do not obtain large stockpiles of sugar, guarantees that sugar prices remain high enough for the loans to be repaid by manipulating the market and setting import tariffs. The governmental manipulation of the sugar market (i.e. supply / demand) has created a niche for HFCS as a low priced alternative to beet and cane sugar, thus as sugar prices remain high, more HFCS is produced and consumed with the resultant increase in non-nutritive calories and then the increase in childhood obesity.

Graph 4.45: Females Age 9-12 BMI Trend vs. Per Capita Consumption of Corn Products
Graph 4.46: Males Age 6-9 BMI Trend vs. Sugar Prices (FY1967 dollars)

Graph 4.47: Males Age 9-12 BMI Trend vs. Sugar Prices (FY1967 dollars)

Graph 4.48: Females Age 6-9 BMI Trend vs. Sugar Prices (FY1967 dollars)
HFCS was first introduced in the food supply in the 1970's because of high tariffs on sugar imports, which increased sugar prices; HFCS consumption grew at a rapid pace and eventually supplanted sugar as the predominant sweetener in beverages. But HFCS prices have become volatile during the early 2000s because of ethanol production which is diverting corn from HFCS production; prices have changed from 14.46 cents/ lb in 1994 to 13.30 cents/ lb in 2004 to 24.50 cents/ lb in 2008 ("Food Availability: Spreadsheets," 2009; Table 2.3.3. Real Personal Consumption Expenditures by Major Type of Product, Quantity Indexes [Data File]," 2008; United States Department of Agriculture NASS Quick Stats (Crops)," 2007).
Graph 4.50: Males Age 6-9 BMI Trend vs. Per Capita Consumption of Sweeteners

Graph 4.51: Males Age 6-9 BMI Trend vs. Per Capita Consumption of Sugar and High Fructose Corn Syrup Sweeteners
Graph 4.52: Males Age 9-12 BMI Trend vs. Per Capita Consumption of Sweeteners

Graph 4.53: Males Age 9-12 BMI Trend vs. Per Capita Consumption of Sugar and High Fructose Corn Syrup Sweeteners
Graph 4.54: Females Age 6-9 BMI Trend vs. Per Capita Consumption of Sweeteners

Graph 4.55: Females Age 6-9 BMI Trend vs. Per Capita Consumption of Sugar and High Fructose Corn Syrup Sweeteners
Finally, corn crop prices have not fallen during the century and have been artificially supported by government subsidies since the 1930s through the Agricultural Adjustment Acts, Soil Conservation Programs and Commodity Credit Programs ("Agricultural Act of 1948," 1948; Agricultural Act of 1956," 1956; Agricultural Act of 1970," 1970; Agricultural Adjustment Act," 1938; Agricultural Adjustment Act of 1933," 1933; Agricultural Credit Act of 1978 ", 1978; Agricultural Credit Act of 1987 ", 1987; Agricultural Programs Adjustment Act of
1984, 1984; Agriculture and Food Act of 1981, 1981; Federal Agriculture Improvement and Reform Act of 1996 (FAIR), 1996; Feed Grain Adjustment Act of 1961, 1961; Food and Agricultural Act of 1962, 1962; Food and Agriculture Act of 1977, 1977; Food Security Act of 1985. A Bill to extend and revise agricultural price support and related programs, to provide for agricultural export, resource conservation, farm credit, and agricultural research and related programs, to continue food assistance to low-income persons, to ensure consumers an abundance of food and fiber at reasonable prices, and for other purposes. 1985; Food, Agriculture, Conservation, and Trade Act of 1990 (Mickey Leland Memorial Domestic Hunger Relief Act), 1990. The translation between corn crop prices and retail prices for products that use corn as a major component is difficult to unravel since prices for products that use corn have not been consistently tracked throughout the century. However, because HFCS production uses a significant portion, over one-third of the corn crop and the resultant residue then enters the food stream as feed grain for animals, there is a dual use for virtually the same corn crop. This dual usage of the crop creates a net cost reduction; basically the price for using corn is half of the actual price. Federal policy has created a situation where governmental crop support has created inexpensive high caloric junk food industry with the resultant increase in both caloric consumption and children’s BMIs.
Graph 4.58: Males Age 6-9 BMI Trend vs. Corn Crop Prices (FY1967 dollars)

Graph 4.59: Males Age 9-12 BMI Trend vs. Corn Crop Prices (FY1967 dollars)

Graph 4.60: Females Age 6-9 BMI Trend vs. Corn Crop Prices (FY1967 dollars)
It was expected that as candy consumption increased and prices fell, a resultant increase in obesity would be exhibited. Candy consumption has increased during the century, from ~ 1 pound to almost 4 pounds per capita and is described by the per capita consumption of cocoa. Government educational policies, especially in school nutrition programs in the 1990s did not appear to slow the rising consumption of candy ("Nutrition Labeling and Education Act", 1990). Chocolate candy prices have fallen from the beginning of the century but then leveled off mid century as exhibited by Hershey Bar prices which may be due the expanding array of candy choices available after the advent of HFCS as an alternative to the higher priced sweeteners (sugar, honey and maple syrup); chocolate was supplanted as the one of the few candy alternatives and thus retained its price. From mid century to the end, it appears that children’s obesity patterns did follow a similar pattern to candy consumption.
Graph 4.62: Males Age 6-9 BMI Trend vs. Per Capita Consumption of Cocoa

Graph 4.63: Males Age 9-12 BMI Trend vs. Per Capita Consumption of Cocoa

Graph 4.64: Females Age 6-9 BMI Trend vs. Per Capita Consumption of Cocoa
Graph 4.65: Females Age 9-12 BMI Trend vs. Per Capita Consumption of Cocoa

Graph 4.66: Males Age 6-9 BMI Trend vs. Hershey Bar Prices (Chained FY1967$)

Graph 4.67: Males Age 9-12 BMI Trend vs. Hershey Bar Prices (Chained FY1967$)
Fat types consumption and retail prices, and federal policies that regulated protein in the U.S. food supply will be displayed in the next series of Graphs (4.70 – 4.85). Fat is required for healthy growth and body maintenance (i.e. to maintain optimal fatty acid composition in the tissues), in a healthy non growing human, approximately 5% of the total dietary intake should be fat (R. Kleinman, 2004; Otten et al., 2006). In children from 1 to 18 years old, many studies have shown that as long as caloric intake is adequate there is no effect of fat intake on
growth; therefore there is only a recommended upper limit for fat intake which is less than 30% of total caloric intake (IOM, 2005). Fat is defined by the USDA Economic Research Service as added fats comprised of food stuffs such as: butter, margarine, lard, edible beef tallow, shortening (both animal and vegetable), salad and cooking oils and other ‘added’ fats contained in foods such as high fat cheese, red meat, eggs, etc. ("Food Availability: Spreadsheets," 2009). Cheese and whole milk account for almost 25% of the saturated fat in the U.S. diet (Putnam et al., 2002). The rise in dietary fat consumption is postulated to be associated with the rise in meals eaten that were not prepared at home and in adults dietary fat consumption significantly increased as more meals were eaten outside of the home (McCrorry et al., 1999). When further looking at individual components of fats, shortening exhibited a constant rise during the century that became exponential increase from the 1960s to the end of the century, leading to an inexpensive fat that could be used in the fast food and junk food industries, leading to more ‘good tasting’ non nutritive calorie consumption and then to more weight gains. The definition of shortening changed during the century with components changing from animal to vegetable by products as the predominant fat; there are actually two trend curves collapsed into one category, shortening, but because of the data collected, the two components, animal shortening versus hydrogenated vegetable shortening cannot be teased apart. The advent of hydrogenated vegetable fat, Crisco, from cottonseed and soybeans, which have been subsidized since the 1930s, in the early part of the century created a new shortening that had longer shelf life and was cheaper to
produce than animal shortening (Beach et al., 2002). However, hydrogenation of fat creates trans fats which the IOM (2005) states “Trans fatty acids are not essential and provide no known benefit to human health” and should not be consumed because of the positive link between trans fatty and the increased risk of cardiovascular disease (Institute of Medicine. Committee on the Scientific Evaluation of Dietary Reference Intakes, 2005). It was expected that fat prices would decline and as total fat consumption increased contributing to higher caloric intake; it would lead to an increase in childhood obesity. Finally, as cotton and soybean subsidies lowered prices in the latter half of the century, they contributed to the production of inexpensive hydrogenated fats and the availability of cheap, refined junk food, contributing to an increase in calories and then a subsequent increase in obesity. Looking at BMI vs. per capita consumption of Fats (Graphs 4.70 to 4.81; 4.83; 4.85; 4.87; 4.87) included are Total Fats, High Fat Cheese, Shortening and Lard, and Margarine and Butter; and BMI vs. Fat prices in chained 1967 dollars (Graphs 4. 82; 4.84; 4.86; 4.88) included are Margarine and Butter was expected to show that BMI increased along with rising consumption of the cheaper fats and that BMI increases occurred when the composition of fats consumed changed from natural fats (lard and butter) to refined fats (hydrogenated vegetable shortening and margarine) in the latter half of the century. Fat consumption shows a continual and steady increased throughout the century, just like the increase in BMIs.
Graph 4.70: Males Age 6-9 BMI Trend vs. Total Fat Consumption

Graph 4.71: Males Age 9-12 BMI Trend vs. Total Fat Consumption

Graph 4.72: Females Age 6-9 BMI Trend vs. Total Fat Consumption
Lard exhibited a curvilinear pattern with an increase which reached its peak in 1932 with 14.4 pounds per capita and then a sharp decline by the end of the century to less than 1 pound per capita annually. Hydrogenated vegetable shortening consumption began to increase in the 1960s because of crop subsidies for soybeans and cotton which made the price significantly lower than lard. In the 1950s and 1960s, vegetable shortening was advertized as the healthy alternative to ‘unhealthy animal fats’ with the resultant increase in consumption. Finally, the advent of the fast food industry in the 1960s increased both the production and consumption of shortening leading to the ensuing increase in obesity.

Graph 4.73: Females Age 9-12 BMI Trend vs. Total Fat Consumption
Graph 4.74: Males Age 6-9 BMI Trend vs. Major Fats Consumption

Graph 4.75: Males Age 9-12 BMI Trend vs. Major Fats Consumption

Graph 4.76: Females Age 6-9 BMI Trend vs. Major Fats Consumption
Additionally, the advent of surplus commodity distribution programs in the 1950s and 1960s lead to distribution of high fat cheese products which would again contribute to higher caloric intakes, thus children’s BMIs were expected to increase. Cheese consumption was between 4-6 lbs per capita in the first half of the century but greatly expanded after the 1950s; the increased consumption occurred with the advent of school milk programs (1954) and surplus commodity distribution programs (1960s) that had cheese as one of the major dairy components. The increase in cheese (high fat content) consumption appears to just precede the increase in children’s BMIs.
It was difficult to determine price trends for various fats during the century because the usage pattern and types of fats consumed changed during the century; lard, beef tallow and butter were predominant in the early part of the century, however, by the 1950s vegetable shortening (hydrogenated fat containing significant amounts of trans fat) and oleomargarine were the predominant dietary fats. Prices for beef tallow, lard and vegetable shortening were not tracked consistently throughout the century (and the composition and definition of shortening changed from animal to vegetable fat further leading to confusion regarding exactly what products prices were measured); as a result consistent prices are available only for butter and oleomargarine and should be viewed as not a complete representation of the price for dietary fats during the 20th century. Butter prices have risen through the century with an accelerated rise in price associated with the advent of surplus dairy commodity programs begun in the 1960s; while margarine prices have remained relatively flat. Margarine and Butter trends appear to be interlinked since their patterns are
opposing mirror images of each other; as margarine consumption increased through the century butter declined. Prices for margarine and butter appear to describe a trend that shows consumption shifts to whichever fat was cheaper with the resulting over consumption of the cheaper fat and increased caloric intake creating higher obesity rates.

Graph 4.82: Males Age 6-9 BMI Trend vs. Butter and Margarine Prices (FY1967 dollars)

Graph 4.83: Males Age 6-9 BMI Trend vs. Butter and Margarine Consumption
Graph 4.84: Males Age 9-12 BMI Trend vs. Butter and Margarine Prices (FY1967 dollars)

Graph 4.85: Males Age 9-12 BMI Trend vs. Butter and Margarine Consumption

Graph 4.86: Females Age 6-9 BMI Trend vs. Butter and Margarine Prices (FY1967 dollars)
Graph 4.87: Females Age 6-9 BMI Trend vs. Butter and Margarine Consumption

Graph 4.88: Females Age 9-12 BMI Trend vs. Butter and Margarine Prices (FY1967 dollars)
The USDA budget, Federal Nutritional Programs and Commodity Price Support Programs will be displayed in the next series of Graphs (4.90 – 4.97). BMI vs. USDA Program Budgets (Graphs 4.90 – 4.93), and BMI vs. Commodity price supports will be presented separately (Graphs 4.94 – 4.97).

Graphs 4.90 – 4.93 display children’s BMI trends versus the Federal Budget for the USDA and the portion of the USDA budget specifically earmarked for Nutritional Programs. It was expected that as USDA programs increased, the food supply would expand providing more calories at a lower prices which would then result in an increase in caloric intake with the result being rising BMIs. The USDA budget trend is polynomial in nature and fluctuates with authorizing legislation: rising with the New Deal Commodity Programs in the 1930s ("Agricultural Adjustment Act," 1938; Agricultural Adjustment Act of 1933," 1933; Soil Conservation and Domestic Allotment Act," 1935; Soil Conservation and Domestic Allotment Act," 1936), Steagall Commodity Credit Amendment, 1942 ("Steagall Amendment of 1941 (Steagall Commodity Credit Act)," 1941); Post

However, beginning in the 1970s, the nutritional component of the USDA budget was separately reported and authorized (still as part of the omnibus Farm Bill but separately accounted for) which accounts for the fluctuation that describes the ‘falling’ USDA budget during the remainder of the 1970 and 80s ("Agricultural Act of 1970," 1970). The rising fluctuation was due to the 1996 Farm Bill which included significant increases because of emergency producer price supports("Federal Agriculture Improvement and Reform Act of 1996 (FAIR)," 1996). USDA Nutritional programs have been in existence since the Agricultural Adjustment Act of 1933, Section 32, which created distribution programs where large quantities of surplus food were distributed to needy households and to school lunch programs ("Agricultural Adjustment Act of 1933," 1933), budgets for these programs have normally been included in the USDA budget appropriation, with a few exceptions during WWII, until the 1970s when
budgets for nutritional programs were separated from the main USDA appropriation. The USDA Nutritional Programs Budget trend is polynomial in nature and fluctuates with authorizing legislation. The large rise in the budget from the mid 1960s to 1995 was due to the rapid expansion of School Lunch (and Breakfast) Programs, Food Stamp Programs and WIC (Women, Infant and Children’s Supplemental Food Program). In 1996, the Personal Responsibility and Work Opportunity Reconciliation Act began the reduction of food program benefits to adults ("Personal Responsibility and Work Opportunity Reconciliation Act ", 1996) and the 1998 School Lunch Act was expanded to assure that children had appropriate nutrition in the school programs ("An Act to Amend the National School Lunch Act and the Child Nutrition Act of 1966 to Provide Children with Increased Access to Food and Nutrition Assistance," 1998); these two legislative acts created the downward fluctuation at the end of the century.

Graph 4.90: Males Aged 6-9 BMI Trend vs. Federal USDA Spending
Graph 4.91: Males Aged 9-12 BMI Trend vs. Federal USDA Spending

Graph 4.92: Females Aged 6-9 BMI Trend vs. Federal USDA Spending
Commodity Price Supports (Graphs 4.94 – 4.97) are associated with both the composition of the food supply and the retail prices of foods. Thus, it is expected as commodity price supports increase, the food supply will expand and retail prices will fall, creating cheaper and more abundant calories thereby increasing obesity rates. However, there are large swings from year to year that affect the commodity price supports; the swings are caused not only by crop failures but the changing Farm policy legislation. Crop commodity program funding has become more volatile as Farm Bill legislation has been adopted to either discontinue the program (1970s) or reign in the costs (late 1980s). As commodity programs have expanded especially in the latter half of the century, they had a large effect on the foods supply and in turn it appears that increasing BMI have a similar pattern as the increases in commodity program support.
Graph 4.94: Males Age 6-9 BMI Trend vs. Crop Subsidy (FY1967 dollars)

Graph 4.95: Males Age 9-12 BMI Trend vs. Crop Subsidy (FY1967 dollars)

Graph 4.96: Females Age 6-9 BMI Trend vs. Crop Subsidy (FY1967 dollars)
BMI vs. Percent Household Income Spent on Food will be displayed in the next series of Graphs (Graphs 4.98 – 4.101). It was expected that as percent of income spent on food declined over the century that households would choose healthier more nutritious food generating better health and optimal weights, thus it would be expected that weights would increase until mid century and then level off. However, this is not what happened, as the percentage of income declined the obesity rate increased, and once the percent of income spent on food fell below 15%, the obesity rate increased more rapidly. The percentage of household income has declined; in the early part of the century approximately 25% of a household’s income was spent on food. The percent of income spent on food peaked during WWII (27.67% in 1945), and by the early 1960s had dropped to below 20%. Food costs to households continued to drop through the end of the century and had declined to 12.1% by 2000.
Graph 4.98: Males Age 6-9 BMI Trend vs. % Household Income Spent on Food

Graph 4.99: Males Age 9-12 BMI Trend vs. % Household Income Spent on Food

Graph 4.100: Females Age 6-9 BMI Trend vs. % Household Income Spent on Food
Inherited Factors to describe Children's BMIs

RQ2) The expression of the 'thrifty' phenotype in the current food environment has created the large spike in obesity that has been seen in the last 30 years in the United States.

RQ2 will be measured by graphs of school children's BMI overlaid with their mother's generation's BMI when the mothers were also school age, respectively. Patterns will emerge between the linear equations that describe the generational trend lines.

Graphs 4.102 to 4.117 display the descriptors for RQ#2. The following series of Graphs will describe the generation with two date series; the first date series describe when the children were born (b. xxxx-xxxx) and the second date series describe when the actual anthropometric measurements were collected [BMI during xxxx-xxxx]. The x-axis is a year time span from 0 to 16 which
describes the actual year in the time span the anthropometric measurement was taken, for example if the time span was 1900 to 1915, 0 would correspond to 1900 and 16 would correspond to 1915, and so forth. The x-axis was standardized in this manner so all graphs would display the same axis for ease of comparison. The y-axis displays BMI units from 15 to 20 and was chosen to optimize comparability between graphs. Each generational data series is described by a linear trend line which is presented along with the $R^2$ value on the graph. Graphs are paired the same mother’s generational BMI trend is presented twice for clarity, once in a graph with sons and then in another graph with daughters generational BMI trend.

“Thrifty” phenotypes are expressed as high BMIs and high percent body fat compositions are heterogeneous phenotypes which results from the combined effects of genes, environmental factors, and their interactions (Borecki et al., 1991). It appears that there is prominent heterogeneity between generations suggesting that there may be significant developmental genetic or environmental effects during early childhood growth to adult pattern phenotypes, particularly for the complex indicators of body composition (Borecki et al., 1991). The most current research evidence (2005) has shown that there are 176 human obesity single-gene mutations (alleles) in 11 different genes and 50 allele loci related to human obesity with causal genes or strong candidates identified for most (Rankinen et al., 2006). It is expected if the thrifty phenotype is not expressed that during times of food scarcity for the mother’s generation, the mother’s generational BMI trend will be similar to both their mother’s and children’s
generational BMI trend; and in times of food plenty for both generations, the mother's, their mother's and their children's BMI trends will be similar. However, if the thrifty phenotype is expressed then in times of food scarcity for the mother's generation, they will be smaller than their mothers and their children will be larger; and in times of food plenty the generations will become successively larger.

In the generational graphs, during periods of food scarcity the mother's generational BMI (4.109) was smaller than their mother's BMI (Graphs 4.105) and their children's BMI (Graphs 4.111 - 4.112). The scenario of food plenty, post WWII and during the 1970s, was also exhibited (Graphs 4.112 – 4.115) where the mother's generational BMI was either similar or larger than their children's BMI, and the generations kept getting successively larger. It is interesting to note that mothers born just prior or just after federal policies to regulate food safety (FDA Act, 1906) and establishment of maternal infant hygiene programs designed to prevent infant mortality (Sheppard-Towner Act, 1921) were actually larger than their offspring who were born during the depression (Graphs 4.104 – 4.105). BMI trends appeared to be most affected by the food supply rather than health policies. There is no indication that the Influenza pandemic (1918-19) had any effect on BMIs; however the composition of the population did change and birth rates were effected (Gernhart, 1999).

Patterns exhibited of different line slopes (i.e. mother decreasing; children increasing) occurred when the children were born during times of food scarcity, such as the great depression and the war years,(Graphs 4.106 – 4.107) and
when the mothers were born just after a period of food scarcity (Graphs 4.112 – 4.113). Additionally, generations appear to have similar linear equations during times of multigenerational food stability (Graphs 4.102 - 4.103; 4.109 - 4.110; 4.114 - 4.115).

BMI Graphs to Describe Relationships Between the Generations

The following graphs display the multigenerational growth patterns allowing a visualization of generational comparisons and growth line slopes.

Graph 4.102: Generational Pattern Mother’s BMI trends (b. 1888-1911) vs. Son’s BMI Trends (b.1912- 1935)
Graph 4.103: Generational Pattern Mother’s BMI trends (b. 1888-1911) vs. Daughter’s BMI Trends (b.1912-1935)

Graph 4.104: Generational Pattern Mother’s BMI trends (b. 1903-1917) vs. Son’s BMI Trends (b.1922-1939)
Graph 4.105: Generational Pattern Mother’s BMI trends (b. 1903-1917) vs. Daughter’s BMI Trends (b.1922-1939)

Graph 4.106: Generational Pattern Mother’s BMI trends (b. 1907-1927) vs. Son’s BMI Trends (b.1933-1947)
Graph 4.107: Generational Pattern Mother’s BMI trends (b. 1907-1927) vs. Daughter’s BMI Trends (b. 1933-1947)

Graph 4.108: Generational Pattern Mother’s BMI trends (b. 1921-1936) vs. Son’s BMI Trends (b. 1941-1957)
Graph 4.109: Generational Pattern Mother's BMI trends (b. 1921-1936) vs. Daughter's BMI Trends (b.1941-1957)

Graph 4.110: Generational Pattern Mother's BMI trends (b. 1930-1946) vs. Son's BMI Trends (b.1957-1973)
Graph 4.111: Generational Pattern Mother’s BMI trends (b. 1930-1946) vs. Daughter’s BMI Trends (b. 1957-1973)

- **Equation**: \( y = 0.0186x + 16.931 \) \( R^2 = 0.0072 \)
- **Equation**: \( y = 0.0253x + 17.041 \) \( R^2 = 0.011 \)

Graph 4.112: Generational Pattern Mother’s BMI trends (b. 1941-1957) vs. Son’s BMI Trends (b. 1963-1985)

- **Equation**: \( y = -0.0943x + 18.204 \) \( R^2 = 0.1504 \)
- **Equation**: \( y = 0.0419x + 17.28 \) \( R^2 = 0.0284 \)
Graph 4.113: Generational Pattern Mother’s BMI trends (b. 1941-1957) vs. Daughter’s BMI Trends (b. 1963-1985)

Graph 4.114: Generational Pattern Mother’s BMI trends (b. 1951-1969) vs. Son’s BMI Trends (b. 1979-1994)
Further exploration of the inheritability of the Thrifty phenotype is displayed in the following three-generational graphs (Graphs 112-113). It is implausible to look at more than one generation of sons, consequently, sons were not included in the three-generational graphs, especially since gestational effects do not directly affect the offspring's, when it is a son, child-bearing capacity (i.e. womb physiology for pregnancy). Widespread evidence shows that inherited genetic factors predispose an individual to obesity (O'Rahilly & Farooqi, 2006) and Li et al (2009) postulates that because of the genetic link to obesity that reductions in the incidence of childhood obesity in the current population may reduce obesity in their children, i.e. future generations (L. Li et al., 2009). It has been assumed that inherited genetic factors would influence either metabolic
rate or the selective conversion of excess calories into fat; but as evidence accumulates “. . . it is notable that, thus far, all monogenic defects causing human obesity actually disrupt hypothalamic pathways and have a profound effect on satiety and food intake” rather than on metabolism and “it seems that from an aetiological/genetic standpoint, human obesity appears less a metabolic than a neuro-behavioural disease” (O'Rahilly & Farooqi, 2006).

Females (b.1922-1948) born just before or during the depression and WWII when there was food scarcity and rationing, were smaller than their mothers (b. 1898-1918) (Graph 4.116). These females also had larger offspring (b. 1951-1971). The slope of the BMI trend lines for all the generations was similar (m= -0.03; -0.05; -0.02, respectively). This is an exhibition of the Thrifty phenotype (Graph 4.116).

To further look at the exhibition of the Thrifty phenotype, both the daughters (b. 1951-1971) and their daughters (b. 1989-1994) of the females (b.1922-1942) born and raised during a time of food scarcity were graphed (Graph 4.117). Again, the females (b.1922-1948) born during food scarcity were smaller than their daughters (b. 1951-1971) and their daughter’s offspring (b. 1989-1994) and each successive generation became larger but still retained similar slopes for the BMI trend lines (m= -0.05; -0.02; -0.03; respectively). Graph 4.117 again shows expression of the Thrifty phenotype that is retained in future generations which is exhibited initially in the grandmothers and then continues in the daughters and granddaughters.
Graph 4.116: Three Generational Overlay early to mid century Grandmothers (b. 1898-1918), Mothers (b. 1922-1942), and Daughters (b. 1951-1971) BMI Trends
Graph 4.117: Three Generational Overlay mid to late century Grandmothers (b. 1922-1942), Mothers (b. 1951-1969), and Daughters (b. 1989-1994)

Summary of Findings

Findings support earlier research which concluded that children’s BMI was increasing exponentially since the 1970s (C. L. Ogden et al., 2002), however this study found that children’s BMI changes appeared to happen around the same time periods that there were changes in the composition of the food supply over
the course of the century. As commodity supports increased, households spent less on food and children’s BMI increased. Not all food prices declined during the century but in most cases the prices of food trends did not readily describe BMI trends, but food commodity price supports did describe both increased food consumption of meat, fat and corn products and children’s BMI trends. The widespread public health programs for childhood vaccinations during the 1960s did not appear to affect the BMI trends at all. BMI trends appears to response more to food supply changes such as shortages during the depression and war years and the expanded feeding programs and food plenty coupled with lower prices during the 1970s. Additionally, there appeared to be generational changes that could be described as exhibition of the ‘thrifty’ phenotype with generational BMIs responding to changes in the food supply. Conceptually, the life course theory of the health appears to adequately describe the generational BMI changes that occurred during the century.

BMI in young males (age 6-9) did not change to a great extent during the century. This might be due to younger male’s body composition which has more lean muscle mass than girls and older boys (Maynard et al., 2001); their metabolism is less efficient in storage of excess calories, therefore they would not gain as much weight even with caloric overconsumption. Interestingly even at the beginning of the century, their BMI was above the WHO normal midpoint range for 6-9 year old males; it continuously remained above the range throughout the entire century. Their BMI trend remained below the WHO overweight range for most of the century (1900-1990) but did cross into the
overweight range after 1990. The young males aged 6-9 years actually had the lowest absolute BMI of the 4 sub-groups. The 6-9 year old male sub-group had the lowest relative BMI of the 4 groups meaning that they were the leanest (i.e. lowest adipose to lean muscle mass), but they were also the furthest of the sub-groups from what the WHO would define as normal midpoint BMIs for their relative age and gender. Additionally, the 6-9 year old male sub-group also had the smallest absolute BMI change (< 0.4 BMI units) during the century. Two conclusions can be drawn from the results: U.S. males aged 6-9 were always larger than their international counterparts (used for the WHO ranges) and because they have the lowest adipose tissue to lean muscle mass are not as affected by changes in the food supply and diet that affect adipose tissue as other sub-groups. In contrast, BMI in older males (9-12), and young females (6-9) was in the WHO normal midpoint range until the mid-1950s. By the 1970s in older males (9-12) BMIs crossed and stayed in the overweight range, however the young females (6-9) did not cross into the overweight range until the 1990s. It appears that changes in the food supply affected the older males more than the younger females, possibly because the males are closer to beginning the puberty growth spurt and would be consuming more food so the composition of the food supply would have a greater effect on them. BMI in older females (9-12) did not cross out of the WHO normal midpoint range until the 1960s when it crossed and then remained in the overweight range. However, females aged 9-12 experienced an exponential increase in BMI during the century with a change of more than 3 BMI units. The normal midpoint range for older females is much
larger because there is wide variation in the onset of the puberty growth spurt (Cooper, Kuh, Egger, Wadsworth, & Barker, 1996). Also the female body has more adipose tissue than the males (Maynard et al., 2001; Treuth et al., 2000; Treuth, Figueroa-Colon et al., 1998; Wong et al., 1999) thus more susceptible to obesity because adipose tissue efficiently stores excess calories compare to lean tissue; so the consumption of excess calories in someone with more adipose tissue effectively inclines them toward higher weight gain. Additionally, females are heavier than males at the prepubescent age and again would have more weight variation. The combination of more initial adipose tissue and an earlier growth spurt would predispose the females to be more affected by the changing composition of the food supply. But the BMI trends for 9-12 year old females do not confirm the previous supposition; growth has changed throughout the century, but females aged 9-12 growth was exponentially increasing before federal policies directly impacted the food supply, mid century, so other factors (such as healthcare, physical activity, etc.) are also involved in the increase. The BMI trend results must be tempered with the acknowledgement that only white children were included in the analysis and that multi-ethnic children’s growth is different (Saha et al., 2005; Wong, Stuff, Butte, Smith, & Ellis, 2000), however a reasonable supposition is that multi-ethnic children would also have increases in BMI, but not the same trend patterns, occurring across the century.

Intergenerational BMI patterns appeared with a generations’ BMI associated with either the historical prevalence or absence of an abundant cheap food supply when they were very young. Intra-generational patterns appear to have emerged
and again appear to be associated with the historical food supply prevalence. Thus, the food supply during periods of the life course appears to affect future growth patterns; therefore changes in food policy will have a long period where the policy effect cannot be measured.

Overall, the implementation timing of most federal health programs/policies did not appear affect or cause changes points in the BMI trends in children (i.e. 1921, Sheppard Towner Act; 1935 Social Security Act; 1941 Antibiotics Mass Availability; 1955 Vaccination Programs) except possibly for the 1975 permanent authorization of WIC which changed the caloric intake of the enrolled children.

Consumption of meat (beef, chicken, lamb, pork) increased after WWII due to the establishment of the Commodity Credit Corporation, PL 80-806 which was designated to carry out price-support and income support activities for the many basic (i.e. storable) commodities such as feed grains ("Commodity Credit Corporation Charter Act," 1948). This bill kept grain prices constant and low enough so it was economical to use grain for animal feed; which in turn fostered higher meat production and lower prices for meat products. The drop in protein prices follow a similar curvilinear trend as the drop in percentage household income spent on food; meaning that it was cheaper to consume more calories from protein at the end of the century versus than at the beginning. Poultry consumption increased much more than the other meats and after 1970 there was an exponential increase in consumption. In that same time period prices for all protein products (meat, poultry, and eggs) drastically declined. Other
research determined that as prices fell for beef and chicken fast food items, consumption increased and the overall nutritional quality of the diet declined (Beydoun et al., 2008). As the health consciousness of a person increased so did poultry consumption, including fast food poultry, (Moon & Ward, 1999); concern for health meant diet changes, the substitution of poultry for other meats, but did not mean dietary restraint, or caloric restriction. The exponential increase in poultry consumption equating to a ‘healthy choice’ might explain the rapid rise in BMIs beginning in the 1970 and 80s when healthy living was becoming a mainstream concern. Also egg consumption has fallen drastically in the second half of the century, again probably related to cholesterol with eggs being promoted as an ‘unhealthy choice’ and is an indicator of the changing diet of U.S. children. However, the large overall increase in protein consumption coupled with the large price reductions meant more calories per individual, creating an increase in obesity.

Milk consumption peaked in 1945 and has been declining ever since. It is difficult to explain the trends for milk consumption which peaked in 1945 and is on the decline ever since; the dramatic fall in milk consumption occurred even with large increases in milk promotion in the surplus food programs, school lunch programs and WIC which distribute milk to the population during the 1950s-70s (Robinson & United States. Food and Nutrition Service., 1978). Oddly enough, high fat content cheese consumption greatly increased in the latter half of the century with the consumption increases appearing to begin at the advent of federal surplus food programs. Even with the increase in health consciousness
at the end of the century, milk consumption has not rebounded; thus the profitability of dairy farming is also on the decline with dairy herds being sold for meat; resulting in lower meat prices and more meat in the food supply (Cone, 2009). Milk prices have also experienced a large decline since the 1950s. In 2009, the retail price of milk was approximately half what it costs to produce the milk, with milk consumption continuing to fall it is predicted that one-third of the nation's milking cows could be slaughtered in 2009 as dairy farmers try to cut costs and remain profitable. Beginning in the 1970s there was a large increase in drink choices (which might have supplanted milk as the primary beverage) for children thus leading to the consumption downturn. As milk consumption declined, children's BMI increased which might be partially explained by the large increase in consumption of fruit juice and soda products (Hawkins & Law, 2006; Kral et al., 2008). The falling prices of milk did not affect consumption but the lower prices suggest that high fat, high calorie milk products are cheaper and more readily available. Cheese (high fat content) consumption greatly increased in the latter half of the century after the surplus food programs were implemented; it appears that the increase in cheese consumption began around the same time as the sharp increase in children's BMIs. Most of the milk products consumed at the end of the century were processed alternatives to unprocessed liquid milk. Cheese and high fat milk product account for 25% of the saturated fat in the U.S. diet and more than half of the milk and milk products consumed was either whole milk, or the highest fat varieties of cheese, yogurt, and ice cream ("Food Availability: Spreadsheets," 2009). Cheaper prices for
product containing higher calories might have an inverse affect on children’s BMIs, therefore milk consumption would not need to increase to provide more calories contributing to an increase in calories consumed with the resulting increase in obesity.

Carbohydrate consumption followed similar patterns as protein, with steady or declining consumption from the beginning to mid century and then a large increase at the end of the century; once again leading to an over consumption of calories and possible part of the explanation of increasing BMIs. But carbohydrate consumption trends were primarily composed of substantial increases in sweetener and snack food consumption (Bray et al., 2004; Competitive foods and beverages available for purchase in secondary schools-selected sites, United States, 2004, 2005; Fisher & Birch, 2002; Gross et al., 2004); snack food and sweetened soda are the primary source of non nutritive calories in American diets. The burgeoning expansion of total corn consumption can be explained by the increase in HFCS use. Corn consumption does exhibit a pattern consistent with the expectation that inexpensive corn [products] would contribute to the creation of a niche market for low-cost, refined non-nutritive ‘junk’ food (i.e. refined corn products and high fructose corn syrup), which sequentially contributed to an increase in calories consumed with the ensuing increase in obesity. Wheat consumption steadily declined through most of the century reaching its nadir in 1972 and then began an increase that predates by approximately 5-years an increase in children’s BMIs in the late 1970s. The increase in both wheat and corn consumption is probably due to the large
increase in the varieties of snack foods that were beginning to be marketed in the
1970s. Prices of wheat and bread have been relatively stable throughout the
century; however corn crop prices have been volatile and have increased as
commodity price supports have increased. Sugar prices have dramatically
increased in the last half of the century and consumption has dropped with a
corresponding increase in HFCS consumption. Chocolate consumption has
increased throughout the century, but in the 1970s there was a decline with a
rebound increase in the 1980s. Chocolate consumption has increased
throughout the century, but in the 1970s there was a decline with a rebound
increase in the 1980s, the decline might have been due to HFCS and
introduction of so many new choices of candy and snack foods.

Total fat consumption has steadily risen throughout the century; the rise
did not accelerate at the end of the century like protein and carbohydrates (i.e.
beef, chicken, corn) that are linked to fast food and snack food. Patterns of
consumption for different types of fats have changed with the change in fat types
accelerating at the end of the century. The composition of fats consumed has
changed from naturally produced fats (lard, butter) at the beginning of the century
to refined fats (hydrogenated vegetable shortening and margarine) at the end; as
the consumption of refined fats increased so did children’s BMIs.

Percentage of Income spent on food began a steady decline in mid
century and by 1965 had fallen below 20% and by 1980 below 15% of income;
the rise in BMI appears to have occurred at the same time suggesting an inverse
linkage between food costs for a household and the BMI of its children.
Explanation of the generational patterns is easily grounded in historical events during the early life course of the generation. An example of expression of the Thrifty phenotype is the generation of children born during the depression; they are smaller than both their parents and their children. Additionally, their grandchildren are also larger; the depression generation has offspring generations that get successively larger gaining approximately 1 BMI unit per respective generation. Historically, the depression generation (1930s) experienced food scarcity and limited calories either in utero and/or during their early years thus triggering a metabolic response, the thrifty phenotype, which would slow metabolism and store extra calories (as adipose tissue). The metabolic changes would then change their uterine environment and their offspring would be predisposed to a lower metabolism and a propensity to store calories as fat. This offspring generation (1960s) grew up in a time of food plenty and changing food supplies created by crop production subsidies that lowered prices (corn/wheat, meat), commodity surplus programs that provided reduced price or free foods (dairy) and tariffs that created niche markets for cheap alternative foods (HFCS and hydrogenated vegetable oils) during the 1960s and 70s. There were more cheap calories in the food supply with high fat, high carbohydrate fast food and snack food becoming a fixture in the U.S. diet. These children eating this higher caloric diet then gained weight and as they aged were predisposed to higher incidence of Adult type II diabetes, hypercholestrimia, and heart disease. Their uterine environment was again different from their mothers’ and predisposed their offspring (1990s) to not only the thrifty phenotype but to
other metabolic phenotypical changes. These phenotypical changes are exhibited in the millennial generation (1990s) who are developing metabolic syndrome, type II diabetes, hypercholestrimia, and heart disease as children and weigh more than any previous generation. It will be interesting to see their offspring’s characteristics and food metabolism phenotypes in 30 years. The millennial generation also lives in a period of food plenty; however the food supply in the beginning of the 21st century is again being changed by federal food policies.
“Every day you may make progress. Every step may be fruitful. Yet there will stretch out before you an ever-lengthening, ever-ascending, ever-improving path. You know you will never get to the end of the journey. But this, so far from discouraging, only adds to the joy and glory of the climb.”
Sir Winston Churchill

Purpose and Objectives

The purpose of this study was to describe a 100-year time span for the growth of U.S. school children during the 20th century overlaid upon changes in federal agricultural policies that directly affected the composition of the U.S. food environment. The objectives of the study were as follows:

1) Describe trends in children’s weights following adoption of different federal agricultural policies as measured by commodity component’s availability and price in the U.S. food supply.

2) Describe generational trends in children’s weight status overlaid with historical context such as adoption of significant U.S. health policies and medical practices (such as immunizations and wide spread
antibiotic usage) which contribute to a healthy childhood environment, food shortages during various periods, and food supply changes due to adoption of significant U.S. agricultural policies (such as school lunches, crop subsidies and various commodity production programs).

The research questions were:

RQ1) Did the aggregation of U.S. agricultural and nutrition policies throughout the past century create a selection process for that is biased towards obesity?

RQ2) Was the large spike in obesity that has been seen in the last 30 years in the United States created by expression of the thrifty phenotype across the generations?

Procedures

This descriptive retrospective study described the intersection between U.S. agricultural policy which shape the nutrient composition of the U.S. food supply and anthropometric measurements regarding body typology related to obesity in American children during the 20th century. Descriptions of the food and BMI trends were tempered with historical events that might illuminate the direction and strength of the trends. The study described trends in the 20th century for pediatric BMIs and the composition of the U.S. food supply. Pediatric BMI trends and the availability of selected nutrient components of the U.S. food
supply were graphed. The U.S. food environment was described in terms of
‘food availability’ defined as both the per capita retail food consumption and price
(i.e. cost per unit of food adjusted for inflation). The measures provide a picture
of overall amounts of food available for human consumption annually and the
actual accessibility of the food to the average person. To complete the trend
picture federal spending on agricultural and nutrition programs and the percent of
household income spent annually on food was also displayed.

Discussion

Federal manipulation of agricultural production, distribution, price and the
overall food supply accelerated after WWII, sharp increases in nutritional
programs occurred in the 1960s and so did exponential increase in crop subsidy
programs during the same time period creating a food supply that was very
different in both which crops were produced and prices from the beginning of the
century. The food supply, across all categories, protein, carbohydrates and fats,
showed distinct changes from the beginning to the end of the century that could
be historically explained by federal food policies. The food trends all describe the
changing nutritional composition of the U.S. food supply which then supplied
more calories per capita. Consumption patterns of types of food and nutritional
diet quality also changed in the time period when the sharp rise in children’s
BMIs were occurring. Additionally, the percent of household income spent on
food dropped dramatically in the latter part of the century and appears to be an inverse of the BMI pattern. The combination of increasing affluence and food production changes suggests an effect on both consumption patterns and BMIs.

Politics greatly influenced U.S. agricultural policies after the New Deal by determining which crops were supported and the resulting market price. Corn, sugar, and cattle were the most heavily subsidized crops which lead to a lower consumer price for added sugars, meat and fats and the abundance and low price of these products at the end of the century slowly changed food consumption patterns in the nation (Drewnowski & Barratt-Fornell, 2004; Pollan, 2003).

Unintended Consequences of Policy

During the mid 1950s, there was a continuing need to address the conundrum of record levels of production and declining prices. Therefore much emergency legislation was passed to bringing supplies into balance with demand and stabilizes prices. For example, when the federal government repealed the oleomargarine tax in the 1950s, oleomargarine consumption increased and then surpassed butter consumption. Arguments against the tax repeal predicted that oleomargarine would be priced enough under butter to give it a price appeal causing it to overtake butter as the food of choice (Issues of Oleomargarine Tax Repeal, 1948). Increasing consumption of oleomargarine would undermining the
product viability of natural less processed alternative, butter and is what did happen (Graph 78-81). This federal policy change (in the financial policy stream) shifted the eating patterns of the nation, by lowering the price of oleomargarine. The milk industry had less market for butter so butter prices rose, consumers shifted from butter to oleomargarine, this shift in eating patterns was an unintentional occurrence caused by change in tax structure. The arguments regarding the repeal of the oleomargarine tax foreshadowed the current agricultural policy debates regarding highly processed foods. “The real issue . . . legalizing of imitation foods, about which little would be known by the consumer . . . Man cannot fully duplicate the products of nature, the market for which could be wrecked by imitation products” (Issues of Oleomargarine Tax Repeal, 1948) p80. Federal taxation policies, affected the production of agricultural products, changing the composition of the food supply, changing food production/processing, and then changed dietary consumption patterns. Unintended consequence of policies in a finance policy stream (repeal of the oleomargarine tax) that is completely unrelated to the agriculture policy stream had a direct effect on the food supply creating a niche for cheaper alternative processed foods and appears to have an indirect effect on the health of the nation because of the change in consumption patterns (Issues of Oleomargarine Tax Repeal, 1948).

Another unintended policy consequence that changed the food supply at the end of the century occurred because of sugar tariffs which created a niche market for another imitation food product, high fructose corn syrup, HFCS. U.S.
agricultural policies on sugar production and import tariffs increased the price of sugar to assure viability of domestic production, which then made the low price of HFCS (subsidized by the corn crop program) more attractive for use in food products (Jurenas, 2007). U.S. sugar commodity programs were and are structured to protect domestic sugar producers to assure a sugar supply at a market price that will not lead to surplus stock that might be forfeited to the government instead of loan repayment. Because of the commodity programs U.S. consumers and food manufacturers pay more for sugar than they would if imports were allowed to enter without any restriction and constitute an indirect subsidy to sugar producers (Jurenas, 2007). The tariff program was intended to balance available sugar supply (i.e., domestic output plus imports) with food demand so that sugar market prices did not fall below price support levels, however, the consequence of the programs was to encourage substitution of a cheaper imitation sweetener, HFCS (Jurenas, 2007).

Sugar tariffs not only affect sugar prices but directly affect HFCS and meat prices. As sugar prices rose, more HFCS is produced more byproducts used for animal feed are also produced and the prices for HFCS and meat fall. HFCS production uses a significant portion, over one-third of the corn crop and the resultant residue then enters the food stream as feed grain for animals, there is a dual use for virtually the same corn crop. This dual usage of the crop creates a net cost reduction setting the price for using corn as a raw material at half of the actual price. A cycle has been created since the 1980s by federal sugar tariffs
that have fostered more HFCS and meat production (Rendleman, 1991), creating more high calories foods in the food supply that could be linked to rising BMIs.

Another well meaning subsidy program for cotton and soybeans has also created another imitation food, the hydrogenated vegetable oil (i.e. Trans-fat). The first vegetable based hydrogenated shortening was produced at the turn of the century as an alternative to meat based fats for candle making, however, since electricity was quickly becoming the predominate light source, and the oil was quickly re-branded as an alternative to lard. The product did not become popular until the late 1950, early 1960s because the commodity programs had amassed huge stocks of soybean and cotton commodities (Beach et al., 2002) which because of their low cost were processed into hydrogenated oils to replace the more costly natural fats, butter and lard. Hydrogenation creates a more dense fat has a consistency like butter and is a much cheaper imitation food. The problem with hydrogenation is that the essential fatty acids are no longer active, thus again interfering with the satiety response and causing overeating. Hydrogenation also creates trans fats which have been causally linked to high cholesterol levels and coronary heart disease (Institute of Medicine. Committee on the Scientific Evaluation of Dietary Reference Intakes, 2005). Because of many changes to commodity subsidy programs, prices for hydrogenated vegetable shortening are lower than what they would be without federal intervention creating another niche for an ‘imitation’ food (Beach et al., 2002).

Hydrogenated vegetable oil is an example like HFCS, of a niche being created for a food product as a cheaper alternative to less processed alternative
food products because of a well meaning federal policy (federal commodity program to support farmers). Price supports for soybeans and cotton where not intended to create a new cheaper food but the policies that created the commodity subsidy program however, had unintended consequences on the food supply and then indirectly on health.

Federal policies have created a situation where governmental crop support created an inexpensive high caloric junk food industry with the resultant increase in both caloric consumption and children’s BMIs. The increase of ‘imitation’ foods in the American diet appeared to occur in the early life course of the mothers of the current generation where pediatric obesity is rampant (Graph 113); thus could possibly be another trigger for the turn on of the ‘Thrifty phenotype. Policy streams that are not remotely related and that traditionally do not interact definitely affect multi modal issues such as obesity. The major problem with policy streams that do not talk to each other, such as the health and agricultural streams are that they can create policies that conflict and are at cross purposes with each other.

Unintended consequence resulting from federal commodity programs have effectively subsidized the snack and fast food industry by creating new imitation food products, HFCS and hydrogenated vegetable oil that can be used in production/ processing because of their extremely low price and because of the raw materials price, the final processed product also has an extremely low price. It is interesting that the examples of unintended policy consequences occurred in policy streams as diverse as finance, agriculture and health because
these policy streams generally are segregated from each other with little or no cross communication occurring. Since the 1970s with more and more changes to all the policy streams the decoupled nature of these streams appears to be worsening the pediatric obesity epidemic through unintended policy consequences. Wildavsky (2002) warns that “More and more public policy is about coping with consequences of past policies. . . The more we do, therefore, the more there is for us to do, as each program bumps into others and sets off consequences. . . In this way past solutions. . . turn into future problems (Wildavsky, 2002) p4.”

"The Food Stamp Act of 1964," 1964), Feed Grain Adjustment Act, 1961 ("Feed Grain Adjustment Act of 1961 ", 1961; Food and Agricultural Act of 1962," 1962), Food and Agriculture Act, 1965 ("Food and Agricultural Act of 1965," 1965), and the various augmentations to the child nutrition programs ("An Act to Amend the National School Lunch Act ", 1968; An Act to Amend the National School Lunch Act of 1946," 1970; Child Nutrition Act ", 1966b; Child Nutrition Act of 1966 Amended Permanent Authorization of WIC as National Health and Nutrition Program," 1975; Child Nutrition Act of 1966 Breast feeding Promotion Requirements Pilot Project," 1972). The rising USDA budget fluctuations at the end of the century was due to the 1996 Farm Bill which included significant increases because of emergency producer price supports ("Federal Agriculture Improvement and Reform Act of 1996 (FAIR)," 1996). USDA Nutritional programs have been in existence since the Agricultural Adjustment Act of 1933, Section 32, which created distribution programs where large quantities of surplus food were distributed to needy households and to school lunch programs ("Agricultural Adjustment Act of 1933," 1933). The large rise in the budget from the mid 1960s to 1995 was due to the rapid expansion of School Lunch (and Breakfast) Programs, Food Stamp Programs and WIC (Women, Infant and Children’s Supplemental Food Program). In 1996, the Personal Responsibility and Work Opportunity Reconciliation Act began the reduction of food program benefits to adults ("Personal Responsibility and Work Opportunity Reconciliation Act ", 1996) and the 1998 School Lunch Act was expanded to assure that children had appropriate nutrition in the school programs ("An Act to Amend the

Federal manipulation of agricultural production, distribution, price and the overall food supply accelerated after WWII, sharp increases in nutritional programs began in the 1960s with exponential increase in crop subsidy programs also occurring during the same time period. Agricultural policies established in from 1954-1970 rapidly changed the commodity programs which formed the core of agricultural policy and became out of sync with the modern farm market (Cochrane & Runge, 1992). Because of rapid technological advances in both mechanical equipment and in chemicals (fertilizers and pesticides), farmers were able to produce more with less effort and commodity unit costs dropped. Farmers who did not keep up with the rapid technical advances, sustained enough financial losses to be driven out of farming, thus agricultural production changed from numerous small farms to a few hundred thousand very large agribusiness farms (Cochrane & Runge, 1992). But as policies changes were implemented, instead of fixing the problems, the policies made things worse because economic assistance predominately went to the large agribusiness farmers and ignored the subsistence farmer on the verge of poverty, the policies ignoring the burgeoning poverty in rural America, created competition barriers for entrance to international markets for smaller farm operations, and drove more
part-time farmers out of farming (Cochrane & Runge, 1992). Thus the bulk of U.S. agriculture was concentrated in fewer and fewer farmers (Cochrane & Runge, 1992). In 1940, 17% of the population was engaged in farming but by 1960, only 6% of the population were farming (The United States Senate Committee on Agriculture, Nutrition, and Forestry 1825-1998. Members, Jurisdiction, and History, 1999).

The increases in federal programs appear to slightly precede and inversely correspond with the decline in the percent of household income spent on food. The timing of increases in federal food program trends and the decreased percentage of income spent on food trends are similar to the rise in BMI trends. Notably, since the 1970s, BMI trends and commodity program funding have very similar lines and slopes leading to the postulate that commodity policies are the factor that has the greatest effect on obesity. At the beginning of the 21st century, U.S. school children’s BMI is continuing to increase, federal commodity policies for corn, sugar and cotton are continuing to (indirectly) subsidize both HFCS and hydrogenated vegetable shortening, percent of income spent on food is beginning to stabilize around 10%, and the amount of excess calories in the food supply is continuing to expand.

Interconnectedness of Policies has created a Web whereas you change one and there is a reactionary change or effect in another. Policies to address pediatric obesity did not begin to impact the United States policy agenda until the 1990s (United States Department of Health and Human Services, 1991). Prior to the 1990’s nutrition and food policies were designed to address food
insufficiency, food insecurity, and under/ mal-nutrition (Garvue et al., 1971; Gould, 1972; Gunderson, 1971; Joint Committee on Health Problems in Education., 1957, 1962; National School Lunch Act,” 1946; Smith et al., 1971; United States Congress Senate. Select Committee on Nutrition and Human Needs, 1972; Young, 1971; Young & Nokkeo, 1970) and health policies were designed to reduce disease transmission and severity. However, obesity is a disease of mal-nutrition, unhealthy quality and quantity of the diet, so policy makers need to re-define food policy to encompass both ends of the nutrition spectrum, under and over nutrition (Current Status and Activities to Decrease the Prevalence of Obesity Among U.S. Children and Adolescents. Statement of William H. Dietz, MD, PhD, Director Division of Nutrition, Physical Activity, and Obesity National Center for Chronic Disease Prevention and Health Promotion Centers for Disease Control and Prevention, 2009).

Two main policy streams affect childhood obesity, the health and the agricultural streams; each stream flows independently and is driven by different forces. In addition, other discrete policy streams also can affect population health and the food supply. The financial stream influences population health by manipulation of funding stream for health care and research and by taxation of various food products. The transportation stream does affect health directly because of its control of the built environment such as city design that is appropriate for physical activity and transport of food to market.

However, when looking at pediatric obesity, the health stream appears to be the primary stream that attempts to set policies regarding pediatric obesity.

226
The other streams do not even consider unintended effects the policies would have on obesity during their agenda setting (Wildavsky, 2002). Many policies and programs outside the health policy stream that directly affect the food supply and population physical activity are developed with blinders on in regard to unintended policy consequences linked to obesity. For example, the School Lunch and School Milk Programs were touted as social welfare programs to assure the health of the America’s school children. However, the programs were never designed as a health program per se, they were designed to stabilize crop prices by eliminating surpluses and to introduce American produced foods to children to boost life time consumption and provide new markets for U.S. farmers (Levine, 2008). The School Lunch legislation was popular for both Republicans and Democrats because it was touted as either a subsidy for southern (white) farmers or a way to help poor children (Levine, 2008). In the 1970s, there were 15 amendments to the child nutrition programs (Martin & Oakley, 2007) creating entitlements which guaranteed that schools would be able to fund all meals. The series of policy adjustments actually adjusted funding levels and assured markets for farm commodities rather than assuring that appropriate nutrition was delivered to children. The unintended consequence of developing childhood feeding programs that were markets for farm commodities was to fuel consumption of calories rather than to assure a balanced diet. Providing calories without regard to nutrition only fueled the expanding obesity epidemic in children. Obesity is a multi-faceted problem with multiple causations and does not lend itself neatly to a conceptual framework (Exworthy, 2008). There is a belief that
because obesity cannot be defined as a single problem, policy solutions will be ineffective because the problem is personal to an individual rather than as a societal problem needing a societal policy solution (Exworthy, 2008). There has often been “no policy response” to obesity, making the problem even worse because of lack of consensus among researchers, policy specialist sub groups, and policy makers about the policy solutions required (Exworthy, 2008), and accordingly created more disconnection in already disconnected policy streams.

Policy disconnects between multiple streams have created an obesity epidemic that cannot be solved by policy changes in only one stream. The Multiple Streams Framework explains that coalition building by scientific leadership in governmental agencies is going to be necessary to solve the obesity problem by proposing cross-cutting policy agendas that link the multiple policy streams to assure that one policy stream is not at cross purposed with the others when trying to solve the dilemma of expanding population obesity.

Obesity is a life course issue and policies that affect a life course are long-term and conflict with political realities which reinforce short time spans to show policy affects. The characteristics and health of an infant and influences from the environment during infancy, toddlerhood, and early childhood are associated with the development of adult obesity (Eriksson et al., 2003; J. Eriksson, T. Forsen, J. Tuomilehto, C. Osmond, & D. J. Barker, 2001), so policy changes need to be made early in the life course with the pay off of better health (i.e. not developing obesity) coming to fruition until years later. The reality of life course policy to combat multifaceted problems is currently entering the policy arena regarding
childhood nutrition programs during testimony for the 2009 reauthorization of the
WIC program even though it becomes difficult to politically justify that the
absence of disease, obesity, as a policy outcome, the recognition that some
problems will only be solved by life course policies is beginning. Chairwoman
McCarthy opened the hearing by stating, the purpose was “. . . to explore how we
can reduce obesity through the child nutrition reauthorization. . . We know that
change for adults is hard, but if we start to educate our kids early enough, we can
establish lifelong habits and the values of healthy living and wellness for the
future (Chairwoman McCarthy Statement Subcommittee Hearing On "Improving
Child Nutrition Programs to Reduce Childhood Obesity", 2009).” It is difficult to
draft policies where individuals are treated as products of social interaction and
shared decision making within families, personal networks and governments, so
policies have to effect the environments that create later life individual disease
but still be linked to the goals (both short and long term) of the society at large
(Institute of Medicine. Committee on the Health and Safety Needs of Older
Workers. Board on Behavioral Cognitive and Sensory Sciences, 2004). Thus,
life course policies require policy action across different organizations and policy
domains, however, most health policy responses are disease specific and other
policy streams (agricultural and financial) are not concerned with addressing
health policies. The life course perspective on health (Barker, 1995a) delivers a
conundrum to policy makers because policy time scales are not measured over a
life span, 50-70 years, but are measured in terms of election cycles, 4 -8 years
(Exworthy, 2008). Formulating long-term coalitions between the divergent policy
streams has become the critical component for successful cross-cutting policy formulation and implementation that will significantly impact obesity.

M. Patterson, First Lady of New York, testimony before the U.S. House Education and Labor Committee Subcommittee on Healthy Families and Communities in May 2009, stated “…We need federal policies, standards, regulations and commitments that help make the healthy option the easy choice. . . [such as] federal legislation that raises the nutrition standards of all food and beverages available in our schools. . . improve the nutritional quality of surplus government commodities offered to schools. . . To accomplish this, policy, systems and environmental changes . . . [need to be] pursued in collaboration with a wide range of organizations and community members across a variety of settings (Hearing to examine how improvements to child nutrition programs can help fight the nation's childhood obesity crisis. Statement of Michelle Patterson, First Lady of New York State., 2009).” The realization has occurred that cross cutting life course policies are necessary to reduce obesity in children and policy entrepreneurs who can span politics and healthcare, such as Chairwoman McCarthy, Senator Bingaham, and Dr. Dietz are beginning to emerge. Federal policies have affected both food prices and the composition of the food supply. Current food prices are on the rise and many policies are being proposed to reduce non-nutritive calories in the food.
Implications

Policies that cover a range of different types of options are necessary and diverse policy streams will need to be coupled to enact policies for real transformation to occur in the obesigenic food supply. The childhood obesity epidemic took three plus generations to develop into a critical problem, therefore it is going to take generations for any policies to effect real population level change. There is little evidence to justify the efficacy of policy changes to affect the obesity epidemic in children, thus it is critical that appropriate evaluations are performed to monitor the policy effects. Most policies to moderate childhood obesity trends are untested and would need consistent and continual monitoring, evaluation, review, and re-design. However, since obesity is a life course problem, there are no short term solutions. Hence the policy cycle timeline would need to be generational rather than administrational in length. Thus enters the dual problems of sustainability and feasibility for policies that need to remain in effect and be monitored for generations. America’s children did not get fat overnight, so sustainability of obesity policies throughout the life course of successive generations in the absence of short term results will become a politically difficult item to keep on the changing political administration’s policy agendas. To understand the development of the current obesigenic environment in the United States, multiple policies need to be reviewed: Agriculture Policy, Transportation Policy and Health Policy combined with increasing population affluence (Economic Policy) conjoined to create a national food environment that
is inherently obesigenic. Successful policies will have to involve the participation of a wide variety of sectors and stakeholders, establishing a broad constituency that supports the policies. Policies that place undue burdens on individuals making changes to their behaviors will not be sustainable. A policy approach that includes long term population wide strategies that can be broadly implemented is required. As policies change in one stream, outcomes in other policy streams are affected. Policy streams are linked in a push-pull web of outcomes when responding to a multimodal issue such as obesity. Additionally, political feasibility need to be considered because politics is irrevocably woven into the policy making process. The U.S. food environment has been shaped by economic and political forces and by agribusiness and food processing companies which control which commodities are produced, processed and sold; thus economics and politics direct food prices, amounts, availabilities, and choices; the politicization of food has created an obesigenic food supply in the USA (Tillotson, 2003a, 2003b). Also the influence of politics in both medicine and science which is partly due to the anticompetitive nature of discipline in which peer standards of conduct are the primary considerations by which scientific evidence is judged so individual values and judgments creep into the evaluation of health policies (Fowkes, 1996). But politics does creep into science and medicine because scientists and doctors are people, and people are social and political animals (Fowkes, 1996). Thus,

Policies can influence the development of childhood obesity and its precursors through legislation, regulation, fiscal policy, or through direct
recommendations targeted at children, parents, service organizations, and local communities. However, there is limited evidence on any direct relationship between policies and obesity in children (Wang & Lobstein, 2006).

Recommendations

Policy options that are currently being debated in the 111th U.S. Congress will be individually discussed and critiqued regarding each policy’s effectiveness in light of the findings from this study. Recommendations will be made for revision of the policy if appropriate.

The Soda Tax (federal excise tax on sweetened beverages)

A federal tax of one cent per 12-ounces on soda and other sweetened beverages is proposed to pay for the comprehensive overhaul of the nation’s healthcare system and would raise about $1.5 billion annually (M. Jacobson, 2009). The tax per beverages item is too small to significantly reduce consumption, but collectively, the tax would generate over a billion dollars annually for healthcare expansion but the revenue generated from the tax is only a fraction of the cost needed to expand health-insurance coverage to all Americans. Also in 2009, a bill was introduced in New York State to tax soda and sweetened beverages ("A Budget Bill; state fiscal plan for the 2009-2010 state fiscal year," 2009) but after a few months of public outcry the Governor
decided not to pursue the soda tax. However, the Health Commissioner of New York City re-proposed the soda tax as an obesity tax that would reduce obesity in the city (Brownell & Frieden, 2009). As Stone (2002) found, the act of labeling a policy has a profound impact on how the policy is perceived and the level of political support generated (Stone, 2002), thus an obesity tax may have a better reception than a soda tax. Currently, 17 States and 2 cities have sweetened beverage taxes but the taxes are just a part of general food sales taxes rather than a direct tax on sweetened beverages, Maine however does directly tax snack foods. Historically, there have been 9 State and 3 regional soda taxes repealed during the 20th century because of intense lobbying pressure from the food industry (M. F. Jacobson & Brownell, 2000). For example in 1993, the Ohio General Assembly passed a $.0008 tax per ounce of soda and by 1994 the soft drink industry was able to get a constitutional amendment to repeal the soft drink tax on the ballot and after spending over $7 million in advertising managed to have the tax repealed. However, a soda tax is actually a revenue generating tax, and it will do what it is designed to do, generate revenue. But as far as controlling obesity, it falls far short of accomplishing that objective. An excise tax on soft drinks would not prohibit people from buying sweetened beverages unless the price hike was so dramatically that the beverages became prohibitively expensive. This type of tax would face strong opposition from the beverage industry and could also create a backlash from consumers who would have to pay significantly more for a soft drink. The sweetened beverage tax itself would raise money for healthcare reform but because the price hike was too low
to be an effective strategy to combat obesity. A better policy recommendation would be to disassemble the commodity supports for the portion of the corn crop that is used in HFCS production. This policy will change the manufacturing price point of products using non-nutritive sweeteners and could begin to erode the competitive advantage of HFCS as a raw material compared to natural sweeteners.

The Sin Tax (federal excise tax on non nutritive foods)

Selective federal taxation on food manufacturers to change production and food processing procedures is proposed. It would radically raise taxes on alcohol, largely eliminate artificial trans-fat (hydrogenated vegetable oil) from all food, and reduce sodium content in packaged and restaurant food. This type of tax is touted as a way to pressure food companies to make healthier products. It is proposed that the tax would lower consumption, reduce health problems and save medical costs, thereby lowering obesity. Excise taxes on food are normally regressive, thus low income consumers would bear the disproportionate burden of the tax because they spend more of their income on food than do middle and upper income consumers. For consumers who are not overweight and enjoy snack foods, there are only costs associated with the tax which could lead to a political backlash against the tax. The combination of beverage and snack food industry’s opposition and consumers who do not want special taxes on their snack foods have caused 12 jurisdictions (cities, counties, states) to either reduced or repealed their snack taxes (M. F. Jacobson & Brownell, 2000). In
1992 in Maryland a snack tax was enacted, by 1997, it was repealed by the State legislature because Frito-Lay basically threatened not to build a manufacturing and distribution center in the State if the snack tax was not repealed (M. F. Jacobson & Brownell, 2000). Again for this tax to have a dramatic effect on diets, the price rises would have to be so prohibitively large that consumers would be forced to respond (i.e. stop buying) to the change in retail price. Experiments using vending machines at a university, high school and work place manipulated prices to reduced the prices of all products containing less than three grams of fat found that the percentage of low fat snacks purchased increased significantly but the purchase of high fat snacks was only modestly reduced (French et al., 2001; French, Jeffery, Story, Hannan, & Snyder, 1997). The increase in low fat snack food purchases was tied to price, as the price dropped by more than 25%, the increases in purchasing of low fat alternatives were higher; a 25% reduction in price caused a 39% consumption increase and a 50% reduction in price caused a 93% consumption increase (French et al., 2001; French et al., 1997). Jacobson and Brownell (2000) found that a small tax on soda, candy, gum, and snack foods was supported by 45% of Americans as long as the revenue was used to support health programs, thus would be politically feasible (M. F. Jacobson & Brownell, 2000). But a small tax (< 5%) is estimated to result in only a 2% decline in sales and would probably be mitigated by price reductions by producers, so would not really change consumption patterns with a resulting effect on obesity rates (M. F. Jacobson & Brownell, 2000). Food costs to households continued to drop through the end of the century and had declined
to 12.1% by 2000. Thusly, increases in food costs must be substantial to affect consumption because of how small the cost of food is relative to the other costs of living. Food retail prices, fats, oils, cereal, and bakery products, increased 6.4%, while restaurant food prices rose 4.4% in 2008 (Clauson, 2008). Even though food prices rose by approximately 5.5% overall between 2007-08, consumption patterns have not slowed or changed, but shopping patterns have changed with consumers switching to lower cost discount retail stores rather than traditional grocery stores to purchase the bulk of their groceries and sales at these stores has risen by over 20% (Clauson, 2008) showing that prices have to change dramatically before consumption can be effected. Looking at French et al's experimental data the price of food would have to rise ~ 50% to change consumer behavior (French et al., 2001; French et al., 1997). Since approximately 10% of household expenditures are currently spent on food as compared to 25% in the early part of the century, that means food would need to increase to approximately 30% of household income expenditures before you would see a definite shift in consumption patterns. Thus, it is unlikely that obesity will be greatly influenced by this type of tax. Better policy alternatives would be: 1) to change federal food subsidy programs to disallow purchase of any type of ‘snack foods’ with program funds, and 2) to change the commodity price support system to subsidize fruit and vegetable production so their retail prices are far below actual costs (i.e. so fruit and vegetables are the cheapest available food).
Redesign the National School Lunch Program

The main reason the School Lunch Program legislation passed was because of the support from Southern Democrats who represented the agricultural lobby who were convinced that commodity over production surpluses (that did not exist during WWII) would reoccur so it was necessary to develop a program to ‘dispose’ of excess commodities that would not affect food prices in the retail market (Levine, 2008). Very little was done in the 1950s to augment the nutrition programs that were put in place in the 1940s, however, the Special Milk Program 1954 (United States) P. L. 86-478 was implemented to encourage fluid milk consumption by selling milk to students at the lowest possible price and serving milk free to students determined to be eligible ("The Special Milk Program 1954 ", 1954). There was an ulterior motive to this program however, and that was the reduction of perishable commodity stocks due to over production (Levine, 2008). Since the inception of the child feeding programs school lunchrooms became a major outlet for surplus meat, milk, eggs, cheese and citrus products (Levine, 2008). Politics dictated what commodities were ‘in surplus’ and food industry groups strongly lobbied their Congressmen for the ‘in surplus’ designation when market prices were low (Levine, 2008). Buying commodities and then giving them to schools acted as an insurance plan to assure that food prices did not fall after the war; the main advocates of the program were The Dairymen’s League, The Grange, The American Farm Bureau Federation and The Independent Grocers Association (Levine, 2008). Social welfare proponents lobbied that the School Lunch program should be part of a
broad social safety net welfare agenda and that it did not make sense for the
permanent program to administered by the USDA rather it should be located in a
federal department that understood the health needs of children (Levine, 2008).
They lost the fight because the program was never designed as a health program
per se, it was designed to stabilize crop prices by eliminating surpluses (Levine,
2008). The legislation was popular for both republicans and democrats because
it could be touted as either a subsidy for southern (white) farmers or a way to
help poor children both black and white, it was also popularly touted as a way to
expand demand for American commodities by introducing children (through the
school lunch rooms) to new and different foods (United States. Congress. House.
Committee on Education and Labor. Subcommittee on General Education.,
1960). In a 2008 review of school children’s diets, the IOM found that for all
children 5–18 years old, the mean intakes of total vegetables (45%), fruit (80%),
whole grains (24%), total meat and beans (70%), and milk (80%) were less than
the DRI recommendations (Institute of Medicine. Food and Nutrition Board,
2008). Additionally, the children consumed larger than recommended amounts
of solid fats and added sugars, over 400 calories per day of fat and between 19
to 29 teaspoons of added sugars each day (Institute of Medicine. Food and
Nutrition Board, 2008). In school year 2008, the USDA purchased 1.1 billion lbs
of food for the child nutrition programs, National School Lunch Program, the
School Breakfast Program, the Summer Food Service Program, and the Child
and Adult Care Food Program ("Food Availability: Spreadsheets," 2009),
therefore the USDA purchasing should be redesigned to only offer healthy non-
processed foods that meet the DRI guidelines. Some schools have found
themselves in a Catch 22 situation when trying to provide healthy meals.
Schools that attempt to bring high-nutrient food to children have run afoul of the
USDA’s School Meal Guidelines for failing to deliver enough calories per meal
(Dahdah, 2004; Gleason & Suitor, 2001). The wheat, corn, soy, and meats in
school meals are subsidized by the government, then the raw products are
processed and sold back to the government as part of a nationwide nutrition
program. There is a distinct feedback loop occurring in the acquisition and
processing of food that is very profitable for food producers making School
Nutrition Programs very lucrative business for the producers who also have very
powerful lobbies. The USDA offers over 180 products for schools to select from,
however, many of the products still contain trans fats, many of the canned
vegetables still do not meet the Food and Drug Administration’s “healthy"
standard for sodium, many fruits are still offered in sweetened syrups, albeit not
heavy syrup, and many foods are processed rather than fresh (Daft, Westfall,
Abel Daft & Early., 1992; Dahdah, 2004; United States. Food and Nutrition
Service., 1991). In addition many States and School Districts further process the
foods obtained from the USDA programs (i.e. whole chicken into breaded
chicken patties) for ease of onsite preparation at the schools (Daft et al., 1992;
Dahdah, 2004; United States. Food and Nutrition Service., 1991). In addition,
currently USDA oversight extends only to the financial and administrative details
related to processing, however regulations to tighten processing standards are critical to assure the nutrient value of the food is not compromised. This expansion of and nutritional content tightening of the USDA School food programs would be costly and would also require additional local contributions for increased onsite food preparation by already cash strapped School Districts. Out of the $1.173 billion budget for School Nutrition Programs, the 2008 Farm Bill required that $50 million be used annually to purchase fresh fruits and vegetables for distribution to schools meaning less than 5% of the budget is allocated for fresh fruits and vegetables ("Food, Conservation, and Energy Act of 2008; Agricultural Security Improvement Act of 2008," 2008). The required policy change in schools would be to eliminate the sale of sweetened beverages, candy bars, any food with hydrogenated vegetable fat, and foods high in calories, fat, or sugar in all school buildings and to require that any foods that compete with school meals be consistent with federal DRI guidelines for fat, saturated fat, cholesterol, sugar, and sodium content. Schools did begin to restrict soda during the 1990s, but the purchases of sweetened fruit drinks increased by 180% negating virtually any effect that the restriction of soda might have caused (Nestle, 2007). A policy change of this magnitude will be difficult to enact since the 2008 Farm Bill will not be renewed until 2018 and to obtain the necessary political consensus will be difficult. Administratively, the USDA could change program regulations, however, the USDA’s historical track record (i.e. Redesign of The Food Pyramid) on bowing to political pressure even in the face of established scientific data does not suggest that they will adopt the sweeping
changes that would be required. However, the USDA has developed pilot
nutrition programs with educational materials and onsite experts to help schools
develop school lunch programs, but after a few years of operation, the USDA
Secretary Freedman stated to Congress “If Congress wanted his Department to
adequately feed the nation’s poor children, it would have to vote new
appropriations and provide new authority for the use of surplus food and federal
resources” (Levine, 2008). Thus, the aforementioned policy changes are unlikely
to be adopted, however if they were obesity would be influenced because >40%
of US schoolchildren are eligible for the program with even more children paying
for and eating their lunches at school. A better recommendation would be to set
mandatory food standards for the types of food available at schools in both the
lunch room and in the vending machines and if a school chooses to not follow the
mandatory guidelines then they should lose their federal funding.

Include Mandatory Nutrition and Health Education in the Primary and Secondary
School Curriculum

Nutrition and health education in the schools is necessary and should be
improved; it has sometimes been neglected in favor of more ‘academic’ subjects.
It is not enough just to provide nutrition and health education in schools but also
sufficient physical education and physical activity throughout the school day are
necessary (Chairwoman McCarthy Statement Subcommittee Hearing On
"Improving Child Nutrition Programs to Reduce Childhood Obesity", 2009;
Current Status and Activities to Decrease the Prevalence of Obesity Among U.S.

242
Children and Adolescents. Statement of William H. Dietz, MD, PhD, Director Division of Nutrition, Physical Activity, and Obesity National Center for Chronic Disease Prevention and Health Promotion Centers for Disease Control and Prevention, 2009; Hearing to examine how improvements to child nutrition programs can help fight the nation's childhood obesity crisis. Statement of Michelle Patterson, First Lady of New York State., 2009). Daily physical education and sports programs would need to be expanded in primary and secondary schools even if they extended the school day. Nutrition and health education are more than just subjects on the curriculum of a school because it involves more than just the children; both the teachers and parents have to be included and involved for the curricular changes to work (Burdette & Whitaker, 2004). There will have to be provisions to require instruction in nutrition and weight management for all health-education teachers. Finally, it is at home that the child receives the bulk of their food and it is not known if nutrition education of the children can influence food buying and food preparation practices of the parents. Interestingly, as the health consciousness of a person increased so did poultry consumption, including fast food poultry, (Moon & Ward, 1999); concern for health meant diet changes, the substitution of poultry for other meats, but did not mean dietary restraint, or caloric restriction.

To ensure that the policies are sustainable, nutrition education must be provided on a continuous basis to teachers, children and parents creating a burden on the local School Districts. Therefore, these policies which are federally mandated would need to be funded by the federal government; a
sustainable funding stream will need to be secured before mandatory implementation of these policies occurs. It is not known what the effect would be on obesity, however, it is likely that they could change children's and parent's attitudes toward nutrition; thus these policies would need to be regularly evaluated.

Nutrition Labeling Guidelines

Extend the Nutrition Labeling and Education Act which gives the FDA authority to require nutrition labeling of most foods regulated by the FDA and requires that all nutrient content claims (i.e. 'high fiber', 'low fat', etc.) and health claims meet FDA regulations ("Nutrition Labeling and Education Act ", 1990) to restaurants because the original bill did not require restaurants to comply to the standards. Currently, restaurants voluntarily provide much of the information; however, not every restaurant participates or provides nutrition information in writing onsite in the restaurant. Harnack, et al. (2008) in a randomized control trial showed that calorie and dietary intake information printed on a fast food menu did not significantly change consumption patterns and postulated that long term education was necessary rather than just exposure to caloric information on a menu (Harnack et al., 2008). The policy would requires that any food service establishment selling food as part of a chain that operates 20 or more establishments must provide consumers access to detailed nutrition information about the food they serve in writing in the restaurant. The policy would increase the availability of nutrition information but as a strategy to combat obesity would
probably not work unless it was combined with educational programs to increase nutritional awareness, thus this policy needs to be coordinated with public health departments for mass media nutrition educations programs.

**Controls on the Advertising and Promotion of Food and Beverages**

The IOM recommended in 2005 that policies be implemented to limit food marketing to children because of research that indicates a possible link between TV advertisements and food preferences (McGinnis et al., 2005). Awareness of targeted advertising to children has grown in the past few years and policies have been implemented in March 2009 to change the current voluntary system. The 2009 omnibus appropriations bill included a provision to evaluate standards on marketing of foods to children under 17 years of age ("Financial Services and General Government Appropriations Act, 2009," 2009). The Federal Trade Commission will establish an Interagency Working Group (Directors of the FDA, and CDC and the Secretary of Agriculture, and other child nutrition experts) to recommend standards for the marketing of food targeted to children and report to Congress by July 2010. This is a step in the right direction to reduce obesity by multiple modalities; however, it is the beginning of a strategy to reduce obesity and is likely to have limited effect.

**Redesign of the WIC Program Food Packages**

2009. After the 2005 IOM report calling for sweeping changes in WIC food packagers (Institute of Medicine. Committee to Review the WIC Food Packages, 2005), in December 2007, the USDA revised the food packages to encourage changes in participant behavior and outcomes, and while maintaining program cost neutrality ("Department of Health Services - WIC Branch Homepage," 2007; Special Supplemental Nutrition Program for Women, Infants and Children (WIC): miscellaneous vendor-related provisions. Final rule, 2008; Taylor, 2006).

Because WIC program obesity rates are well over the national average rates of obesity for children in the U.S. population; the federally subsidized food program has a noteworthy effect on obesity in its enrollees (Health, United States, 2006 with Chartbook on Trends in the Health of Americans, 2006). Using the WIC Program as an example of federal food subsidy programs shows that obesity rates in WIC enrolled children are consistently higher than the general population thus there must be something in the WIC food policies that create an obesigenic environment (Bartlett et al., 2006). The obesity rate in WIC served children by the time they are 2 years old is higher (15.4% are overweight and 22.3% are obese) than the general population rate of 13.9% for both overweight and obesity (Bartlett et al., 2006; Health, United States, 2006 with Chartbook on Trends in the Health of Americans, 2006). Obesity rates are even worse for minority children participating in WIC, in 2004, at 1 year of age obesity rates range from: 42.7% overweight, 34.9% obese in Native Americans; 37.3% overweight, 29.6% obese in Hispanics; 32.1% overweight, 25.2% obese in African Americans; to 31.3% overweight, 24.0% obese in Whites (Survey of the Public Health Nutrition
Workforce 1999-2000., 2003). White children enrolled in WIC were 48% more likely to become overweight at age 5 than non WIC enrolled white children (Rose et al., 2006). The substantive changes in the WIC program will have an effect on U.S. pediatric obesity rates because over half the infants in the United States live in households eligible for WIC (Bogen et al., 2004) but the magnitude of the effect is still unknown. The revisions will take effect in October, 2009 and are the most significant changes to the WIC program since its founding; the revisions include reductions in amounts of saturated fats, cholesterol, total fats, sodium, and sugar, and increases in amounts of whole grains. Demonstration projects in the WIC programs have shown significant improvements in childhood nutrition (Rose, Habicht, & Devaney, 1998; Siega-Riz et al., 2004) which should hopefully translate to a healthy diet and healthy weights. However, the impact of the revised food packages on childhood obesity is unknown at this time but it is likely that there will be a significant effect because of the caloric reduction in the food packages.

Dismantling of Commodity Price Programs and Import Tariffs

U.S. farming affects not only the U.S. food supply but with price supports can distort the world trade balance; affect the health of the population because price supports artificially encourage low prices for non-nutritious food products (such as those based on HFCS, hydrogenated vegetable oils and high fat meat and milk products); and affects the environment because production methods have direct impacts on air, soil and water quality (Food and Agricultural Policy
Taking Stock for the New Century, 2000; USDA, 2007). Additionally, the United Nations has accused the U.S. of artificially manipulating global commodity prices through the dumping of subsidized crops at low prices rather than letting market forces establish commodity prices. Farm subsidies transfer income from consumers and taxpayers to food producers, impose deadweight losses on society, and impede international commodity trade (Alston & James, 2002). Farm subsidy programs have not been found to stabilize agricultural market volatility nor do they ensure food security, but they do provide needed income stabilization to farmers who are marginalized (Alston & James, 2002). Farm commodity programs were designed to combat disastrously low food prices and to support farmers who were in financial trouble so that they could continue to farm (Cochrane & Runge, 1992); they were not designed as long term food price controls. President Roosevelt extended the Agricultural Adjustment Act of 1933 to make it a “. . . long-time and more permanent adjustment program [that] will provide positive incentives . . . that will encourage individual farmers to adopt sound farm management, crop rotation and soil conservation methods. . . [and] The crop insurance feature . . . will help farmers to maintain these beneficial systems of farming without interruption in poor crop years” (Woolley & Peters, 2009). However, there are large swings from year to year that affect the commodity price supports; the swings are caused not only by crop failures but the changing Farm policy legislation. Crop commodity program funding has become more volatile as Farm Bill legislation has been adopted to discontinue
the program (1970s), reign in the costs (late 1980s) or redesign the program (1990s-present).

Commodity programs focused on farming and farm production, the changes in the composition of the food supply was an unintended consequence. Because commodity programs have become an ingrained as part of the nation’s food production apparatus, it is going to be politically difficult to change the commodity programs or design other types of major policy shifts. However, most of the crops that are subsidized are major export crops and also are crops that have created niche products such as HFCS and hydrogenated vegetable oils. Subsidies for one commodity may harm other crops that would provide a more healthy diet; accordingly the commodity subsidy programs should be changed to subsidize the costs of nutritious foods that promote a healthy diet for all Americans. “Obesity is not a symptom of eating well but an indicator of poor diet (Current Status and Activities to Decrease the Prevalence of Obesity Among U.S. Children and Adolescents. Statement of William H. Dietz, MD, PhD, Director Division of Nutrition, Physical Activity, and Obesity National Center for Chronic Disease Prevention and Health Promotion Centers for Disease Control and Prevention, 2009).”

The federal food assistance programs began during the 1930s and 40s; Congress passed legislation creating programs that were supposed to help meet the nutritional needs of poor people but there was a political ulterior motive to these nutrition programs. The main purpose of the federal food assistance programs were to diminish the excess supply of farm products so that commodity
prices would remain high (Luna, 2004). However, federal food assistance programs need to be designed to respond to the nutritional needs of the poor. Thus, fresh food should be significantly subsidized which would make it an economically viable alternative to the cheaper processed food for the poor. Subsidy programs that lower the costs of fruits and vegetables would be more consistent with the USDA DRI nutrition goals set by the government but likely would meet significant resistance from farmers and the food industry. It is going to be very difficult to change U.S. agriculture policies and commodity programs because of the legacy of extreme politicization of the policy process and massive effect the resultant policies have on all sectors of the economy. Changes in commodity subsidy programs would likely have the largest effect on creating a healthy food environment in the United States and would likely have a large effect on reducing obesity but the political feasibility is still an issue that needs to be surmounted.

*Obesity Prevention, Treatment and Research Act of 2009*

The Act would develop a United States Council on Overweight & Obesity Prevention which would “coordinate interagency cooperation and action related to the prevention, treatment, and reduction of overweight and obesity in the United States” and create a national strategy to combat childhood and adult obesity ("Obesity Prevention, Treatment and Research Act of 2009," 2009). It would administer a grant program for coordination of both translation and research efforts and provide a forum for each Cabinet Department Secretary,
Department of Agriculture, Department of Education, Department of Housing and Urban Development, the Department of the Interior, Federal Trade Commission, Department of Transportation, and other Federal agencies Heads, such as the Federal Communications Commission, the Centers for Disease Control and Prevention, the National Institutes of Health, the Agency for Healthcare Research and Quality, the Centers for Medicare and Medicaid Services, and the Food and Drugs Agency, to coordinate policy, regulation, and implementation thereby providing a multi-faceted approach to solving the obesity epidemic ("Obesity Prevention, Treatment and Research Act of 2009," 2009). It does expand the treatment paradigms for obesity treatment, nutrition counseling, and expands federal health programs to pay for obesity counseling and treatment. The Act also updates and reforms Federal oversight of food and beverage labeling. This Act would definitely coordinate efforts on obesity programs and would merge many of the policy streams that effect population obesity and health risk but does not address the commodity programs that affect the food supply. Political feasibility of the Act is questionable since this is a re-introduction of an Act that died before it was assigned to a Committee in the previous Congressional Session. Thus, if political support could be generated for enactment, it would likely have a large effect on coordinating obesity treatment and a possibility of effecting obesity rates.
Limitations

Limitations of the current study need to be considered. First, the descriptive nature of the study does not provide causal data or relational data. Thus, it is difficult to ascertain how or if there are relationships between the food supply and children’s body size. There appears to be trends that indicate a possible relationship but validation of the existence of or significance of the relationships cannot be determined from this study. Second, the study depends on 100 years of secondary data that was not collected for the study purposes. The use of secondary data limited the research because proxy measures of specific variables had to be used and in many cases was not found for the entire time span. The data was collected for other purposes and in some cases there was little or no documentation about the data collection process, and the data varied from individual to aggregate data depending upon the year of data collection. It is always difficult to obtain data across such a prolonged time span but even with the incomplete data, trends were able to be described across the century. However, discrete comparison between the relationships of the variables was not possible with the data in this study.

Third, trends in body weight were described along with trends in the food supply that were caused by federal agricultural policy. Body weight equals energy input (i.e. calories consumed) minus energy expended (i.e. physical activity). Consequently, the major limitation of this study is that the energy expenditure side of the equation was not measured. However, energy intake,
from appropriate diet and not energy expenditure, from physical activity was the primary determinant of obesity status in one to two year old children (Stunkard et al., 2004). Two to three year old children of mothers who skipped breakfast and did not eat fruits and vegetables had the highest incidence of poor nutritional status (Lee et al., 2005). Thirty year trends (1970 to 2000) in children’s food choices correspond with the national food supply and are influenced by taste, television, and cultural norms (R. E. Allen & Myers, 2006). Home environments where energy-dense foods are predominant may create an obesigenic feedback loop predisposing the child for future obesity (Birch & Fisher, 1998). United States economic and agriculture policies influence the home food environment by constraining the availability of affordable food choices. Many affordable foods contain significant amounts of non-nutritive calories at artificially lowered prices because of government intervention in either agricultural commodity production or supply.

The inability to measure any type of surrogate for energy expenditure was the main limitation in the data, because there was no good proxy measure that was consistent over the century for children’s physical activity. Television viewing was considered as a proxy measure for physical activity but, the rising trends in obesity began in the 1950s 20 to 30 years before television was identified as a factor that reduced children’s physical activity levels (Coon & Tucker, 2002; Mendoza et al., 2007). Of U.S. children aged 4 to 11 years, 37.3% had low levels of active play, 65.0% had high screen time use (television and computer), and 26.3% had both these behaviors (Anderson et al., 2008).
Characteristics associated with a higher probability of simultaneously having low active play and high screen time were older age, female gender, non-Hispanic black race/ethnicity, and having a BMI-for-age $\geq 95^{th}$ percentile of the CDC growth reference (Anderson et al., 2008). Many young children in the U.S. are reported to have physical activity and screen time behaviors that are inconsistent with recommendations for healthy pediatric development. Children who are overweight, approaching adolescence, girls, and non-Hispanic blacks may benefit most from public health policies and programs aimed at behaviors changes to increase physical activity (Anderson et al., 2008). However new data [released May 2009] indicates that physical activity may not be as important as previously thought as causing the appreciable weight gain in American children during the previous 40 years. Swinburn (2009) found that increases in children’s energy intake, alone, from 1970 to 2000 were able to explain the entire weight increase for that time period (Swinburn, 2009). This is contrary to longstanding views that a decrease in physical activity because of higher TV viewing and video game playing coupled with an increase in consumption contributed to the increase in weight of American children from 1970 to 2000 (Andersen et al., 1998; Coon & Tucker, 2002; Dietz & Gortmaker, 1985; Expert Committee Recommendations on the Assessment, Prevention, and Treatment of Child and Adolescent Overweight and Obesity 2007; Mendoza et al., 2007). Swinburn (2009) states "To return to the average weights of the 1970s, we would need to reverse the increased food intake of about 350 calories a day for children . . . Alternatively, we could achieve similar results by increasing physical activity by
about 150 minutes a day of extra walking for children . . . but realistically, although a combination of both is needed, the focus would have to be on reducing calorie intake (Swinburn, 2009).” The latest findings from the Swinburn (2009) study lower expectations regarding what types of weight reduction can be achieved with exercise so public health policy will need to shift its focus toward eating less rather than physical activity. But Brownson, Boehmer, & Luke, 2005 found that physical activity was on the decline in the U.S. during the previous 50 years. However, the following trends were observed: there was relatively stable or slightly increased levels of leisure-time physical activity, declining work-related activity, declining transportation activity, declining activity in the home, and increased sedentary activity resulting in an overall trend of declining total physical activity (Brownson, Boehmer, & Luke, 2005). Large decline were found in the rates of walking as a means of transportation across urban areas over the past half century, and that trend was coupled with a strong and consistent trend toward Americans living in suburbs (Brownson et al., 2005). It is apparent that a combination of changes to the built environment and increases in the proportion of the population engaging in sedentary activities places the majority of the American population at high risk of physical inactivity (Brownson et al., 2005), thus changes to both the built environment through the HUD and Transportation policy streams could have an effect on increasing physical activity and changing obesity trends.
Future Research

Limited evidence supports the short-term efficacy of medications and lifestyle interventions, but the long-term efficacy and safety of pediatric obesity treatments remain unclear, thus policies should be carefully developed to assure they are based on sound evidence (McGovern et al., 2008). The next series of studies would create a dynamic model by cataloging the food supply at various points in the century, describing the policies that created the food supply at that discrete point and then relating the children’s BMI trends at that discrete point forward with that type of food supply held constant. Thus, a model could be built that describes the food supply and then predicts children’s BMI giving policy makers a way to posit legislation changes and possible effects on both the food supply and children’s body size. Building the postulated model will require access to further historical body size data so that the data can be consistent throughout the century, rather than as it was in this study which used both individual and aggregate historical weight data. Additionally, a mathematical process to anticipate policy effects magnitude and lag time will also need to be developed. Future research that models the effects of changing agricultural commodity availability through various policies and the effects on pediatric obesity rates will provide opportunities for forecasting the long term policy consequences before a new policy is adopted. A dynamic modeling approach should be able to determine the combinations of factors that created food environments that were pivot points for children’s BMI trends.
Conclusion

A model of the agricultural production and policy streams should be able to provide a glimpse into each policy cycle’s respective pediatric obesity rate. This study has set the groundwork for modeling by describing 100 years of agricultural policies in conjunction with the food environment and children’s body sizes. It was important to understand the changing food environment and accurately describe children’s body sizes before policy effects can be evaluated.

From the New Deal to the present day, an underlying political bias in agricultural policy making favors agricultural interests and accordingly tethers the needs of the hungry to the agri-business political agenda (Duniform & Kowaleski-Jones, 2002, 2004; Gunderson, 1971; Luna, 2004). Agriculture policy effects agriculture production, food prices and the availability of food. Thus, agriculture policy needs to incorporate data and evidence from the health policy stream so effect obesity policies can be developed. However, data that links the actual availability of food and eating behavior in the population is lacking. Methods for accurately tracking individual and family food consumption are problematic and need to be re-evaluated so a better link between consumption patterns and body size can be determined. This study proved a better understanding of the U.S. food producers and marketplace when various policy options were adopted, however further study needs to be undertaken so policies can be informed by more complete data that would allow discovery of relations between the food environment and children’s body size. This study described the broad effects
that diverse policy streams (agricultural, health) has on U.S. farm production. It shows the need for policies to be developed collaboratively across multiple policy streams rather than politically so the policies: reflect the changing nature of agricultural production, provide a food supply that provides affordable appropriate nutrition to the entire population and creates a health food environments that fosters appropriate body weights. However, population obesity is multi-factorial so a multi-faceted policy stream is necessary to develop effective solutions. The policy stream cannot be limited just to agriculture and health policy but needs to merge across other streams including the Financial, Transportation, Housing and Urban Development, and Education policy streams for synthesis of policy to be effective.
BIBLIOGRAPHY


An Act to Amend the National school lunch act and the Child nutrition act of 1966 in order to revise and extend the summer food program, to revise the special milk program, U.S. Congress, 95 Sess.(1977).

An Act to Amend the National School Lunch Act and the Child Nutrition Act of 1966 to Better Assist Children in Homeless Shelters, to Enhance Competition among Infant Formula Manufacturers and to Reduce the Per Unit Costs of Infant Formula for the Special Supplemental Food Program for Women, Infants, and Children (WIC), and for Other Purposes, U.S. Congress, 102 Sess.(1992).


An Act to Extend and Amend the special supplemental food program and the child care food program, and for other purposes, U.S. Congress, 95 Sess.(1978).


ADA supports USDA School Meals Initiative for Healthy Children but recommends more improvements for child nutrition. (1994). J Am Diet Assoc, 94(8), 841-842.


A Budget Bill; state fiscal plan for the 2009-2010 state fiscal year, New York Assembly A00160 and S00060 (2009).


Center for Disease Control. (2008). *Epidemiology and Prevention of Vaccine Preventable Diseases* (10th ed.). Atlanta, GA.


Commodity Credit Corporation Charter Act, U.S. Congress, 80 Sess.(1948).

Cone, T. (2009). Dairy cows head for slaughter as milk prices sour. The Buffalo News,


Food Security Act of 1985. A Bill to extend and revise agricultural price support and related programs, to provide for agricultural export, resource conservation, farm credit, and agricultural research and related programs, to continue food assistance to low-income persons, to ensure consumers an abundance of food and fiber at reasonable prices, and for other purposes. , United States Congress, 99 Sess.(1985).


Soil Conservation and Domestic Allotment Act, U.S. Congress, 74 Sess., 590(e); 590(e)(1); 590(g); 590(h); 590(p)(1); and 590(q)(3) Cong. Rec.(1936).


Steagall Amendment of 1941 (Steagall Commodity Credit Act), U.S. Congress, 77 Sess.(1941).


United States. Department of Commerce. Bureau of Economic Analysis. Table 2.3.3. Real Personal Consumption Expenditures by Major Type of Product, Quantity Indexes [Data File] (Publication. (2008). Retrieved October, 10, 2008, from http://www.bea.gov/national/hipaweb/TableView.asp?SelectedTable=63&ViewSeries=NO&Java=no&Request3Place=N&3Place=N&FromView=YES&Freq=Year&FirstYear=1929&LastYear=2000&3Place=N&Update=Update&JavaBox=no


APPENDICES
APPENDIX A

LISTING OF HISTORICAL EVENTS AND POLICIES PERTINENT TO
POPULATION HEALTH AND THE US FOOD SUPPLY FROM 1900 TO 2000

<table>
<thead>
<tr>
<th>Year</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1906</td>
<td>Federal Food and Drug Act of 1906: Established federal control and</td>
</tr>
<tr>
<td></td>
<td>regulation of food; additionally coordinated federal and state efforts</td>
</tr>
<tr>
<td></td>
<td>for food safety and sanitation.</td>
</tr>
<tr>
<td>1918-19</td>
<td>Influenza Pandemic: More than 550,000 Americans died; primarily</td>
</tr>
<tr>
<td></td>
<td>affected the 20-40 years of age group thusly having an impact on</td>
</tr>
<tr>
<td></td>
<td>birth rates (Gernhart, 1999).</td>
</tr>
<tr>
<td>1921</td>
<td>Sheppard-Towner Act 1921: Established federal grants to state</td>
</tr>
<tr>
<td></td>
<td>health departments for maternal infant hygiene programs to reduce</td>
</tr>
<tr>
<td></td>
<td>maternal infant mortality.</td>
</tr>
<tr>
<td>1928-38</td>
<td>Great Depression</td>
</tr>
<tr>
<td>1933</td>
<td>Federal Surplus Relief Corporation: Established to divert</td>
</tr>
<tr>
<td></td>
<td>agricultural farm surpluses from the open market and to distribute</td>
</tr>
<tr>
<td></td>
<td>the commodities to destitute families (&quot;Archival Research Catalog.</td>
</tr>
<tr>
<td>Year</td>
<td>Event</td>
</tr>
<tr>
<td>------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>1935</td>
<td>Social Security Act of 1935</td>
</tr>
<tr>
<td>1938</td>
<td>Agricultural Adjustment Act</td>
</tr>
<tr>
<td>1941</td>
<td>Mass production and distribution of first antibiotic, Penicillin</td>
</tr>
<tr>
<td>1941</td>
<td>Steagall Commodity Credit Act</td>
</tr>
<tr>
<td>1941-45</td>
<td>World War II</td>
</tr>
<tr>
<td>1946</td>
<td>National School Lunch Act 1946</td>
</tr>
<tr>
<td>Year</td>
<td>Event Description</td>
</tr>
<tr>
<td>------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>1955</td>
<td>Polio vaccination program begins (Stern &amp; Markel, 2005).</td>
</tr>
<tr>
<td>1966</td>
<td>Child Nutrition Act 1966: Extended, expanded, and strengthened the program to meet more effectively the nutritional needs of children (&quot;Child Nutrition Act &quot;, 1966a).</td>
</tr>
<tr>
<td>1969-79</td>
<td>Widespread Smallpox vaccination and smallpox eradicated (Stern &amp; Markel, 2005).</td>
</tr>
<tr>
<td>1985</td>
<td>Food Security Act of 1985: Revise agricultural price support and related programs, to provide for agricultural export, resource conservation, farm credit, and agricultural research and related programs, to continue food assistance to low-income persons, to ensure consumers an abundance of food and fiber at reasonable prices, and for other purposes</td>
</tr>
</tbody>
</table>
1988 Hunger Prevention Act of 1988: increased the maximum food stamp benefit, allowed private nonprofit organizations to take over school breakfast and lunch programs during the summer vacation, extended by two years the Temporary Emergency Food Assistance Program, by which surplus cheese, honey are distributed to the poor and to soup kitchens and food banks. Since those surpluses are now exhausted, the bill would require the Department of Agriculture to buy needed commodities to continue the distribution.

1989 School Breakfast Program and Reauthorization
APPENDIX B

MAJOR HISTORICAL POLICIES AND EVENTS