

Georgia State University

ScholarWorks @ Georgia State University

Public Health Theses

School of Public Health

8-9-2016

**Adverse Childhood Experiences and the Association with
Childhood Obesity: A Cross-Sectional Study of the U.S. National
Survey of Children's Health (NSCH), 2011-2012.**

Omarwalid Noorzada

Follow this and additional works at: https://scholarworks.gsu.edu/iph_theses

Recommended Citation

Noorzada, Omarwalid, "Adverse Childhood Experiences and the Association with Childhood Obesity: A Cross-Sectional Study of the U.S. National Survey of Children's Health (NSCH), 2011-2012.." Thesis, Georgia State University, 2016.

doi: <https://doi.org/10.57709/8805831>

This Thesis is brought to you for free and open access by the School of Public Health at ScholarWorks @ Georgia State University. It has been accepted for inclusion in Public Health Theses by an authorized administrator of ScholarWorks @ Georgia State University. For more information, please contact scholarworks@gsu.edu.

ABSTRACT

INTRODUCTION: Studies on the topic of adverse childhood experiences (ACEs) and childhood obesity collectively indicate an association, but there is a lack of replication in nationally representative sample of children aged 10-17 years. This study aims to expand on the definition of ACEs to include: socio-economic hardship, racial discrimination, witness or victim of neighborhood violence, and bereavement, and to examine their individual and joint association with BMI levels, especially childhood obesity (primary outcome).

METHODS: The 2011-2012 National of Children's Health (NSCH) was used for this study (N=45,309). One child interview weight was produced; hence, the estimates are generalized to all non-institutionalized children 10-17 years of age in the US and each state. Statistical methods used included descriptive statistics and multivariable multinomial logistic regression models.

ACEs examined included: (1) Socioeconomic hardship, (2) Parental divorce or separation, (3) Bereavement, (4) Incarcerated family member, (5) Witness to domestic violence, (6) Victim/witness of neighborhood violence, (7) Household mental illness, (8) Household substance abuse, (9) Racial discrimination.

BMI for the same sex and age (10-17 years) percentile relative measurement, using growth charts recommended by CDC, among children and teens were used as indicators of BMI. BMI-95th percentile or greater was considered obese.

RESULTS: The prevalence of childhood obesity and ACE exposure was higher for boys compared to girls. Controlling for gender, among those who were obese, White-non-Hispanic children had the highest prevalence of obesity compared to other races for both genders. Southern States constituted 80% and 60 % of top 10 states with the highest prevalence of childhood obesity and ACE, respectively.

Approximately 25.4 million (89.5%) children aged 10-17 years had experienced 3 or less ACE. The most prevalent ACE category of nine asked about for child was-living with parents who were either divorced or separated after his/her birth (26.77%) and- the least prevalent was living with a parent who died (4.84 %). ACEs were not mutually exclusive, and all nine categories of ACEs were interrelated.

The adjusted odds ratio of covariates to their reference groups that were only statistically significant for childhood obesity relative to healthy weight encompassed: a) Place of residence in metropolitan statistical area, b) two or more chronic health conditions of 18 asked about, c) Watching TV, videos, or playing video games across categories >1 to <4 hours and ≥ 4 hours, d) family members in the household eat a meal together 7 days of the week, e) and computer, cell phone or electronic device use ≤ 1 hour.

Moreover, the explanatory variables, namely, age, sex, physical health status of parents, and physical activity, were strongly related to childhood obesity (associated both with higher odds and lower odds of outcome) compared to overweight and underweight BMI categories.

CONCLUSIONS: This is the first study to explore the co-occurrence, individual and joint association of ACEs with childhood obesity using nationally representative sample of children aged 10-17 years in the U.S. Having childhood obesity, BMI-95th percentile or above was strongly related to ACE dichotomy, ACE score ≥ 2 and two ACE types (socioeconomic hardship and bereavement) than the probability of overweight, BMI-85th to 94th percentile. Underweight-BMI less than 5th percentile had only statistically significant association with socioeconomic hardship ACE category. Sociodemographic, parental, and childhood related factors were also independently associated with childhood obesity.

KEYWORDS: adverse childhood experience, child abuse, child maltreatment, household dysfunction, pediatric obesity, child of impaired parents, interrelationship, weight management, United States



ADVERSE CHILDHOOD EXPERIENCES AND THE ASSOCIATION WITH CHILDHOOD
OBESITY: A CROSS-SECTIONAL STUDY OF THE U.S. NATIONAL SURVEY OF CHILDREN'S
HEALTH (NSCH), 2011-2012.

by

OMARWALID NOORZADA

M.D., KABUL MEDICAL UNIVERSITY

A Thesis Submitted to the Graduate Faculty
of Georgia State University in Partial Fulfillment
of the
Requirements for the Degree

MASTER OF PUBLIC HEALTH

ATLANTA, GEORGIA
30303

APPROVAL PAGE

ADVERSE CHILDHOOD EXPERIENCES AND THE ASSOCIATION WITH CHILDHOOD
OBESITY: A CROSS-SECTIONAL STUDY OF THE U.S. NATIONAL SURVEY OF CHILDREN'S
HEALTH (NSCH), 2011-2012.

by

OMARWALID NOORZADA

Approved:

Dr. Shanta Rishi Dube
Committee Chair

Dr. Matthew Hayat
Committee Member

June 23, 2016
Date

Author's Statement Page

In presenting this thesis as a partial fulfillment of the requirements for an advanced degree from Georgia State University, I agree that the Library of the University shall make it available for inspection and circulation in accordance with its regulations governing materials of this type. I agree that permission to quote from, to copy from, or to publish this thesis may be granted by the author or, in his/her absence, by the professor under whose direction it was written, or in his/her absence, by the Associate Dean, School of Public Health. Such quoting, copying, or publishing must be solely for scholarly purposes and will not involve potential financial gain. It is understood that any copying from or publication of this dissertation which involves potential financial gain will not be allowed without written permission of the author.

OMARWALID NOORZADA

Signature of Author

DEDICATION

This thesis research project would not have been possible without Allah's grace and the love and support from my family. I would like to dedicate this work to all my family members who are the equator and prime meridian of my love. My father who is the hero of my life, my mother where paradise lies beneath her feet, my two sisters who are the angels on earth, and my small brother who is my best friend. I also want to dedicate this work to my late grandfather who is my role model in life.

ACKNOWLEDGMENTS

THESIS COMMITTEE CHAIR: Dr. Shanta Rishi Dube

THESIS COMMITTEE CO-CHAIR: Dr. Matthew Hayat

GSU-SCHOOL OF PUBLIC HEALTH ADVISOR: Gina Sample

GSU-SCHOOL OF PUBLIC HEALTH: Instructors and Administration

GSU-INTERNATIONAL STUDENT AND SCHOLAR SERVICES ADVISOR: Mike Townsend

INTERNATIONAL INSTITUTE OF EDUCATION ADVISOR: Tom Koerber

DATA RESOURCE CENTER: Child and Adolescent Health Measurement Initiative

THE FULBRIGHT SCHOLARSHIP PROGRAM FOUNDER: Senator J. William Fulbright

THE FULBRIGHT SCHOLARSHIP PROGRAM FOR CITIZENS OF AFGHANISTAN:

The U.S. Department of State's Bureau of Educational and Cultural Affairs

LIST OF TABLES

TABLE 2.1. Potentially Reversible Causes of Weight Gain.....	21
TABLE 2.2. Literature Review Table of Causes of Childhood Obesity.....	23
TABLE 2.3. Literature Review Table of Consequences of Childhood Obesity.....	29
TABLE 2.4. Original ACE Study-ACE Burden, Scores and Associated Health Problems (N=17,337).....	36
TABLE 2.5. Total Lifetime Costs of Child Maltreatment 2008 United States.....	38
TABLE 3.1. Adverse Childhood Experiences Among Children in the NSCH 2011-2012. ACEs Definitions are Based on after the Child’s Birth.....	55
TABLE 3.2. BMI-Class for Children of the Same Sex and Age.....	60
TABLE 3.3. Glossary and Explanation of Variables (2011-2012 NSCH.....	66
TABLE 4.1. Distribution of Age Categories by Sex.....	79
TABLE 4.2. Prevalence of Each Category of ACE, ACE Scores, RACE And Household Poverty Status by Gender Among U.S. Children 10-17 Years Old. The 2011-2012 National Survey of Children’s Health (N=45,309).....	80
TABLE 4.3. Frequency Distribution of Number of ACEs.....	82
TABLE 4.4. Childhood Obesity Distribution by and RACE Controlling for Gender among Children Aged 10-17 Years in the U.S.....	84
TABLE 4.5. Prevalence of Reporting of Additional Categories of ACEs Among U.S. Children 10-17 Years Old, Who Reported Exposure to First Category of ACE. The 2011-2012 National Survey of Children’s Health (N=45,309).....	87
TABLE 4.6. Tetrachoric Correlation Of Reporting of Additional Categories Of ACEs Among U.S. Children 10-17 Years of Age Who Reported Exposure To First Category Of ACE. The 2011-2012 National Survey of Children’s Health (N=45,309).....	88
TABLE 4.7. Distribution and Association (Unadjusted Odds Ratio) of Participants’ Characteristics by their ACE Status among U.S. Children Aged 10-17 Years. The 2011-2012 National Survey of Children’s Health (N=45,309).....	91
TABLE 4.8. Distribution And Association (Unadjusted Odds Ratio) of Participants’ Characteristics by their BMI for Age Status among U.S. Children Aged 10-17 Years. The 2011-2012 National Survey of Children’s Health (N=45,309).....	96
TABLE 4.9. Crude And Covariate Adjusted Odds Ratios of Obesity; Overweight and Underweight Relative to Healthy Weight Among U.S. Children Aged 10-17 by Selected Demographic and Behavioral Characteristics. The 2011-2012 National Survey Of Children’s Health (N=45,309).....	104
TABLE 5.1. Effect of ACE Missing Values on Childhood Obesity.....	114

LIST OF FIGURES

FIGURE 1.1 - Adverse Childhood Experiences Measured in NSCH 2011-2012.....	08
FIGURE 2.1 - Age-Standardized Prevalence of Overweight and Obesity, and Obesity Alone (Based On IOTF Cutoffs), Ages 2-19 Years, by Sex, 1980-2013.....	13
FIGURE 2.2 - Prevalence of Overweight And Obesity (BMI>=25) And Obesity (BMI>=30), By Age And Sex, 2013.....	14
FIGURE 2.3 - The ACE Pyramid: Conceptual Framework for the Ace Study.....	37
FIGURE 2.4 - Consequences of Lifetime Exposure to Violence and Abuse.....	44
FIGURE 3.1 - National Survey of Children's Health Sampling and Survey Administration.....	51
FIGURE 4.1 - Distribution of Age Categories by Sex.....	79
FIGURE 4.2 - Weighted Total % of ACES.....	81
FIGURE 4.3 - Frequency Distribution of Number of Aces.....	82
FIGURE 4.4 - Top 10 Highest Levels of Obesity by States (Including DC) in the U.S. among Children 10-17 Years. The 2011-2012 National Survey of Children's Health.....	83
FIGURE 4.5 - Top 10 Highest Levels of ACE by States (Including DC) in the U.S. among Children 10-17 Years. The 2011-2012 National Survey Of Children's Health.....	83
FIGURE 4.6 - Comparison of BMI Class Frequencies (Unweighted) by Sex.....	84
FIGURE 4.7 - Comparison of BMI Class % (Weighted) by Sex.....	84
FIGURE 5.1 - BMI Class Distribution among U.S. Children Aged 10-17 Years.....	108
FIGURE 5.2 - Top 10 Highest ACES % (Weighted) and its Correlation with its Corresponding Obesity % (Weighted) by States (Including DC) in The U.S. Among Children 10-17 Years. The 2011-2012 National Survey of Children's Health.	109
FIGURE 5.3 - The ACE Pyramid: Conceptual Framework for the ACE Study.....	117
FIGURE 5.4 - Potential Mechanisms of Action of ACE Leading To Childhood Obesity..	118
FIGURE 5.5 - Reasons for the Early Prevention and Control of Childhood Obesity.....	120
FIGURE 5.6 - Association Between ACES And Childhood Obesity in Individualistic Vs. Collectivistic Societies.....	121
FIGURE 5.7 - Conclusion of ACES Association with Childhood Obesity.....	122

TABLE OF CONTENTS

DEDICATION.....	III
ACKNOWLEDGMENTS.....	IV
LIST OF TABLES.....	V
LIST OF FIGURES.....	VI
CHAPTER 1: INTRODUCTION	1
1.1 BACKGROUND	2
1.2 PURPOSE OF RESEARCH/RATIONAL	8
CHAPTER 2:REVIEW OF LITERATURE.....	11
2.1 CHILDHOOD OBESITY.....	12
2.1.1 <i>Epidemiology/Burden</i>	<i>12</i>
<i>MALNUTRITION-THE DOUBLE BURDEN:</i>	<i>18</i>
2.1.2 <i>Risk Factor and Etiology</i>	<i>19</i>
2.1.3 <i>Prognosis and Consequences</i>	<i>26</i>
2.2 THE CONTRIBUTION OF ADVERSE CHILDHOOD EXPERIENCES TO OBESITY	31
2.2.1 <i>Origins of ACE Study:.....</i>	<i>31</i>
2.2.2 <i>Epidemiology/Burden</i>	<i>32</i>
2.2.3 <i>Risk Factor and Etiology</i>	<i>34</i>
2.2.4 <i>Prognosis and Consequences.....</i>	<i>35</i>
2.2.5 <i>Overview of ACES Association with Childhood Obesity:.....</i>	<i>39</i>
CHAPTER 3:METHODS AND PROCEDURES	49
3.1 STUDY DESIGN AND SAMPLE	50
3.2 INCLUSION AND EXCLUSION CRITERIA	52
3.3 DEFINITION OF TERMS/ MEASUREMENT	53
3.3.1 <i>Adverse Childhood Experiences (ACE) - Main Independent Variable)</i>	<i>53</i>
3.3.2 <i>Childhood Obesity (Main Outcome).....</i>	<i>56</i>
3.3.3 <i>COVARIATES</i>	<i>61</i>
3.4 STATISTICAL ANALYSIS	69
3.4.1 <i>Characteristics of the Study Population By Sex, ACE Status, BMI-CLASS and State:</i> <i>.....</i>	<i>71</i>
3.4.2 <i>Relationship Between Categories of ACE:</i>	<i>74</i>
3.4.3 <i>Covariate Adjusted (Final Model) Analysis of BMI-Classes Relative to Healthy</i> <i>Weight: (Multivariate Analysis):.....</i>	<i>74</i>
3.4.4 <i>Missing Information on Exposure and Outcome Variables (Unadjusted OR):</i>	<i>76</i>
3.5 ETHICAL STATEMENT.....	77

CHAPTER 4: RESULTS	78
4.1 CHARACTERISTICS OF THE STUDY POPULATION:	79
4.3 ACE AND CHILDHOOD OBESITY DISTRIBUTIONS BY STATES IN THE UNITED STATES:	83
4.4 CHILDHOOD OBESITY DISTRIBUTION BY GENDER AND RACE IN THE UNITED STATES:.....	84
4.5 RELATIONSHIPS BETWEEN CATEGORIES OF ADVERSE CHILDHOOD EXPERIENCES:	85
4.6 DISTRIBUTION AND ASSOCIATION (UNADJUSTED ODDS RATIO) OF PARTICIPANTS’ CHARACTERISTICS BY THEIR ACE STATUS:	86
4.7 DISTRIBUTION AND ASSOCIATION (UNADJUSTED ODDS RATIO) OF PARTICIPANTS’ CHARACTERISTICS BY BMI-CLASS:	93
4.8 DISTRIBUTION AND ASSOCIATION (UNADJUSTED ODDS RATIO) OF PARTICIPANTS’ ACE STATUS, ACE SCORE AND ACE CATEGORIES BY BMI-CLASS:.....	94
4.9.1 CRUDE AND COVARIATE ADJUSTED (FINAL MODEL) ASSOCIATION OF BMI-CLASSES RELATIVE TO HEALTHY WEIGHT:	100
4.9.2 ACE AND CHILDHOOD OBESITY:	102
CHAPTER 5: DISCUSSION	106
5.1 DISCUSSION OF RESEARCH QUESTIONS:.....	107
5.2 STUDY STRENGTHS AND LIMITATIONS:	112
5.2.1 Strengths:.....	112
<i>Note: Odds Ratios of Obesity for those with ACE vs. No ACE are unadjusted and are calculated from unweighted frequencies.</i>	114
5.2.2 Limitations:	114
5.3 IMPLICATIONS OF FINDINGS:	116
<i>Potential Mechanisms by Which ACES Lead to Childhood Obesity:.....</i>	117
5.4 RECOMMENDATIONS AND PREVENTION STRATEGIES:.....	119
5.5 FURTHER RESEARCH QUESTIONS:	120
5.6 CONCLUSION:.....	122
REFERENCES.....	123

CHAPTER 1: INTRODUCTION

1.1 Background

Obesity is one of the 21st centuries public health issues that lies at the other end of the spectrum of malnutrition, which has transcended geographical boundaries and now has inscribed itself as a global epidemic “globesity”(World Health Organization [WHO], 2015). It affects individuals of all ages, both sexes, wealthy and poor, developed and developing countries around the world (WHO, 2015).

It is a [disease] (AMA News Room, 2013) that has been “one of the greatest neglected health problems of our time” that grave repercussions “as great as that of smoking”, stated by The World Health Organization press release in 1997 (In K. M. Goel & D. K. Gupta, P.438).

Over and above, recently under the campaign of *lets' move*, led by the first lady of the United States Michelle Obama, the issue of childhood obesity was highlighted in the United States. On the first day of her campaign, she contended, *"The physical and emotional health of an [entire generation] and the economic health and security of our nation is at stake."* (Michelle Obama, 2010).

Childhood obesity is a critical public health issue for the new generation of children in the US and not only has reached an epidemic level, but also the children are fatter and heavier today compared to decades before (Kohn & Booth, 2003). The authors urge that although an epidemic of non-communicable disease such as obesity seems “benign”; however, they are as detrimental as their “contagious infectious cousins” to individuals’ health.

Based on World Health Organization estimates, 42 million pre-school children (under five years) have been overweight in the world, of which approximately 74 % were in developing countries (WHO, 2015). There is evidence of an increase in the number of obese children in low and middle countries, that is, the rate of childhood overweight and obesity indicates a 30 % increase compared to that of developed countries.

This overweight paves the ground for their obesity and eventually leads to an increased risk for adulthood obesity, early death or disability due to different related chronic non-communicable diseases compared to those who are non-overweight (WHO, 2014).

In a systematic review “Global, regional and national prevalence of overweight and obesity in children and adults 1980-2013” by Ng et al. (2014) indicated that in the year 2013 there was a considerable increase in the number of overweight and obese among children and adolescents both in the developed and developing countries. The authors reported that there were 23.8% (22.9-24.7) of boys and 22.6% (21.7-23.6) of girls who were overweight or obese in the developed countries compared to 12.9% (12.3-13.5) of boys and 13.4% (13.0-13.9) of girls who were overweight and obese in the developing countries.

Moreover, based on the National Health and Nutrition Examination Survey (NHANES, 2011-2012), 8.1 % of infants and toddlers, 16.9% of 2-19-year-olds and, 34.9% of adults were obese in the United States (Ogden, Carroll, Kit, & Flegal, 2014).

The authors argue that the global prevalence of overweight and obesity between 1980 and 2013 has increased 27.5 % for adults and 47.1% for children, that is, from 921 million to 2.1 billion.

Based on the literature it seems that different factors at Micro, Meso, and Macro level interplay with one another or independently increases the risk of childhood obesity with various pathophysiological mechanisms. The individual and proximal elements are either: **A)** behavioral, viz. Taking fat-energy-rich food and energy imbalance between caloric intake and expenditure not for a day but over time (DHHS, AIM for a Healthy weight, 2014), Medication use (CDC, 2015), Decrease in physical activity due to sedentary lifestyles, urbanization and shift in transportation modes (WHO Fact sheet, 2015), Technological advancement, watching TV and computer use (K. M. Goel & D. K. Gupta, 2012; Singh, Kogan, Van Dyck, & Siahpush, 2008), **B)** Genetic (Comuzzie & Allison, 1998; Rankinen et al., 2006; K. M. Goel & D. K. Gupta, 2012), **C)** Biologic and evolutionary hereditary traits either by mismatch pathway (Hanson & Gluckman, 2014) or developmental pathway from paternal (McPherson, Fullston, Aitken, & Lane, 2014) and maternal health conditions (Whitaker, 2004; Reynolds, Osmond, Phillips, & Godfrey, 2010; Fraser et al., 2010; Woo Baidal et al., 2016; Eisenman, Sarzynski, Tucker, & Heelan, 2010), **D)** Metabolic or endocrine disorders (J. Webster-Gandy, A. Madden, & M. Holdsworth, 2012; Chatterjea, M. N., & Shinde, R, 2012).

At the Meso level *Fetal programming*, as suggested by Thrifty Phenotype Hypothesis, which is also known as Barker Hypothesis and Developmental origins hypothesis, may lead to childhood obesity among children undernourished in the womb (Barker & Osmond, 1986).

There is myriad scientific evidence of distal factors correlation to childhood obesity¹, that is, these upstream factors include: Advertising less healthy food (CDC, Children's Food Environment State Indicator Report, 2011; McGinnis, J. M., Gootman, J. A., & Kraak, V. I. (2006), differences in state child care licensing regulations related to nutrition, physical activity, and media use (Pathways and Partnerships for Childcare Excellence, 2012; Kaphingst & Story, 2009), No Safe and appealing place, in many communities, to play or to be active (CDC, State Indicator Report on Physical Activity, 2014), disparities in access to healthy foods (Larson, Story, & Nelson, 2009), Greater availability of dietary-energy-dense foods and sugar-sweetened beverages (L. Johnson, Mander, Jones, Emmett, & Jebb, 2008; Laura Johnson, Mander, Jones, Emmett, & Jebb, 2008; Reedy & Krebs-Smith, 2010; Vartanian, Schwartz, & Brownell, 2007; Wang, Bleich, & Gortmaker, 2008), Increasing portion sizes (Orlet Fisher, Rolls, & Birch, 2003; Orlet Fisher et al., 2003), “Lack of breastfeeding support” (Arenz, Ruckerl, Koletzko, & Kries, 2004; Owen, Martin, Whincup, Smith, & Cook, 2005), culture (K. M. Goel & D. K. Gupta, 2012), and Race/ethnicity, SES (Singh et al., 2008).

The study by Singh et al. (2008) indicated that both individual and social factors are associated with Childhood and adolescence obesity. The authors in the 2003 National Survey of Children’s Health found that “Racial/Ethnic, Socioeconomic, and Behavioral” determinants are not only independently but also jointly associated with childhood and adolescence obesity.

Over and above, B. R. Walker, N. R. Colledge, S. H. Ralston, & I. D. Penman (2014) urge that some causes of obesity on occasions are reversible, that is, diagnosable and treatable.

¹ Headings adopted from CDC, <http://www.cdc.gov/obesity/childhood/causes.html>

The authors argue that, these causes are differentially distinguished from the others by their short history and a recently pronounced weight gain. These causes are either due to endocrine factors (Hypothyroidism, Cushing's syndrome, Insulinoma, Hypothalamic tumors or injury) or drug treatments (Atypical antipsychotics [e.g. olanzapine], Sulphonylureas, thiazolidinediones, insulin, Pizotifen, Corticosteroids, Sodium valproate and β -blockers).

One of the predisposing factors to childhood obesity is Adverse childhood experiences and/or childhood maltreatment (Danese & Tan, 2014; Helton & Liechty, 2014; Li, Chassan, Bruer, Gower, & Shelton, 2015; Power, Pinto Pereira, & Li, 2015; Whitaker, Phillips, Orzol, & Burdette, 2007; Felitti et al., 1998; Lodhia et al., 2015; Fuemmeler, Dedert, McClernon, & Beckham, 2009; Burke, Hellman, Scott, Weems, & Carrion, 2011), which is defined by the Centers for Disease Control and Prevention (CDC) as "any act or series of acts of commission or omission by a parent or other caregiver (e.g., clergy, coach, teacher) that results in harm, potential for harm, or threat of harm to a child" (CDC, 2015).

Power, Pinto Pereira, & Li, 2015 based on prospective 50-year British cohort urged that life course of exposure to childhood maltreatment had a significant effect on obesity and body mass index. They found that the effect of physical abuse in both genders, sexual abuse in females was significant and positively associated with lifetime BMI gains, that is, they were more likely to be at faster risk for obesity. Psychological commission and omission were less consistent with the findings of this study.

Moreover, the original ACE study examined seven categories of adversaries a child experience in his/her childhood, which included 3 subtypes types of abuse - (physical, sexual and psychological) and 4 subtypes of household dysfunction (household members who were substance abusers, mother or stepmother treated violently, mentally ill or suicidal, or ever imprisoned) (Felitti et al., 1998). Publications from ACE study have denoted a statistically significantly positive association between adverse childhood experiences and obesity “Body weight and obesity in adults and self-reported abuse in childhood” (Williamson, Thompson, Anda, Dietz, & Felitti, 2002). The authors urge that if this association turns to be causal-prevention of childhood abuse may lead to a modest reduction of adulthood obesity and at the same time might help to discover the mechanisms that lead to adult obesity, and contribute to developing therapeutic remedies.

Studies have shown that adverse childhood experience affects the results of treatments for obesity. In a prospective case series study of 223 adult and mostly female patients, who undergone bariatric surgery determined, a positive relationship between ACE and postoperative BMI (Lodhia et al., 2015) .Those who were having a high ACE score (≥ 6) vs. patient with lower score had significantly higher levels of postoperative BMI both 6-months and 12 months after surgery (36.9 vs. 33.4 kg/m²), $p = 0.03$) and (34.5 vs. 30.5 kg/m², $p=0.07$), respectively. Therefore, the authors urge that is sagacious to tackle this issue preoperatively through counseling.

What is more, it is noteworthy to mention that it is also likely that children who are maltreated, to be at higher risk of visceral obesity that those of non-childhood maltreated group (1,1366 \pm 160 vs. 836 \pm 116 g, $P<0.05$), but have the normal body mass

index (Li, Chassan, Bruer, Gower, & Shelton, 2015). The authors assert, *perturbation of the hypothalamic–pituitary–adrenal axis activity and activation of the immune system*, as probable pathophysiologic mechanisms. This conditional was coined initially as *Thin-on-the-outside fat-on-the-inside* and individuals having this issue are found to be a higher risk for metabolic disorders (Thomas et al., 2012).

1.2 Purpose of Research/Rational

The research gap indicates that more investigations are needed to elucidate the associations between categories of ACEs and childhood obesity (V. J. Felitti et al., 1998; Williamson et al., 2002). Therefore, this study aims to expand on the definition of ACEs to include: socio-economic hardship, racial discrimination, witness/victim of neighborhood violence, and bereavement and to examine their individual and joint association with BMI levels among 45,309 U.S. children and adolescents aged 10-17 years in the 2011-2012 National Survey of Children’s Health dataset (2011-2012 NSCH).

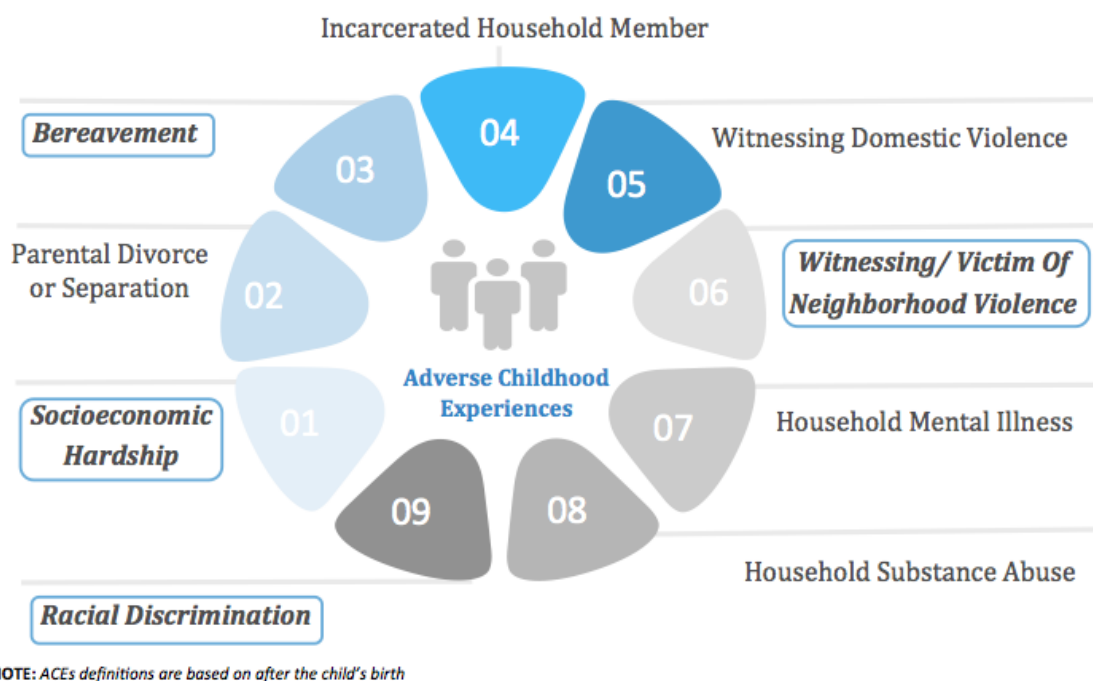


Figure 1.1 - Adverse Childhood Experiences Measured in NSCH 2011-2012.

Figure 1.1, above, shows the adverse childhood experiences that are examined in the present study which includes individual and family-level risk factors: (1) Socioeconomic hardship, (2) Parental divorce or separation, (3) Bereavement, (4) Incarcerated family member, (5) Witness to domestic violence, (6) Victim/witness of neighborhood violence, (7) Household mental illness, (8) Household substance abuse, (9) Racial discrimination (NSCH, 2011-2012). We use the NSCH 2011-2012, which is a dataset with a nationally representative sample of U.S. children 0-17 years of age.

Studies on the topic of childhood abuse and childhood obesity collectively indicate an association, but there is the lack of replications in nationally representative sample of children from 10-17 years of age.

The following research questions guide the study:

- 1) What is the relationship between the prevalence of ACEs and Childhood obesity in a nationally representative sample (NSCH 2011-2012) of children 10-17 years age in the United States?

Hypothesis 1: It is hypothesized that there is a statistically significant difference in prevalence of BMI of the given age and sex among children age (10-17years) who have adverse childhood experiences vs. No ACE in the US (National Survey of Children's Health 2011/12 NSCH).

- 2) What is the contribution of individual and joined categories of childhood adversity on levels of BMI among children 10-17 years of age with adverse childhood experience compared to those without ACE in the United States?

Hypothesis 2: The effect of ACEs on childhood obesity and may be higher than their individual impacts and follow a gradient pattern. Hence, an ACE score ≥ 2 would be associated with an increased odds of childhood obesity than ACE score =1 compared

to those without a history of adverse childhood experience.

- 3) Which ACE exposures have stronger associations with childhood obesity in a 45,309 nationally representative sample of children 10-17 years of age with ACE compared to those with no ACE in the United States?

Hypothesis 3: ACEs associated with childhood obesity are of similar magnitude.

Hence, by providing the findings of this study we aim to fill the research gap that our study aims to explore.

CHAPTER 2:
REVIEW OF LITERATURE

2.1 CHILDHOOD OBESITY

2.1.1 Epidemiology/Burden

Obesity is one of the 21st centuries public health issues. It lies at the other end of the spectrum of malnutrition. It has transcended geographical boundaries and now has inscribed itself as a global epidemic [globesity](WHO, 2015). It affects individuals of all ages, both sex, wealthy and poor, and developed and developing countries around the world (WHO factsheets, 2015).

Not only adolescence obesity has reached an epidemic level in the world, but also they are fatter and heavier today compared to decades before (Kohn & Booth, 2003). The authors urge that although epidemics of noncommunicable disease such as obesity seems [benign]; however, they are as detrimental to health as their [contagious infectious cousins]. It has reached pandemic level yet entirely not appropriately addressed and as Kaye K. Gaines addressed it as the “Elephant in the room-Pandemic Obesity” (Gaines, 2015).

Abnormal or excess accumulation of fat that leads to overweight or obesity and possess a health risk to the individual (WHO, 2016). In 2008, based on WHO estimates, it was reported that there were 1.4 billion overweight and more than 500 million obese adults. In 2014, the number of overweight reached to 1.9 billion adults (38% men and 40% women) of which more than half a billion were obese (11 % men and 15% women), which indicates that women have higher rates of overweight and obesity (WHO: Obesity and Overweight, 2015).

Besides, the prevalence of obesity has more than doubled between 1998-2014. On average it takes the life of 2.8 million people worldwide (WHO: 10 facts on obesity, 2014). For instance, in more than half of the high and middle-income country it takes more lives than does underweight. “44 % of diabetes, 23 % IHD and 7 to 41% of certain cancers are attributable to overweight and obesity” (WHO: 10 facts on obesity, 2014).

42 million pre-school children (under five years) have been overweight in the world of which 31 million of them were in developing countries. There is evidence of an increase in the number of obese children in low and middle countries, that is, the rate of childhood overweight and obesity indicates a 30 % increase compared to that of developed countries. This overweight paves the ground for their obesity and eventually led to elevated prevalence of adulthood obesity, early death or disability due to different related chronic non-communicable diseases compared to those who are non-overweight (WHO: 10 facts on obesity, 2014).

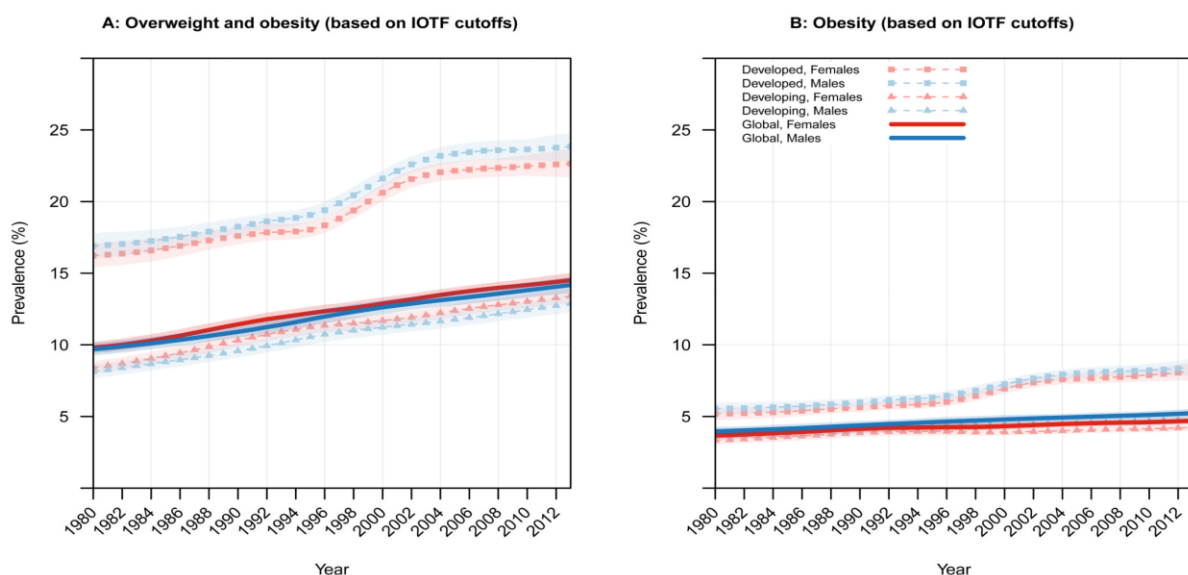


Figure 2.2 - Age-standardized prevalence of overweight and obesity, and obesity alone (based on IOTF cutoffs), ages 2–19 years, by sex, 1980–2013 (Ng et al., 2014)

In a systematic review “Global, regional and national prevalence of overweight and obesity in children and adults 1980-2013” by Ng et al. (2014) indicated that in the year 2013 there was a marked surge in the number of overweight and obese among -children and adolescents both in the developed and developed countries (Figure 2.1)

There were 23.8% (22.9-24.7) of boys and 22.6% (21.7-23.6) of girls who were overweight or obese in the developed countries compared to 12.9% (12.3-13.5) of boys and 13.4% (13.0-13.9) in girls who were overweight and obese in the developing countries (Figure 2.2).

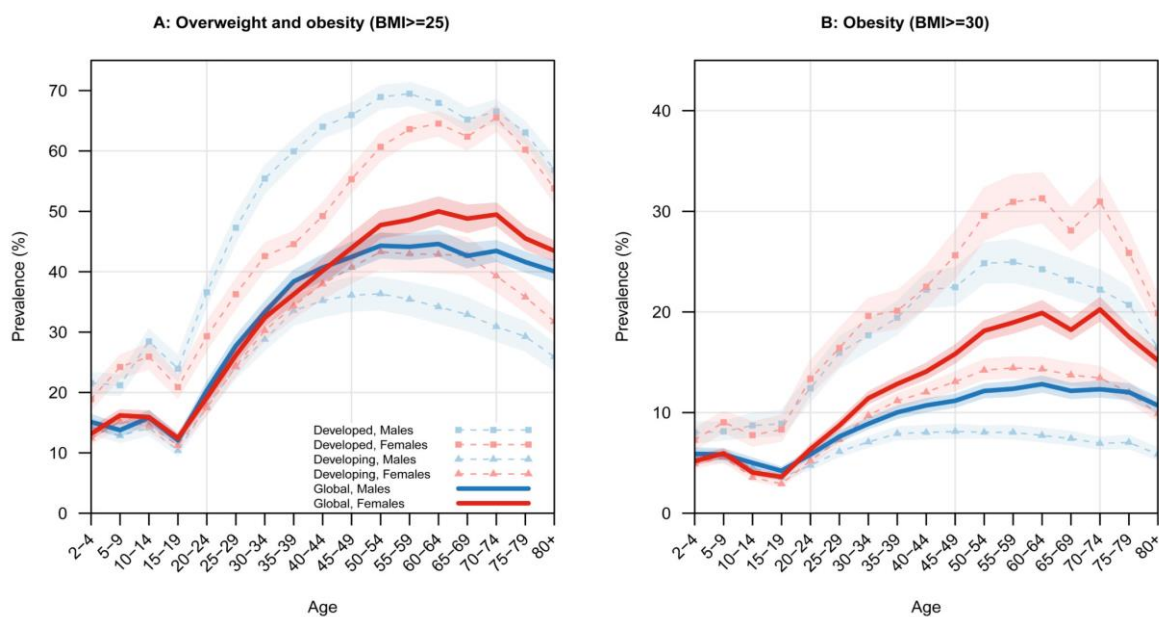


Figure 2.3 - Prevalence of overweight and obesity (BMI >= 25) and obesity (BMI >= 30), by age and sex, 2013 (Ng et al., 2014)

The authors argue that the global prevalence of overweight and obesity between 1980 and 2013 has increased 27.5 % for adults and 47.1% for children, that is, from 921 million to 2.1 billion.

These estimates among the children and adolescents category, indicates higher prevalence of heavier weight among boys vs. girls (Ng et al., 2014), but these estimates-

were opposite for adult age group estimates of WHO where women had higher overweight and obesity prevalence compared to men (WHO: Obesity and Overweight, 2015).

In the year 2012, a group of retired American Generals, Admirals, and Civilian Military Leaders issued a report “Too Fat to Fight” that urged the prominent reason that 75% of young Americans ranging from age 17 to 24 years cannot join military forces is [being too overweight and obese] (Mission: Readiness. Military Leaders for Kids, 2012). This report rang a different type of bell for the American people that not only their overall health is in danger but also, it is a significant threat to [national security] of the US.

Another alarming point of consideration is the medial costs attributable to obesity. Finkelstein, Trogon, Cohen, & Dietz (2009) found that rise in obesity prevalence and medical expenses cannot be irrelevant. The results indicate that in 2006 medical costs of obesity have soared up to \$147 billion dollars a year based on the - National Health Expenditure Accounts (NHEA) data or \$86 billion dollars based on Medical Expenditure Panel Survey (MEPS) data, which constitutes almost 10% of all medical expenditures in the US. Astonishingly, in 1998, these expenses were \$42 and- \$74 billion, respectively. It was highlighted by the researchers that “Across all payers, obese people had medical spending that was \$1,429 greater than spending for normal-weight people in 2006.” This spending shows a 41.5% difference compared to normal weight individuals. Moreover, 8.5%, 11.8%, and 12.9 % are the costs incurred by Medicare, Medicaid, and private payer spending respectively, attributable to obesity in 2006.

However, if obesity trends in the U.S. were lowered by reducing the average adult BMI by only 5 %, millions of Americans could be spared from serious health problems- and preventable diseases, and the country could save \$29.8 billion in five years, \$158 billion in 10 years and \$611.7 billion in 20 years (Trust for America's Health & Robert Wood Johnson Foundation, 2014).

Childhood obesity has physical and psychological health consequences during childhood, and can contribute to behavioral and emotional difficulties, and reduces educational attainment (Pizzi & Vroman, 2013). It is one of the alarming public health challenges of the 21st century. However, childhood obesity and its related diseases are preventable. WHO has developed the "Global Action Plan for the prevention and control of non-communicable diseases 2013-2020", one of its aims is to decrease the global obesity levels to those of 2010 (WHO, Global Action Plan for the Prevention and Control of NCDs 2013-2020, 2013).

In a systematic review by Woo Baidal et al. (2016) comprising 282 original quantitative studies on "risk factors for childhood obesity in the first 1,000 days" found several categories of modifiable risk factors associated with childhood obesity. The authors urge that these 1,000 days, from conception to 2 years, form a critical window to prevent modifiable risk factors that are related to childhood obesity. The enumerated list of factors that imperils children to obesity were, namely, higher maternal pre-pregnancy BMI, prenatal tobacco exposure, maternal excess gestational weight gain, high infant birth weight, and accelerated infant weight gain, gestational diabetes, child care attendance, low strength of maternal- infant relationship, low SES, curtailed infant sleep, inappropriate bottle use, introduction of solid food intake before age 4 months, and infant antibiotic exposure.

The UN sustainable development goals (SDGs) set 17 priority goals to be achieved by 2030 of which goal 3 “Ensure healthy lives and promote well-being for all ages”, and one of the deliverables by 2030 to achieve the target goal is to prevent and treat one-third of early deaths from non-communicable diseases and to promote mental health and well-being (Sustainable development goals - United Nations, 2015).

Similarly, the Office of Disease Prevention and Health Promotion (ODPHP) within the U.S. Department of Health and Human Services (HHS) in December 2010 set forth the “Healthy people 2020” initiative targeting around 43 topics (Health People 2020, 2010). One of the objectives in Nutrition and weight status domain is the 10 % reduction in the proportion of children and adolescents’ aged 2-19 years who are obese by the year 2020 (NSW10-Healthy people 2020, 2010).

The report of World Health Organization “ The Commission on Ending Childhood Obesity” (ECHO) formed in 2014 indicates the magnitude of the issue of childhood obesity and the dire need for its control and prevention (WHO, ECHO, 2016). The main points of this report, which is the product of 2 years exhaustive expert working - groups, elucidates the multidimensional facet of the issue: The roles of obesogenic environments promoting weight gain, energy imbalance (as a result of unhealthy food-consumption and marketing, availability of healthy food, sedentary lifestyle with less physical engagement), the biological and behavioral responses of child being formed prenatally and fortified by encountering obesogenic environment after birth.

The committee [ECHO] urges that there is no one size fits all solution for childhood obesity, and there is a need for considering a) contextual conditions b) consideration of three critical life course periods, namely, preconception and pregnancy, infancy and early childhood and older childhood and adolescence c) treatment of children who are already obese.

The ECHO commission final report has a comprehensive, integrated and multisectoral (including: WHO, International organizations, Member States, NGOs, - Philanthropic foundations, and Academic Institutions), non-vertical (in alignment with governmental and non-governmental initiatives) set of recommendations to address childhood obesity epidemic, namely, promotion of high nutrient value food intake by children and adolescents; put into practice programs that physically engage them; perinatal care and health education on prevention of noncommunicable diseases; proper diet, sleep and physical activity management for children at their early childhood; increase health and nutrition literacy and physically activity among school-age children; and proper weight management of children and adolescents who are obese.

MALNUTRITION-THE DOUBLE BURDEN:

The World Health Organization has let off the siren of [malnourishment] in developing countries struggling to deal with the issue of [under nutrition] for a long time-they are in the hot zone of overweight and obesity (WHO, Obesity and overweight, 2015). WHO contends that the two-lane road of malnutrition is moving in the same direction- endangering both the children's health and increasing the probability of

many non-communicable diseases. Lack of access to food during perinatal and - childhood period from one hand and access to reduced nutrient value and cheap food (rich with high fat, high sugar, and high salt) on the other are doubling the burden of malnutrition diseases in low and middle-income countries. It is not odd to find simultaneously in families, communities and nations at large where one has undernutrition while the other suffers from overweight and obesity.

2.1.2 Risk Factor and Etiology

Technological advancements have been one of the culprits of childhood obesity (K. M. Goel & D. K. Gupta, 2012), that is, Watching TV and computer has induced a reduction in physical activities and hence leads to accumulation of energy in the body that is not consumed by physical activities. For instance, around 28% of all children in the United States are watching TV more than 4 hours per day (NHANES 3 survey). Moreover, the authors underpin that genetics and culture are also as primary determining factors for childhood obesity, with estimated contributions of 25 % and 30%, respectively.

Over and above, physical activity is one of the most determining factors in the prevention of overweight and obesity; however, no more than 49 % and 20 % of Americans are active at moderate and vigorous levels, respectively (S. J. McPhee, M. A. Papadakis, & M. W. Rabow, 2). Besides, it is noted that only three out of every hundred - Americans follow the four of the five recommendations of the food guide pyramids when it comes to eating high nutrient value food such as, grains, fruits, vegetables, dairy products and meat, and merely a quarter of Americans consume the suggested five or more vegetables and fruits per day.

Besides, the Human Obesity Gene Map in 2005 urged that there are 20 to 30 genes in human DNA that are responsible for obesity in humans (Rankinen et al.,2006).

The authors contended that:

[176 human obesity cases due to single-gene mutations in 11 different genes have been reported...The number of human obesity QTLs derived from genome scans continues to grow, and we now have 253 QTLs for obesity-related phenotypes from 61 genome-wide scans...The obesity gene map shows putative loci on all chromosomes except Y.] (p. 529)

B. R. Walker, N. R. Colledge, S. H. Ralston, & I. D. Penman (2014) indicates twin and adoption studies have confirmed the genetic contribution in obesity that can be either polygenic or single gene disorders. “The pattern of inheritance suggests a polygenic disorder, with small contributions from a number of different genes, together accounting for 25–70% of variation in weight.”

Although single gene disorders are known to be rare, yet still they cause severe obesity among children, namely, mutations of the melanocortin-4 receptor (MC4R), which account for approximately 5% of serious early-onset obesity, the authors argued; defects in the enzymes processing proopiomelanocortin (POMC, the precursor for adrenocorticotrophic hormone [ACTH]) in the hypothalamus; and mutations in the leptin gene. Childhood obesity can be a feature of genetic syndromes, such as Prader-Willi and Lawrence-Moon-Biedl syndromes.

Besides, the authors urge that on occasions some causes of obesity are reversible (diagnosable and treatable). These causes are differentially distinguished from the others by their short history and a recently pronounced weight gain (Table 2.1).

TABLE 2.1. Potentially reversible causes of weight gain

Endocrine factors	Drug treatments
<ul style="list-style-type: none">• Hypothyroidism• Cushing's syndrome• Insulinoma• Hypothalamic tumors or injury	<ul style="list-style-type: none">• Atypical antipsychotics (e.g. olanzapine)• Sulphonylureas, thiazolidinediones, insulin• Pizotifen• Corticosteroids• Sodium valproate• β-blockers

Other mechanisms that lead to childhood obesity has been postulated (D. L. Longo, A. S. Fauci, D. L. Kasper, S. L. Hauser, J. L. Jameson, & J. Loscalzo, 2013):

- Increase nutrient absorption in the intestine depending on food composition, [sleep deprivation], and [unfavorable gut flora].
- Polygenic, monogenic (mutations) and syndromic obesity pathways
- Secondary causes of obesity include hypothalamic injury, hypothyroidism, Cushing's syndrome, and hypogonadism. Drug-induced weight gain is also common in those who use antidiabetes agents (insulin, sulfonylureas, thiazolidinediones), glucocorticoids, psychotropic agents, mood stabilizers (lithium), antidepressants (tricyclics, monoamine oxidase inhibitors, paroxetine, mirtazapine), or antiepileptic drugs (valproate, gabapentin, carbamazepine). Insulin-secreting tumors can cause overeating and weight gain.

Biological factor has also been found to be a risk factor for obesity by different pathophysiologic mechanisms:

1) MISMATCH PATHWAY:

The evidence in the field of developmental origins of health and disease (DOHaD) indicates “the result of the physiological processes of developmental plasticity, which may have potential adverse consequences in terms of NCD risk later” and “operate across the normal range of development and are largely physiological rather than pathophysiological” (Hanson & Gluckman, 2014). The authors urge that the field of DOHaD explains how [conditionings mechanisms] represents physiological processes in early life, but how later health status is endangered. The authors argue that one of the reasons that NCDs, such as obesity, have increased is due to a [mismatch], where neither evolutionary traits nor physiologies of individuals are prepared to attune to. The [mismatch] resulting from westernization, socio-economic development, change in nutritional habits, sedentary life style, etc. results in nonadaptive consequences.

2) DEVELOPMENTAL PATHWAY:

Paternal overweight/obesity has shown to have genetic and epigenetic effects on sperm function, embryo development and damage of offspring health subsequently (McPherson, Fullston, Aitken, & Lane, 2014). Moreover, many studies have also indicated a relationship between adverse maternal health conditions and childhood obesity: (Whitaker, 2004; Reynolds, Osmond, Phillips, & Godfrey, 2010; Fraser et al., 2010; Woo Baidal et al., 2016; Eisenman, Sarzynski, Tucker, & Heelan, 2010).

Table 2.2, below, summarizes the potential causes of Childhood Obesity.

TABLE 2.2. Literature Review Table of Causes of Childhood Obesity

TYPE	SUBTYPES	EXAMPLES	DESCRIPTION	RESOURCE
<p>A) DOWNSTREAM/ MICRO</p>	<p>Behavior</p>	<p>Energy imbalance between caloric intake and expenditure.</p>	<p>Healthy behavior is determined by a healthy diet and regular physical activity. Based on Dietary guidelines for Americans a healthy eating includes: various vegetables of all subgroups, whole fruits, grains, fat free or low fat dairy, various protein foods including meats, poultry, eggs legumes, nuts, seeds and soy products, and Oils. On the other hand the physical Activity Guidelines for Americans suggest a 1 hour or more daily physical activity that includes: Aerobic activity, Muscle strengthening and Bone strengthening.</p>	<p>Aim for a healthy weight: Maintaining a Healthy Weight On the Go A Pocket Guide. (2014, August). Retrieved June 9, 2016, from http://www.nhlbi.nih.gov/files/docs/public/heart/AIM_Pocket_Guide_tagged.pdf</p> <p>World Health Organization (WHO). <i>Obesity and overweight</i>. (2015, January). Retrieved April 03, 2016, from http://www.who.int/mediacentre/factsheets/fs311/en/</p> <p>Centers for Disease Control and Prevention (CDC). <i>Childhood Obesity Causes & Consequences</i>. (2015, June 19). Retrieved June 09, 2016, from http://www.cdc.gov/obesity/childhood/causes.html</p> <p>U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015 – 2020 Dietary Guidelines for Americans. 8th Edition. December 2015. Available at http://health.gov/dietaryguidelines/2015/guidelines/. http://health.gov/paguidelines/guidelines/</p>
		<p>Fat-energy-rich food intakes,</p>		
		<p>Decrease in physical activity due sedentary life styles, urbanization and shift in transportation modes.</p>		
	<p>Metabolic Factors</p>	<p>Endocrine disorders</p>	<p>Cushing syndrome, hypothyroidism, Prader-Willi Syndrome, Congenital Leptin Deficiency, Frölich’s Syndrome, hyperinsulinism</p>	<p>Chatterjea, M. N., & Shinde, R. (2012). <i>Diet and Nutrition/Obesity</i>. In <i>Textbook of medical biochemistry</i> (8th ed., pp. 763-769). New Delhi: Jaypee Brothers Medical Publications (P).</p> <p>Nutritional problems of children and adolescents. (2012). In J. Webster-Gandy, A. Madden, & M. Holdsworth (Eds.), <i>Oxford handbook of nutrition and dietetics</i> (p. 262). Oxford: Oxford University Press, USA.</p>
	<p>Genetics</p>	<p>Developmental pathway Paternal and maternal conditions</p>	<p>Genetic and epigenetic effects on sperm function, embryo development and damage to offspring health subsequently</p>	<p>McPherson, N. O., Fullston, T., Aitken, R. J., & Lane, M. (2014). Paternal obesity, interventions, and mechanistic pathways to impaired health in offspring. <i>Annals of Nutrition & Metabolism</i>, 64(3-4), 231–238. http://doi.org/10.1159/000365026</p> <p>Whitaker, 2004; Reynolds, Osmond, Phillips, & Godfrey, 2010; Fraser et al., 2010; Woo Baidal et al., 2016; Eisenman, Sarzynski, Tucker, & Heelan, 2010</p>

<p>B) MESO</p>	<p>Fetal Programing</p>	<p>Barker Hypothesis, Thrifty Phenotype Hypothesis, Developmental origins hypothesis</p>	<p>“The hypothesis (proposed in 1990 by the British epidemiologist David Barker) proposing that an undernourished baby becomes thrifty. It maintains high levels of sugar in the bloodstream to benefit the brain but less sugar in muscles. Muscle growth may be “traded off” to protect the brain. Once adopted, this thrifty behavior becomes permanent and, combined with adiposity in later life, leads to type 2 diabetes.” But this under nutrition graves for the other repercussions in middle ages and leads to programing ill health such as, cardiovascular diseases and type 2 diabetes.</p>	<p>Porta, M. S. (Ed.). (2008). A dictionary of epidemiology (5th ed.). Oxford: Oxford University Press.</p>
-----------------------	-----------------------------	--	---	---

TYPE	SUBTYPES	EXAMPLES	DESCRIPTION	RESOURCE
C) UPSTREAM/ MACRO	Environment Education Food Promotion	Variation in licensure regulations among child health care centers	<p>More than half of US middle and high schools offer sugar drinks and less healthy foods as competitive foods.</p> <p>High caloric diets enriched with high sugar, low healthy nutrients, and saturated fats are predisposing the children and the adult for different diseases later on in life such as CVD and type 2 diabetes.</p>	Johnson L, Mander AP, Jones LR, Emmett 28. PM, Jebb SA. A prospective analysis of dietary energy density at age 5 and 7 years and fatness at 9 years among UK children. <i>Int J Obes (Lond)</i> 2008;32(4):586—593.
		No Safe and appealing place, in many communities, to play or to be active	<p>“More than 12 million American children regularly rely on child care to support their healthy development and school success.”</p>	Johnson L, Mander AP, Jones LR, Emmett 29. PM, Jebb SA. Energy-dense, low-fiber, high-fat dietary pattern is associated with increased fatness in childhood. <i>Am J Clin Nutr</i> 2008;87:846—854.
		Limited access to healthy affordable foods	<p>Most of the US states don’t enforce and regulate their childcare centers for Nutrition, physical activity and media use. Only 12 regulated food of low nutritional value, 36 promoted physical outdoor activities in CCCs and only 8 states regulated amount of time to be spent on screen per day in the week at Small family child care homes.</p>	Reedy J, Krebs-Smith SM. Dietary sources of energy, solid fats, and added sugars among children and adolescents in the United States. <i>J Am Diet Assoc</i> 2010;110(10):1477—84.
		Greater availability of high-energy-dense foods and sugar sweetened beverages.	<p>Only 27 states have street safety policies for everyone including pedestrians and bicyclists.</p>	Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. <i>Am J Public Health</i> 2007;97(4):667—675.
		Increasing portion sizes	<p>United States society has been the named the capital of fast food or "fast food nation"² that pioneers in “obesogenic” unhealthy food. The environments have less walkability, inaccessible, and thus making healthy choices difficult for children across Macro socio-ecological levels.</p>	Wang YC, Bleich SN, Gortmaker SL. 5. Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among US children and adolescents, 1988–2004. <i>Pediatrics</i> 2008;121(6):e1604—1614.
		Lack of breastfeeding support		Fisher JO, Rolls BJ, Birch LL. Children's bite size and intake of an entrée are greater with large portions than with age-appropriate or self-selected portions. <i>Amer J Clin Nutr</i> 2003;77(5):1164—1170.
			Arenz S, Ruckerl R, Koletzko B, von Kries R. Breast-feeding and childhood obesity – a systematic review. <i>Int J Obes Relat Metab Disord</i> 2004; 28:1247—1256.	
			Owen CG, Martin RM, Whincup PH, et al. Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. <i>Pediatrics</i> 2005; 115:1367—1377.	

² Adopted from Eric Schlossel book title.

2.1.3 Prognosis and Consequences

Childhood obesity has profound detrimental effects on children's health not only in short term but also in a long run (J. Webster-Gandy, A. Madden, & M. Holdsworth, 2012):

- **Immediate Effects on health:**
 - It leads to non-communicable mostly chronic consequences such as cardiomyopathy, pancreatitis, orthopedic disorders, upper airway obstruction, or chest wall restriction.

- **Effects on well-being:**
 - Children who suffer from overweight and obesity face adverse repercussions, namely, physical, social (low self-esteem and social interaction), educational (poorer academic achievements), and earlier puberty.

- **Long-term effect on health:**
 - If overweight and obesity are combined with a diet with low nutritional values that is high in saturated fat, low Calcium and coupled with sedentary lifestyle-there is a higher risk of getting Diabetes Mellitus type 2, CVD, osteoporosis, gallstones, cancers related to diet and suboptimal peak bone mass compared to those with healthy weight. The risk is higher among children who retain their obesity into their adulthood. However, all obese children do not become obese adults.

Childhood overweight and obesity have been shown to lead to different adverse and detrimental health aftermaths, in multiple systems of the body by different pathophysiologic pathways (Daniels, 2006). The list of *body organs and systems* that were considered to be mostly affected by childhood obesity included: cardiovascular, endocrine, respiratory, gastrointestinal, skeletal, nervous and urogenital systems. The author urges that obesity-related repercussions that were pertinent to adults are [now threatening children's health] not only in short term but also in a long run. In summary, the children compared to their parents will have a shorter life span and possess less healthy lifestyles.

Central obesity is frequent among men and is correlated with diabetes Mellitus type 2, CVD and metabolic syndrome (B. R. Walker, N. R. Colledge, S. H. Ralston, & I. D. Penman, 2014). The most noteworthy point between these two types (Central and General Obesity) is their vasculature anatomy—intra abdominal fat is drained by portal vein to liver and thus increasing the products and by products of adipocytes in the liver (free fatty acids; 'adipokines' such as, tumor necrosis factor- α and adiponectins; steroid hormone) and as a result give rise to metabolic syndromes.

Moreover, S. J. McPhee, M. A. Papadakis, & M. W. Rabow (2013) claims that upper body obesity has severe consequences compared to lower body, and obese men and women with a higher abdominal circumference (> 102 cm in men and 88 cm in women) or increased waist-hip ratios (> 1.0 in men and > 0.85 in women), compared to [equally] obese men and women with reduced rates, are at greater risk for noncommunicable diseases (diabetes mellitus, stroke, coronary artery disease) and premature death.

On top of that, consequences of childhood obesity are diverse. It is a direct cause of morbidities in childhood including gastrointestinal, musculoskeletal and orthopedic complications, sleep apnea, and the accelerated onset of cardiovascular disease and -type-2 diabetes, as well as the comorbidities of the latter two non communicable diseases (Lobstein & Jackson-Leach, 2006).

Also, Obesity in childhood can contribute to behavioral and emotional difficulties, such as depression, and can also lead to stigmatization and poor socialization and reduce educational attainment (Pizzi & Vroman, 2013) (Miller, Lee, & Lumeng, 2015).

Critically, childhood obesity is a strong predictor of adult obesity, which has well-known health and economic consequences, both for the individual and society as a whole (Litwin, 2014; Nader et al., 2006).

Table 2.3, below, summarizes the consequence of Childhood obesity.

TABLE 2.3. Literature Review Table of Consequences of Childhood Obesity

TYPE	SUBTYPES	EXAMPLES	DESCRIPTION	RESOURCE
A) NOW	Cardiovascular System	High blood pressure and high cholesterol	<p>Childhood Obesity affects multiple systems with different patho-physiologic pathways and at different time periods.</p> <p>Children who are suffering from obesity are more likely to obese adults. Severity of the disease and its consequences are worse.</p>	Freedman DS, Mei Z, Srinivasan SR, Berenson GS, Dietz WH. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: the Bogalusa Heart Study. J Pediatr. 2007;150(1):12—17.e2.
	Gastro-intestinal system	Impaired glucose intolerance, Insulin resistance, and type 2 diabetes Fatty liver disease Gallstones Gastro-esophageal reflux Colon cancer		Whitlock EP, Williams SB, Gold R, Smith PR, Shipman SA. Screening and interventions for childhood overweight: a summary of evidence for the US Preventive Services Task Force. Pediatrics. 2005;116(1):e125—144
	Respiratory System	Apnea and Asthma		Han JC, Lawlor DA, Kimm SY. Childhood obesity. Lancet. May 15 2010;375(9727):1737—1748.
	Genito-urinary System	Breast cancer Infertility (men and women) Polycystic ovary syndrome		Sutherland ER. Obesity and asthma. Immunol Allergy Clin North Am. 2008;28(3):589—602, ix.
	Musculoskeletal System	Joint problems and musculoskeletal discomfort Osteoarthritis Chronic back pain		Nutritional problems of children and adolescents. (2012). In J. Webster-Gandy, A. Madden, & M. Holdsworth (Eds.), Oxford handbook of nutrition and dietetics (p. 409). Oxford: Oxford University Press, USA.
	Nervous System/ Psychosocial	Depression Behavioral problems Issues in School Low self esteem and low self-reported quality of life Social isolation Impaired relationships Poor employment		Han JC, Lawlor DA, Kimm SY. Childhood obesity. Lancet. May 15 2010;375(9727):1737—1748.
	Taylor ED, Theim KR, Mirch MC, et al. Orthopedic complications of overweight in children and adolescents. Pediatrics. Jun 2006;117(6):2167—2174. Morrison, Katherine M., et al. "Association of depression & health related quality of life with body composition in children and youth with obesity." Journal of affective disorders 172 (2015): 18-23. Mustillo, Sarah, et al. "Obesity and psychiatric disorder: developmental trajectories." Pediatrics 111.4 (2003): 851-859. Halfon, Neal, Kandyce Larson, and Wendy Slusser. "Associations between obesity and comorbid mental health, developmental, and physical health conditions in a nationally representative sample of US children aged 10 to 17." Academic pediatrics 13.1 (2013): 6-13.			

TYPE	SUBTYPES	EXAMPLES	DESCRIPTION	RESOURCE
NOW			Childhood obesity impairs children's physical, mental and social well-being.	<p>Schwimmer, Jeffrey B., Tasha M. Burwinkle, and James W. Varni. "Health-related quality of life of severely obese children and adolescents." <i>Jama</i> 289.14 (2003): 1813-1819.</p> <p>Taylor, Valerie H., et al. "The impact of obesity on quality of life." <i>Best Practice & Research Clinical Endocrinology & Metabolism</i> 27.2 (2013): 139-146.</p>
	Integumentary System	Skin Problems		<p>Nutritional problems of children and adolescents. (2012). In J. Webster-Gandy, A. Madden, & M. Holdsworth (Eds.), <i>Oxford handbook of nutrition and dietetics</i> (p. 409). Oxford: Oxford University Press, USA.</p>
	Other	<p>Impaired social, physical and emotional functioning</p> <p>Mobility and accidents</p>		<p>Morrison, Katherine M., et al. "Association of depression & health related quality of life with body composition in children and youth with obesity." <i>Journal of affective disorders</i> 172 (2015): 18-23.</p>
B) LATER	Physical	<p>Adult obesity and its consequences</p> <p>"Early puberty and tall stature, Gynaecomastia or adipomastia, Oligomenorrhoea and hyperandrogenism"</p>		<p>Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. <i>Pediatrics</i> 2005;115:22-7.</p> <p>Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. Risk factors and adult body mass index among overweight children: the Bogalusa Heart Study. <i>Pediatrics</i>. 2009;123:750-57.</p> <p>Biro FM, Wien M. Childhood obesity and adult morbidities. <i>Am J Clin Nutr</i>. May 2010;91(5):1499S—1505S.</p> <p>Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. Racial differences in the tracking of childhood BMI to adulthood. <i>Obes Res</i> 2005;13:928-35.</p> <p>Endocrine Disorders/Obesity. (2012). In K. M. Goel & D. K. Gupta (Eds.), <i>Hutchison's paediatrics</i> (2nd ed., pp. 438-439). New Delhi, India: Jaypee Brothers Medical.</p>

2.2 THE CONTRIBUTION OF ADVERSE CHILDHOOD EXPERIENCES TO OBESITY

2.2.1 Origins of ACE Study:

ACE study traces its history to an obesity clinic of Kaiser Permanente in San Diego, California. Dr. Vincent Felitti, “who was working Permanente’s revolutionary Department of Preventive Medicine in San Diego, CA, [couldn’t figure out why, each year for the last five years, more than half of the people in his obesity clinic dropped out].” (Redding, 2003; Stevens, 2012).

Jane Ellen Stevens founder of “acestoohigh” further elaborates that clinic was specially designed for those who were 60 to 100 pounds overweight. The preventative medicine department was aiming to diagnose individuals’ diseases at an early stage before they were symptomatic. Roughly 50,000 patients were being screened annually.

It was in 1980 that Dr. Vincent Felitti got puzzled finding that around 50% of the participants dropped out of the obesity clinic, and quite astonishingly he found that those who were losing weight were the ones who left the program initiated for overweight and obesity management. It was the quest for the answer to this question that ACE study made its hallmarking beginnings in the years to come (1995-1997). To find an explanation for this issue the equation to ACE was formed-Dr. Felitti along with 17,000 members of Kaiser Permanente’s San Diego care program and Researchers from CDC (Redding, 2003; Stevens, 2012).

But before the ACE study begun, he wanted to learn more about the demographics and characteristics of dropouts. Quite contrary to customary beliefs, he noticed that “many had been unconsciously using obesity as a shield against unwanted-

sexual attention, or as a form of physical, sexual and emotional protective factor, and that many of them had been sexually and/or non-sexually abused as children.” (Felitti, 1991, 1993).

Moreover, Dr. Vincent Felitti discovers that all of the dropout had normal birth weight and didn’t gain weight gradually over years (V. J. Felitti, 1991) and obesity provided the patients [marital stability] by reducing spousal jealousy (V. J. Felitti, 1993).

It was also noticed that most of the obese patients had prior exposure to precarious health behaviors such as smoking, alcohol, and injected drug use that challenged the prevailing notion of addiction as a substance related phenomenon, but forecasted it as [experience-dependent during childhood] (Vincent J. Felitti, 2003).

These findings were confirmed in the ACE study cohort of over 17,000 health maintenance members (Felitti et al, 1998; R. F. Anda et al., 1999; Hillis, Anda, Felitti, & Marchbanks, 2001; S. R. Dube et al., 2001; Robert F. Anda et al., 2002; Shanta R. Dube, Anda, Felitti, Edwards, & Croft, 2002; Williamson, Thompson, Anda, Dietz, & Felitti, 2002; Shanta R. Dube et al., 2003; Shanta R. Dube et al., 2006; Edwards, Anda, Gu, Dube, & Felitti, 2007; Ford et al., 2011)

2.2.2 Epidemiology/Burden

The ACE study is an ongoing study that was undertaken from 1995 to 1997 to measure the association between childhood maltreatment and its health outcomes later on among 17,000 study participants. It is an ongoing collaborative research between Centers for Disease control and Prevention and Kaiser Permanente’s Health Appraisal-

Clinic in San Diego. This study was aimed to study the association between exposures of different categories of adverse childhood experiences, viz. physical, social or emotional - abuse or physical neglect, family dysfunctions and social problems, and different outcomes simultaneously that are not only the leading causes of death but also predictors of health-related behaviors and poor quality of life in the U.S.

Based on the 2014 Child Maltreatment report series of the U.S. Department of Health and Human Services from the National Child Abuse and Neglect Data System (NCANDS)-there were around 702,000 children who were victims of abuse in 2014, namely, physical (17%), sexual (8.3%), neglect (75%) and psychological maltreatment (6%): White children constituted the majority of the victims (44%) followed by Hispanic (22.7%) and African American (21.4%) (USDHHS, 2016).

However, death rates were 88.4% among white children, 43% among African American, and 15.1 % in Hispanic children. Approximately 1,580 lives were taken as a result of abuse (2.13/100,000 nationally), of which Almost three out each four children were younger than three years of age. Besides, boys had a higher fatality rate compared to girls (2.48 vs. 1.82 per100, 000 in the population), but a lower victimization rate (9.0 vs. 9.8 per 1,000). In addition, among children who died around three-quarters of them had suffered from neglect and 41.3% from physical abuse alone or in combination with other types of maltreatment (USDHHS, 2016).

A cross-sectional study of the National Survey of Children's Exposure to Violence (NatSCEV2) among children aged 1 month to 17 years found that in general that the life victimization of children by a caregiver is 25.6% in the US (Finkelhor, Turner, Shattuck, & Hamby, 2013). Moreover, the results of this study demonstrated that 41,2 % of -

children had suffered from physical abuse, 2 % from sexual abuse, 6.5% from neglect - and 8% from emotional abuse³. This prevalence study indicated that children who were at the oldest subgroup (14-17 years) had highest rate of maltreatment (20.6%); this findings is in opposition to HHS' finding from The National Child Abuse and Neglect Data System (NCANDS)-which indicated that children <1 years are the most vulnerable (24.4%) (USDHHS, 2016).

2.2.3 Risk Factor and Etiology

These adverse Family Experiences that were included in our study to capture psychosocial risk factors that affect children at the individual and familial level with their respective measures were (NSCH, 2011-12):

(1) Socioeconomic hardship: How often has it been hard to get by on your family's income - hard to cover basics like food or housing?

(2) Divorce/separation of parent: Child lived with a parent who got divorced/separated after he/she was born?

(3) Death of parent: Child lived with a parent who died?

(4) Parent served time in jail: Child lived with parent who served time in jail after he/she was born?

(5) Witness to domestic violence: Child saw parents hit, kick, slap, and punch or beat each other up?

³ Among those are 2 years of older

(6) Victim of neighborhood violence: Child was a victim of violence or witness violence in his/her neighborhood?

(7) Lived with someone who was mentally ill or suicidal: Child lived with anyone who was mentally ill or suicidal, or severely depressed for more than a couple weeks?

(8) Lived with someone with alcohol/drug problem: Child lived with anyone who had a problem with alcohol or drugs?

(9) Treated or judged unfairly due to race/ethnicity: Child was ever treated or judged unfairly because of his/her race or ethnic group?

These ACEs were initially developed in the original ACE study (Felitti et al., 1998) which includes ACE2, ACE4, ACE5, ACE7, and ACE8. Remarks from the Technical Expert Panel (TEP)⁴, and of general population, led to the development of four new items in the list of ACEs after review of *life course stressors in children's life*, namely, socioeconomic hardship [ACE1], bereavement [ACE2], witness/victim or neighborhood violence [ACE 6] and racial discrimination [ACE9]).

2.2.4 Prognosis and Consequences

ACEs scoring system was developed to measure their prevalence before age 18 since researchers discovered participants' exposure to multiple categories in their childhood (Table 2.4). It was contended that increase in ACE scores increased the health

⁴ Representative group of experts in the field of survey methodology, children's health, community organizations, and family leaders were members of this TEP

risks for-multiple causes of death -such as alcohol abuse, obesity, physical inactivity, smoking, use of illicit drugs, promiscuity, and suicide attempts.

TABLE 2.4. *Original ACE study-ACE burden, Scores and associated health problems(n=17,337)

ACE burden and Score			Health Problems ⁵
Abuse	Emotional	10.6%	<ul style="list-style-type: none"> • Alcoholism and alcohol abuse • Chronic obstructive pulmonary disease (COPD) • Depression • Fetal death • Health-related quality of life • Illicit drug use • Ischemic heart disease (IHD) • Liver disease • Risk for intimate partner violence • Multiple sexual partners • Sexually transmitted diseases (STDs) • Smoking • Suicide attempts • Unintended pregnancies • Early initiation of smoking • Early initiation of sexual activity • Adolescent pregnancy
	Physical	28.3%	
	Sexual	20.7%	
Neglect**	Emotional	14.8%	
	Physical	9.9%	
ACE score	0	36.1%	
	1	26.0%	
	2	15.9%	
	3	9.50%	
	4 or more	12.50%	

Note:

* Collected between 1995 and 1997, the prevalence (%) presented below are estimated from the entire ACE Study sample (n=17,337). Individual research papers that use only Wave 1 data or Wave 2 data will contain slightly but not significantly different prevalence estimates for individual ACE.

** Collected during wave 2 only (N=8629)

Source: Centers for Disease Control and Prevention, Kaiser Permanente. The ACE Study Survey Data [Unpublished Data]. Atlanta, Georgia: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention; 2016.

Childhood maltreatment is associated with various adverse health outcomes. In a systematic review, childhood maltreatment was related to chronic inflammatory states independent of preexisting health comorbidities (L Daruy-Filho, 2011).

⁵ These health problems increases in strong and graded fashion

ACE study uses a whole life perspective model that considers periods-from conception to death which is illustrated below in figure 2.3 (V. J. Felitti et al., 1998).

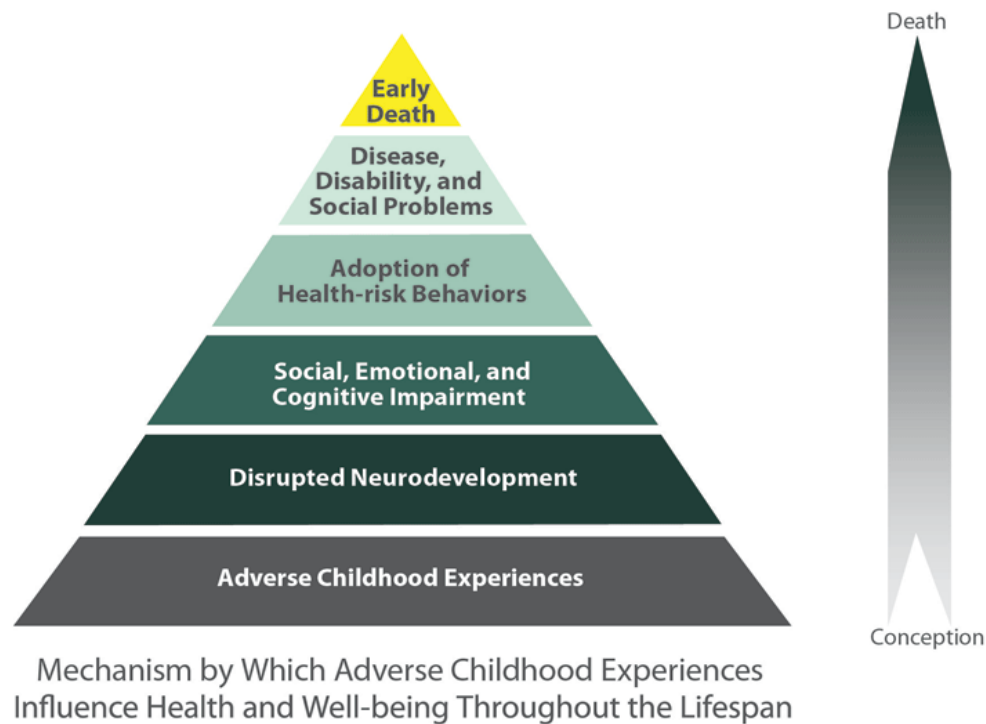


Figure 2.3 - The ACE Pyramid: Conceptual Framework for the ACE Study

Childhood maltreatment not only predisposes children to adverse clinical outcomes proximally but also distally. One of the most staggering consequences of adverse childhood experiences is the economic burden. In a cross-sectional study, Fang, Brown, Florence, & Mercy (2012) argued that the economic difficulties of childhood maltreatment (CM) is comparable to other public health costly conditions, such as stroke and type 2 diabetes. This study found that lifelong cost for all the incidence cases of non-fatal (N=579,000) and fatal (N=1,740) CM in the year 2008 summed up to \$124 billion, that is, by and large [\$210,012] for one who experienced nonfatal CM and [\$1,272,900] for every fatal case of CM (Table 2.5).

The authors studied different types of cost associated with childhood maltreatment depending whether it was non-fatal or fatal child maltreatment:

- The average lifetime cost per victim (in 2010 dollars) of non-fatal child maltreatment:
 - \$32,648 - childhood health care costs (physical and mental)
 - \$10,530 - adult medical costs (physical and mental)
 - \$144,360 - productivity losses
 - \$7,728 - child welfare costs
 - \$6,747 - criminal justice costs
 - \$7,999 - special education costs
 - **Total** = \$210, 012
- The average lifetime cost per victim (in 2010 dollars) of fatal child maltreatment:
 - \$14,100 – medical costs
 - \$1,258,800 – productivity losses
 - **Total** = \$1,272,900

TABLE 2.5. Total lifetime costs of child maltreatment 2008 United States (Based on substantiated cases of child maltreatment)

Source of cost	Total lifetime costs (in 2010 dollars)	
	Discounted at 3%	Discounted at 7%
Nonfatal		
<i>Incidence (cases)</i>	\$579, 000	\$579,000
<i>Short-term health care costs</i>	\$18,903,192,000	\$15,669,477,000
<i>Long-term health care costs</i>	\$6,096,870,000	\$2,193,831,000
<i>Productivity losses</i>	\$83,584,440,000	\$28,410,372,000
<i>Child welfare costs</i>	\$4 474 512,000	\$4,474,512,000
<i>Criminal justice costs</i>	\$3,906,513,000	\$2,234,940,000
<i>Special education costs</i>	\$4,631,421,000	\$3,730,497,000
<i>Total</i>	\$121,596,948,000	\$56,714,208,000
Fatal		
<i>Incidence (cases)</i>	1,740	1 740
<i>Medical costs</i>	\$24,534,000	\$24,534,000
<i>Productivity losses</i>	\$2,190,312,000	\$565,964,580
<i>Total</i>	\$2,214,846,000	\$590,498,580
Total costs (including both fatal and nonfatal cases)	\$123,811,794,000	\$57,304,706,580

Note:

Source: (Fang, Brown, Florence, & Mercy, 2012)

In summary, the authors' urge that based on their findings the costs of child maltreatment are enormous, and it is sagacious and favorable to focus on prevention rather on treatment programs.

2.2.5 Overview of ACES Association with Childhood Obesity:

Meta-analysis of 41 studies has indicated that ACE increases the risk of obesity over the lifespan (odds ratio.1.36; 95% confidence interval.1.26–1.47) (Danese & Tan, 2014). Besides, the experience of Childhood abuse is related to the development of health risk behaviors and diseases among adults (Springs & Friedrich, 1992).

At the macro level, race/ethnicity, SES, and behavioral factors are related “independently and jointly” to childhood and adolescent obesity in the United States (Singh, Kogan, Van Dyck, & Siahpush, 2008).

The first publication of ACE study was “Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults”(V. J. Felitti et al., 1998). A questionnaire was mailed to 13,494 participants that had participated at the baseline of the study for completed medical evaluation. This study had a 70% response rate (9,508). The exposure of interest in this study was adverse childhood experiences: psychological, physical, or sexual abuse; violence against mother; or living with household members who were substance abusers, mentally ill or suicidal, or ever imprisoned.

The authors of this study found a graded statistically significant association ($p < .001$) between ACE and several risk factors that comprise the leading causes of death among adults. For instance, the authors urged that adults who experienced 4 or more ACEs in their childhood compared to those who had none were more at risk of physical inactivity and obesity (1.4 to 1.6 fold), ≥ 50 sexual intercourse partners, 12 fold increase in alcoholism, drug abuse, depression and suicide attempt and two to four fold increase in smoking and poor self-reported health status. Similarly, this study found that as the number of ACEs increase so does the risk of adult diseases presence, namely, IHD, cancer, chronic lung disease, skeletal fractures and liver disease. What is more, this study signified that those adults who experienced multiple ACEs were more likely to have myriad health risk factors.

Another publication from the ACE study was the study by Williamson, Thompson, Anda, Dietz, & Felitti (2002).⁶ The authors investigated the association of childhood abuse and adult weight and risk factors for obesity among sample of 13,177 adults age 19-92 years. The data were collected through a questionnaire asking participants regarding their childhood experiences (prior age 18 years).

The primary predictor variables of the study were four types of child abuse, viz. social, verbal, fear of physical and physical. BMI was measured at the time of their physical examination in the Kaiser Permanente HMO in San Diego California. The results of the study suggested a positive association between the four types of childhood abuse and increase in body weight and risk of obesity in middle age, that is, they were on average 0.6-4kg heavier than adults who didn't experience abuse in their childhood,

⁶ The ACE study sampled all adult members aged ≥ 19 y examined at the clinic during two time periods: August 1995 – March 1996 (wave 1) and June 1997 – October 1997 (wave 2).

8% and 17% of cases of BMI \geq 30 and BMI \geq 40 were attributable to childhood abuse exposure, respectively. Physical and verbal abuses were strongly associated with BMI with a relative risk of 1.4(1.2-1.6) of body mass index \geq 30 for those who experience physical abuse to those who didn't and 1.9 (1.3-2.7) of body mass index of \geq 40 for those who were often verbally abused to those who didn't. Similar to the study by Felitti et al. (1998) this study indicated that violence types aren't mutually exclusive and risk of outcome increases as number and severity of the childhood abuses increased.

In another prospective cohort in the United Kingdom (England, Scotland and Wales) the researchers studied the " Childhood maltreatment and BMI Trajectories to Mid-Adult life (Power, Pinto Pereira, & Li, 2015). This study intended to elucidate how abuse and neglect influences body weight by a life cycle approach at different intervals from 7y-50y (7, 11,16, 23, 33, 45, and 50y) among the birth cohort of 1958 (n~15,000).

The results of the study indicated a positive linear association between physical abuse and BMI gain (~0.006/y for male and ~0.007 for females) and obesity " in males by 1.03 (1.003,1.05) fold/y, from an OR_{adjusted} at 7y of 0.47, increasing to 0.71 at 23y, to 1.25 at 45y and 1.42 at 50y and "For females, the OR for obesity associated with physical abuse increased by 1.04 (1.02,1.06) fold/y from an OR_{adjusted} at 7y of 0.34, to 0.61 at 23y, 1.39 at 45y to 1.67 at 50y". Sexual abuse demonstrated faster BMI gain (~0.0034/y) among females. However, neglect and psychological abuse didn't corroborate a consistent correlation (Power, Pinto Pereira, & Li, 2015).

Whitaker, Phillips, Orzol, and Burdette (2007) in a national study using the data from the Fragile Families and Child Wellbeing Study, in a sample of 2412, 3-year-old children from the birth cohort of 1998 and 2000 from 75 hospitals in 20 cities of 15-

states in the US, examined “the association between childhood maltreatment and obesity among preschool children.” In this study three types of maltreatment-neglect, corporal punishment and psychological aggression were self-reported by mothers. Childhood obesity ($\text{kg}/\text{m}^2 \geq 95^{\text{th}}$ percentile) had statistically significant association with neglect (adjusted OR 1.5, confidence interval, 1.14-2.14) after controlling for socioeconomic status, maternal obesity and birth weight, but neither with psychological-aggression nor corporal punishment. However, the prevalence of psychological aggression and corporal punishment were higher compared to neglect, 84%, 93% and 11% respectively. The results of this study imply that factors that are not directly related to children’s eating habit and activity viz. *neglect*-might increase the risk of childhood obesity. The authors urge that preventing childhood neglect before age 3 may conceivably lead to *healthy weight status*.

Similarly, in another cross-sectional study by Helton & Liechty (2014), using the National Survey of Child and Adolescent Well-being 2 (NSCAW2) data, the relationship between maltreatment and obesity were studied among children age 2 to 17 ($n \sim 2948$). NSCAW is the only nationally representative Child Protective Services (CPS)-investigated-children-survey in the US that is funded federally and comprises child weight measurements. The results of the study indicated higher obesity prevalence among boys compared to girls (30.0% vs. 20.8 %), based on race African American boys were at lower risk compared to white boys (OR = 0.28,95%CI [0.08,0.94]), girls of 2-5 years were at higher risk obesity if they had experienced sexual abuse compared to those who were victims of neglect (OR = 3.54,95%CI [1.01,12.41]).

Besides, among the boys, those who were victims of physical abuse were at lower risk for obesity compared to those who suffered from childhood neglect (OR = 0.24, 95%CI [0.06, 0.99]). The authors concluded that the childhood obesity prevalence among CPS-investigated children using NSCAW II was almost 8 % higher compared to the general population children using NHANES data on BMI-for-age (Ogden & Carol, 2010), that is, 25% and 17% respectively.

The exact mechanism through which ACEs lead to Childhood obesity is yet to be understood. However, in a systematic review by Danese & Tan (2014) the authors found that childhood maltreatment is associated with a chronic inflammatory state, increase proinflammatory cytokines and C-reactive protein, independent of other comorbid factors. However, the authors urged that the precise mechanism by which chronic inflammatory state mediates the association between childhood maltreatment and adverse health consequences (morbidity and mortality) is yet to be understood.

Incest, molestation, and rape have indicated to grave for undesirable health implications in the long run (V. J. Felitti, 1991). In this retrospective cohort study individuals who had given a positive history of sexual abuse during their childhood and adolescence (n~131) were compared to a random control group decades after the incidence. Study participants were chosen after going to their medical records. Both the study group and counterfactual groups were matched by age and sex and were sampled from the same health maintenance organization (HMO).

The findings of this study provide the statistically significant evidence of higher chronic adverse health conditions and outcomes among study group compared to control group. For instance ≥ 10 or more doctor offices visits (DOVs) (22% vs. 6%), $P < .01$, recurrent gastrointestinal distress (64% vs. 39%, $P < .01$), chronic headaches of all types (45% vs. 25%, $P < .05$), Asthma (13 % vs. 8%, $P < .05$), and marital instability, viz. number of times married or divorced ($P = .003$ for marriage; $P < .001$ divorce). The striking outcome of the study was chronic depression decades after an incidence of sexual abuse among the study group. The author contends “ This chronic depression is associated with a strong predisposition to obesity, particularly morbid obesity.”

The “COLEVA PROJECT”, Figure 2.4, which stands for the “Consequences of Lifetime Exposure to Violence and Abuse” has gathered a visual illustration of the effects of child maltreatment on different organs and systems of the body (David McCollum, 2011).

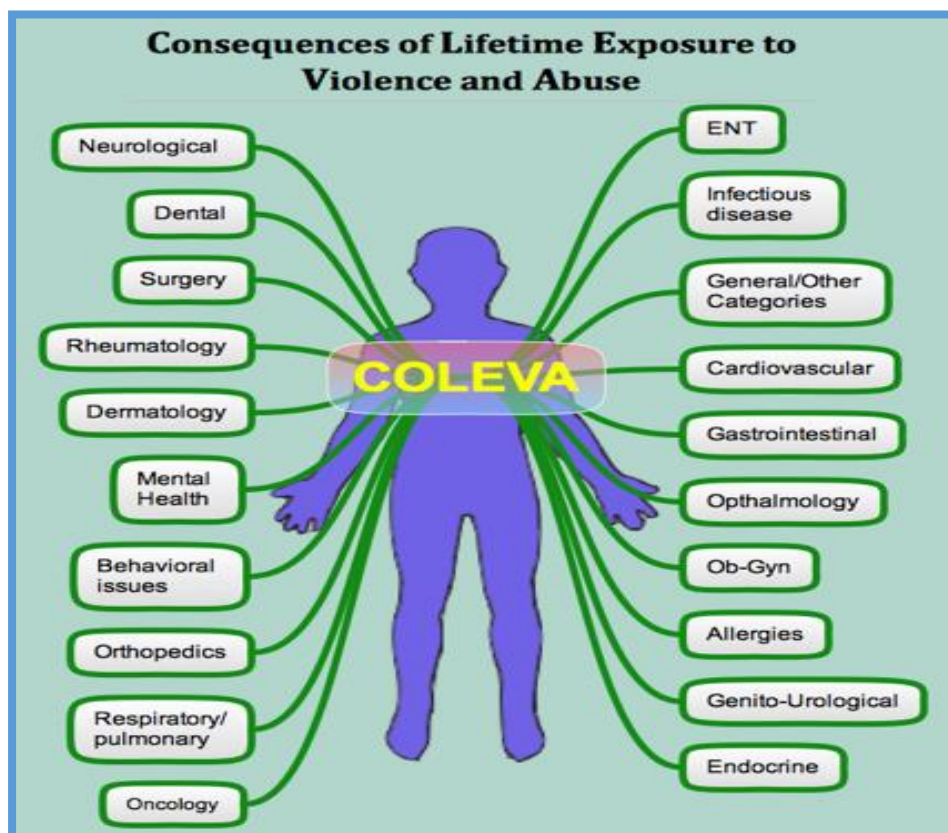


Figure 4.4 - Consequences of Lifetime Exposure to Violence and Abuse

It has been indicated that ACE affects learning/behavior and obesity in low-income urban setting among children (Burke, Hellman, Scott, Weems, & Carrion, 2011). In a cross-sectional study in San Francisco records of 701 subjects from Bayview Child Health Center was looked retrospectively for ACEs to determine its correlation with learning/behavior and obesity issues.

The results indicated a correlation between ACE and increased risk for learning/behavior problems and obesity. The odds of learning/behavior problem among those who had ≥ 1 ACE was statistically significant (OR=10.30[4.66-22.77], $P < .001$) compared to those who had no ACE; however, it wasn't true in the case of overweight and obesity, but for the number of ACEs ≥ 4 the association become statistically significant for both obesity and learning/behavior problems [OR=32 (13.00-81.78), $P < 0.001$] and [OR=2 (1.11-3.55), $P < 0.02$], respectively. Another significant finding from this study was that 45.2% of individuals who were exposed to ACEs ≥ 4 had BMI $\geq 85\%$ compared to 31.3% among those with zero ACEs.

Effects of ACEs on surgical outcomes has also been studied (Lodhia et al., 2015). The sample of this included 223 adult patients, BMI >40 or >35 kg/m² with two or more comorbid conditions, and have undergone any of the three weight loss surgical methods [RYGB (laparoscopic Roux-en-Y gastric bypass), SG (sleeve gastrectomy), or AGB (adjustable gastric band)]. The researchers studied the treatment success rate in a short and long term among those who had prior ACEs to those who didn't.

Astonishingly, the result indicated that patients with ACEs ≥ 6 VS. Low ACEs score had higher values of BMI (were heavier) not only in the short term (6-months) but also in the long run (12-months) after their bariatric surgery (36.9 vs. 33.4 kg/m², $p=0.03$) and (34.5 vs. 30.5 kg/m², $p=0.07$), respectively. Similarly, 12 months after operation individuals with higher ACEs compared to those with low ACEs had higher levels of total cholesterol and low density lipoproteins (191 vs. 169 mg/dL, $p=0.02$) and (116 vs. 94 mg/dL, $p=0.02$), respectively.

Correlation of ACEs and obesity by sex shows different results. Fuemmeler, Dedert, McClernon, & Beckham (2009) studied “disordered eating” by a sample from a nationally representative sample of National Longitudinal Study of Adolescent Health (Add Health) (N~15,197). This cross-sectional study found that only sexual abuse is associated with overweight and obesity only among men [OR=1.66 (1.03-2.70), $p<. 05$] and it was not related to physical abuse and neglect. Childhood physical abuse (CPA), childhood sexual abuse (CSA), and neglect were not associated with overweight or obesity among women. The authors had adjusted for race, parental education level, age, and depressive symptoms. Moreover, the percentage of women who were told by doctor have eating disorder were significant among those who were exposed to childhood sexual abuse, neglect and physical violence compared to those who did not experience these adversities. Women who were physically abused in their childhood were more like to miss their meals (22.3 % vs. 16.6%, $p<. 05$) and afraid to eat because of the fact they might lose control of their weight, compared to those that did not experience physical abuse.

The finding of this study is different from previous studies that indicated a positive association of ACEs among men and women (V. J. Felitti, 1991,1993) and studies merely among women((Alvarez, Pavao, Baumrind, & Kimerling, 2007). Alvarez et al. (2007) found that obesity was statistically significant among women who reported child abuse (adjusted OR_ 1.27, 95% CI_ 1.13–1.40) after adjusting for age, race/ethnicity, education, food insecurity, inadequate fruit and vegetable consumption, physical inactivity, and perceived stress. The study participants were chosen from California Women’s Health Survey (CHWS) who were 18 years and older (N~11,115) and not pregnant.

Family level stressors trigger childhood overweight and obesity among children of different age differently (Garasky, Stewart, Gundersen, Lohman, & Eisenmann, 2009). The researchers studied this topic using nationally representative Child Development Supplement (CDS 2) of the Panel Study of Income Dynamics (PSID) data among children 5-17 years of age. Among children 5-11years “lack of cognitive stimulation and emotional support” and among children 12-17years living in families with higher financial struggles, physical and mental health problems were found to be associated with their overweight and obesity status.

On the other hand, studies have shown that childhood maltreatment is merely correlated with visceral fat mass (L. Li, Chassan, Bruer, Gower, & Shelton, 2015). Researchers at the University of Alabama at Birmingham discovered this in a sample (N~75) of participants recruited by the Office of Psychiatric Clinical Research. Different indicators of body fat mass were used, viz. “body mass index, waist to hip ratio, total body fat, android fat and visceral fat” of which only visceral fat mass was found to be correlated with CM. Dual-energy X-ray absorptiometry measured visceral fat mass.

However, childhood maltreatment subtype analysis of this study indicated a different association between adverse childhood experiences and visceral fat mass comparing CM group (n~37) to Non-CM group. Among CM subtypes (physical neglect, emotional neglect, emotional abuse, physical abuse and sexual abuse) only physical abuse had statistically significant association with visceral fat mass ($r=0.22$, $P=0.04$). The authors categorized the potential mechanism through which CM leads to obesity:

- a) "Suppression of Hypothalamic pituitary axis functioning" leading to decrease in-cortisol availability that subsequently ends up to visceral obesity through pathophysiological mechanisms.

- b) Immune system through hyperactivation of inflammatory markers.

However going through to the literature a study by Schneiderman, Mennen, Negri, & Trickett (2012) indicate an inverse association of maltreatment with "overweight and obesity among maltreated young adolescents". It was found that maltreatment didn't predict the BMI and reduced the odds of having a high BMI in adolescents group.

CHAPTER 3:
METHODS AND PROCEDURES

3.1 STUDY DESIGN AND SAMPLE

The data set used for this cross-sectional study was the 2011-2012 National Survey of Children's Health Indicator Data Set (NSCH, 2011-2012). This cross-sectional telephone survey was administered nationally in the US between February 28, 2011 and June 25, 2012, among households in the 50 states including District of Columbia (DC) with at least one child 0-17 years of age during the time of interview.

CDC's National Center for Health Statistics (NCHS), State and Local Area Integrated Telephone Survey program with the financial support of United States Department of Health and Human Services (DHHS), Health Resources and Services Administration, Maternal and Child Health Bureau conducted it. Besides, specific questions were funded by DHHS, Office of the Assistant Secretary for Planning and Evaluation.

The survey information was collected by *list-assisted random digit dialing* (RDD) of landline telephones coupled with independent RDD sample of cell phone numbers. There were 95,677 Observations, 31,972 from cell phone sample interviews and 63,705 from landline sample interviews, and 637 variables; the detail of sampling strategy is summarized in Figure 3.1.

Only one child was randomly selected in households with more than one child to participate in the survey. Each record contains all interview data for the child and the household in which the child resides, including the child's health and health care, family functioning, parental health, neighborhood and community characteristics, health insurance coverage, and demographics (CDC, 2011-2012 NSCH, FAQs, 2013).

2011/12 National Survey of Children's Health (2011/12 NSCH) Sampling and Survey Administration

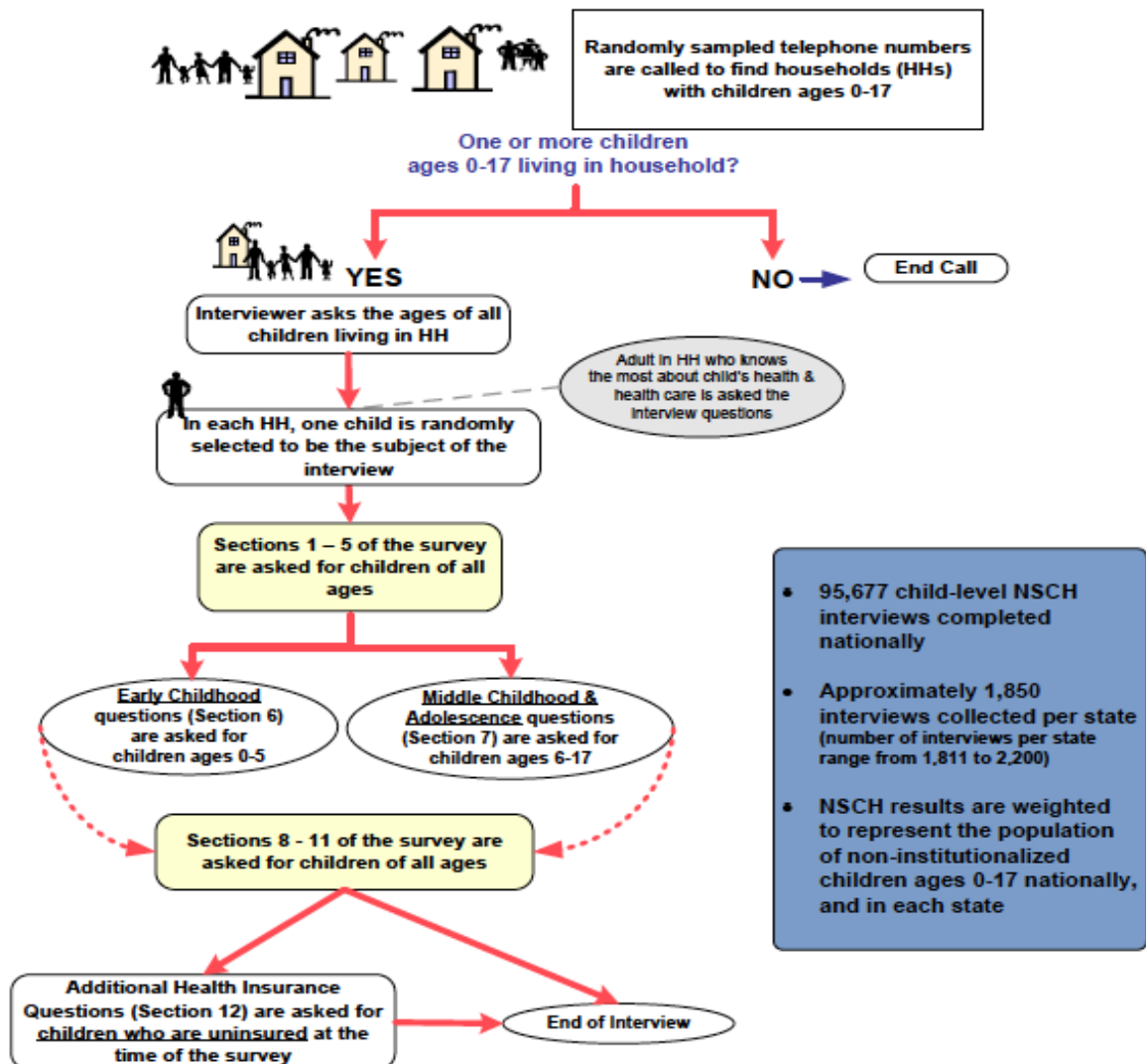


Figure 3.1 - Child and Adolescent Health Measurement Initiative (2012). "2011/12 National Survey of Children's Health (2012), Sampling and Survey Administration." Data Resource Center, supported by Cooperative Agreement 1-U59-MC06980-01 from the U.S. Department of Health and Human Services, Health Resources and Services Administration (HRSA), Maternal and Child Health Bureau (MCHB). Available at www.childhealthdata.org

Respondents of this survey were the mother, father or other relatives who knew about health status of the sampled child. Besides, One child interview weight was produced; hence, the estimates are generalized to all non-institutionalized children 0-17 years of age in the US and each state.

However, the weighted approximates aren't representative of the population of parents, mothers, or pediatric health care providers.

3.2 INCLUSION AND EXCLUSION CRITERIA

For this study we present the analysis of ACEs association with BMI classes for children, specifically childhood obesity (primary outcome) relative to healthy weight for the subpopulation of non-institutionalized U.S. children aged 10- 17 years (N=45,309, 45.16%) in 2011-2012 NSCH.

Eligible households for the NSCH were the households with at least one child aged 0-17 years at the time of telephone interview, and if there were more than one child in the house, only one child was randomly selected for the interview. In the first sample collected during 1st quarter of 2011, households who were contacted by cell phone were considered eligible if they didn't have a landline at house or could not be reached given that they had a landline (CDC, 2011-2012 NSCH, FAQs, 2013).

However, the sample gathered after the 1st quarter of 2011, a [take all approach] considered all respondents eligible (irrespective if their phone type use) if there were any child age 0-17 years in the house.

187,422 households were considered eligible for this survey after screening 847,881 homes in 50 states and DC for children aged 0-17 years. 95,677 were the final sample of age-eligible children who were interviewed from 187,422 households.

But, data collected from age-eligible children from the United States Virgin Islands (USVI) were excluded from this data set and wasn't included in the final analysis.

An interview was complete if the section 6 (for children 0-5 years of age) and section 7 (for children 6 -17 years age) of the questionnaire was completed by the respondents. Thus, the interview completion rate for landline sample was (54.1%) while it was (41.2%) for the cell phone use. The national response rate was 38.2% for the landline sample, 15.5% for the cell phone sample, and 23.0% for the combined dual-frame sample.

32% of all eligible households for the survey also were also qualified for an incentive (ranging from \$11-\$15) to complete the interview. 18,728 households received incentives upon completion of the interview. The NSCH questionnaire was translated into six different languages, namely, English, Spanish, Mandarin, Cantonese, Vietnamese, and Korean. A total of 4,905 interviewers were Spanish language speakers, and 229 of them were Asian-language speakers (CDC, 2011-2012 NSCH, FAQs, 2013).

3.3 DEFINITION OF TERMS/ MEASUREMENT

3.3.1 Adverse Childhood Experiences (ACE) - Main Independent Variable)

Adverse childhood experiences are early life exposures of abuse, neglect, and serious dysfunction that an individual experience in his/her childhood. V. J. Felitti et al., (1998) in the seminal ACE study used the ACE study questionnaire to measure all these categories of adverse childhood experiences-both at the individual and family level. These unfavourable conditions included three subtypes types of abuse (physical, sexual and psychological) and four subtypes of household dysfunctions (household members who were substance abusers, mother or step mother treated violently, mentally ill or suicidal, or ever imprisoned,) (V. J. Felitti et al., 1998).

The first wave of ACE study was conducted between August-November 1995 and January-March 1996, but the second wave was carried out between June-October 1997. The questionnaire of the second wave of ACE study had 2 extra categories of ACEs (S. R. Dube et al., 2001) , viz. emotion neglect and physical neglect which was found to be of importance in previous ACE publication using wave 1 (V. J. Felitti et al., 1998; Dietz et al., 1999).

In the original Adverse Childhood Experience (ACE) Study, there were significant associations between childhood abuse (except neglect which was not included in wave 1 of the ACE Study), and exposure to violence with adult health problems (Felitti et al., 1998).

An expanded list of definitions for the nine adverse childhood experiences was developed for the National Survey of Children's Health (NSCH 2011-2012; Section 9, subdomain 5) to capture psychosocial risk factors that affect children, namely, (1) socioeconomic hardship, (2) parental divorce or separation, (3) child lived with a parent who died (bereavement) (4) incarcerated household member, (5) witness to domestic violence, (6) victim/witness of neighborhood violence, (7) lived with someone who was mentally ill or suicidal, (8) substance abuse in the household, (9) treated or judged unfairly due to race/ethnicity (racial discrimination). These Nine adverse Family Experiences are summarized in Table 3.1 (2011-2012 NSCH).

TABLE 3.1. Adverse Childhood Experiences among children in the NSCH 2011/12. ACES definitions are based on after child's birth.⁷

Socioeconomic Hardship	Since the [CHILD] was born, how often has it been very hard to get by on your family's income – hard to cover the basics like food or housing? Would you say very often, somewhat often, often, rarely, or never?
Parental Divorce Or Separation	Did the [CHILD] ever live with a parent or guardian who got divorced or separated after [CHILD] was born?
Bereavement	Did the [CHILD] ever live with a parent or guardian who died?
Incarcerated Household Member	Did the [CHILD] ever live with a parent or guardian who served time in jail or prison after [CHILD] was born?
Witnessing Domestic Violence	Did the [CHILD] ever see or hear any parents or adults in (his/her) home slap, hit, kick, punch, or beat each other up?
Witnessing Neighborhood Violence	Was the [CHILD] ever the victim of violence or witness any violence in (his/her) neighborhood?
Household Mental Illness	Did the [CHILD] ever live with anyone who was mentally ill or suicidal, or severely depressed for more than a couple of weeks?
Household Substance Abuse	Did the [CHILD] ever live with anyone who had a problem with alcohol or drugs?
Racial Discrimination	Was the [CHILD] ever treated or judged unfairly because of (his/her) race or ethnic group?

Note:

Variable Name: Adverse family experiences

ACE Categories: ACE1; ACE2; ACE3; ACE4; ACE5; ACE6; ACE7; ACE8; ACE9;

Denominator: Children age 10-17 years

Numerator: It can take various values based on the aim for the analysis type; Children with no adverse family experiences; Children with 1 adverse family experience; Children with 2 or more adverse family experiences

Source: 2011/12 National Survey of Children's Health. Maternal and Child Health Bureau in collaboration with the National Center for Health Statistics. 2011/12 NSCH [SAS] Indicator Data Set prepared by the Data Resource Center for Child and Adolescent Health, Child and Adolescent Health Measurement Initiative. www.childhealthdata.org

⁷ All these ACE numbers were recoded as original survey entry didn't have ACE2 (coded as ACE1; ACE3-ACE10)

As mentioned earlier, these ACEs were initially developed in the original ACE study (Felitti et al., 1998) which includes ACE2, ACE4, ACE5, ACE7, and ACE8. Remarks from the Technical Expert Panel (TEP)⁸, and of general population, led to the development of four new items in the list of ACEs after review of *life course stressors in children's life*, namely, socioeconomic hardship [ACE1], bereavement [ACE2], witness/victim or neighborhood violence [ACE 6] and racial discrimination [ACE9]) (NSCH, 2011-2012).

ACE2-9 are dichotomous 'Yes/No' response options, but socioeconomic hardship had: (1) VERY OFTEN (2) SOMEWHAT OFTEN (3) RARELY (4) NEVER (77) DON'T KNOW (99) REFUSED options. A response of 'somewhat often' or 'very often' was coded as an adverse family experience (NSCH, 2011-2012).

3.3.2 Childhood Obesity (Main Outcome)

The most widely used method for measuring obesity and overweight is the body mass index (BMI), which is an individual's weight in kilograms divided by his/her height in squared meters (Garrow & Webster, 1985). The Quetelet Index for body mass index in adults (BMI), W/H^2 , is one of the convenient and reliable methods to measure body fat. Body mass index (BMI) is the recommended method of body fat screening among children and adolescents (Kuczmarski et al., 2000; Krebs, Jacobson, & American Academy of Pediatrics Committee on Nutrition, 2003; Koplan, Liverman, Kraak, & Committee on Prevention of Obesity in Children and Youth, 2005; "Obesity," 2000).

⁸ Representative group of experts in the field of survey methodology, children's health, community organizations, and family leaders were members of this TEP

Similarly, It is the prescribed method of body fat screening by the American Medical Association, the American Academy of Pediatrics, and International Task Force on Obesity (Himes & Dietz, 1994). BMI has been found to be a good indicator of body growth from childhood to adulthood (Freedman, Khan, Dietz, Srinivasan, & Berenson, 2001).

Individuals who have a BMI of 30 or more are considered obese, and those with BMI of 25 or more are considered overweight. However, BMI for children and Teenagers are sex and age specific due to the fact their body is progressively growing as they age and varies based on their sex, that is, a children or teen whose BMI fall between 85th percentile and below 95th percentile is considered overweight, and at or above 95th percentile is defined as obese among children and teens of the same age and sex (CDC, 2015).

Also, while assessing body fat further individual anthropometric (such as skinfolds and girth measurements) and other techniques of body fat measurements that considers the triads of diagnosis (medical history, laboratory examination and physical examination) needs to be reviewed, urged by Kohn & Booth (2003).

Similarly, obesity should not merely be defined by adipose tissue as there are individuals who are muscular and based on the arbitrary measures of body weight might be classified as overweight and obese without having excess body fat (D. L. Longo, A. S. Fauci, D. L. Kasper, S. L. Hauser, J. L. Jameson, & J. Loscalzo, 2013).

However, the “Expert Committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity” suggests that BMI by sex at a given age for children and adolescents needs to be plotted on - growth charts and be used routinely by physician and [allied] health care providers compared to skinfold thickness and waist circumference methods (Barlow & Expert Committee, 2007).

Similarly, in an evidence synthesis study by (Whitlock, Williams, Gold, Smith, & Shipman, 2005) found that identification of overweight older teens by BMI measurement method helps to identify individuals who have higher probability of developing obesity in their adulthood. The researchers argued that despite the fact that BMI relatively measures body weight to one’s height, and it does not directly measure an individual’s body fat level, it is the most common measurement method of total body adipose tissue not only among children but also adults.

The researchers affirmed the clinical validity of BMI to be [moderate or very well] when it was used to track adult obesity among those who were classified as overweight by this measure in their childhood and adolescents and lived with ≥ 1 obese parent. Moreover, the authors asserted that children >13 years who are obese (BMI \geq 95th percentile for age and sex) are $\geq 50\%$ at risk of developing adult obesity (BMI >30 kg/m²); therefore, treatment at this period may plummet the risk.

However, the authors urge that, the question that whether those who are classified as being either overweight or obese through BMI has increased body fat, or *fat-free mass* (muscle, bone, and fluids) needs to be scrutinized. Therefore, the credibility of BMI-based body fat categorization can be questioned among individuals of

different race/ethnicity due to their *unique body composition* with age, gender, sexual maturation, etc. Correspondingly, it was recommended that children who are less than 13 years old and do not have *clinical weight-related comorbidities* be labeled as at risk or high-risk as per their BMI with reduced emphasis on classifying them as overweight. It is sagacious to use BMI among this group of children as growth monitoring tool for that given age and sex. The summary of evidence compiled from this study indicated that screening of children under the age 12 or 13, compared to those who are above that age, is not a good predictor of risk of adult obesity. Besides, treatment of overweight adolescents is unusual and not shown to be clinically significant.

Nonetheless, recently K. Li et al. (2016) contended that irrespective of which adolescent weight criteria is used, that is, CDC growth charts, WHO growth references and International Obesity Task Force (IOTF) standards, they yield almost identical associations and more than 90% of agreement in weight assessment and classification of adolescents.

There are different classifications for obesity depending on its distribution and mechanisms of pathophysiology. Obesity based on distribution of adipose tissue in the body can be classified into: a) Central obesity (abdominal, visceral, android or apple-shaped) due to increasing intra-abdominal fat tissue in the body, b) Generalised obesity (gynoid or pear-shaped) as a result of subcutaneous fat stock in body (B. R. Walker, N. R. Colledge, S. H. Ralston, & I. D. Penman, 2014).

Chatterjea, M. N., & Shinde, R. (2012) based on mechanisms of pathophysiology involved in advent of obesity has classified it into different categories:

1. Immediate causes that lead to positive energy balance:

- Exogenous: due to gluttony and overeating coupled with less physical activity
- Endogenous: Endocrinal, metabolic and hypothalamic lesion

2. Pathologically:

- a. Hyperplastic: This is the life-long type of obesity that results from increase not only in fat cells size but also their number. The size of adipocytes might decrease after treatment; however, the number will remain high. Their distribution can be peripheral and central and doesn't have a good therapeutic response.
- b. Hypertrophic: This is adult onset type that renders adipocytes to have merely greater size. They have a central fat distribution and unlike hyperplastic has a good response to treatment.

Therefore, in our study BMI for the same sex and age (10-17 years) percentile relative measurement among children and teens were used as indicators of BMI by using growth charts recommended by CDC (Kuczmarski et al., 2000). BMI for children and Teenagers are sex and age specific due to the fact their body is progressively growing as they age and varies based on their sex. The gender specific BMI-for-age is classified into four categories, children or teen whose BMI fall between 85th percentile and below 95th percentile is considered overweight, and at or above 95th percentile is defined as obese among children and teens of the same age and sex; summarized in Table 3.2 (CDC, 2015).

TABLE 3.2. BMI-CLASS for Children of the same sex and age

Weight Status Category	Percentile Range
Underweight	Less than the 5th percentile
Normal or Healthy Weight	5th percentile to less than the 85th percentile
Overweight	85th to less than the 95th percentile
Obese	95th percentile or greater

BMI levels estimation for children aged 10-17 years are based on the parents self-reports.⁹ The BMI for children less than 10 years of age is not reported in 2011-2012 NSCH.

3.3.3 COVARIATES

Covariates were selected at different levels based on the literature a priori and during analysis; they are summarized in table 3.3.

Parental Factors:

- *Physical health status of child's mother:* It was based on the question “ Of children living with fathers in the household, in general, what is the mother’s physical health status?” There responses were coded into the Linkert scale of 1 (Excellent or very good), 2 (good) and 3 (fair or poor). The respondent of this question was the child’s mother (biological, step, foster, adoptive) who rated herself her overall physical health status. If the mother wasn’t at home, but was living in the household, the father or other relatives were the respondents of the mother’s physical health status. Whether the respondent was the mother herself, or any other family member other than the child’s mother their responses were combined.

⁹ BMI measurements (child’s height and weight) were not independently ascertained by measurements, health records or, etc.

- *Physical health status of child's father:* It was based on the question “ Of children living with fathers in the household, in general, what is the father’s physical health status?” There responses were coded into the Linkert scale of 1 (Excellent or very good), 2 (good) and 3 (fair or poor). The respondent of this question was the child’s father (biological, step, foster, adoptive) who rated himself his general physical health status. If the father wasn’t at home, but was living in the household, the mother or other relatives were the respondents of the father’s physical health status. Whether the respondent was the father himself, or any other family member other than the child’s mother their responses were combined.

*Perinatal Factors And Infancy:*¹⁰

- *Low birth weight,* that is, child weighed less than 2500 grams when they were born. It was coded as 1 (Child was born with low birth weight (<2500g) and 2 (Child had normal birth weight).
- *Prematurity,* that is, birth of the child 3 weeks or more before his or her due date. It was structured as 1(child was born premature) and 2 (child was not born premature).

Sociodemographic Factors:

- *Age* (was not normally distributed, so it was categorized into three different age groups: 10-11 years, 12-14 years, 15-17 years).
- *Race/Ethnicity* (race/ethnicity classification was nominal): It was arranged as Hispanic, White non-Hispanic, Black non-Hispanic and Multiracial/other, non-Hispanic. The other race group comprised of non-Hispanic children who were either

¹⁰ “22 completed weeks (154 days) of gestation and ends seven completed days after birth (WHO)”

multiracial or belonged to other distinctive categories, that is, Asian, American Indian or Alaska Native, and Native Hawaiian or Other Pacific Islander).¹¹

- *Gender of the child* was dichotomous variable: Male and Female
- *Place of residence was measured as a dichotomous variable*: Child living in Metropolitan Statistical Area (MSA), only in states that met the 500,000 thresholds vs. child living outside of MSA. Data for MSA status was only available for 36 states and the remaining 15 states with missing details for MSA included: AK, CT, DE, HI, ID, MA, MD, MT, ND, NH, NV, RI, SD, VT, WY, and ME.
- *Family eats together*: It was based on the question “how many days of the week members of the household had a meal together during past week?” It was coded as an open-ended question, where responses could range from 0-7 days and it for analysis it was categorized into four categories (No days, 1-3 days, 4-6 days, Everyday). This variable was used as a proxy for healthier food choices. It is conjectured that children who eat meals more often in a week with family members have lower odds for childhood obesity compared to those who doesn’t eat any meal in the week with members of the family in the household.

Childhood Factors:

- *Ill health*: It was based on the question “How many children currently have one or more chronic health conditions from a list of 18 conditions?” which was coded as 0, 1, and ≥ 2 . These chronic health conditions from the list of 18 conditions asked about comprised: learning disability, depression, anxiety problems, behavioral or conduct problems, autism or autism spectrum disorder, developmental delay, intellectual-

¹¹ Race/ethnicity was not treated as a covariate as it would lead to bias estimation of Racial discrimination ACE category of childhood obesity, it was merely used for to show its frequency distribution by sex

disability, cerebral palsy, speech problems, asthma, diabetes, Tourette Syndrome, epilepsy or seizure disorder, hearing problems, vision problems, bone or joint problems, brain injury or concussion and ADD.

These chronic non-communicable comorbidities were coded as positive responses, if the respondents mentioned that the selected child currently has the condition after they responded positively that the listed condition was ascertained by health care professional. The positive reply to question, “whether they have ever been told by a health care professional that the child has the condition?” doesn’t not indicate current prevalence. It is noteworthy that all the responses regarding the child’s health condition were solely based on parent’s reports.

- *Physical activity* It was based on the question “During the past week, on how many days did [child name] exercise, play a sport, or participate in physical activity for at least 20 minutes that made [him/her] sweat and breathe hard?” The responses were assembled in four levels (0 days; 1-3 days; 4-6 days and Everyday)
- *Time spent watching TV, videos, or playing video games:* This variable response was based on the question “On an average weekday, about how much time does the selected child usually spend in front of a TV watching TV programs, videos, or playing video games?” The responses include four options (none; 1 hour or less; more than 1 hour but less than 4 hours; 4 hours or more).
- *Time spent with a computer, cell phone, or electronic device:* It was based on the question “On an average weekday, about how much time does [child name] usually spend with computers, cell phones, handheld video games, and other electronic devices?” The responses were compiled into four categories (Does not use electronic

devices; Uses devices 1 hour a day or less; Uses devices more than 1 hour but less than 4 per day; Uses devices more than 4 hours per average weekday).

- *Adequate amount of sleep:* The question for this variable in the survey was “ Nights in the previous week on which children had adequate sleep for their age?” This question was open-ended type and respondents’ answer could range from 0 to 7, that is, the numerator options included four groups (no nights; 1-3 nights; 4-6 nights; every night).

TABLE 3.3. Glossary And Explanation Of Variables (2011-2012 NSCH)

CONSTRUCT	MEASUREMENT	VALUE	LABEL	MISSING
AGE CATEGORIES*	How many children/youth of different ages are in the 10-17 year old population? (3 Groups)	1 = 10-11 years old 2 = 12-14 years old 3 = 15-17 years old	U.S. children in 3 age groups	Legit skip =< 10 years of age
SEX*	How many males and females is in the 10-17 year old population?	1 = Male 2 = Female	Sex of child	. M = Don't know Refused
RACE/ETHNICITY	How many children of different races or Hispanic ethnicity are in the 10-17 year old U.S. populations? Non- Hispanic children reporting only one race category of Asian, American Indian, Alaska Native, Native Hawaiian, or Pacific Islander, or who are more than one race (multi-racial) are grouped as Other, non-Hispanic because of small sample sizes in many states. Non-Hispanic children who reported more than one race are categorized as Other, non-Hispanic.	1 = Hispanic 2 = White, non-Hispanic 3 = Black, non-Hispanic 4 = Multiracial/Other, non-Hispanic	Race and ethnicity distribution of the child population	. M = DK/Ref/Missing in error to HISPANIC or RACER or both
HOUSEHOLD POVERTY STATUS	How many children live in households with incomes above/below the federal poverty level (FPL)? Derived. Household income level based on DHHS guidelines - Imputed; single imputation using version 3	1 = < 100% FPL 2 = 100-199% FPL 3 = 200-399% FPL 4 = 400% or more FPL	Income level of child's household	
BMICLASS	What is the weight status of children based on Body Mass Index (BMI) for age? 4 categories Assessment of body fat in children and teenagers is approached differently than for adults. Children's body fat composition changes as they grow, and growth patterns are different for boys and girls. Consequently, measurement of body mass for children, known as BMI-for-age, is age and gender specific BMI-for-age. In the NSCH BMI-for-age is based on parents' recollections of the selected child's height and weight.	1 = Underweight-less than 5th percentile 2 = Healthy weight-5th to 84th percentile 3 = Overweight -85th to 94th percentile 4 = Obese - 95th percentile or above	Childhood weight status in 4 categories, age 10-17 years	M = Missing in error . N = Skip: Less than 10 yrs.
ACE*	Child experienced any Adverse Childhood Experiences for child, of 9 asked about? A response of 'somewhat often' or 'very often' was coded as an adverse family experience for ACE1. ACE2-19 are dichotomous 'Yes/No' response options.	ACE1 = Socioeconomic Hardship ACE2 = Parental Divorce or Separation ACE3 = Bereavement ACE4 = Incarcerated Household Member ACE5 = Witnessing Domestic Violence ACE6 = Witnessing Neighborhood Violence ACE7 = Household Mental Illness ACE8 = Household Substance Abuse ACE9 = Racial Discrimination	Adverse Childhood Experiences	. M = DK/Ref/Missing in error/Partial interview
ACESCORE*	Child experienced how many of the Adverse Childhood Experiences for child, of 9 asked about?	0 = Child experienced no adverse family experiences, of 9 asked about 1 = Child experienced one adverse family experience 2 = Child experienced two or more adverse family experiences	ACE Score	. M = DK/Ref/Missing in error/Partial interview
SOCIOECONOMIC HARDHSIP*	How often has it been hard to get by on your family's income - hard to cover basics like food or housing?	0 = Never/Rarely hard to get by on family income 1 = Somewhat Often/Very Often hard to get by on family income	ACE1- Socioeconomic Hardship	. M = DK/Ref/Missing in error/Partial interview
PARENTAL SEPARATION OR DIVORCE*	Child lived with parent who got divorced/separated after he/she was born?	0 = Child did not experience the Adverse Family Experience 1 = Child experienced the Adverse Family Experience	ACE2-Parental Separation or Divorce	. M = DK/Ref/Missing in error/Partial interview
BEREAVEMENT*	Child lived with parent who died?	0 = Child did not experience the Adverse Family Experience 1 = Child experienced the Adverse Family Experience	ACE3- Bereavement	. M = DK/Ref/Missing in error/Partial interview;
INCARCERATED HOUSEHOLD MEMBER*	Child lived with parent who served time in jail after he/she was born?	0 = Child did not experience the Adverse Family Experience 1 = Child experienced the Adverse Family Experience	ACE4- Incarcerated Household Member	. M = DK/Ref/Missing in error/Partial interview

WITNESSING DOMESTIC VIOLENCE*	Child saw parents hit, kip, slap, punch or beat each other up?	0 = Child did not experience the Adverse Family Experience 1 = Child experienced the Adverse Family Experience	ACE5- Witnessing Domestic Violence	. M=DK/Ref/Missing in error/Partial interview
WITNESSING NEIGHBORHOOD VIOLENCE*	Child was a victim of violence or witness violence in his/her neighborhood?	0 = Child did not experience the Adverse Family Experience 1 = Child experienced the Adverse Family Experience	ACE6- Witnessing Neighborhood Violence	. M=DK/Ref/Missing in error/Partial interview
HOUSEHOLD MENTAL ILLNES*	Child lived with anyone who was mentally ill or suicidal, or severely depressed for more than a couple weeks?	0 = Child did not experience the Adverse Family Experience 1 = Child experienced the Adverse Family Experience	ACE7- Household Mental Illness	. M=DK/Ref/Missing in error/Partial interview
HOUSEHOLD SUBSTANCE ABUSE*	Child lived with anyone who had a problem with alcohol or drugs?	1 = Child experienced the Adverse Family Experience	ACE8- Household Substance Abuse	. M=DK/Ref/Missing in error/Partial interview
RACIAL DISCRIMINATION*	Child was ever treated or judged unfairly because of his/her race or ethnic group?	0 = Child did not experience the Adverse Family Experience 1 = Child experienced the Adverse Family Experience	ACE9-Racial Discrimination	. M=DK/Ref/Missing in error/Partial interview
PLACE OF RESIDENCE*	Child is living in a Metropolitan area? Metropolitan Statistical Area (Only in states that meet the 500,000 threshold). The MSA status information is available for the 35 states in which the population is at least 500,000 in both categories (MSA and non-MSA). "Metropolitan statistical areas (MSA) are geographic entities defined by the Office of Management and Budget (OMB) using published standards that are applied to Census Bureau data. An MSA is a county or group of contiguous counties that contains at least one urbanized area of 50,000 or more population. In addition to the county or counties that contain all or part of the urbanized area, an MSA may contain other counties that are economically and socially integrated with the main city as measured by work commuting." For more information, please visit http://www.census.gov/population/www/metroareas/metrodef.html .	1- Located within Metropolitan Statistical Area (MSA) 2- Located outside of a Metropolitan Statistical Area (MSA)	Metropolitan Statistical Area	L-legitimate skip . M= Missing in Error Data for MSA status was Only available for 36 states and missing states include; AK, CT, DE, HI, ID, MA, MD, MT, ND, NH, NV, RI, SD, VT, WY, and ME.
PHYSICAL HEALTH STATUS OF MOTHER*	Of children living with mothers in the household, in general, what is the mother's physical health status? If the survey respondent was the selected child's mother (biological, step, foster, adoptive), she rated her own physical health. Respondents who were not the child's mother (e.g. father or other relative) gave a rating of the mother's physical health if the mother was living in the household. All responses were combined regardless of whether the person answering was the mother herself or another respondent.	1 = Excellent/very good 2 = Good 3 = Fair/poor	Physical health status of child's mother	. M=DK/Ref/missing in error/partial interview
PHYSICAL HEALTH STATUS OF FATHER*	Of children living with fathers in the household, in general, what is the father's physical health status? When the respondent was the target child's father (biological, step, foster, adoptive), he rated his own physical health. Respondents who were not the target child's father (e.g. mother or other relative) gave a rating of the father's physical health. All responses were combined regardless of whether the person answering was the father himself or another respondent.	1 = Excellent/very good 2 = Good 3 = Fair/poor	Physical health status of child's father	. M=DK/Ref/missing in error/partial interview
FAMILY EATS TOGETHER*	During the past week, on how many days did all the family members who live in the household eat a meal together?	0 = No days 1 = 1-3 days 2 = 4-6 days 3 = Every day		. M=DK/Ref/Missing in error/Partial interview

LOW BIRTH WEIGHT*	How many children had a low birth weight? That is, they weighed less than 2500 grams when they were born?	1 = Child was born with low birth weight (<2500g) 2 = Child had normal birth weight . M = Missing	Children born at low birth weight	. M= Missing
PREMATURTIY*	Children were born premature, that is 3 weeks or more before his or her due date?	0 = No, child was not born premature 1 = Yes, child was born premature	Children born premature	. M=DK/Ref/Missing in error
PHYSICAL ACTIVITY*	How many days during the past week did [child name] exercise, play a sport, or participate in physical activity for at least 20 minutes that made [him/her] sweat and breathe hard?	1 = 0 days 2 = 1-3 days 3 = 4-6 days 4 = Everyday	Physical activity, age 10-17 years	. M=DK/Ref/Missing in error . L = Legit Skip: age less than 10 yrs.
AMENITIES*	Children live in neighborhoods that contain certain amenities -- parks, Recreation centers, sidewalks or libraries?	Park Recreation center Sidewalk Library	Presence of neighborhood amenities	. M=DK/Ref/Missing in error/Partial interview to any of the questions
TELEVISION WATCHING, VIDEOS OR PLAYING VIDEO GAMES*	On an average weekday, about how much time does [child name] usually spend in front of a TV watching TV programs, videos, or playing video games?	0 = Does not watch T 1 = Watches TV 1 hour or less per day 2 = Watches TV more than 1 hour but less than 4 hours per day 3 = Watches TV 4 hours or more per day	Time spent watching TV, videos, or playing video games, age 10-17 years	. M=DK/Ref/Missing in error/Does not own TV . L = Legit Skip: Child is less than 10 year of age
COMPUTER, CELL PHONE, OR ELECTRONIC DEVICE*	On an average weekday, about how much time does [child name] usually spend with computers, cell phones, handheld video games, and other electronic devices, doing things other than school work, age 10-17 years	0 = Does not Use Electronic Devices 1 = Uses Electronic Devices 1 hour or less per day 2 = Uses Electronic Devices more than 1 hour but less than 4 hours per day 3 = Uses Electronic Devices 4 hours or more per day	Time spent with a computer, cell phone, or electronic device, age 6-17 years	. M=DK/REF/Missing in error/Does not own Electronic Devices . L = Legit Skip: Child is less 10 years
ADEQUATE AMOUNT OF SLEEP*	During the past week, on how many nights did [child name] get enough sleep for a child [his/her] age?	0 = No nights 1 = 1-3 nights 2 = 4-6 nights 3 = Every night	Adequate amount of sleep, age 10-17 years	. M=DK/Ref/Missing in error/Partial interview . L = Legit skip: age 0-9 years
ILL HEALTH (CHRONIC HEALTH CONDITIONS FROM A LIST OF 18 CONDITIONS)*	How many, children currently have (none, one or more) chronic health conditions from a list of 18 conditions? Learning disability, ADD or ADHD, depression anxiety problems, Behavioral or conduct problems, autism or other autism spectrum disorder, developmental delay, intellectual disability, cerebral palsy, speech problems, asthma, diabetes, Tourette Syndrome, epilepsy or seizure disorder, hearing problems, vision problems, bone or joint problems, brain injury or concussion. For each condition, conditions were named and parents were asked to respond for each condition whether they had ever been told by a health professional that their child had the condition. If yes, they were asked whether the child currently had the specific condition. Children were then grouped according to the number of conditions they currently have: none, one, or two or more.	1 = Does not have any current chronic health conditions from the list of 18 asked 2 = Currently has 1 chronic health conditions from the list of 18 asked 3 = Currently has 2 or more chronic health conditions from the list of 18 asked	Children with 1 or more current chronic health conditions	. M=DK/Ref/Missing in error to all

Notes:

*Indicates covariates included in final model

Source:

-2011/12 National Survey of Children's Health. Child and Adolescent Health Measurement Initiative (CAHMI), "2011- 2012 NSCH: Child Health Indicator and Subgroups SAS Codebook, Version 1.0" 2013, Data Resource Center for Child and Adolescent Health, sponsored by the Maternal and Child Health Bureau. www.childhealthdata.org.

-Glossary of Terms--Data Resource Center for Child and Adolescent Health. (n.d.). Retrieved June 29, 2016, from <http://childhealthdata.org/help/glossary>

3.4 STATISTICAL ANALYSIS

The analysis was performed using the Statistical Analysis System (SAS) software (v9.3; Cary, NC: SAS Institute Inc. 2011), specially SAS survey procedures [SURVEYMEAN, SURVEYFREQ, AND SURVEY LOGISTICS, which considers the complexity of survey sample design during analysis, on the sample data of children aged (10-17years) in the National Survey of Children's Health (NSCH, 2011/12). We used Numbers (version 3.6.2 [25771], U.S.: Apple Inc. 2008-2015) and Infographics (version 2.7 [2534], U.S.: Jumsoft. 2016) computer soft wares for graphical display of the results.

CDC'S National Center for Health Statistics (NCHS), State and Local Area Integrated Telephone Survey Program conducted the survey. Hence, the data set has already been checked and thoroughly cleaned, and sampling weights were calculated to get national and state specific statistics. Besides, sampling weights are adjusted for potential non-response biases and non-coverage of non-telephone households. Sub setting of data set (e.g., SAS sub setting if statement and deleting unneeded records) was not performed as it was urged that it would remove completely the [primary sampling units] from the sample design; and to precisely estimate standard errors the software should have the complete observation in the sample (CDC, 2011-2012 NSCH, FAQs, 2013).

Therefore, domain statements were used to analyze the weighted results of children aged 10-17 year from study sample of the 0-19 years old.

Besides, variables with missing values were set to “.”. There were unique [missing data codes] in order distinguish different types of missing data in the dataset (CDC, 2011-2012 NSCH, FAQs, 2013):

- (.A) Added question—A variable was missing when it was added after completion of interview or after data collection had begun.
- (.L) Legitimate skip—A Variable was considered missing if it was already answered by a [root question] in the questionnaire.
- (.M) Missing in error—A variable was regarded as missing because of the system, interviewer errors or when the answer for a variable was not available in the questionnaire (blank)
- (.N) Not in universe—A Variable was missing when the child was not eligible for a question at any section of the questionnaire, (for example, children ages 0-5 were not eligible for section 7 and children ages 6-17 were not eligible for section 6).
- (.P) Partially completed interview—Variable was missing because the respondent ended the interview after completing Sections 6 or 7 (depending on the age of the child) but before completing the full interview.
- Missing data because the respondents of the survey refused to answer or did not know the answer was coded differently. [Don't Know] was coded as DK = 6, 96, 996, 9996 and [Refused] answers was coded as RF = 7, 97, 997, 9997.

3.4.1 Characteristics of the Study Population By Sex, ACE Status, BMI-CLASS and State:

To take into consideration the complex sampling design of our study sample, SAS SURVEY procedures were used to get weighted, unbiased, representative population parameter estimates. The SAS SURVEYFREQ and SURVEYLOGISTIC procedures were utilized with and without stratifications to estimate the weighted prevalence and odds ratio estimates, respectively.

In the Introductory analysis, SAS SURVEYFREQ procedures were used with and without stratifications to detect the weighted prevalence estimates. More precisely, these procedures were used:

- a) To assess the distribution (weighted column % and unweighted column frequencies) of primary exposure variable (ACE exposure=Yes and ACE exposure=No), ACE Scores (0, 1 or ≥ 2), each category of ACE, and selected socio-demographic factors by gender of the study participants among children 10-17 years old (N=45,309).
- b) To estimate the likelihood of BMI class frequency distribution (especially childhood obesity [primary outcome of interest]) with selected covariates,
- c) To detect the likelihood of ACE exposure (primary predictor variable) prevalence with selected covariates including outcome variable (obesity [primary outcome of interest], overweight, healthy weight and underweight).

Besides, The SAS SURVEYFREQ procedures were used to produce: one-way, two-way and multi-way cross tabulation weighted percentages (both total and column weighted %), and unweighted frequency distribution tables (for covariates by ACE status and covariates by BMI class) with standard errors and 95 % confidence intervals.

For the primary analysis, the bivariate SURVEYLOGISTIC procedures were used to estimate separately the individual association of covariates with discrete categories of exposure variable ACE status (probabilities modeled as ACE=1 [exposed]).

For the response variable [BMI-class], Parallel regression assumption or proportional odds assumption was rejected ($p < .0001$); therefore, bivariate (for primary bivariate analysis of covariates association with childhood obesity and other BMI categories) and multinomial multivariate generalized survey logistic models (main analysis) were used to model BMI-classes as nominal outcome variable with probabilities modeled as BMI class=Healthy weight (Reference group).

The results of these bivariate survey logistic regression analysis show the effect of each covariate on each category of the outcome (specially childhood obesity as outcome of interest) and exposure (child experienced ACE) variables, relative to their reference groups. The results of measures of association included parameter estimates from:

- 1) Analysis of maximum likelihoods for the regression coefficient parameters,
- 2) Unadjusted Odds ratio estimates with 95 % Confidence intervals,
- 3) And type 3 analyses of effects for the Wald-Chi-Square test of independence at 5 % significance level.

In the SAS survey procedures ID Number was used as sampling cluster variable (clusters are nested within the strata), State as sampling strata variable and NSCHWT as sampling weight.

“One child interview weights were produced,” therefore, the results are generalized to all non-institutionalized U.S. children aged 10-17 years. However, these weighted estimates are not representative of parents or health care providers; more details about the weighting procedure are provided elsewhere (CDC, 2011-2012 NSCH, frequently asked questions [FAQS], 2013).

Because, BMI level (outcome variable) were measured only for children age 10-17 years among children 0-17 years in 2011-2012 NSCH, domain statement were used to request for analysis of the weighted estimation for the subpopulation of children age 10-17 years in addition to overall study population estimations with specified missing options.

The goodness of fit test for normality was used to determine the distribution of continuous variable age. As the assumption of normality was not met, Wilcoxon test for equality of median age for ACE status and Kruskal Wallis test for age on BMI class (obese, overweight and underweight relative to healthy weight) was used. Thus, median and interquartile ranges (IQR) are reported for age.

On top of that, among our study participants we determined the prevalence of ACE and childhood obesity by 50 states, including District of Columbia, in the U.S. Only states with top 10 highest distributions (weighted column percentages) were reported.

Also, U.S. nationally representative weighted childhood obesity distributions (unweighted frequencies and weighted percentages) were reported both by gender and race (controlling for gender) for children aged 10-17 years.

3.4.2 Relationship Between Categories of ACE:

To answer our third research question, we analyzed the prevalence (weighted column percentages and unweighted column frequency distributions) and interrelationship of additional categories of ACEs among those who were exposed to one category of ACE of 9 ACEs asked about, with Tetrachoric Correlation Test, Chi-Square test of independence of association, and cross-tabulated bivariate frequency column distributions. We have reported polychoric correlation and Wald Chi-Square tests results at 5% significant level.

3.4.3 Covariate Adjusted (Final Model) Analysis of BMI-Classes Relative to Healthy Weight: (Multivariate Analysis):

In the main analysis in order to establish whether children exposed to ACE vs. unexposed had significant difference or not for the BMI class (especially childhood obesity) relative to healthy weight, adjusted odds ratio estimates and 95 % confidence interval (CI) were calculated.

As the proportional odds assumption was equivocal for the BMI, for the final models analysis, adjusted OR, 95 % CI estimates were obtained from multinomial multivariate generalized logistic regression models [GLOGIT function] that assessed adjusted strength and significance of the associations between:

- a) ACE status (1=exposed and 0=reference category) for each class of BMI relative to healthy weight

- b) Dose-response relationship of ACES (0=reference group, 1 or ≥ 2) for each category of BMI relative to healthy.
- c) In addition, separate adjusted odds ratios for each one of the category of ACES for BMI-class relative to healthy was also calculated in order to determine the most strong and significant category of ACE that predicts the odds of childhood obesity relative to healthy weight.

Similar to bivariate logistic regression analysis in the final models, sample cluster variable (ID Number), Strata variable (State), domain statement (for children aged 10-17) and sampling weight variable (NSCHWT) were used.

Therefore all these estimates own not only internal validity but also external validity to the general population of U.S. children aged 10-17 years. The final models show the effect of each variable for BMI class relative to healthy weight (healthy weight was used as the reference category in model for the outcome variable BMI)

The variables that had statistically significant associations ($p < .05$) in the bivariate analysis, both with the exposure of interest (ACE dichotomized status) and outcome of interest, were treated as lurking variables and therefore included in the final model.

The covariates in the model: the final models considered different ACE types, yes/no and ace score, select socio-demographic (age, sex, place of residence, family eats together), parental (overall physical health status of mother and father), perinatal infancy (low birth weight and prematurity) and childhood factors (Time spent watching TV, videos, or playing video games; using electronic devices; Time spent with a

computer, cell phone, or electronic device; Adequate amount of sleep for the child's age; physical activity; ill health).

3.4.4 Missing Information on Exposure and Outcome Variables (Unadjusted OR):

Children and adolescents with missing information (coded as DK/Ref/Missing in error/Partial interview to all 9 ACES) for ACE and BMI class (Missing in error) were considered separate categories in the analysis (N [unweighted] = 514; 1.27% [weighted] and N=1,445[unweighted]; 4.77% [weighted], respectively).

To assess the role of missing values in differential misclassification-unadjusted odds ratios were calculated for missing values, for ACE exposure, when they were once treated as positive (ACE=1) and then as negative exposure to ACE (ACE=0) for all BMI-classes, respectively. Those who were classified as missing for exposure but would have been unexposed (ACE=missing, ACE2=0) and exposed (ACE=missing, ACE3=1) were analyzed separately. To assess and compare this potential effect, we performed our analysis after treating those with missing information on any category of ACES as unexposed (didn't have exposure to any category of ACE of nine asked about) and exposed (experience any of the nine categories of ACES for child asked about). In all the repeated analysis, there wasn't a vast and notable difference in the results.

3.5 ETHICAL STATEMENT

Research projects using secondary data set (NSCH public dataset) are designated as not human subjects research; hence this thesis dataset use was exempted from IRB approval (Georgia State University: Policy for Publicly Available, Archival, and Secondary Data).

The publicly available dataset used for this thesis was provided by the Data Resource Center for Child and Adolescents Health (DRC), which is a “project of the Child and Adolescent Health Measurement Initiative (CAHMI)” (2011-2012 NSCH).

DRC, CAHMI also provided the codebooks that only included SAS codes used creating *child health indicators* (CHI) and NSCH variables (2011-2012 NSCH: CHI and Subgroups SAS Codebook, Version 1.0' 2013).

CHAPTER 4: RESULTS

4.1 Characteristics of the Study Population:

The target population of our study consisted of the subpopulation of U.S. children 10-17 years old, which is further divided into three age categories, from the 2011-2012 National Survey of Children’s Health (NSCH). NSCH is a nationwide survey in the U.S. that includes all 50 states including District of Columbia. All percentages calculated from this study are weighted to be representative of the U.S. population of children aged 10-17 years. This nationally representative survey has a sample size of 95,677 of non-institutionalized U.S. children aged 0-17 years, 45,309 (45.16%) participants between age 10-17 years (target study population) and 50,368 (54.83%) between ages 0-9 years of age. The median age (IQR) was 13 (11-15) for both genders.

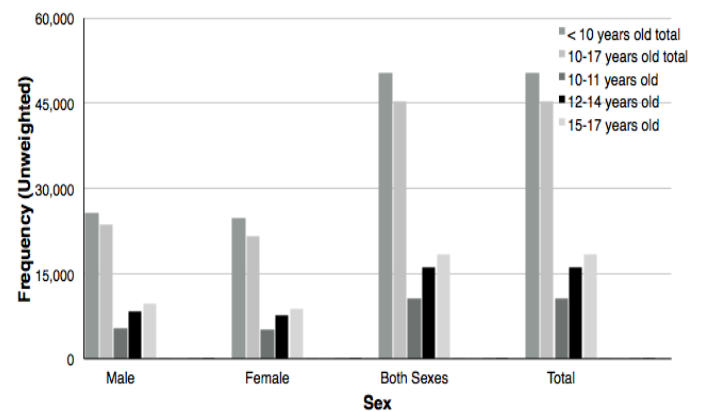


Figure 4.1 - Distribution of Age Categories by Sex

The participants of our study consisted of 21,658 (51.16%) boys and 21,658 (48.72%) of girls. There were more participants in age category 15-17 years old (N=18,444, 38.26 %) compared to age categories 10-11 years old (N=10,708, 24.58%) and 12-14 years old (N=16,157, 37.16 %), respectively; the detail of distribution is summarized in Figure 4.1 and Table 4.1.

TABLE 4.1. Distribution of Age Categories by Sex

	Male		Female		Both Sexes		Total		Missing	
	N	% (weighted)	N	% (weighted)	N	% (weighted)	N	% (weighted)	N	% (weighted)
< 10 years old total	25,622	27.99	24,691	26.79	50,368	54.78	50,368	54.83	55	0.06
10-17 years old total	23,597	23.10	21,658	22.00	45,255	45.100	45,309	45.16	54	0.05
10-11 years old	5,487	12.56	5,205	11.98	10,692	25.540	10,708	24.58	16	0.03
12-14 years old	8,411	19.09	7,734	18.05	16,145	37.150	16,157	37.16	12	0.01
15-17 years old	9,699	19.49	8,719	18.68	18,418	38.180	18,444	38.26	26	0.08

Note: The percentage values represent weighted percentages. For <10 and >10 years-old-totals, percentages are calculated out of weighted frequency of age 0-17 years, but for 10-11, 12-14 and 15-17 years old age categories percentages are calculated out of weighted frequency of age 10-17 years.

TABLE 4.2. Prevalence of Each Category of ACE, ACE Scores, Race and Household Poverty Status by Gender Among U.S. Children 10-17 years old. The 2011-2012 National Survey of Children's Health (N=45,309)

Characteristics	Prevalence (%)					
	Male		Female		Both sexes combined	
	N	% (Weighted)	N	% (Weighted)	N	% (Weighted)
Total population*	23,597	51.16	21,658	48.72	45,255	99.88
Socioeconomic hardship	4,903	25.54	4,495	24.76	9,398	25.17
Parental divorce Or separation	5,760	26.98	5,318	26.55	10,808	26.77
Bereavement	1,081	4.92	993	4.75	2,074	4.84
Incarcerated household member	1,619	8.43	1,442	7.87	3,061	8.16
Witnessing domestic violence	1,888	9.64	1,665	8.78	3,553	9.22
Witnessing neighborhood violence	2,907	13.84	2,269	12.17	5,176	13.02
Household mental illness	2,663	10.97	2,474	10.93	5,137	10.95
Household substance abuse	3,280	14.18	2,965	14.49	6,245	14.33
Racial discrimination	1,288	6.20	1,174	6.45	2,462	6.32
ACE Score						
0	11,411	43.61	10,664	43.51	22,075	43.56
1	5,704	25.11	5,183	25.91	10,887	25.50
≥2	6,197	30.11	5,586	29.22	11,783	29.67
ACE						
No	11,411	43.61	10,664	43.51	22,075	43.56
Yes	11,901	55.22	10,769	55.13	22,670	55.17
Race/ethnicity						
Hispanic	2,750	21.25	2,463	20.32	5,213	20.79
Non-Hispanic white	15,930	54.23	14,528	53.44	30,458	53.85
Non-Hispanic black	2,197	13.61	2,045	14.35	4,242	13.97
Multi-racial/Other, non-Hispanic	2,195	8.59	2,168	9.48	4,363	9.02
Household poverty status						
<100% FPL	3,133	19.93	2,878	19.86	6,011	19.89
100-199% FPL	4,137	21.62	3,602	20.62	7,739	21.13
200-399% FPL	7,325	28.42	6,707	29.32	14,032	28.86
400% or more FPL	9,002	30.03	8,471	30.19	17,473	30.11

Note: The weighted percentages are computed out of the total number of observations (both missing and non-missing) for every variable and only non-missing column percentages and unweighted frequencies of the cross tabulated values by sex are represented. *The total missing values for sex in the sample is [N (Unweighted)=54 and % (weighted) =0.12

Table 4.2, above, shows overall (both sexes combined) and gender specific differences in the prevalence of race, poverty, Adverse Childhood Experience and ACE Score. The race of the majority of the children was non-Hispanic White (53.85%) followed by Hispanic (20.79), non-Hispanic Black (13.97%), and Multi-racial/other, non-Hispanic (9.02%), respectively. Approximately, 20 % of children between both genders were living in households with incomes <100% federal poverty level.

4.2 Distribution Of Adverse Childhood Experiences:

Approximately 55.15% of the study participants of had exposure to any ACE for a child, of 9 asked about (Figure 4.2). The prevalence of Adverse Childhood Experiences varied as the ACE Score increased. For instance, for those who didn't experience any ACE of 9 asked about (ACE Score=0) it was 43.56%, those with ACE Score=1, 25.50 % and for the ACE Score ≥ 2 , 29.67%, respectively. ACE exposure (Yes/No) and ACE score percentages were almost the same between both genders (Table 4.2).

Moreover, approximately 25.4 million (89.5%) children aged 10-17 years experienced 3 or less ACE (Figure 4.3, Table 4.3).

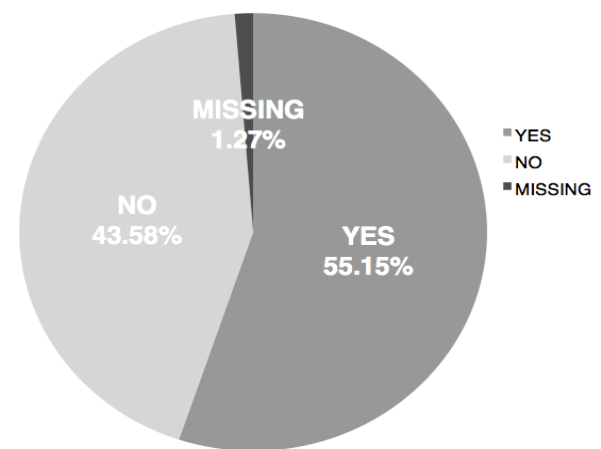


Figure 4.2- Weighted Total % of ACE

	% (WEIGHTED)	N (WEIGHTED)	N (UNWEIGHTED)
YES	55.15	18,360,592	22,690
NO	43.58	14,510,501	22,105
MISSING	1.27	422,729	514

The most prevalent ACE category of 9 asked about for child was living with parents who were either divorced or separated after his/her birth (26.77%) and the least prevalent was living with a parent who died (4.84 %)(Table 2).

However, the prevalence of specific category of ACE exposure varied slightly across genders. In general, of 9 ACEs asked about, seven of them were reported slightly higher for boys compared to girls, namely: living in households finding it difficult to cover their basic needs like food or housing due to economic hardship (25.54% vs. 24.76%), living with parents who were either divorced or separated after his/her birth (26.98% vs. 26.55%), lived parent who died (4.92% vs. 4.75%), lived with a parent who was imprisoned after his/her birth (8.43% vs. 7.87%), saw parents hit, kick, slap, punch or beat one another(9.64% vs. 8.78%), witnessed or was victim of neighborhood violence (13.84% vs. 12.17%), and lived with someone who suffered from mental illness, was suicidal or severely depressed for few weeks (10.97% vs. 10.93%)(Table 4.2).

TABLE 4.3. Frequency Distribution of number of ACEs

Number of ACE for child, of 9 asked about	N (UNWEIGHTED)	N (WEIGHTED)	% (WEIGHTED)
0	22,105	14,510,501	43.58
1	10,899	8,482,678	25.48
2	5,261	4,362,321	13.10
3	2,875	2,452,842	7.37
4	1,684	1,406,884	4.22
5	1,040	919,811	2.76
6	625	483,407	1.45
7	241	205,331	0.62
8	60	32,221	0.09
9	5	15,097	0.04

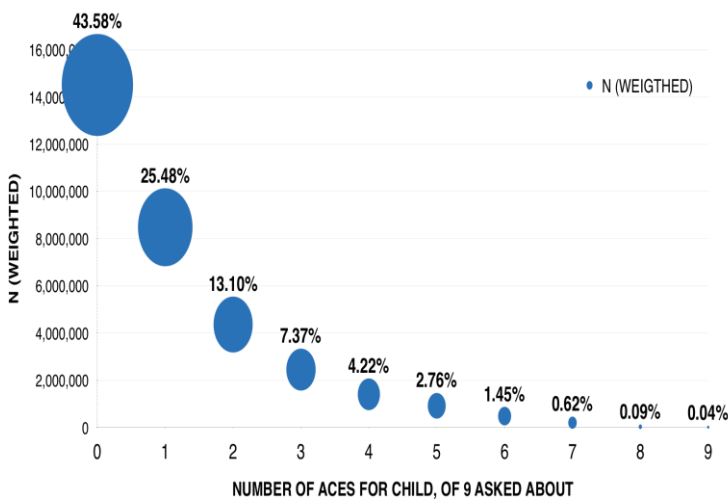


Figure 4.3- Frequency Distribution of number of ACEs

4.3 ACE and childhood obesity distributions by States in the United States:

Among 50 states in the U.S. including District of Columbia, eight out top ten obese states for children aged 10-17 years of age were located in the Southern region, including District of Columbia (Figure 4.4). Of these states, South Carolina had the highest prevalence of obesity among children 10-17 years old 21.09%¹² [95 % CI (17.31-25.14, SE=2.07)].

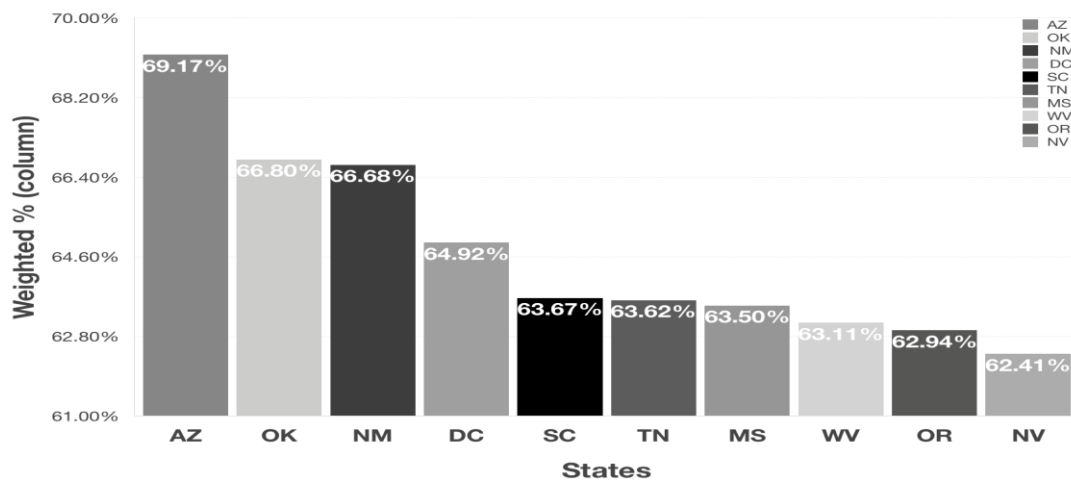


Figure 4.4 - Top 10 highest levels of Obesity by states (including DC) in the U.S. among children 10-17 years. The 2011-2012 National Survey of Children’s Health

Similarly, it was discovered that 60 % of top 10 states with adverse childhood experience were in Southern states of the US (including DC). However, the state with the highest percentage of ACE was a western state, the state of Arizona 69.17% [95 % CI (64.8273.53), SE=2.33]] (Figure 4.

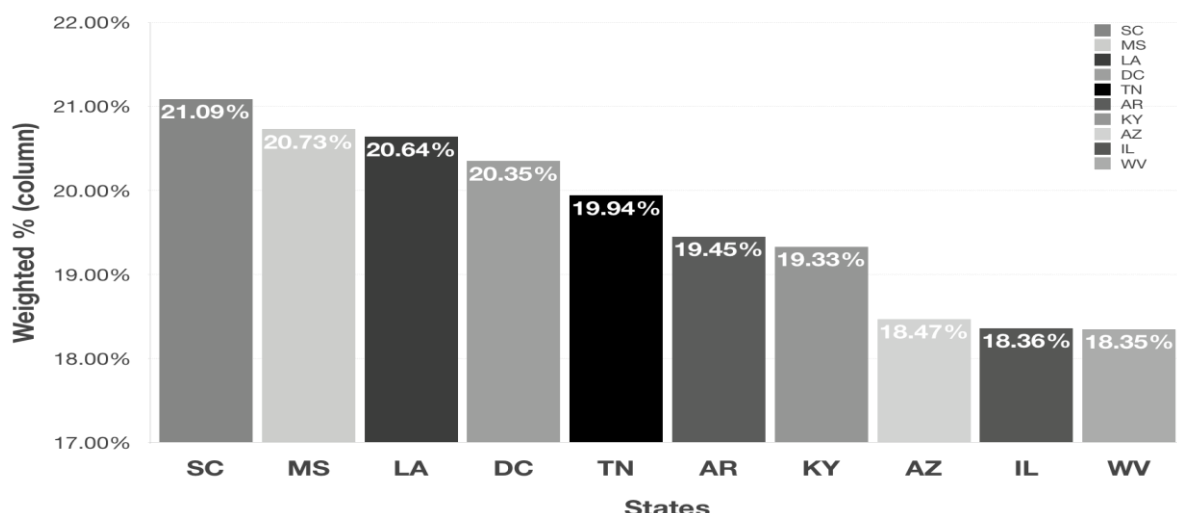


Figure 4.5 - Top 10 highest levels of ACE by states (including DC) in the U.S. among children 10-17 years. The 2011-2012 National Survey of Children’s Health

¹² Weighted column percentage of BMI-classes by states

4.4 Childhood Obesity Distribution by Gender and Race in the United States:

The distribution of gender among our study participants, aged 10-17 years, by obesity level varied between boys and girls. Those who had obesity were mostly boys compared to girls, that is, 60.20% [(95% CI (57.31-63.06), SE=1.47] vs. 39.82% [(95% CI (36.94-42.70) SE=1.47], respectively (Figure 4.6 and Figure 4.7).¹³

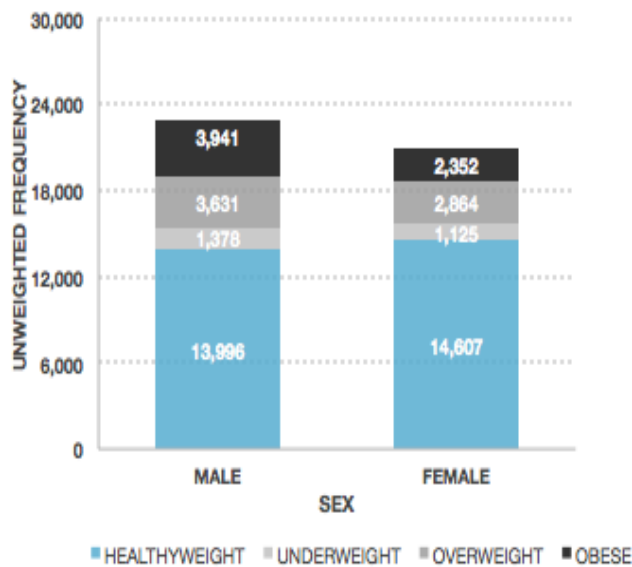


Figure 4.6 - Comparison of BMICLASS Frequencies (unweighted) by SEX

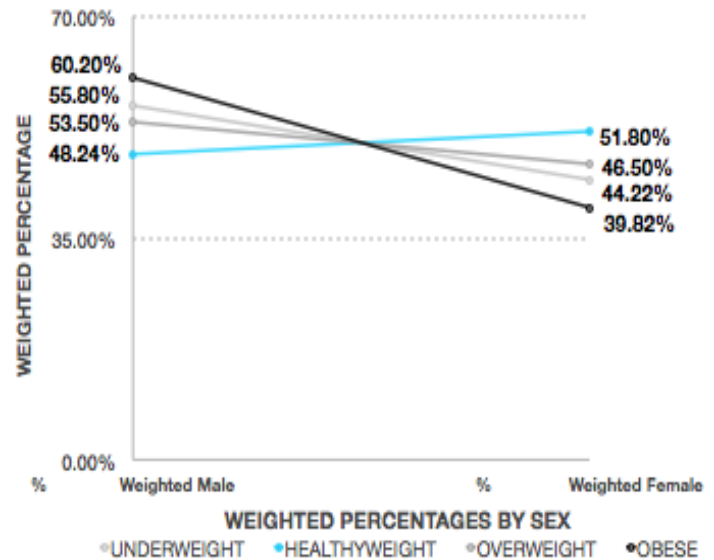


Figure 4.7 - Comparison of BMICLASS % (weighted) by SEX

Controlling for gender, among those who were obese, White-non-Hispanic children had the highest prevalence of obesity compared to other races for both genders (Table 4.4). The total prevalence of childhood obesity for all races adds up to 17.58% for boys and 12.21% for girls.

TABLE 4.4. Childhood Obesity Distribution by and Race controlling for Gender Among Children Aged 10-17 years in the U.S. ¹⁴

CATEGORIES	N (UNWEIGHTED)	% (WEIGHTED)	STD ERROR OF %
BOYS ALL	3,941	17.58%*	0.58
HISPANIC	595	4.61%	0.42
WHITE, NON-HISPANIC	2,330	7.84%	0.34
BLACK, NON-HISPANIC	528	3.30%	0.27
MULTIRACIAL/OTHER/NON-HISPANIC	374	1.31%	0.16

¹³ The % is column percentages for Sex by BMI-class.

¹⁴ **Note:** (% weighted total); * includes the obesity prevalence for missing race 0.52 %; ** includes the obesity prevalence for missing race 0.15%

GIRLS ALL	2,352	12.21%**	0.55
HISPANIC	350	3.23%	0.39
WHITE, NON-HISPANIC	1,232	4.91%	0.30
BLACK, NON-HISPANIC	470	2.98%	0.26
MULTIRACIAL/OTHER/NON-HISPANIC	257	0.94%	0.15

4.5 Relationships between Categories of Adverse Childhood Experiences:

The frequency distribution of reporting additional categories of ACE among those who were exposed to any one of the nine categories of ACE and ACE score (1 and 2 or more) is represented in Table 4.5.

It seems that all nine categories of ACEs are interrelated. Astonishingly, if a child had any one of the nine categories of ACEs (except racial discrimination), he/she was also simultaneously exposed mostly to parental divorce or separation (% ranges from 38.77 for socioeconomic hardship to 66.15% for Witnessing domestic violence); however, if a child was discriminated because of his/her race or ethnicity or lived with parent who was divorced or separated after his/her birth was at the same time for the most part living in households with socioeconomic hardship to finance their food or housing (34.43% and 36.46%, respectively) (Table 4.5).

35.29% of the children who were subject to any one of the 9 ACEs (ACE score=1), irrespective of the particular type, were those living in families with socioeconomic hardship; 64,31 % of children with ACE Score=2, were living with parent who got divorced or separated after the child's birth (Table 4.5).

Similarly, the results of the Tetrachoric correlation matrix between ACE categories verified the positive and statistically significant (p value $< .05$) correlation between ACE categories (Table 4.6). The values of Tetrachoric correlation coefficients matrix for ACE categories ranged from 0.092 to 0.710 ($p < .05$), and this value for the ACE score covers 0.610 to 0.884 ($p < .05$). Besides, the hypotheses of independence of association among the nine ACE categories were strongly rejected by Chi-Square test for independence ($p < .0001$). Therefore, these results pinpoint that ACE categories are interrelated with one another (Table 4.5 and Table 4.6).

4.6 Distribution and association (unadjusted odds ratio) of participants' characteristics by their ACE status:

Table 4.7 shows the distribution and association (unadjusted odds ratio) of participants' characteristics by their ACE status (Yes=exposed, No=unexposed) among U.S. children aged 10-17 years ($N=45,309$). The Goodness of Fit Tests for normal distribution revealed that age is not normally distributed ($p < .01$). Besides, the Wilcoxon Rank Sums Test indicated that median for those with and without ACE categories was significantly different between the groups ($p < .0001$). The median age (IQR) for children with ACE was one year higher compared to those without any ACE, that is 14(12-16) vs. 13 (11-15).

TABLE 4.5. Prevalence of Reporting of Additional Categories of ACEs among U.S. Children 10-17 years old, Who Reported Exposure to First Category of ACE. The 2011-2012 National Survey of Children's Health (N=45,309)

First Category of ACE	Socioeconomic hardship	Parental divorce or separation	Bereavement	Incarcerated household member	Witnessing domestic violence	Witnessing neighborhood violence	Household mental illness	Household substance abuse	Racial discrimination	Additional ACEs; ACE Score	
										1	≥2
Unweighted N Weighted %	N %	N %	N %	N %	N %	N %	N %	N %	N %	N %	N %
Socioeconomic hardship	—	3,560 36.46%	682 38.80%	1,328 46.25%	1,593 49.85%	2,037 44.93%	1,973 42.82%	2,271 40.63%	793 34.43%	3,324 35.29%	6,085 54.53%
Parental divorce or separation	3,560 38.77%	—	813 39.42%	1,981 61.75%	2,383 66.15%	2,408 49.58%	2,583 53.17%	3,681 61.16%	807 33.88%	3,298 30.18%	7,515 64.31%
Bereavement	682 7.45%	813 7.11%	—	396 13.35%	399 11.48%	447 9.11%	475 9.34%	674 10.72%	163 6.89%	535 4.07%	1,540 12.78%
Incarcerated household member	1,328 14.97%	1,981 18.79%	396 22.52%	—	1,270 33.99%	1,180 25.04%	1,082 22.67%	1,956 33.93%	318 13.23%	178 1.73%	2,883 25.97%
Witnessing domestic violence	1,593 18.24%	2,383 22.75%	399 21.88%	1,270 38.42%	—	1,711 34.08%	1,417 29.31%	2,101 35.02%	426 16.38%	200 2.07%	3,353 29.26%
Witnessing neighborhood violence	2,037 23.22%	2,408 24.09%	447 24.53%	1,180 39.99%	1,711 48.15%	—	1,628 35.24%	1,971 34.39%	730 31.25%	1,004 7.62%	4,173 37.30%
Household mental illness	1,973 18.62%	2,583 21.73%	475 21.15%	1,082 30.46%	1,417 34.83%	1,628 29.64%	—	2,355 34.24%	695 16.98%	879 6.72%	4,264 31.12%
Household substance abuse	2,271 23.11%	3,681 32.71%	674 31.76%	1,956 59.62%	2,101 54.45%	1,971 37.85%	2,355 44.79%	—	582 19.73%	734 5.05%	5,513 43.92%
Racial discrimination	793 8.64%	807 7.99%	163 9.01%	318 10.26%	426 11.24%	730 15.18%	512 9.80%	582 8.70%	—	747 7.26%	1,719 15.06%
Total N Weighted %	9,409 27.15%	10,813 26.77%	2,075 4.83%	3,061 8.15%	3,553 9.21%	5,177 13.00%	5,143 10.94%	6,247 14.32%	2,466 6.32%	10,899 25.48%	11,791 29.67%

Note: The weighted percentages are computed out of the total number of observations (both missing and non-missing) for every category of ACE and only column percentages, and unweighted frequencies of the cross-tabulated values by other categories of ACE are represented.

TABLE 4.6. Tetrachoric Correlation of Reporting of Additional Categories of ACEs among U.S. Children 10-17 Years of Age Who Reported Exposure to First Category of ACE*. The 2011-2012 National Survey of Children’s Health (N=45,309)

First Category of ACE	Socioeconomic hardship	Parental divorce or separation	Bereavement	Incarcerated household member	Witnessing domestic violence	Witnessing neighborhood violence	Household mental illness	Household substance abuse	Racial discrimination	Number of ACEs, (Of 9 asked about)**
Socioeconomic hardship	1.000	0.296	0.182	0.341	0.374	0.329	0.313	0.299	0.173	0.785
Parental divorce or separation	0.296	1.000	0.210	0.545	0.590	0.374	0.425	0.571	0.130	0.850
Bereavement	0.182	0.210	1.000	0.313	0.279	0.207	0.232	0.313	0.092	0.623
Incarcerated household member	0.341	0.545	0.313	1.000	0.658	0.495	0.455	0.710	0.187	0.840
Witnessing domestic violence	0.374	0.590	0.279	0.658	1.000	0.634	0.538	0.692	0.246	0.860
Witnessing neighborhood violence	0.329	0.374	0.207	0.495	0.634	1.000	0.460	0.489	0.345	0.787
Household mental illness	0.313	0.425	0.232	0.455	0.538	0.460	1.000	0.598	0.201	0.803
Household substance abuse	0.299	0.571	0.313	0.710	0.692	0.489	0.598	1.000	0.230	0.884
Racial discrimination	0.173	0.130	0.092**	0.187	0.246	0.345	0.201	0.188	1.000	0.610
Number of ACEs, (Of 9 asked about)**	0.785	0.850	0.623	0.840	0.860	0.787	0.803	0.870	0.610	1.000

Note: *All the variables are dichotomous, ** classified as (0, 1 and ≥2); the correlations are significant at p < .05.

**For example 0.85 % of the variation among children who have bereavement among different classes of ACE can be predicted from the relationship between those who have bereavement and Racial Discrimination (r=0.092, p<.05).

There was a statistically significant association between all the covariates (X^2 ; $p < .0001$) and adverse childhood experience status, except gender [$OR_{unadjusted}$ 1.00(0.97-1.04)] and $X^2(2)=0.032$; $p=0.98$]. The odds ratio for children aged 15-17 years to 10-11 years for having ACE was 1.19(1.13-1.25, $p < .0001$)(Table 4.7).

Among the covariates the risk of having adverse childhood experience was lower among children who were living in a metropolitan area compared to non-metropolitan [$OR_{unadjusted}$ 0.72(0.68-0.76), $p < .0001$], ate more meals together with family [everyday vs. No days, $OR_{unadjusted}$ 0.63(0.56-0.70), $p < .0001$], engaged in rigorous physical activities many days in a week [everyday vs. no days, $OR_{unadjusted}$ 0.72(0.62-0.84), $p < .0001$], lived in neighborhoods with two or more amenities compared to none [$OR_{unadjusted}$ 0.72(0.68-0.76), $p < .0001$], and had an adequate amount of sleep for his/her age most nights of the week [every night vs. no night, [$OR_{unadjusted}$ 0.59(0.45-0.76), $p < .0001$].

However, the odds ratio estimates of having ACE relative to without, was higher comparing older age categories of children vs. lower [15-17 years old vs. 10-11 years old [$OR_{unadjusted}$ 1.19(1.13-1.25), $p < .0001$], fair/poor overall health status of mother vs. excellent/very good [$OR_{unadjusted}$ 5.05(4.70-5.42), $p < .0001$], fair/poor overall health status of father vs. excellent/very good [$OR_{unadjusted}$ 4.52(4.15-4.93), $p < .0001$], low birth weight vs. healthy weight [$OR_{unadjusted}$ 1.35(1.27-1.44), $p < .0001$], premature vs. not premature [$OR_{unadjusted}$ 1.23(1.16-1.30), $p < .0001$], children with ≥ 2 chronic health conditions of 18 asked about vs. none [$OR_{unadjusted}$ 3.20(2.98-3.45), $p < .0001$]; children who on average spent more hours daily in front of TV watching TV, videos or playing video games vs. lower [≥ 4 hours per day vs. doesn't watch TV, $OR_{unadjusted}$ 2.76(2.49-3.05), $p < .0001$]; children who spent more time with computers, cell phones, handheld

video games, and other electronic devices for purposes other than school work vs. lower [≥ 4 hours per day vs. doesn't use electronic devices, $OR_{unadjusted}$ 1.82(1.49-2.23), $p < .0001$]; and children who had heavier weight vs. healthy weight [obese-95th percentile or above vs. healthy weight-5th to 84th percentile, $OR_{unadjusted}$ 1.95(1.84-2.06), $p < .0001$](Table 4.7).

Among those with adverse childhood experiences category and positive response to the risk factors enumerated in Table 4.7, the prevalence vary from 4.81% (underweight-less than 5th percentile) to 86.94% (neighborhoods with two or more amenities in which the child was living). The percentage point difference in mothers overall poor physical health status compared to the father of the children was 7.65%(17.47 vs. 9.82).

Children who were exposed to ACE compared to those who were not, watched more TV per day (17.37%, ≥ 4 hours per day) spent more hours using his/her electronic device (19.59%, ≥ 4 hours per day), more physically active during the week in vigorous exercises for 20 minutes (24.34%, everyday), had more chronic comorbidities (17.06%, $2 \geq$), were more premature birth (11.33%, ≥ 3 weeks before his/her due date), more low birth weight (8.46%, weighed less than 2500 grams), had higher percentage of old age category (39.86%, 15-17 years old), and were more obese(17.35%, obese-95th percentile or above of weight for age). The percentage point difference of obesity among those with ACE relative to control group was almost 5.5 % (17.35 vs. 11.94%).

Surprisingly, children who belong to the group without ACE exposure had higher prevalence of: residence in metropolitan areas (76.35%), access to ≥ 2 amenities in the neighborhood of four asked about, namely, park, recreational center, sidewalk and

library (89.47%), every night adequate sleep for a child of his/her age (55.64%), percentages of underweight and healthy weight (6.38% and 63.72%, respectively).

TABLE 4.7. Distribution and Association (unadjusted odds ratio) of Participants' Characteristics by their ACE Status among U.S. Children Aged 10-17 years. The 2011-2012 National Survey of Children's Health (N=45,309)

Characteristics	ACE (NO) N= 22,105 % (Weighted)=43.58		ACE (YES) N=22,690 % (Weighted)=55.15		OR (95% CI)	Pr>ChiSq
	N	% (Weighted)	N	% (Weighted)		
Age, yr.* Median (IQR)	13 (11-15)		14 (12_16)		1.09(1.08-1.010)	<.0001
Age, yr.* 10-11 12-14 15-17 Missing (None)	5,448 7,970 8,687	25.64 30.08 36.28	5,114 8,000 9,576	23.56 36.58 39.86	1.0(Referent) 1.07(1.02-1.13) 1.19 (1.13-1.25)	<.0001
Sex Male Female Missing	11,411 10,664 30	51.19 48.64 0.17	11,901 10,769 20	51.23 48.70 0.07	1.0 (Referent) 1.00(0.97-1.04)	>.05
Place of residence *, ° Non-metropolitan Metropolitan Missing	2,979 11,754 118	12.42 76.35 1.15	3,790 11,702 127	15.43 74.11 1.43	1.0 (Referent) 0.72(0.68-0.76)	<.0001
Physical health status mother* Excellent/very good Good Fair/poor Missing	16,463 4,007 1,183 30	70.86 19.79 7.03 0.14	10,500 5,475 3,604 30	44.69 25.09 17.47 0.07	1.0 (Referent) 2.29(2.19-2.41) 5.05(4.70-5.42)	<.0001
Physical health status father* Excellent/very good Good Fair/poor Missing	15,707 4,094 1,012 18	68.24 18.47 5.39 0.08	8,412 4,050 2,179 17	36.16 17.98 9.82 0.06	1.0 (Referent) 2.12(2.02-2.24) 4.52(4.15-4.93)	<.0001
Family eats together (No. Of days/wk) * 0 1-3 4-6 7 Missing	667 4,712 9,275 7,424 27	2.84 21.82 39.82 35.41 0.09	1,271 5,315 7,970 8,081 53	5.47 23.50 34.69 36.00 0.34	1.0 (Referent) 0.73(0.64-0.82) 0.56(0.49-0.63) 0.63(0.56-0.70)	<.0001
Low birth weight (<2500G) * No Yes Missing	19,623 1,566 916	86.91 8.39 4.69	18,771 2,045 1,871	82.88 8.46 8.65	1.0 (Referent) 1.35(1.27-1.44)	<.0001
Prematurity (≥ 3wks before his/her due date)* No Yes Missing	19,866 2,088 151	89.44 9.98 0.58	19,689 2,656 345	87.48 11.33 1.19	1.0 (Referent) 1.23(1.16-1.30)	<.0001

Ill health (chronic health conditions from a list of 18 conditions) *, *						
0	17,009	77.32	14,210	62.77	1.0(Referent)	<. 0001
1	3,275	14.57	4,242	20.16	1.87(1.76-1.98)	
≥2	1,817	8.09	4,237	17.06	3.20(2.98-3.45)	
Missing	4	0.00	1	0.01		
Physical activity (No. of days/wk) *						
0	1,625	8.27	2,588	12.65	1.0(Referent)	<. 0001
1-3	5,822	27.77	6,103	26.03	0.61 (0.52-0.72)	
4-6	9,862	42.30	8,488	35.94	0.54 (0.46-0.63)	
7	4,656	20.59	5,270	24.34	0.72(0.62-0.84)	
Missing	140	1.05	241	1.04		
Neighborhood Amenities*, #						
0					1.0(Referent)	<. 0001
1	782	3.08	980	4.22	1.00(0.88-1.13)	
≥2	1,340	5.64	1,608	6.78	0.69(0.63-0.77)	
Missing	19,687	89.47	19,772	86.94		
	296	1.80	330	2.06		
Television watching, videos or playing video games (No. Of hr/day) *						
0	1,388	6.88	1,198	5.37	1.0 (Referent)	<. 0001
≤1	9,528	42.81	7,873	34.12	1.30 (1.20-1.42)	
>1-<4	9,082	40.34	9,892	42.54	1.78 (1.64-1.93)	
≥4	2,022	9.53	3,585	17.37	2.76(2.49-3.05)	
Missing	85	0.44	142	0.60		
Computer, cell phone, or electronic device (No. Of hr./day) *						
0	1,499	8.65	1,790	8.99	1.0 (Referent)	<. 0001
≤1	12,125	53.65	9,481	40.17	0.86 (0.81-0.93)	
>1-<4	6,064	26.82	6,974	29.66	1.56(1.41-1.73)	
≥4	2,224	9.92	4,165	19.59	1.82(1.49-2.23)	
Missing	193	0.95	280	1.57		
Adequate amount of sleep (No. Of nights previous week) *						
0	498	2.46	771	3.51	1.0 (Referent)	<. 0001
1-3	1,438	6.22	1,914	8.24	1.01 (0.74-1.37)	
4-6	8,336	34.83	8,018	33.59	0.55 (0.42-0.72)	
7	11,664	55.64	11,732	53.25	0.59 (0.45-0.77)	
Missing	169	0.84	255	1.028		
BMI CLASS*, †						
Healthy weight	14,943	63.72	13,348	57.04	1.0 (Referent)	<. 0001
Underweight	1,329	6.38	1,154	4.81	0.97 (0.89-1.05)	
Overweight	2,846	13.29	3,566	16.14	1.44(1.36-1.52)	
Obese	2,316	11.94	3,893	17.35	1.95(1.84-2.06)	
Missing	671	4.67	729	4.65		

Note:

ACE (YES/NO) indicates presence or absence of any of the 9 ACEs asked about

Missing value for ACE categories [N (unweighted)=514, % (weighted)=1.27]

OR= Crude Odds Ratio, CI= 95 % Confidence Interval

‡Underweight is BMI for age less than 5th percentile, Healthy weight is BMI 5th to 84th percentile, Overweight is BMI 85th to 94th percentile, and Obesity is BMI≥95 percentile

The 0 or No categories of covariates are used as reference group for calculation of odds ratios

°Metropolitan Statistical Area (Only in states that meet the 500,000 threshold)

*The Wald Chi-square test for independence between the covariates and ACE prevalence indicated significant association at significant level (p<0.001)

**The trend for increasing ORs as the BMICLASS increases is significant at (p<0.001)

•Learning disability, ADD or ADHD, depression anxiety problems, Behavioral or conduct problems, autism or other autism spectrum disorder, developmental delay, intellectual disability, cerebral palsy, speech problems, asthma, diabetes, Tourette Syndrome, epilepsy or seizure disorder, hearing problems, vision problems, bone or joint problems, brain injury or concussion.

#Neighborhood amenities include sidewalks, parks, recreation center or libraries

The results of both unadjusted odds ratios and weighted percentages indicate that children who belong to the group with ACE exposure are heavier, especially more obese, compared to the group without ACE.

4.7 Distribution and association (unadjusted odds ratio) of participants' characteristics by BMI-CLASS:

Table 4.8 indicates the distribution and relationship of proximal and distal risk factors with childhood obesity including other BMI-classes. The results of multinomial generalized logistic regression indicate the effect of each predictor variable on each category of response variable, namely, obesity -95th percentile or above (primary outcome), overweight-85th to 94th percentile and underweight-less than 5th percentile, compared to the reference category of healthy weight-5th to 84th percentile. The Wald Chi-Square of association detected statistically significant association between all the covariates and BMI-levels ($p < .0001$). The median age (IQR) was higher for those with healthy weight compared to other BMI categories 14 (12-16).

The odds of being obese relative to the healthy weight was 0.62 times lower among those who were female compared to men [$OR_{unadjusted}=0.62(0.54-0.70)$], 0.76 times lower among children who resided in metropolitan areas compared to non-metropolitan [$OR_{unadjusted}=0.76(0.66-0.88)$, $p < .0001$], and 0.72 times lower among those children who had ≥ 2 neighborhood amenities vs. none [$OR_{unadjusted}=0.72(0.53-0.97)$, $p < .0001$]. Similarly, children who were engaged most days of the week in sports or physical activities for at least 20 minutes their risk of being obese compared to healthy weight decreased significantly, that is, the odds ratio of children who were physical active for 4-6 days or everyday of the week to those who were not physically active any day of the week for obesity relative to healthy weight decreased by a factor of 0.61 [$OR_{unadjusted}=0.61(0.49-0.75)$, $p < .0001$] and 0.59, [$OR_{unadjusted}=0.59(0.47-0.76)$, $p < .0001$], respectively.

Interestingly, the odds of obesity relative to healthy weight significantly decreased among children who used their electronic for fewer hours during the day. For instance, children who used electronic devices for one hour to less than four hours and one hour or less compared to those who do not use electronic devices, had 42% [OR_{unadjusted}=0.58(0.46-0.73), p < .0001] and 26 % [OR_{unadjusted}= 0.74(0.59-0.93), p < .0001] lower odds of obesity relative to healthy weight, respectively (Table 4.8).

4.8 Distribution and association (unadjusted odds ratio) of participants' ACE status, ACE score and ACE categories by BMI-CLASS:

For adverse childhood experience, the probability of obesity relative to healthy weight in the group with adverse childhood experience (any of 9 asked about) was 62% higher compared to the reference group, with the true population effect between 58.5% and 65% [OR_{unadjusted}=1.62(1.41-1.86), p < .0001]. This probability lowers to 57.6% for those with overweight relative to healthy weight, but for those with underweight BMI class relative to healthy weight it doesn't reach statistically significant level (p < .09)(Table 4.8).

Based on the ACE score, the trend for increase in odds ratios for obesity relative healthy weight as the ACE score increases is highly statistically significant at (p< .0001). The trend was statistically significant for obese and overweight groups, but the strength of association was stronger for obese group relative to healthy. For instance, children with ACE score ≥ 2 compared to those without any ACE of 9 asked about, had almost 2 fold odds of obesity relative to healthy weight [OR_{unadjusted}= 1.91(1.64-2.23), p < .0001] (Table 4.8).

It was also discovered that the only BMI class that was statistically associated with all ACE categories was obese-95th percentile or above category ($p < .0001$). The odds of childhood obesity relative to healthy weight was highest for children with the ACE category of bereavement compared to those without [$OR_{unadjusted} = 1.90(1.45-2.48)$, $p < .0001$], followed by the ACE category of socioeconomic hardship [$OR_{unadjusted} = 1.91(1.64-2.23)$, $p < .0001$], and Incarcerated household member [$OR_{unadjusted} = 1.63(1.33-1.99)$, $p < .0001$]. Therefore, these results indicate that the exposure to adverse childhood experiences in general and specifically by category are associated with higher and statistically significant odds (unadjusted) of outcome of childhood obesity relative to healthy weight in comparison to the other BMI categories.

TABLE 4.8. Distribution and Association (unadjusted odds ratio) of Participants' Characteristics by their BMI for Age Status among U.S. Children Aged 10-17 Years. The 2011-2012 National Survey of Children's Health (N=45,309).

CHARACTERISTICS	HEALTHY WEIGHT (REFERENCE GROUP) N=28,573 %(Weighted)=59.89		OBESE (MAIN OUTCOME) N=6,293 %(Weighted)=14.94			OVERWEIGHT N=6,495 %(Weighted)=14.89			UNDERWEIGHT N=2,503 %(Weighted)=5.50			
	N	% (Weighted)	N	% (Weighted)	OR OBESE VS. REF (95% CI)	N	% (Weighted)	OR OVERWT VS. REF (95% CI)	N	% (Weighted)	OR UNDERWT VS. REF (95% CI)	
Age, yr. Median (IQR)*	14 (12-16)		13 (11-15)			0.86(0.83-0.88)	13 (11-15)		0.89(0.87-0.92)	13 (11-15)		0.87(0.83-0.91)
Age, yr. *												
10-11	5,496	19.81	2,096	32.56	1.0 (Referent)	1,761	27.24	1.0 (Referent)	807	34.65	1.0 (Referent)	
12-14	10,089	36.72	2,242	37.58	0.62(0.53-0.73)	2,520	41.01	0.81(0.69-0.95)	837	33.69	0.52(0.41-0.66)	
15-17	12,988	43.47	1,955	29.86	0.42(0.35-0.49)	2,214	31.74	0.53(0.45-0.62)	859	33.66	0.42(0.32-0.54)	
Missing	0	0	0	0		0	0		0	0		
Sex*												
Male	13,966	48.24	3,941	60.18	1.0 (Referent)	3,631	53.49	1.0 (Referent)	1,378	55.76	1.0 (Referent)	
Female	14,607	51.76	2,352	39.82	0.62(0.54-0.70)	2,864	46.50	0.81(0.72-0.92)	1,125	44.22	0.74(0.60-0.89)	
Missing	0	0	0	0		0	0		0	0		
Place of residence*, °												
Non-Metropolitan	4,070	13.46	1,203	16.98	1.0 (Referent)	1,054	15.33	1.0 (Referent)	294	11.30	1.0 (Referent)	
Metropolitan	14,992	74.82	3,198	71.92	0.76(0.66-0.88)	3,272	73.79	0.87(0.74-1.02)	1,374	77.91	1.24(0.98-1.57)	
Missing	204	1.75	60	2.11		45	1.21		21	1.54		
Physical health status mother*												
Excellent/very good	18,396	61.19	2,864	42.71	1.0 (Referent)	3,537	51.29	1.0 (Referent)	1,604	60.32	1.0 (Referent)	
Good	5,592	20.45	1,608	28.71	2.01(1.71-2.36)	1,457	22.33	1.30(1.11-1.52)	493	21.93	1.08(0.84-1.41)	
Fair/poor	2,432	10.06	1,065	17.62	2.51(2.09-3.01)	818	16.31	1.93(1.60-2.34)	213	9.30	0.94(0.64-1.38)	
Missing	219	0.83	59	0.79		72	0.89		15	1.13		
Physical health status father*												
Excellent/very good	16,584	54.89	2,420	36.35	1.0 (Referent)	3,117	45.07	1.0 (Referent)	1,492	57.12	1.0 (Referent)	
Good	4,940	17.04	1,246	20.08	1.78(1.48-2.13)	1,232	18.73	1.34(1.13-1.59)	428	17.58	0.99(0.74-1.33)	
Fair/poor	1,741	6.48	645	9.56	2.23(1.76-2.81)	502	8.64	1.62(1.28-2.05)	151	7.41	1.09(0.69-1.75)	
Missing	205	0.84	55	0.69		61	0.85		14	1.04		
Family eats together (No. of days/wk) *												
0	1,205	4.24	303	4.49	1.0 (Referent)	280	3.99	1.0 (Referent)	96	4.49	1.0 (Referent)	
1-3	6,546	24.26	1,308	18.76	0.73(0.54-0.98)	1,433	22.17	0.97(0.74-1.27)	533	21.46	0.83(0.55-1.27)	
4-6	11,534	39.03	2,112	31.95	0.77(0.58-1.03)	2,409	36.35	0.99(0.76-1.29)	949	37.49	0.91(0.61-1.35)	
7	9,218	32.24	2,552	44.68	1.31(0.98-1.74)	2,357	37.12	1.22(0.94-1.59)	921	35.47	1.04(0.69-1.55)	
Missing	70	0.24	18	0.11		16	0.37		4	1.08		
Low birth weight (<2500G) *												
No (Normal weight)	24,764	86.07	5,371	85.82	1.0 (Referent)	5,600	85.62	1.0 (Referent)	2,069	88.08	1.0 (Referent)	
Yes	2,175	7.84	570	8.51	1.09(0.88-1.34)	505	8.59	1.10(0.89-1.37)	269	13.10	1.77(1.28-2.47)	
Missing	1,634	6.09	352	5.67		390	5.79		165	5.81		

Prematurity (≥ 3wks before his/her due date) *												
No	25,390	81.17	5,456	87.55	1.0 (Referent)	5,727	87.14	1.0 (Referent)	2,194	85.48	1.0 (Referent)	
Yes	2,863	9.94	786	11.94	1.22(1.01-1.48)	697	11.65	1.19(0.99-1.46)	280	13.46	1.41(1.02-1.96)	
Missing	320	0.89	51	0.51		71	1.20		29	1.06		
Ill health (chronic health conditions from a list of 18 conditions) *, *												
0	20,587	70.84	3,843	63.85	1.0 (Referent)	4,345	66.17	1.0 (Referent)	1,725	66.91	1.0 (Referent)	
1	4,658	17.29	1,169	17.97	1.15(0.97-1.36)	1,170	19.15	1.19(1.01-1.39)	393	19.29	1.18(0.89-1.56)	
≥2	3,324	11.86	1,281	18.17	1.70(1.44-2.00)	980	14.67	1.32(1.11-1.57)	384	13.79	1.23(0.96-1.58)	
Missing	4	0.01	0.00	0.00		0.00	0.00		1	0.00		
Physical activity (No. of days/wk) *												
0	2,369	9.42	812	13.41	1.0 (Referent)	610	9.97	1.0 (Referent)	244	12.56	1.0 (Referent)	
1-3	7,245	25.24	1,908	30.24	0.84(0.67-1.05)	1,776	27.60	1.03(0.82-1.29)	684	30.35	0.90(0.58-1.39)	
4-6	12,131	40.71	2,328	35.19	0.61(0.49-0.75)	2,670	39.80	0.92(0.74-1.15)	961	34.11	0.63(0.42-0.94)	
7	6,614	23.80	1,189	20.22	0.59(0.47-0.76)	1,397	22.12	0.88(0.69-1.11)	599	22.75	0.72(0.47-1.09)	
Missing	214	0.82	56	0.92		42	0.52		15	0.24		
Neighborhood Amenities*, #												
0	1,026	3.22	310	4.31	1.0 (Referent)	276	3.71	1.0 (Referent)	80	2.30	1.0 (Referent)	
1	1,729	5.80	520	7.76	1.00(0.69-1.45)	442	6.76	1.02(0.68-1.53)	162	5.93	1.43(0.81-2.53)	
≥2	25,190	88.11	5,287	84.97	0.72(0.53-0.97)	5,608	87.02	0.86(0.61-1.19)	2,213	88.96	1.41(0.89-2.24)	
Missing	628	2.90	176	2.95		169	2.51		48	2.80		
Television watching, videos or playing video games (No. of hr./day) *												
0	1,829	6.45	228	4.23	1.0 (Referent)	274	4.92	1.0 (Referent)	181	6.44	1.0 (Referent)	
≤1	11,872	40.03	1,886	30.33	1.15(0.79-1.67)	2,285	35.43	1.16(0.80-1.67)	988	41.50	1.04(0.64-1.66)	
>1-<4	11,657	40.85	2,949	44.90	1.67(1.64-2.41)	2,988	43.30	1.39(0.97-1.99)	1,005	37.62	0.92(0.57-0.90)	
≥4	3,088	12.28	1,194	19.80	2.48(1.69-3.63)	910	15.85	1.70(1.16-2.50)	321	14.31	1.18(0.71-1.95)	
Missing	127	0.50	36	0.80		38	0.51		8	0.15		
Computer, cell phone, or electronic device (No. Of hr./day) *												
0	1,825	7.45	577	10.65	1.0 (Referent)	505	9.97	1.0 (Referent)	241	8.60	1.0 (Referent)	
≤1	14,105	47.54	2,688	39.64	0.58(0.46-0.73)	3,093	45.15	0.71(0.56-0.90)	1,293	50.64	0.92(0.69-1.23)	
>1-<4	8,369	28.51	1,873	30.15	0.74(0.59-0.93)	1,898	28.67	0.75(0.59-0.96)	662	26.20	0.79(0.58-1.08)	
≥4	4,020	15.47	1,079	18.10	0.82(0.63-1.05)	932	14.84	0.72(0.55-0.94)	285	13.37	0.75(0.51-1.10)	
Missing	254	1.03	76	1.47		67	1.37		22	1.21		

Adequate amount of sleep (No. of nights previous week) *											
0	799	2.78	188	3.48	1.0 (Referent)	181	3.59	1.0 (Referent)	72	2.94	1.0 (Referent)
1-3	2,206	7.45	452	6.68	0.72(0.46-1.11)	445	7.75	0.81(0.51-1.28)	181	7.66	0.97(0.51-1.85)
4-6	10,815	35.30	2,108	33.25	0.75(0.51-1.22)	2,255	31.64	0.69(0.46-1.05)	881	33.71	0.90(0.53-1.55)
7	14,506	53.34	3,493	55.77	0.84(0.56-1.24)	3,551	55.87	0.81(0.54-1.22)	1,348	55.37	0.98(0.58-1.66)
Missing	247	1.12	52	0.81		63	1.15		21	0.32	
Socioeconomic hardship*											
No	22,972	76.24	4,240	63.56	1.0 (Referent)	4,817	68.99	1.0 (Referent)	1,991	80.40	1.0 (Referent)
Yes	5,107	21.96	1,915	34.41	1.88(1.63-2.16)	1,531	28.87	1.45(1.26-1.67)	466	17.54	0.76(0.61-0.94)
Missing	494	1.79	138	2.03		147	2.14		46	2.06	
Parental divorce or separation*											
No	21,726	71.95	4,329	67.95	1.0 (Referent)	4,607	69.15	1.0 (Referent)	1,954	74.81	1.0 (Referent)
Yes	6,414	26.38	1,840	30.42	1.22(1.06-1.40)	1,765	28.86	1.14(0.99-1.30)	517	23.58	0.86(0.68-1.08)
Missing	433	1.67	124	1.63		123	1.98		32	1.60	
Bereavement*											
No	27,047	94.56	5,797	91.18	1.0 (Referent)	6,046	92.70	1.0 (Referent)	2,395	95.11	1.0 (Referent)
Yes	1,176	4.16	400	7.62	1.90(1.45-2.48)	350	5.63	1.38(1.05-1.81)	82	3.50	0.84(0.52-1.35)
Missing	350	1.28	96	1.20		99	1.67		26	1.39	
Incarcerated household member*											
No	26,530	91.33	5,532	87.40	1.0 (Referent)	5,823	87.77	1.0 (Referent)	2,317	91.42	1.0 (Referent)
Yes	1,635	7.19	639	11.21	1.63(1.33-1.99)	556	10.33	1.49(1.19-1.87)	154	6.93	0.96(0.69-1.34)
Missing	408	1.48	122	1.39		116	1.90		32	1.65	
Witnessing domestic violence*											
No	26,011	89.51	5,420	89.93	1.0 (Referent)	5,740	87.54	1.0(Referent)	2,283	87.96	1.0 (Referent)
Yes	2,000	8.37	711	12.12	1.51(1.23-1.84)	599	10.06	1.23(1.00-1.50)	169	9.69	1.18(0.81-1.72)
Missing	562	2.12	162	1.95		156	2.39		51	2.34	
Witnessing neighborhood violence*											
No	25,110	85.85	5,156	81.96	1.0 (Referent)	5,468	81.99	1.0 (Referent)	2,207	87.41	1.0 (Referent)
Yes	2,936	12.18	989	15.97	1.37(1.15-1.64)	872	15.44	1.33(1.11-1.58)	256	10.62	0.86(0.62-1.18)
Missing	527	1.97	148	2.07		155	2.57		40	1.97	
Household mental illness*											
No	25,012	88.07	5,283	84.06	1.0 (Referent)	5,543	84.76	1.0 (Referent)	2,184	86.25	1.0 (Referent)
Yes	3,057	10.19	873	13.87	1.42(1.19-1.71)	816	13.06	1.33(1.09-1.61)	283	11.64	1.17(0.87-1.56)
Missing	504	1.73	137	2.07		136	2.18		36	2.10	
Household substance abuse*											
No	24,409	84.54	5,112	82.02	1.0 (Referent)	5,386	82.06	1.0 (Referent)	2,163	82.86	1.0 (Referent)
Yes	3,735	13.72	1,061	16.46	1.24(1.04-1.47)	991	16.03	1.20(1.01-1.43)	311	15.42	1.15(0.85-1.55)
Missing	429	1.73	120	1.51		118	1.91		29	1.71	
Racial discrimination*											
No	26,600	91.88	5,730	90.28	1.0 (Referent)	5,965	90.44	1.0(Referent)	2,347	92.74	1.0 (Referent)
Yes	1,452	6.06	417	7.63	1.28(1.02-1.61)	402	7.24	1.21(0.95-1.55)	117	5.26	0.86(0.59-1.26)
Missing	521	2.06	146	2.09		128	2.31		39	2.00	

ACE (Had any of 9 asked about)*											
No	14,943	46.37	2,316	34.82	1.0 (Referent)	2,846	38.88	1.0 (Referent)	1,329	50.53	1.0 (Referent)
Yes	13,348	52.53	3,893	64.05	1.62(1.41-1.86)	3,566	59.76	1.36(1.19-1.54)	1,154	48.15	0.84(0.69-1.02)
Missing	282	1.09	84	1.12		83	1.36		20	1.32	
ACE Score**											
0	14,943	46.37	2,316	34.82	1.0 (Referent)	2,846	38.88	1.0 (Referent)	1,329	50.53	1.0(Referent)
1	6,669	25.30	1,646	24.99	1.31(1.12-1.54)	1,598	25.47	1.20(1.02-1.40)	577	21.51	0.78(0.62-0.98)
≥2	6,679	27.23	2,247	39.06	1.91(1.64-2.23)	1,968	34.28	1.50(1.30-1.73)	577	26.64	0.89(0.70-1.14)
Missing	282	1.09	84	1.12		83	1.36		20	1.32	

Note: OR= Crude multinomial generalized logistic regression Odds Ratio, CI= 95% Confidence Interval

BMICLASS: Underweight is BMI for age less than 5th percentile, Healthy weight is BMI 5th to 84th percentile, Overweight is BMI 85th to 94th percentile, and Obesity is BMI≥95 percentile

Missing value for BMICLASS [N (unweighted)=1,445, % (weighted)=4.77]; ACE (YES/NO) indicates presence or absence of any of the 9 ACEs asked about

The 0 or No categories of covariates are used as reference group for calculation of odds ratios; °Metropolitan Statistical Area (Only in states that meet the 500,000 threshold)

*Learning disability, ADD or ADHD, depression anxiety problems, Behavioral or conduct problems, autism or other autism spectrum disorder, developmental delay, intellectual disability, cerebral palsy, speech problems, asthma, Diabetes, Tourette syndrome, epilepsy or seizure disorder, hearing problems, vision problems, bone or joint problems, brain injury or concussion.

*The Wald Chi-square test for independence between all the covariates and BMICLASSES prevalence indicated significant association at significant level (p<0.001)

**The trend for increasing ORs for obesity relative to healthy-weight as the ACE score increases is significant at (p<0.001); #Neighborhood amenities include sidewalks, parks, recreation center or libraries

4.9.1 Crude and Covariate Adjusted (Final model) Association of BMI-Classes Relative to Healthy Weight:

Table 9 shows the crude and adjusted final models (adjusted odds ratio) estimates for BMI-classes, especially childhood obesity (primary outcome of interest) relative to healthy weight (reference group), among U.S. children aged 10-17 years. All the variables that had statistically significant association with the exposure of interest (adverse childhood experiences) and outcome of interest (BMI-class, specifically childhood obesity) were treated as confounders and thus included in the final models (Table 4.7; Table4.8).

The fully adjusted odds ratios are the results of multinomial generalized logistic regression analysis ¹⁵models for BMI levels with healthy weight as reference, that is, the odds ratios indicate the effect of each independent variable on each class of BMI for age [specifically childhood obesity (primary outcome), overweight and underweight] among the U.S. children 10-17 years old. After adjustment, the effect of neighborhood amenities, prematurity and adequate amount of sleep at night became statistically insignificant (didn't effect the odds of outcome for BMI-classes), across their all levels, for all BMI-classes relative to healthy weight (Table 4.9).

The adjusted odds ratio of covariates to their reference groups in the final models that were only statistically significant for childhood obesity relative to healthy weight encompassed: a) Place of residence in metropolitan statistical area, b) two or more chronic health conditions of 18 asked about, c) Watching TV, videos, or playing videos across categories >1 to <4 hours and ≥4 hours, d) family members in the

¹⁵ Fitting expected proportion values of obesity, overweight and underweight on healthy weight with a logit link function.

household eat a meal together 7 days of the week, e) and computer, cell phone or electronic device use ≤ 1 hour.

Moreover, the explanatory variables, namely, age, sex, physical health status of parents, physical activity were strongly related to childhood obesity (associated both with higher odds and lower odds of outcome) compared to overweight and underweight BMI categories. For example, girls had 41% lower odds to boys for obesity relative to healthy weight [$OR_{adjusted} = 0.59(0.51-0.68)$, $p < .0001$], 20% lower odds for overweight relative to healthy weight [$OR_{adjusted} = 0.80(0.71-0.91)$, $p = 0009$] and 31% lower odds for underweight relative to healthy weight [$OR_{adjusted} = 0.69(0.57-0.86)$, $p = 0008$]. Besides, the adjusted odds ratio of childhood obesity to Healthy weight for children with younger age (10-11 years) compared to older age categories (15-17 years) increased by a factor of 2.94 [95% CI (2.44-3.57), $p=0.0001$], for overweight outcome relative to healthy weight by 2.08 [95% CI (1.69-2.38), $p=0.0001$], and for underweight outcome relative to healthy weight by 2.70 [95% CI (2.08-3.45), $p= 0.0001$](Table 4.9).

4.9.2 ACE and Childhood Obesity:

Model 1- shows the crude or unadjusted model of obesity relative to healthy weight, model 2-fully adjusted odds ratio for the dichotomized category of ACE (Yes/No of 9 ACEs asked about) as the main independent variable, model 3-fully adjusted odds ratios for ACE score (0, 1, ≥ 2 as the main independent variable) in order to discover the trend of odds ratios for BMI classes (specially childhood obesity) relative to healthy weight as the number of ACEs increase, and finally model 4-fully adjusted odds ratios to determine which ACE category is the highest predictor (of all other categories) childhood obesity relative to healthy weight (Table 4.9).

The Model 2 indicates that the odds of childhood obesity relative to healthy weight is 17% higher among those children who experienced any of the ACE categories for child, of 9 asked about, compared to children who experienced no ACE, of 9 asked about given that all the other covariates are held constant in the model [$OR_{adjusted} = 1.17(1.01-1.35)$, $p < .0001$] (Table 4.9).

Similarly, Model 3 ascertained the dose response relationship of adverse childhood experiences for childhood obesity relative to healthy while adjusting for all the covariates in the model. For instance, children who were subject to ≥ 2 ACEs compared to those without exposure to any ACE category, of nine asked about had 1.27 times higher odds of childhood obesity relative to healthy weight while controlling for all the covariates in the model [$OR_{adjusted} = 1.17(1.01-1.35)$, $p < .006$]. However, for the children who experienced only one ACE (except socioeconomic hardship and bereavement), it didn't affect the odds of outcome and wasn't statistically significant (Table 4.9; Model4).

Model 4 provides evidence of the association specific categories of ACE as predictor of BMI classes, especially childhood obesity, relative to healthy weight while holding other variables constant in the model. Of all nine categories of ACEs for child, that was asked about only socioeconomic hardship and bereavement were statistically significant. Hence, children who lived in families with financial strains to cover their need such as food and shelter compared to those who were not subject to it, the odds for childhood obesity relative to healthy would be expected to increase by a factor of 1.34 [OR_{adjusted}= 1.34(1.15-1.56), p <. 0002]. Correspondingly, children who lived with parent who died compared to reference group, had 46% higher odds for childhood obesity relative to those with healthy weight BMI for age, while controlling for all covariates in the model [OR_{adjusted}= 1.46(1.09-1.94), p <. 009].

TABLE 4.9. Crude and Covariate Adjusted Odds Ratios[□] of Obesity; Overweight and Underweight Relative to Healthy Weight Among U.S. Children Aged 10-17 by Selected Demographic and Behavioral Characteristics. The 2011-2012 National Survey of Children's Health (N=45,309)

Covariate	Model 1 ^{***}	Model 2 ^{****}			Model 3 ^{*****}			Model 4 ^{*****}		
	Unadjusted odds ratio	Fully adjusted odds ratio for ACE (Yes/No)			Fully adjusted odds ratio for ACE Score (Yes/No)			Fully adjusted odds ratio for ACE categories (of 9 asked about)		
	OR OBESE VS. REF (95% CI)	OR OBESE VS. REF (95% CI)	OR OVERWT VS. REF (95% CI)	OR UNDERWT VS. REF (95% CI)	OR OBESE VS. REF (95% CI)	OR OVERWT VS. REF (95% CI)	OR UNDERWT VS. REF (95% CI)	OR OBESE VS. REF (95% CI)	OR OVERWT VS. REF (95% CI)	OR UNDERWT VS. REF (95% CI)
Age, yr.										
10-11	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
12-14	0.62(0.53-0.72)	0.55(0.47-0.65)*	0.77(0.66-0.91)	0.49(0.39-0.63)	0.55(0.47-0.65)*	0.77(0.66-0.91)	0.49(0.39-0.63)	0.55(0.47-0.65)*	0.77(0.66-0.91)	0.49(0.39-0.62)
15-17	0.42(0.35-0.49)	0.34(0.28-0.41)*	0.48(0.42-0.59)	0.37(0.29-0.48)	0.34(0.28-0.41)*	0.49(0.42-0.59)	0.37(0.29-0.48)	0.33(0.28-0.40)*	0.49(0.41-0.58)	0.37(0.29-0.48)
Sex										
Male	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
Female	0.62(0.54-0.70)	0.59(0.51-0.68)*	0.80(0.71-0.91)	0.69(0.57-0.86)	0.59(0.51-0.68)*	0.80(0.71-0.91)	0.69(0.56-0.86)	0.59(0.51-0.68)*	0.81(0.71-0.92)	0.69(0.56-0.85)
Place of residence										
Non-Metropolitan	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
Metropolitan	0.76(0.66-0.88)	0.81(0.69-0.94)*	0.91(0.77-1.07)	1.23(0.97-1.55)	0.81(0.69-0.95)*	0.91(0.77-1.07)	1.23(0.98-1.55)	0.80(0.68-0.94)*	0.91(0.77-1.07)	1.22(0.97-1.53)
Physical health status mother										
Excellent/very good	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
Good	2.01(1.71-2.36)	1.63(1.37-1.95)*	1.18(0.99-1.41)	1.17(0.88-1.56)	1.62(1.36-1.94)*	1.18(0.99-1.40)	1.16(0.88-1.55)	1.59(1.34-1.90)*	1.16(0.98-1.38)	1.18(0.89-1.56)
Fair/poor	2.51(2.09-3.01)	1.71(1.41-2.07)*	1.60(1.29-1.99)	0.96(0.66-1.39)	1.68(1.38-2.04)*	1.59(1.28-1.97)	0.95(0.66-1.37)	1.61(1.32-1.96)*	1.55(1.25-1.92)	0.97(0.67-1.41)
Physical health status father										
Excellent/very good	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
Good	1.78(1.48-2.13)	1.36(1.12-1.65)*	1.19(0.99-1.42)	0.92(0.69-1.24)	1.36(1.12-1.65)*	1.19(0.99-1.42)	0.92(0.69-1.24)	1.35(1.11-1.64)*	1.19(0.99-1.43)	0.91(0.68-1.21)
Fair/poor	2.23(1.76-2.81)	1.56(1.21-2.01)*	1.28(0.99-1.64)	1.14(0.73-1.79)	1.55(1.21-2.00)*	1.28(0.99-1.64)	1.13(0.72-1.77)	1.52(1.18-1.97)*	1.26(0.99-1.62)	1.13(0.73-1.77)
Family eats together (No. Of days/wk)										
0	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
1-3	0.73(0.54-0.98)	0.81(0.59-1.12)	1.02(0.76-1.34)	0.81(0.53-1.23)	0.82(0.59-1.13)	1.03(0.77-1.38)	0.81(0.53-1.24)	0.82(0.59-1.13)	1.02(0.77-1.36)	0.82(0.54-1.25)
4-6	0.77(0.58-1.03)	0.95(0.69-1.31)	1.08(0.82-1.43)	0.85(0.56-1.29)	0.96(0.70-1.32)	1.08(0.82-1.44)	0.86(0.56-1.30)	0.96(0.70-1.32)	1.08(0.82-1.44)	0.87(0.57-1.32)
7	1.31(0.98-1.74)	1.41(1.03-1.93)*	1.22(0.93-1.61)	0.93(0.61-1.42)	1.42(1.04-1.95)*	1.23(0.93-1.63)	0.94(0.61-1.43)	1.40(1.03-1.92)	1.22(0.92-1.61)	0.96(0.63-1.47)
Low birth weight (<2500G)										
No (Normal weight)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
Yes	1.09(0.88-1.34)	0.90(0.69-1.18)	0.97(0.75-1.24)	1.72(1.25-2.36)	0.91(0.69-1.18)	0.97(0.75-1.25)	1.72(1.25-2.36)	0.90(0.69-1.18)	0.97(0.75-1.24)	0.99(0.71-1.39)
Prematurity (≥ 3wks before his/her due date)										
No	1.0(Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0(Referent)	1.0 (Referent)	1.0 (Referent)
Yes	1.22(1.01-1.48)	1.09(0.86-1.39)	1.09(0.87-1.38)	1.06(0.76-1.48)	1.10(0.87-1.40)	1.10(0.87-1.39)	1.07(0.77-1.49)	1.09(0.86-1.39)	1.10(0.87-1.39)	1.07(0.76-1.49)
Ill health (chronic health conditions from a list of 18 conditions) •										
0	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
1	1.15(0.97-1.36)	1.03(0.86-1.23)	1.11(0.94-1.32)	1.18(0.91-1.53)	1.03(0.86-1.23)	1.11(0.94-1.31)	1.18(0.90-1.53)	1.02(0.86-1.22)	1.11(0.94-1.31)	1.17(0.90-1.51)
≥2	1.70(1.44-2.00)	1.19(1.00-1.43)*	1.06(0.88-1.27)	1.14(0.88-1.46)	1.18(0.99-1.41)	1.05(0.87-1.26)	1.12(0.87-1.44)	1.16(0.97-1.39)	1.03(0.85-1.24)	1.12(0.87-1.44)
Physical activity (No. of days/wk)										
0	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
1-3	0.84(0.67-1.05)	0.91(0.72-1.16)	1.07(0.85-1.34)	0.81(0.53-1.25)	0.91(0.72-1.16)	1.07(0.85-1.34)	0.81(0.53-1.25)	0.91(0.72-1.16)	1.06(0.85-1.33)	0.82(0.53-1.26)
4-6	0.61(0.49-0.75)	0.65(0.52-0.83)*	0.95(0.76-1.18)	0.53(0.35-0.81)	0.65(0.51-0.83)*	0.94(0.76-1.18)	0.53(0.35-0.81)	0.66(0.52-0.83)*	0.94(0.76-1.18)	0.53(0.35-0.81)
7	0.59(0.47-0.76)	0.49(0.38-0.64)*	0.79(0.61-1.01)	0.55(0.36-0.85)	0.49(0.37-0.63)*	0.78(0.61-1.00)	0.54(0.35-0.84)	0.49(0.38-0.64)*	0.78(0.61-1.00)	0.55(0.35-0.84)

Neighborhood Amenities ✧ 0 1 ≥2	1.0 (Referent) 1.00(0.69-1.45) 0.72(0.53-0.97)	1.0 (Referent) 1.00(0.69-1.45) 0.85(0.63-1.16)	1.0 (Referent) 1.03(0.69-1.53) 0.94(0.67-1.31)	1.0 (Referent) 1.45(0.82-2.58) 1.39(0.87-2.24)	1.0 (Referent) 1.00(0.69-1.45) 0.85(0.63-1.15)	1.0 (Referent) 1.02(0.68-1.53) 0.94(0.67-1.31)	1.0 (Referent) 1.44(0.81-2.56) 1.39(0.87-2.23)	1.0 (Referent) 1.04(0.72-1.49) 0.89(0.66-1.19)	1.0 (Referent) 1.04(0.69-1.54) 0.95(0.69-1.33)	1.0 (Referent) 1.44(0.81-2.56) 1.39(0.87-2.24)
Television watching, videos or playing video games (No. of hr./day) 0 ≤1 >1-<4 ≥4	1.0(Referent) 1.15(0.79-1.67) 1.67(1.64-2.41) 2.48(1.69-3.63)	1.0(Referent) 1.20(0.82-1.76) 1.46(1.00-2.14)* 1.86(1.25-2.77)*	1.0 (Referent) 1.16(0.79-1.69) 1.31(0.90-1.89) 1.57(1.06-2.33)	1.0 (Referent) 1.00(0.62-1.61) 0.87(0.54-1.38) 1.16(0.69-1.92)	1.0 (Referent) 1.20(0.82-1.76) 1.46(1.00-2.14)* 1.86(1.25-2.76)*	1.0 (Referent) 1.16(0.79-1.69) 1.30(0.89-1.89) 1.56(1.05-2.32)	1.0 (Referent) 0.99(0.62-1.59) 0.86(0.54-1.37) 1.56(1.05-2.32)	1.0 (Referent) 1.23(0.84-1.80) 1.49(1.02-2.19)* 1.88(1.26-2.80)*	1.0 (Referent) 1.17(0.80-1.71) 1.32(0.91-1.92) 1.58(1.06-2.34)	1.0 (Referent) 1.00(0.62-1.61) 0.87(0.54-1.39) 1.19(0.71-1.98)
Computer, cell phone, or electronic device (No. Of hr./day) 0 ≤1 >1-<4 ≥4	1.0 (Referent) 0.58(0.46-0.73) 0.74(0.59-0.93) 0.82(0.63-1.05)	1.0 (Referent) 0.77(0.62-0.98)* 0.99(0.77-1.27) 0.98(0.74-1.30)	1.0 (Referent) 0.83(0.64-1.07) 0.88(0.68-1.15) 0.79(0.59-1.07)	1.0 (Referent) 1.11(0.81-1.52) 1.12(0.79-1.58) 1.08(0.70-1.67)	1.0(Referent) 0.78(0.62-0.98)* 0.98(0.77-1.26) 0.97(0.74-1.29)	1.0 (Referent) 0.83(0.64-1.07) 0.88(0.68-1.15) 0.79(0.59-1.07)	1.0 (Referent) 1.11(0.81-1.53) 1.12(0.79-1.57) 1.08(0.69-1.66)	1.0 (Referent) 0.78(0.62-0.99)* 0.99(0.78-1.28) 0.99(0.75-1.31)	1.0 (Referent) 0.83(0.64-1.08) 0.89(0.68-1.16) 0.79(0.59-1.07)	1.0 (Referent) 1.10(0.80-1.51) 1.11(0.79-1.56) 1.06(0.69-1.63)
Adequate amount of sleep (No. Of nights previous week) 0 1-3 4-6 7	1.0 (Referent) 0.72(0.46-1.11) 0.75(0.51-1.22) 0.84(0.56-1.24)	1.0 (Referent) 0.79(0.51-1.25) 0.87(0.58-1.30) 0.86(0.57-1.29)	1.0 (Referent) 0.84(0.52-1.35) 0.71(0.46-1.09) 0.79(0.52-1.21)	1.0 (Referent) 1.16(0.61-2.21) 0.99(0.57-1.71) 1.01(0.59-1.74)	1.0 (Referent) 0.80(0.51-1.25) 0.87(0.58-1.31) 0.86(0.58-1.29)	1.0 (Referent) 0.84(0.53-1.36) 0.72(0.47-1.10) 0.79(0.52-1.22)	1.0 (Referent) 1.17(0.62-2.22) 0.99(0.58-1.72) 1.02(0.60-1.75)	1.0 (Referent) 0.81(0.52-1.27) 0.88(0.59-1.33) 0.87(0.58-1.30)	1.0 (Referent) 0.85(0.53-1.36) 0.72(0.47-1.10) 0.79(0.52-1.21)	1.0 (Referent) 1.18(0.63-2.21) 1.00(0.59-1.33) 1.04(0.62-1.74)
Socioeconomic hardship ⊙ No Yes	1.0 (Referent) 1.88(1.62-2.16)							1.0 (Referent) 1.34(1.15-1.56)*	1.0 (Referent) 1.18(1.01-1.38)	1.0 (Referent) 0.72(0.58-0.89)
Parental divorce or separation No Yes	1.0 (Referent) 1.22(1.06-1.40)							1.0 (Referent) 0.91(0.78-1.07)	1.0 (Referent) 0.95(0.81-1.12)	1.0 (Referent) 0.85(0.67-1.09)
Bereavement ⊙ No Yes	1.0 (Referent) 1.90(1.45-2.48)							1.0 (Referent) 1.46(1.09-1.94)*	1.0 (Referent) 1.18(0.89-1.58)	1.0 (Referent) 0.88(0.55-1.42)
Incarcerated household member No Yes	1.0 (Referent) 1.63(1.33-1.99)							1.0 (Referent) 1.11(0.89-1.39)	1.0 (Referent) 1.20(0.94-1.54)	1.0 (Referent) 0.90(0.59-1.38)
Witnessing domestic violence No Yes	1.0 (Referent) 1.51(1.23-1.84)							1.0 (Referent) 1.04(0.82-1.31)	1.0 (Referent) 0.91(0.73-1.15)	1.0 (Referent) 1.35(0.90-2.03)
Witnessing neighborhood violence No Yes	1.0 (Referent) 1.37(1.15-1.64)							1.0 (Referent) 0.96(0.78-1.19)	1.0 (Referent) 1.11(0.92-1.35)	1.0 (Referent) 0.84(0.60-1.17)
Household mental illness No Yes	1.0 (Referent) 1.42(1.19-1.71)							1.0 (Referent) 1.13(0.93-1.39)	1.0 (Referent) 1.12(0.90-1.39)	1.0 (Referent) 1.21(0.87-1.69)

Household substance abuse											
No	1.0 (Referent)								1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
Yes	1.24(1.04-1.47)								0.87(0.71-1.07)	0.96(0.77-1.18)	1.23(0.88-1.73)
Racial discrimination											
No	1.0 (Referent)								1.0 (Referent)	1.0 (Referent)	1.0 (Referent)
Yes	1.28(1.02-1.61)								1.19(0.94-1.52)	1.12(0.87-1.44)	0.94(0.64-1.39)
ACE (Had any of 9 asked about)**											
No	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)	1.0 (Referent)							
Yes	1.62(1.41-1.86)	1.17(1.01-1.35)*	1.14(0.99-1.32)	0.86(0.69-1.06)							
ACE Score***											
0	1.0 (Referent)				1.0 (Referent)	1.0 (Referent)	1.0 (Referent)				
1	1.31(1.12-1.54)				1.07(0.91-1.27)	1.08(0.91-1.27)	0.79(0.63-1.01)				
≥2	1.91(1.64-2.23)				1.27(1.07-1.52)*	1.22(1.03-1.44)	0.95(0.73-1.23)				

Note:

☞ Crude odds ratios not adjusted for covariates

☞☞ Fully adjusted odds ratios for covariates with ACE (YES/NO) exposure of 9 asked about

☞☞☞ Fully adjusted odds ratios for covariates with ACE Score (0, 1, ≥2) exposure of 9 asked about

☞☞☞☞ Fully adjusted odds ratios for covariates with ACE categories (Socioeconomic hardship, Parental Divorce or Separation, Incarcerated household member, Witnessing domestic violence, Witnessing neighborhood violence, Household mental illness, Household substance abuse and Racial discrimination) exposure of 9 asked about.

BMICLASS: Underweight is BMI for age less than 5th percentile, Healthy weight is BMI 5th to 84th percentile, Overweight is BMI 85th to 94th percentile, and Obesity is BMI ≥95 percentile

* Odds ratio with 95 % Confidence Interval is statistically significant for childhood obesity relative to healthy weight at $\alpha < .001$.

⊙ Indicates significant category of ACE (after adjusted for the covariates in the model) associated with obesity relative to healthy among those who were exposed to it.

● Learning disability, ADD or ADHD, depression anxiety problems, Behavioral or conduct problems, autism or other autism spectrum disorder, developmental delay, intellectual disability, cerebral palsy, speech problems, asthma, diabetes, Tourette Syndrome, epilepsy or seizure disorder, hearing problems, vision problems, bone or joint problems, brain injury or concussion.

* Neighborhood amenities include sidewalks, parks, recreation center or libraries

□ Parallel regression assumption or proportional odds assumption was rejected ($p < .0001$); therefore multinomial multivariate generalized model was used to model BMI-classes as nominal outcome (obese category=primary outcome of interest), REF=Healthy weight

Based on all the fully adjusted models (model 2,3 and 4) with the healthy weight as reference group, the probability of having childhood obesity, BMI-95th percentile or above was strongly related to ACE dichotomy, ACE score ≥2 and two ACE types (socioeconomic hardship and bereavement) than the probability of overweight, BMI-85th to 94th percentile. In all these above mentioned fully adjusted models, underweight-BMI less than 5th percentile was only found to be associated with socioeconomic hardship ACE category [OR_{adjusted}= 0.72(0.58-0.89), $p < .003$] (Table 4.9, model 4).

CHAPTER 5: DISCUSSION

5.1 DISCUSSION OF RESEARCH QUESTIONS:

Studies on the topic of adverse childhood experiences and childhood obesity collectively indicate an association, but there is the lack of replications in nationally representative sample of children aged 10-17 years.

The 2011-2012 NSCH in the U.S. among children aged 10-17 years data helped us study for the first time the individual and joint associations between adverse childhood experiences, including [new categories of ACEs] expanded on the original ACEs definition, and childhood obesity. The new items included into the definition of ACE were: a) socio-economic hardship, b) racial discrimination, c) witness/victim of neighborhood violence, and d) bereavement. The principle objectives of this study was to assess the relationship between the prevalence of ACEs and Childhood obesity, contribution of individual and joined categories of childhood adversity on levels of BMI, and which ACE exposures have stronger association with Childhood obesity in a 45,309 nationally representative sample of children 10-17 years of age with ACE, compared to those with No ACE in the United States.

Our estimates of the prevalence of childhood obesity, following healthy weight, was higher among children aged 10-17 years in the U.S. compared to overweight and underweight (Figure 5.1); this finding is similar to NSCH 2007. Boys had heavier weight compared to girls, which is consistent with other national estimates(Ogden, Carroll, Kit, & Flegal, 2012; Helton & Liechty, 2014; Ng et al., 2014; Ogden CL, Carroll MD, Kit BK, & Flegal KM, 2014).

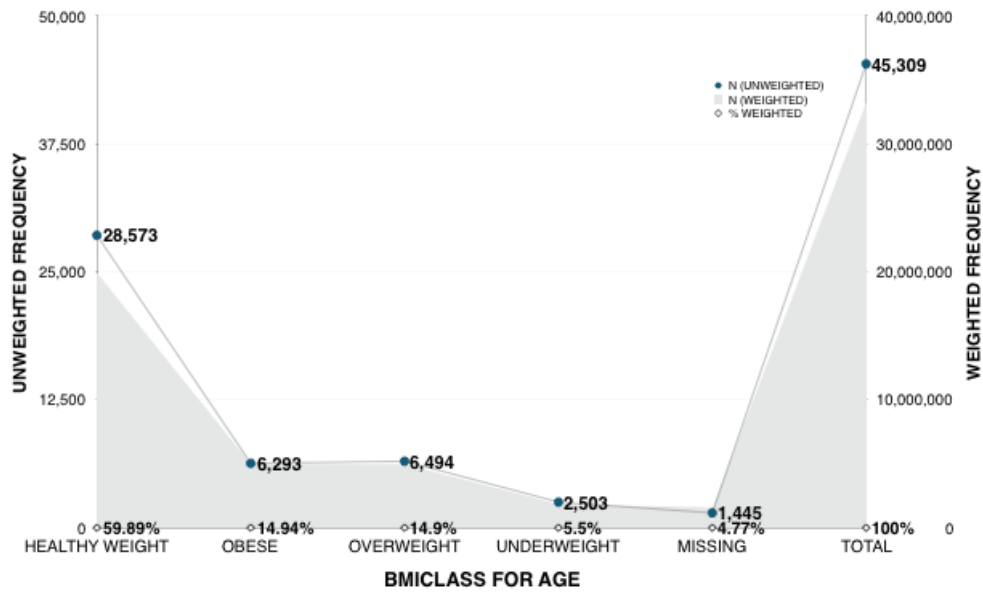


Figure 5.1 – BMI CLASS Distribution among U.S. Children Aged 10-17 years

The total prevalence of childhood obesity for all races adds up to 17.58% for boys and 12.21% for girls. Controlling for gender, among those who were obese, White-non-Hispanic children had the highest prevalence of obesity compared to other races for both genders (Table 4.4). However, this estimate is different compared to other national estimates (Ogden, C., & Carroll, M., 2010; Ogden CL et al., 2014; Fryar, C. D., Carroll, M. D., & Ogden, C. L. 2014). For instance, Ogden CL et al, (2014) results from the 2011-2012 National Health and Nutrition Examination Survey (NHANES) indicated that for the age category 12-19 years girls had higher obesity rates for the Non-Hispanic black followed by non-Hispanic white, and for the Hispanic and Asian race it was boys who had higher prevalence of obesity.

More than 50% of U.S. children had an experience to any ACE of 9 asked about. Almost 1 of every 3 children aged 10-17 was exposed to two or more ACE or in other terms approximately 25.4 million (89.5%) children aged 10-17 years experienced 3 or less ACE (Figure 4.3; Table 4.3).

The most prevalent ACE category of 9 asked about for child was living with parents who were either divorced or separated after his/her birth (26.77%) and the least prevalent was living with a parent who died (4.84 %)(Table 4.2). The distribution ACEs exposure was slightly higher among boys compared to girls. The median age for children who were exposed to adverse childhood experience and those who were obese was 14 and 13, respectively.

8 out of 10 obese and 6 out of 10 highly exposed to ACE [states] were located in the southern region of the United States. Among states with the top 10 highest levels of ACEs there seems to be a positive trend with their obesity distributions (Figure 5.2).

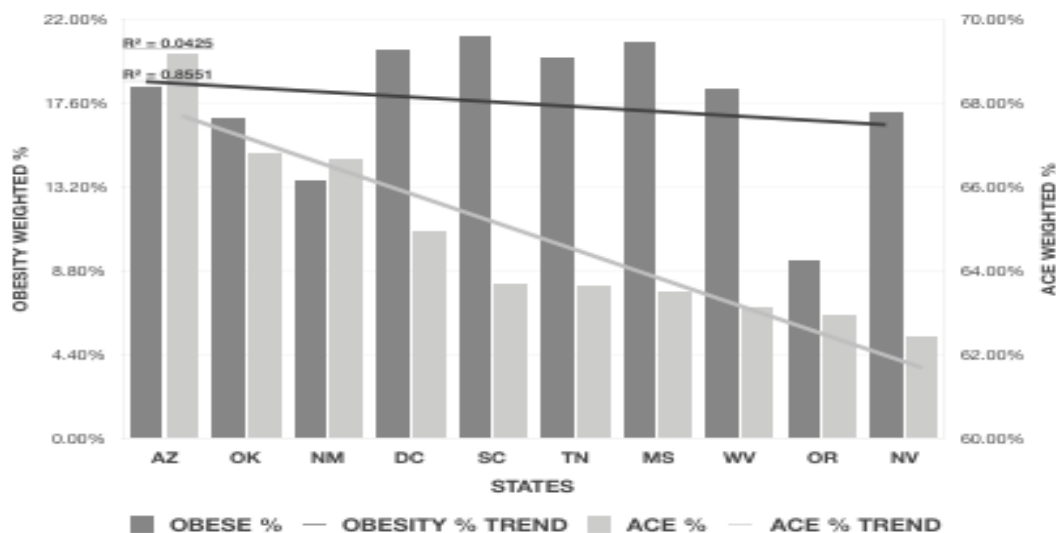


Figure 5.2 - Top 10 highest ACEs % (weighted) and its correlation with its corresponding obesity % (weighted) by states (including DC) in the U.S. among children 10-17 years. The 2011-2012 National Survey of Children’s Health.

Based on all the fully adjusted models (Table 4.9, Model 2,3 and 4) with the healthy weight as reference group, the probability of having childhood obesity, BMI-95th percentile or above was strongly related to ACE dichotomy, ACE score ≥ 2 and two ACE types (socioeconomic hardship and bereavement) than the probability of overweight, BMI-85th to 94th percentile. In all these above mentioned fully adjusted models,

underweight-BMI less than 5th percentile was only found to be associated with socioeconomic hardship ACE category (Table 4.9, model 4).

Therefore, prevalence and likelihood of childhood obesity-BMI \geq 95th was higher among children who experienced any of the ACE categories for child, of 9 asked about, compared to children who experienced no ACE, of 9 asked about, that is, the odds of having childhood obesity, BMI-95th percentile or above relative to healthy weight was strongly related to ACE dichotomy independent of the effect of several sociodemographic, parental, perinatal and infancy, and childhood related intervening variables.

Besides, the effect of ACEs on childhood obesity estimates was higher than their individual impacts and indicated a dose-response relationship, that is, ACEs association with childhood obesity is not mutually exclusive and may co-occur. Hence, an ACE score ≥ 2 would be only associated with an increased odds of childhood obesity, BMI \geq 95th percentile relative to healthy weight, BMI-5th to 95th percentile, than ACE score ≥ 1 compared to those without a history of adverse childhood experience.

The clinical importance of this finding is the cumulative effect of ACEs on childhood obesity. Previous ACE studies have also shown the aggregate effect of multiple ACEs on different detrimental health outcomes ; R. F. Anda et al., 1999; Dietz et al., 1999; S. R. Dube et al., 2001; Hillis, Anda, Felitti, & Marchbanks, 2001; Robert F. Anda et al., 2002; Shanta R. Dube et al., 2003; Dong et al., 2004; Edwards, Anda, Gu, Dube, & Felitti, 2007; Burke, Hellman, Scott, Weems, & Carrion, 2011; Ford et al., 2011).

Of all nine categories of ACEs for child, that was asked about only exposure to socioeconomic hardship and bereavement predicted statistically significant odds for childhood obesity. Hence, children who lived in families with financial strains to cover their need such as food and shelter compared to those who were not subject to it, and children who lived with parent who died compared to reference group, had higher odds for childhood obesity relative to those with healthy weight BMI for age and sex, while controlling for all covariates.

Moreover, ACEs were not mutually exclusive and all nine categories of ACEs were interrelated (Table 4.6, Table 4.5). Therefore, this result indicates that children are not subject to ACEs solitarily recognized the findings reproduced from an ACE study (Dong et al., 2004). Astonishingly, if a child had any one of the nine categories of ACEs (except racial discrimination), he/she was also simultaneously exposed mostly to parental divorce or separation (% ranges from 38.77 for socioeconomic hardship to 66.15% for Witnessing domestic violence); however, if a child was discriminated because of his/her race or ethnicity or lived with parent who was divorced or separated after his/her birth was at the same time for the most part living in households with socioeconomic hardship to finance their food or housing (34.43% and 36.46%, respectively) (Table 4.5).

On the other hand, 35.29% of the children who were subject to any one of the 9 ACEs (ACE score =1), irrespective of the specific type, were those living in families with socioeconomic hardship; 64,31 % of children with ACE Score=2, were living with parent who got divorced or separated after the child's birth (Table 4.5).

The adjusted odds ratio of covariates to their reference groups in the final models that were only statistically significant for childhood obesity relative to healthy weight encompassed: a) Place of residence in metropolitan statistical area, b) two or more chronic health conditions of 18 asked about, c) Watching TV, videos, or playing videos across categories >1 to <4 hours and ≥4 hours, d) family members in the household eat a meal together 7 days of the week, e) and computer, cell phone or electronic device use ≤1 hour.

Moreover, the explanatory variables-age categories, sex, physical health status of parents, physical activity were strongly related to childhood obesity (associated both with higher odds and lower odds of outcome) compared to overweight and underweight BMI categories.

5.2 STUDY STRENGTHS AND LIMITATIONS:

5.2.1 Strengths:

In comparison to our study, most of the retrospective cohort studies of the ACE study were based on the data collected from obesity clinic of Kaiser Permanente in San Diego, California (Felitti et al., 1998). However, we use a nationally representative sample of children and for the first time use the expanded categories of ACEs. To the extent of literature knowledge, our study provides information not available from other sources, that is, the study of new items related to ACEs at the national and state level [50 states including DC] for children aged 10-17 years in the U.S.

Due to the chronic nature of the outcome variable (childhood obesity) and one child survey weights used in our study, our findings are generalizable, both at national and state level (50 states including DC), to all non-institutionalized children aged 10-17 years. Besides, the weighted sample is adjusted for the non-response and non-coverage bias of families without telephones. Hence, the estimates are more reliable and precise. Non-response biases were corrected and adjusted for by application of sampling weights. The maximum estimated bias was 1.14 percentage points (NSCH, FAQs, 2011-2012). As listed random-digit-dialing method was used for this telephone survey, there is no class bias for those unlisted. To consider cultural and linguistic barriers NSCH questionnaire was translated into six languages, namely, English, Spanish, Mandarin, Cantonese, Vietnamese, and Korean.

To assess the role of missing values in differential misclassification-unadjusted odds ratios were calculated for missing values, for ACE exposure, when they were once treated as positive (ACE=1) and then as negative exposure to ACE (ACE=0) for all BMI-classes, respectively. Those who were classified as missing for exposure but would have been unexposed (ACE=missing, ACE2=0) and exposed (ACE=missing, ACE3=1) were analyzed separately. To assess and compare this potential effect, we performed our analysis after treating those with missing information on any category of ACES as unexposed (didn't have exposure to any category of ACE of 9 asked about) and exposed (experience any of the nine categories of ACES for child asked about). In all the repeated analysis, there wasn't a large and notable difference in the results (Table 5.1)

TABLE 5.2. Effect of ACE Missing Values on Childhood Obesity.

ACE BY BMICLASS INCLUDING MISSING					
1 ACE/BMICLASS	HEALTHY WEIGHT	OBESE	OVERWEIGHT	UNDERWEIGHT	OR OBESE VS.HEALTHY WEIGHT
ACE NO	14,943	2,316	2,846	1,329	1.88 (1.78-1.99)
ACE YES	13,348	3,893	3,566	1,154	
ACE BY BMICLASS (MISSING VALUES TREATED AS NEGATIVE)					
2 ACE2/BMICLASS2	HEALTHY WEIGHT	OBESE	OVERWEIGHT	UNDERWEIGHT	OR OBESE VS.HEALTHY WEIGHT
ACE NO	15,941	2,400	2,929	1,349	1.88 (1.78-1.98)
ACE YES	14,077	3,893	3,566	1,154	
3 ACE2/BMICLASS3	HEALTHY WEIGHT	OBESE	OVERWEIGHT	UNDERWEIGHT	OR OBESE VS.HEALTHY WEIGHT
ACE NO	15,225	2,400	2,929	2,065	1.89 (1.79-2.00)
ACE YES	13,348	3,893	3,566	1,883	
4 ACE2/BMICLASS4	HEALTHY WEIGHT	OBESE	OVERWEIGHT	UNDERWEIGHT	OR OBESE VS.HEALTHY WEIGHT
ACE NO	15,225	2,400	3,645	1,349	1.89 (1.79-2.00)
ACE YES	13,348	3,893	4,295	1,154	
5 ACE2/BMICLASS5	HEALTHY WEIGHT	OBESE	OVERWEIGHT	UNDERWEIGHT	OR OBESE VS.HEALTHY WEIGHT
ACE NO	15,225	3,116	2,929	1,349	1.69(1.61-1.78)
ACE YES	13,348	4,622	3,655	1,154	
ACE BY BMICLASS (MISSING VALUES TREATED AS POSITIVE)					
1 ACE3/BMICLASS2	HEALTHY WEIGHT	OBESE	OVERWEIGHT	UNDERWEIGHT	OR OBESE VS.HEALTHY WEIGHT
ACE NO	15,614	2,316	2,846	1,329	1.86 (1.76-1.97)
ACE YES	14,404	3,977	3,649	1,174	
2 ACE3/BMICLASS3	HEALTHY WEIGHT	OBESE	OVERWEIGHT	UNDERWEIGHT	OR OBESE VS.HEALTHY WEIGHT
ACE NO	14,943	2,316	2,846	2,000	1.88 (1.78-1.99)
ACE YES	13,630	3,977	3,649	1,948	
3 ACE3/BMICLASS4	HEALTHY WEIGHT	OBESE	OVERWEIGHT	UNDERWEIGHT	OR OBESE VS.HEALTHY WEIGHT
ACE NO	14,943	2,316	3,517	1,329	1.88 (1.78-1.99)
ACE YES	13,630	3,977	4,423	1,174	
4 ACE3/BMICLASS5	HEALTHY WEIGHT	OBESE	OVERWEIGHT	UNDERWEIGHT	OR OBESE VS.HEALTHY WEIGHT
ACE NO	14,943	2,987	2,846	1,329	1.74(1.66-1.83)
ACE YES	13,630	4,751	3,649	1,174	

Note: Odds Ratios of Obesity for those with ACE vs. No ACE are unadjusted and are calculated from unweighted frequencies.

5.2.2 Limitations:

Our findings of measure of exposure and outcome are proxies of mean distributions at the population level. Therefore, caution is required to prevent erroneous application of the observed associations at individual level. Hence, individual differences are masked. The secondary data set use in our study collected BMI information only on children aged 10-17 years, although other variables were measured for children age 0-17 years; therefore, we used this age category as our study population.

The respondents of each complete NSCH 2011-2012 interview was mostly each child’s mother 68.6% (biological, step, foster, or adoptive), followed by the father 24.2% (biological, step, foster, or adoptive), and other guardians 7.2%, who knew about the health and health care status of the sampled child in the house.

Therefore, recall bias, is a potential limitation due to an erroneous recollection of ACEs exposure and children BMI level (children's weight were not ascertained either directly by a physician or medical records). On the top of recall bias, additional biases may be possible, acquiescence bias "YEA" saying effect, Conformism bias " socially desirable effect", and Prevarication bias, viz. respondents providing answers that might be either not true or could be lies.

The weighted estimates are only generalizable to children age 10-17 years, but not to their parents and pediatric health care providers, and even if the question was referred to child's parents the result are reported "in terms of children"(2011-2012 NSCH: CHI and Subgroups SAS Codebook, Version 1.0' 2013). Nonetheless, to prevent antecedent-consequent bias, it is difficult to provide strong temporal evidence for ACE and Childhood obesity occurrence.

In our study the three abuse categories (physical, sexual and psychological) and two neglect types (physical and emotional) related to ACE were not measured. Hence, the strength of association for the gender difference (female vs. male) of obesity relative to healthy weight, among those with ACE vs. No ACE, would have been weaker than observed had the abuse and neglect ACE groups been included in our study.

Children and adolescents with missing information (coded as DK/Ref/Missing in error/Partial interview to all 9 ACES) for ACE and BMI class (Missing in error) were considered separate categories in the analysis (N [unweighted] = 514; 1.27% [weighted] and N=1,445[unweighted]; 4.77% [weighted], respectively). The detailed description of the survey limitations and strengths used in our study is published elsewhere (NSCH, FAQs, 2011-2012).

5.3 IMPLICATIONS OF FINDINGS:

More than 50% of U.S. children had experience to any ACE of nine asked about. Almost one of every three children aged 10-17 was exposed to two or more ACE or in other terms approximately 25.4 million (89.5%) children aged 10-17 years experienced three or less ACE (Figure 3.3, Table3.3).

In regards to public health Implications - there is no specific regulation for ACEs as an indicator for early detection of childhood obesity and diagnosis, and management or monitoring of adverse childhood experiences (including the new ACEs categories included in our study) either at local or state level. Even though the WHO-commission on ending childhood obesity has recently been published, there isn't any direct endorsement for ACEs relationship to childhood obesity (WHO, ECHO, 2016). Therefore, consideration is sagacious to establish regulatory measures both at the state and country levels in order include ACEs as one of the developmental and child health detrimental factors in the life course.

Researchers studied the treatment success rate in a short and long term among obese children who had prior ACEs to those who didn't. The striking result was that children with higher ACE scores compared to those with low ACE Scores were still more obese (even 1 year after surgery), had higher levels of total cholesterol and low-density lipoprotein (Lodhia et al., 2015).

Potential Mechanisms by Which ACEs Lead to Childhood Obesity:

The exact mechanism through which ACEs lead to Childhood obesity is yet to be understood. However, in a systematic review by Danese & Tan (2014) the authors found that childhood maltreatment is associated with a chronic inflammatory state, increase proinflammatory cytokines and C-reactive protein, independent of other comorbid factors. However, the authors urged that the precise mechanism through which chronic inflammatory state mediates the association between childhood maltreatment and adverse health consequences (morbidity and mortality) is yet to be understood.

However, the authors of original ACE study has developed a socio-ecologic model (also referred to as ACE Pyramid) of ACEs channel of action that leads to different detrimental health outcomes in a long run (Felitti et al., 1998). The authors use a whole life perspective model that considers periods-from conception to death (Felitti et al., 1998). In this model, the first line of invasion by ACEs is nervous system during child's nervous system development period. The aftermaths grave repercussion for disabilities, detrimental health outcomes and eventually premature death (Figure 5.3).

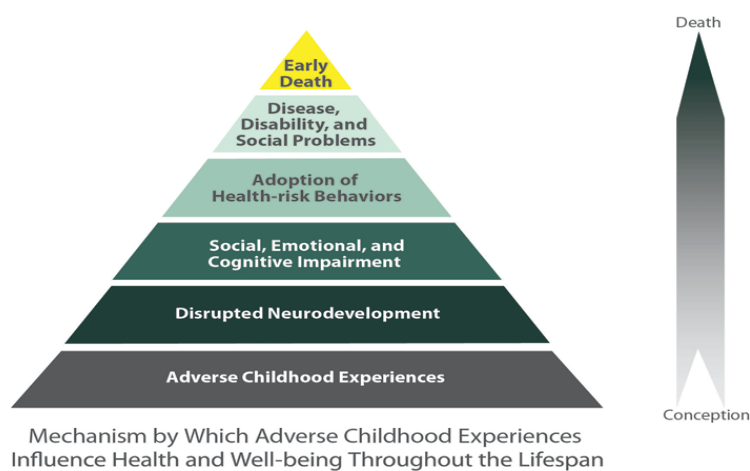


Figure 5.3 - The ACE Pyramid: Conceptual Framework for the ACE Study

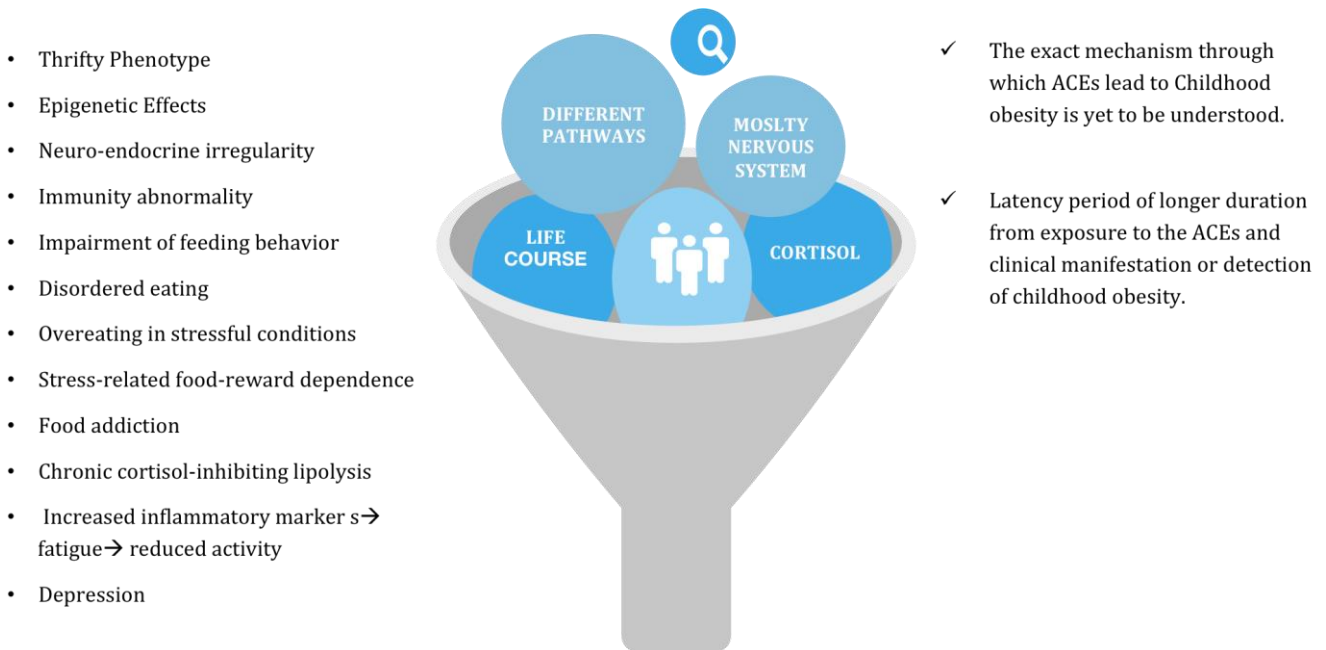


Figure 5.4 - Potential Mechanisms of Action of ACE leading to Childhood Obesity

Figure 5.4, above, summarizes more than 10 mechanisms and pathways through which childhood maltreatment (physical abuse, sexual abuse, emotional abuse, physical neglect, emotional neglect or family violence) compared to control groups (without obesity and maltreatment), may lead to childhood obesity and has been comprehensively reviewed in a Meta-analysis of 41 studies (N=190,285) among children less than 18 years of age (Danese & Tan, 2014).

5.4 RECOMMENDATIONS AND PREVENTION STRATEGIES:

ACEs are generally those families of psychosocial adversaries that are modifiable. ACEs should not merely be considered as an individual but also societal health issues. As ACEs increase so does the risk of childhood obesity. Therefore, it sagacious and advisable to establish evidence-based national guidelines, protocols, or standards for the management of ACEs through a primary care approach; especially prioritizing the southern states due to the higher prevalence.

On the other hand, there is a dire need for perspicacious and multisectoral preventative measures to reduce the burden of [childhood obesity] epidemic resulting from multi-component causal factors, neither necessary nor sufficient, especially familial psychosocial afflictions.

Therefore, there are numerous reasons for the early prevention of childhood obesity (Figure 5.5). Firstly, if current trends continue the number of overweight or obese infants and young children globally will increase to 70 million by 2025 (WHO, facts and figures on childhood obesity, 2014). Secondly, childhood obesity puts children's physical, mental and social well-being at risk. Thirdly, Obese children are more likely to become obese adults. If proper actions are not taken, this will endanger not only current generation but also the generations to come. Finally, if childhood obesity is not properly managed its burden will consequently reach levels beyond control of all stakeholders.

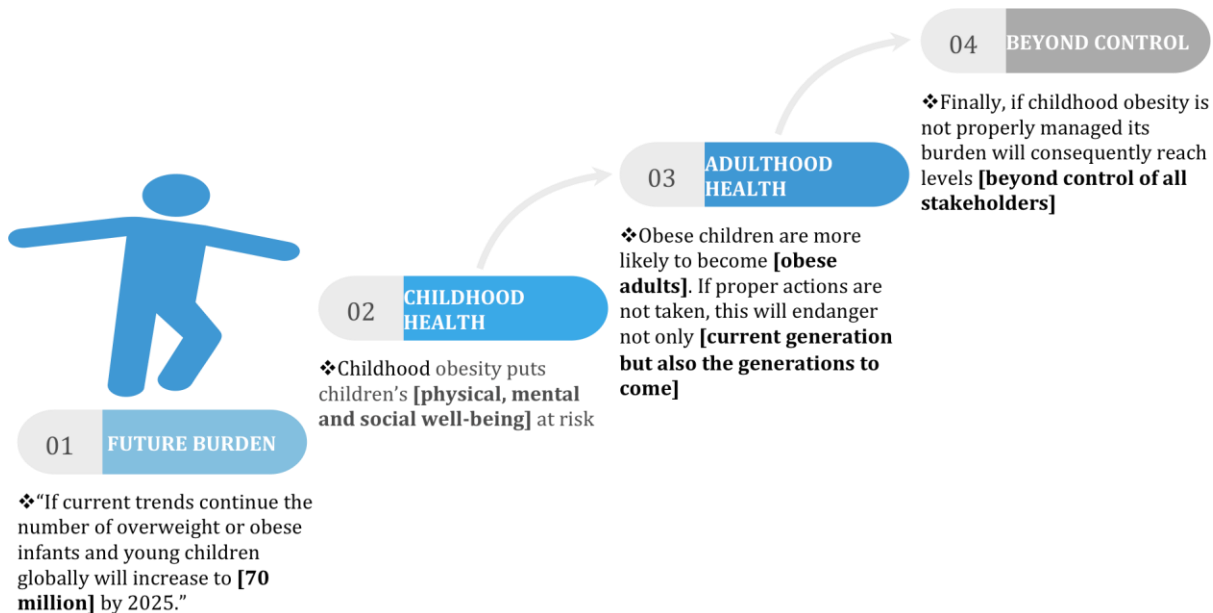


Figure 5.5 - Reasons for the Early Prevention and Control of Childhood Obesity

There is no one size fits all solution for childhood obesity, and there is a need for considering: a) contextual multisectoral and non-vertical integrated approach b) consideration of three critical life course periods, namely, preconception and pregnancy, infancy and early childhood, older childhood and adolescence, c) managing obesogenic environments, and d) treatment of children who are already obese (WHO, ECHO, 2014). Hence, prevention seems the best treatment for childhood obesity.

5.5 FURTHER RESEARCH QUESTIONS:

In the future, it is required to distinguish the pathophysiological pathways through which ACEs causes obesity and what are the best ways to reverse and stop its further systematic damage in the body. Besides, there is a dire need to evaluate the impact of existing and recently established preventative and therapeutic measures for ACEs and its after-effects on childhood obesity.

In addition, experimental studies will help to test the contribution of screening children for ACEs as indicator for early detection and early management of childhood obesity, among other measures? Analytical observational studies, namely, case-control, nested case-control, and cohort studies are needed to provide evidence of cause and effect at the individual level for ACEs (used for this study and original ACE items) and childhood obesity. Besides, qualitative studies in the future will be helpful not only to generate new research questions and areas for research but also scrutinizing the knowledge, attitude and perception of people regarding ACEs and childhood obesity.

Hence, it is advisable that future studies use national surveys that include children both 0-9 and older, to compare and contrast the association of ACEs exposure [both the expanded categories in our included in our study and original ACE categories] to childhood obesity in the U.S. and a new context. For instance: What is the difference in the magnitude of association between ACEs and childhood obesity in countries with collectivistic or socialistic norms compared to individualistic societies (e.g., Afghanistan or China vs. The United States; Figure 5.6).

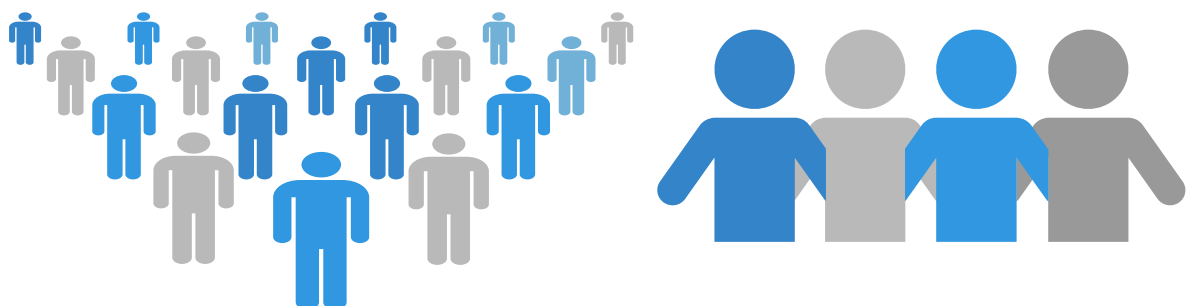


Figure 5.6 - Association between ACEs and Childhood Obesity in individualistic vs. collectivistic societies

Answers to these questions not only help in foundation of public policies but also propound new hypothesis for future research not only at the ecologic level but also individual level regarding the topic ACEs association (including new ACE items in our study) and BMI levels, especially childhood obesity.

5.6 CONCLUSION:

This is the first study to explore the co-occurrence, individual and joint association of ACEs with childhood obesity using nationally representative sample of children aged 10-17 years in the U.S. Having childhood obesity, BMI-95th percentile or above was strongly related to ACE dichotomy, ACE score ≥ 2 and two ACE types (socioeconomic hardship and bereavement) than the probability of overweight, BMI-85th to 94th percentile (Figure 5.7). Underweight-BMI less than 5th percentile had only statistically significant association with socioeconomic hardship ACE category. Sociodemographic, parental, and childhood related factors were also independently associated with childhood obesity.

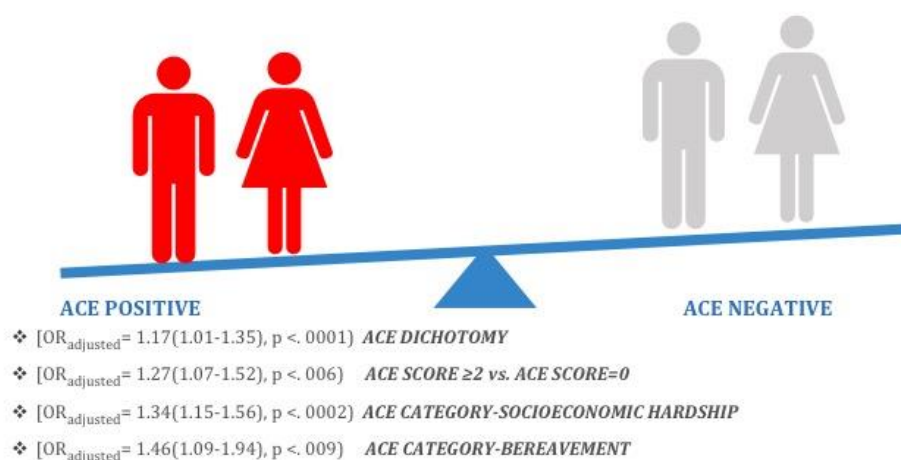


Figure 5.7 – Conclusion of ACEs Association with Childhood Obesity

REFERENCES

- Aim for a healthy weight: Maintaining a Healthy Weight On the Go A Pocket Guide. (2014, August). Retrieved June 9, 2016, from http://www.nhlbi.nih.gov/files/docs/public/heart/AIM_Pocket_Guide_tagged.pdf
- Alvarez, J., Pavao, J., Baumrind, N., & Kimerling, R. (2007). The relationship between child abuse and adult obesity among California women. *American Journal of Preventive Medicine*, 33(1), 28–33. <http://doi.org/10.1016/j.amepre.2007.02.036>
- AMA News Room. (2013, June 18). Retrieved March 07, 2016, from <http://www.ama-assn.org/ama/pub/news/news/2013/2013-06-18-new-ama-policies-annual-meeting.page>
- Anda, R. F., Croft, J. B., Felitti, V. J., Nordenberg, D., Giles, W. H., Williamson, D. F., & Giovino, G. A. (1999). Adverse childhood experiences and smoking during adolescence and adulthood. *JAMA*, 282(17), 1652–1658.
- Anda, R. F., Whitfield, C. L., Felitti, V. J., Chapman, D., Edwards, V. J., Dube, S. R., & Williamson, D. F. (2002). Adverse childhood experiences, alcoholic parents, and later risk of alcoholism and depression. *Psychiatric Services (Washington, D.C.)*, 53(8), 1001–1009. <http://doi.org/10.1176/appi.ps.53.8.1001>
- Arenz, S., Rückerl, R., Koletzko, B., & Kries, R. von. (2004). Breast-feeding and childhood obesity--a systematic review. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, 28(10), 1247–1256. <http://doi.org/10.1038/sj.ijo.0802758>
- Barker, D. J., & Osmond, C. (1986). Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet (London, England)*, 1(8489), 1077–1081.

Barlow, S. E., & Expert Committee. (2007). Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. *Pediatrics*, 120 Suppl 4, S164–192. <http://doi.org/10.1542/peds.2007-2329C>

Body weight and obesity in adults and self-reported abuse in childhood. (2002). ,
Published Online: 16 July 2002; | doi:10.1038/sj.ijo.0802038, 26(8).
<http://doi.org/10.1038/sj.ijo.0802038>

Burke, N. J., Hellman, J. L., Scott, B. G., Weems, C. F., & Carrion, V. G. (2011). The impact of adverse childhood experiences on an urban pediatric population. *Child Abuse & Neglect*, 35(6), 408–413. <http://doi.org/10.1016/j.chiabu.2011.02.006>

Centers for Disease Control and Prevention. Children's Food Environment State Indicator Report, 2011. (2011). Retrieved March 15, 2016, from <http://www.cdc.gov/obesity/downloads/ChildrensFoodEnvironment.pdf>

Centers for Disease Control and Prevention (CDC). Childhood Obesity Causes & Consequences. (2015, June 19). Retrieved June 09, 2016, from <http://www.cdc.gov/obesity/childhood/causes.html>

Centers for Disease Control and Prevention (CDC). *Child Maltreatment: Definitions*. (2015, March 16). Retrieved March 07, 2015, from <http://www.cdc.gov/violenceprevention/childmaltreatment/definitions.html>

Centers for Disease Control and Prevention (CDC). *Controlling the global obesity epidemic*. (n.d.). Retrieved April 03, 2016, from <http://www.who.int/nutrition/topics/obesity/en/>

Centers for Disease Control and Prevention (CDC). *Defining Childhood Obesity*. (2015, June 19). Retrieved April 03, 2016, from <http://www.cdc.gov/obesity/childhood/defining.html>

Centers for Disease Control and Prevention, National Center for Health Statistics, State and Local Area Integrated Telephone Survey. 2011-2012 National Survey of Children's Health Frequently Asked Questions. April 2013. Available from URL: <http://www.cdc.gov/nchs/slaits/nsch.htm>

Centers for Disease Control and Prevention. State Indicator Report on Physical Activity, 2014. Atlanta, GA: U.S. Department of Health and Human Services, 2014. You can see more information about the State Indicator Report on Physical Activity, 2014 at: www.cdc.gov/physicalactivity/resouces/reports.html

Chatterjea, M. N., & Shinde, R. (2012). Diet and Nutrition/Obesity. In Textbook of medical biochemistry (8th ed., pp. 763-769). New Delhi: Jaypee Brothers Medical Publications (P).

Child and Adolescent Health Measurement Initiative (2012). "2011/12 National Survey of Children's Health (2012), Sampling and Survey Administration." Data Resource Center, supported by Cooperative Agreement 1-U59-MC06980-01 from the U.S. Department of Health and Human Services, Health Resources and Services Administration (HRSA), Maternal and Child Health Bureau (MCHB). Available at www.childhealthdata.org

Collum, D. M. (2011, February 02). COLEVA | Consequences of Lifetime Exposure to Violence Abuse. Retrieved June 11, 2016, from <http://www.coleva.net/COLEVA-Main-2-2-2011-v2.html>

Comuzzie, A. G., & Allison, D. B. (1998). The search for human obesity genes. *Science* (New York, N.Y.), 280(5368), 1374–1377.

Danese, A., & Tan, M. (2014). Childhood maltreatment and obesity: systematic review and meta-analysis. *Molecular Psychiatry*, 19(5), 544–554. <http://doi.org/10.1038/mp.2013.54>

- Daniels, S. R. (2006). The consequences of childhood overweight and obesity. *The Future of Children / Center for the Future of Children, the David and Lucile Packard Foundation*, 16(1), 47–67.
- Dietz, P. M., Spitz, A. M., Anda, R. F., Williamson, D. F., McMahon, P. M., Santelli, J. S., ... Kendrick, J. S. (1999). Unintended pregnancy among adult women exposed to abuse or household dysfunction during their childhood. *JAMA*, 282(14), 1359–1364.
- Dong, M., Anda, R. F., Felitti, V. J., Dube, S. R., Williamson, D. F., Thompson, T. J., ... Giles, W. H. (2004). The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect*, 28(7), 771–784.
<http://doi.org/10.1016/j.chiabu.2004.01.008>
- Dube, S. R., Anda, R. F., Felitti, V. J., Croft, J. B., Edwards, V. J., & Giles, W. H. (2001). Growing up with parental alcohol abuse: exposure to childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect*, 25(12), 1627–1640
- Dube, S. R., Anda, R. F., Felitti, V. J., Edwards, V. J., & Croft, J. B. (2002). Adverse childhood experiences and personal alcohol abuse as an adult. *Addictive Behaviors*, 27(5), 713–725.
- Dube, S. R., Felitti, V. J., Dong, M., Chapman, D. P., Giles, W. H., & Anda, R. F. (2003). Childhood abuse, neglect, and household dysfunction and the risk of illicit drug use: the adverse childhood experiences study. *Pediatrics*, 111(3), 564–572.
- Dube, S. R., Miller, J. W., Brown, D. W., Giles, W. H., Felitti, V. J., Dong, M., & Anda, R. F. (2006). Adverse childhood experiences and the association with ever using alcohol and initiating alcohol use during adolescence. *The Journal of Adolescent Health: Official Publication of the Society for Adolescent Medicine*, 38(4), 444.e1–10. <http://doi.org/10.1016/j.jadohealth.2005.06.006>
- Edwards, V. J., Anda, R. F., Gu, D., Dube, S. R., & Felitti, V. J. (2007). Adverse childhood experiences and smoking persistence in adults with smoking-related symptoms and illness. *The Permanente Journal*, 11(2), 5–13.

- Eisenman, J. C., Sarzynski, M. A., Tucker, J., & Heelan, K. A. (2010). Maternal prepregnancy overweight and offspring fatness and blood pressure: role of physical activity. *Pediatric Exercise Science*, 22(3), 369–378.
- Endocrine Disorders/Obesity. (2012). In K. M. Goel & D. K. Gupta (Eds.), *Hutchison's paediatrics* (2nd ed., pp. 438-439). New Delhi, India: Jaypee Brothers Medical.
- Environmental and Nutritional factors in disease/Disorders of altered energy balance. (2014). In B. R. Walker, N. R. Colledge, S. H. Ralston, & I. D. Penman (Eds.), *Davidson's principles and practice of medicine* (22d ed., pp. 115-117). China: Churchill Livingstone Elsevier.
- Fang, X., Brown, D. S., Florence, C. S., & Mercy, J. A. (2012). The economic burden of child maltreatment in the United States and implications for prevention. *Child Abuse & Neglect*, 36(2), 156–165. <http://doi.org/10.1016/j.chiabu.2011.10.006>
- Felitti, V. J. (1991). Long-term medical consequences of incest, rape, and molestation. *Southern Medical Journal*, 84(3), 328–331.
- Felitti, V. J. (1993). Childhood sexual abuse, depression, and family dysfunction in adult obese patients: a case control study. *Southern Medical Journal*, 86(7), 732–736.
- Felitti, V. J. (2003). [Origins of addictive behavior: evidence from a study of stressful childhood experiences]. *Praxis Der Kinderpsychologie Und Kinderpsychiatrie*, 52(8), 547–559.
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ... Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 14(4), 245–258.

- Finkelhor, D., Turner, H. A., Shattuck, A., & Hamby, S. L. (2013). Violence, crime, and abuse exposure in a national sample of children and youth: an update. *JAMA Pediatrics*, 167(7), 614–621. <http://doi.org/10.1001/jamapediatrics.2013.42>
- Finkelstein, E. A., Trogon, J. G., Cohen, J. W., & Dietz, W. (2009). Annual medical spending attributable to obesity: payer-and service-specific estimates. *Health Affairs (Project Hope)*, 28(5), w822–831. <http://doi.org/10.1377/hlthaff.28.5.w822>
- Ford, E. S., Anda, R. F., Edwards, V. J., Perry, G. S., Zhao, G., Li, C., & Croft, J. B. (2011). Adverse childhood experiences and smoking status in five states. *Preventive Medicine*, 53(3), 188–193. <http://doi.org/10.1016/j.ypmed.2011.06.015>
- Fraser, A., Tilling, K., Macdonald-Wallis, C., Sattar, N., Brion, M.-J., Benfield, L., ... Lawlor, D. A. (2010). Association of maternal weight gain in pregnancy with offspring obesity and metabolic and vascular traits in childhood. *Circulation*, 121(23), 2557–2564. <http://doi.org/10.1161/CIRCULATIONAHA.109.906081>
- Fryar, C. D., Carroll, M. D., & Ogden, C. L. (2014, September 19). Prevalence of Overweight and Obesity Among Children and Adolescents: United States, 1963–1965 Through 2011–2012. Retrieved June 15, 2016, from http://www.cdc.gov/nchs/data/hestat/obesity_child_11_12/obesity_child_11_12.htm
- Freedman, D. S., Khan, L. K., Dietz, W. H., Srinivasan, S. R., & Berenson, G. S. (2001). Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics*, 108(3), 712–718.
- Fuemmeler, B. F., Dedert, E., McClernon, F. J., & Beckham, J. C. (2009). Adverse childhood events are associated with obesity and disordered eating: results from a U.S. population-based survey of young adults. *Journal of Traumatic Stress*, 22(4), 329–333. <http://doi.org/10.1002/jts.20421>

- Gaines, K. K. (2015). The Elephant in the Room - Pandemic Obesity. *Urologic Nursing*, 35(4), 161–163.
- Garasky, S., Stewart, S. D., Gundersen, C., Lohman, B. J., & Eisenmann, J. C. (2009). Family stressors and child obesity. *Social Science Research*, 38(4), 755–766.
<http://doi.org/10.1016/j.ssresearch.2009.06.002>
- Garrow, J. S., & Webster, J. (1985). Quetelet's index (W/H²) as a measure of fatness. *International Journal of Obesity*, 9(2), 147–153.
- Georgia State University: Policy for Publicly Available, Archival, and Secondary Data – University Research Services Administration. (n.d.). Retrieved June 10, 2016, from <http://ursa.research.gsu.edu/ursa/compliance/human-subjects/policy-for-publicly-available-archival-and-secondary-data/>
- Glossary of Terms. Data Resource Center for Child and Adolescent Health. (n.d.). Retrieved June 29, 2016, from <http://childhealthdata.org/help/glossary>
- Goel, K.M., & Gupta, K.M. (2012). Endocrine Disorders/Obesity (Eds.), Hutchison's paediatrics (2nd ed., pp. 438-439). New Delhi, India: Jaypee Brothers Medical.
- Hanson, M. A., & Gluckman, P. D. (2014). Early developmental conditioning of later health and disease: physiology or pathophysiology? *Physiological Reviews*, 94(4), 1027–1076. <http://doi.org/10.1152/physrev.00029.2013>
- Healthy People 2020. *Nutrition and Weight Status*. (2010, December 02). Retrieved March 17, 2016, from <https://www.healthypeople.gov/2020/topics-objectives/topic/nutrition-and-weight-status/objectives>
- Helton, J. J., & Liechty, J. M. (2014). Obesity prevalence among youth investigated for maltreatment in the United States. *Child Abuse & Neglect*, 38(4), 768–775.
<http://doi.org/10.1016/j.chiabu.2013.08.011>

- Hillis, S. D., Anda, R. F., Felitti, V. J., & Marchbanks, P. A. (2001). Adverse childhood experiences and sexual risk behaviors in women: a retrospective cohort study. *Family Planning Perspectives, 33*(5), 206–211.
- Himes, J. H., & Dietz, W. H. (1994). Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. The Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services. *The American Journal of Clinical Nutrition, 59*(2), 307–316.
- Kohn, M., & Booth, M. (2003). The worldwide epidemic of obesity in adolescents. *Adolescent Medicine (Philadelphia, Pa.), 14*(1), 1–9.
- Johnson, L., Mander, A. P., Jones, L. R., Emmett, P. M., & Jebb, S. A. (2008). A prospective analysis of dietary energy density at age 5 and 7 years and fatness at 9 years among UK children. *International Journal of Obesity (2005), 32*(4), 586–593. <http://doi.org/10.1038/sj.ijo.0803746>
- Johnson, L., Mander, A. P., Jones, L. R., Emmett, P. M., & Jebb, S. A. (2008). Energy-dense, low-fiber, high-fat dietary pattern is associated with increased fatness in childhood. *The American Journal of Clinical Nutrition, 87*(4), 846–854.
- Kaphingst, K. M., & Story, M. (2009). Child care as an untapped setting for obesity prevention: state child care licensing regulations related to nutrition, physical activity, and media use for preschool-aged children in the United States. *Preventing Chronic Disease, 6*(1), A11.
- Kohn, M., & Booth, M. (2003). The worldwide epidemic of obesity in adolescents. *Adolescent Medicine (Philadelphia, Pa.), 14*(1), 1–9.
- Koplan, J. P., Liverman, C. T., Kraak, V. I., & Committee on Prevention of Obesity in Children and Youth. (2005). Preventing childhood obesity: health in the balance: executive summary. *Journal of the American Dietetic Association, 105*(1), 131–138. <http://doi.org/10.1016/j.jada.2004.11.023>

- Krebs, N. F., Jacobson, M. S., & American Academy of Pediatrics Committee on Nutrition. (2003). Prevention of pediatric overweight and obesity. *Pediatrics*, 112(2), 424–430.
- Kuczmarski, R. J., Ogden, C. L., Grummer-Strawn, L. M., Flegal, K. M., Guo, S. S., Wei, R., ... Johnson, C. L. (2000). CDC growth charts: United States. *Advance Data*, (314), 1–27.
- Larson, N. I., Story, M. T., & Nelson, M. C. (2009). Neighborhood environments: disparities in access to healthy foods in the U.S. *American Journal of Preventive Medicine*, 36(1), 74–81. <http://doi.org/10.1016/j.amepre.2008.09.025>
- Li, K., Haynie, D., Palla, H., Lipsky, L., Iannotti, R. J., & Simons-Morton, B. (2016). Assessment of adolescent weight status: Similarities and differences between CDC, IOTF, and WHO references. *Preventive Medicine*. <http://doi.org/10.1016/j.ypmed.2016.02.035>
- Li, L., Chassan, R. A., Bruer, E. H., Gower, B. A., & Shelton, R. C. (2015). Childhood maltreatment increases the risk for visceral obesity. *Obesity (Silver Spring, Md.)*. <http://doi.org/10.1002/oby.21143>
- Litwin, S. E. (2014). Childhood obesity and adulthood cardiovascular disease: quantifying the lifetime cumulative burden of cardiovascular risk factors. *Journal of the American College of Cardiology*, 64(15), 1588–1590. <http://doi.org/10.1016/j.jacc.2014.07.962>
- Lobstein, T., & Jackson-Leach, R. (2006). Estimated burden of paediatric obesity and co-morbidities in Europe. Part 2. Numbers of children with indicators of obesity-related disease. *International Journal of Pediatric Obesity: IJPO: An Official Journal of the International Association for the Study of Obesity*, 1(1), 33–41.

Lodhia, N. A., Rosas, U. S., Moore, M., Glaseroff, A., Azagury, D., Rivas, H., & Morton, J. M. (2015). Do adverse childhood experiences affect surgical weight loss outcomes? *Journal of Gastrointestinal Surgery: Official Journal of the Society for Surgery of the Alimentary Tract*, 19(6), 993–998. <http://doi.org/10.1007/s11605-015-2810-7>

Longo, D.L, Fauci, A.S., Kasper, D.L., Hauser, S.L., Jameson, J.L., & Loscalzo (2013). *Obesity/Endocrinology and Metabolism* (Eds.), *Harrison's Manual of Medicine* (18th ed., pp. 1134-1137). McGraw-Hill Companies.

McGinnis, J. M., Gootman, J. A., & Kraak, V. I. (2006). *Food marketing to children and youth: Threat or opportunity?* Washington, D.C.: National Academies Press.

McPhee S.J., Papadakis, M.A., & Rabow, M.W. (2013). *Disease Prevention and Health Promotion/Prevention of overweight and obesity* (Eds). *Current medical diagnosis & treatment 2013* (pp. 13-14). New York: McGraw-Hill Medical.

McPherson, N. O., Fullston, T., Aitken, R. J., & Lane, M. (2014). Paternal obesity, interventions, and mechanistic pathways to impaired health in offspring. *Annals of Nutrition & Metabolism*, 64(3-4), 231–238. <http://doi.org/10.1159/000365026>

Miller, A. L., Lee, H. J., & Lumeng, J. C. (2015). Obesity-associated biomarkers and executive function in children. *Pediatric Research*, 77(1-2), 143–147. <http://doi.org/10.1038/pr.2014.158>

Mission: Readiness. *Military Leaders for Kids. Still Too Fat to Fight.* (2012, September). Retrieved June 11, 2016, from <https://www.missionreadiness.org/2012/still-too-fat-to-fight/>

M. O. (2010, February 09). *Let's Move/The Epidemic of Childhood Obesity.* Retrieved March 07, 2016, from <http://www.letsmove.gov/learn-facts/epidemic-childhood-obesity>

- Nader, P. R., O'Brien, M., Houts, R., Bradley, R., Belsky, J., Crosnoe, R., ... National Institute of Child Health and Human Development Early Child Care Research Network. (2006). Identifying risk for obesity in early childhood. *Pediatrics*, 118(3), e594–601. <http://doi.org/10.1542/peds.2005-2801>
- Ng, M., Fleming, T., Robinson, M., Thomson, B., Graetz, N., Margono, C., ... Gakidou, E. (2014). Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet (London, England)*, 384(9945), 766–781. [http://doi.org/10.1016/S0140-6736\(14\)60460-8](http://doi.org/10.1016/S0140-6736(14)60460-8)
- Numbers (version 3.6.2 [25771]. [Computer Software]. U.S., Apple Inc., 2008-2015
- Nutrition: Controlling the global obesity epidemic. (n.d.). Retrieved June 09, 2016, from <http://www.who.int/nutrition/topics/obesity/en/>
- Nutritional problems of children and adolescents. (2012). In J. Webster-Gandy, A. Madden, & M. Holdsworth (Eds.), *Oxford handbook of nutrition and dietetics* (p. 262). Oxford: Oxford University Press, USA.
- Ogden, C., & Carroll, M. (2010). Prevalence of obesity among children and adolescents: United States, trends 1963–1965 through 2007–2008. Centers for Disease Control, National Center for Health Statistics. Retrieved from <http://www.cdc.gov/>
- Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2012). Prevalence of obesity in the United States, 2009-2010. *NCHS Data Brief*, (82), 1–8.
- Ogden CL, Carroll MD, Kit BK, & Flegal KM. (2014). Prevalence of childhood and adult obesity in the united states, 2011-2012. *JAMA*, 311(8), 806–814. <http://doi.org/10.1001/jama.2014.732>
- Orlet Fisher, J., Rolls, B. J., & Birch, L. L. (2003). Children's bite size and intake of an entrée are greater with large portions than with age-appropriate or self-selected portions. *The American Journal of Clinical Nutrition*, 77(5), 1164–1170.

- Owen, C. G., Martin, R. M., Whincup, P. H., Smith, G. D., & Cook, D. G. (2005). Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. *Pediatrics*, 115(5), 1367–1377.
<http://doi.org/10.1542/peds.2004-1176>
- Pathways and Partnerships for Child Care Excellence. (2012). Retrieved March 15, 2016, from http://www.acf.hhs.gov/sites/default/files/occ/pathways_partnerships_v1_0.pdf
- Pizzi, M. A., & Vroman, K. (2013). Childhood obesity: effects on children's participation, mental health, and psychosocial development. *Occupational Therapy in Health Care*, 27(2), 99–112. <http://doi.org/10.3109/07380577.2013.784839>
- Power, C., Pinto Pereira, S. M., & Li, L. (2015). Childhood Maltreatment and BMI Trajectories to Mid-Adult Life: Follow-Up to Age 50y in a British Birth Cohort. *PLoS ONE*, 10(3). <http://doi.org/10.1371/journal.pone.0119985>
- Rankinen, T., Zuberi, A., Chagnon, Y. C., Weisnagel, S. J., Argyropoulos, G., Walts, B., ... Bouchard, C. (2006). The Human Obesity Gene Map: The 2005 Update. *Obesity*, 14(4), 529–644. <http://doi.org/10.1038/oby.2006.71>
- Redding, C. A. (2003). Origins and Essence of the study. *ACE Reporter*. San Diego: ACE Reporter.
- Reedy, J., & Krebs-Smith, S. M. (2010). Dietary sources of energy, solid fats, and added sugars among children and adolescents in the United States. *Journal of the American Dietetic Association*, 110(10), 1477–1484.
<http://doi.org/10.1016/j.jada.2010.07.010>
- Reynolds, R. M., Osmond, C., Phillips, D. I. W., & Godfrey, K. M. (2010). Maternal BMI, parity, and pregnancy weight gain: influences on offspring adiposity in young adulthood. *The Journal of Clinical Endocrinology and Metabolism*, 95(12), 5365–5369. <http://doi.org/10.1210/jc.2010-0697>

- Schneiderman, J. U., Mennen, F. E., Negriff, S., & Trickett, P. K. (2012). Overweight and obesity among maltreated young adolescents. *Child Abuse & Neglect*, 36(4), 370–378. <http://doi.org/10.1016/j.chiabu.2012.03.001>
- Singh, G. K., Kogan, M. D., Van Dyck, P. C., & Siahpush, M. (2008). Racial/ethnic, socioeconomic, and behavioral determinants of childhood and adolescent obesity in the United States: analyzing independent and joint associations. *Annals of Epidemiology*, 18(9), 682–695. <http://doi.org/10.1016/j.annepidem.2008.05.001>
- Springs, F. E., & Friedrich, W. N. (1992). Health risk behaviors and medical sequelae of childhood sexual abuse. *Mayo Clinic Proceedings*, 67(6), 527–532.
- Stevens, J. E. (2012, 10 03). AcesTooHigh. (J. E. Stevens, Editor) Retrieved 02 29, 2016, from ACESTooHigh: <http://acestoohigh.com>
- Trust for America's Health & Robert Wood Johnson Foundation. (2014). *The state of obesity: Better policies for a healthier America 2014*. Retrieved from <http://stateofobesity.org/files/stateofobesity2014.pdf>
- United Nations. *Sustainable development goals*. (September 25, 2015). Retrieved March 05, 2016, from <http://www.un.org/sustainabledevelopment/sustainable-development-goals/>
- U.S. Department of Health & Human Services, Administration for Children and Families, Administration on Children, Youth and Families, Children's Bureau. (2016). Child maltreatment 2014. Available from <http://www.acf.hhs.gov/programs/cb/research-data-technology/statistics-research/child-maltreatment>
- Vartanian, L. R., Schwartz, M. B., & Brownell, K. D. (2007). Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *American Journal of Public Health*, 97(4), 667–675. <http://doi.org/10.2105/AJPH.2005.083782>

- Walker, B. R., Colledge, N. R., Ralston, S. H., & Penman, I. D. (2014). Environmental and Nutritional factors in disease/Disorders of altered energy balance. (Eds.), Davidson's principles and practice of medicine (22d ed., pp. 115-117). China: Churchill Livingstone Elsevier.
- Wang, Y. C., Bleich, S. N., & Gortmaker, S. L. (2008). Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among US children and adolescents, 1988-2004. *Pediatrics*, 121(6), e1604-1614.
<http://doi.org/10.1542/peds.2007-2834>
- Webster-Gandy, J., Gandy, Madden, A., & Holdsworth, M. (2012). Nutritional problems of children and adolescents (Eds). *Oxford handbook of nutrition and dietetics* (p. 262). Oxford: Oxford University Press, USA.
- Whitaker, R. C. (2004). Predicting preschooler obesity at birth: the role of maternal obesity in early pregnancy. *Pediatrics*, 114(1), e29-36.
- Whitaker, R. C., Phillips, S. M., Orzol, S. M., & Burdette, H. L. (2007). The association between maltreatment and obesity among preschool children. *Child Abuse & Neglect*, 31(11-12), 1187-1199. <http://doi.org/10.1016/j.chiabu.2007.04.008>
- Whitlock, E. P., Williams, S. B., Gold, R., Smith, P. R., & Shipman, S. A. (2005). Screening and interventions for childhood overweight: a summary of evidence for the US Preventive Services Task Force. *Pediatrics*, 116(1), e125-144.
<http://doi.org/10.1542/peds.2005-0242>
- Williamson, D. F., Thompson, T. J., Anda, R. F., Dietz, W. H., & Felitti, V. (2002). Body weight and obesity in adults and self-reported abuse in childhood. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, 26(8), 1075-1082.
<http://doi.org/10.1038/sj.ijo.0802038>

- Woo Baidal, J. A., Locks, L. M., Cheng, E. R., Blake-Lamb, T. L., Perkins, M. E., & Taveras, E. M. (2016). Risk Factors for Childhood Obesity in the First 1,000 Days: A Systematic Review. *American Journal of Preventive Medicine*.
<http://doi.org/10.1016/j.amepre.2015.11.012>
- World Health Organization (WHO). Commission on Ending Childhood Obesity. Report of the Commission on Ending Childhood Obesity. Geneva: World Health Organization, 2016. <http://www.who.int/end-childhood-obesity/en/> (accessed Jan 25, 2016).
- World Health Organization (WHO). Diet and physical activity: *Childhood obesity and overweight*. (2015, January). Retrieved June 09, 2016, from <http://www.who.int/dietphysicalactivity/en/>
- World Health Organization (WHO). *Facts and figures on childhood obesity*. (2014, October 29). Retrieved June 09, 2016, from <http://www.who.int/end-childhood-obesity/facts/en/>
- World Health Organization (WHO). *Global Action Plan for the Prevention and Control of NCDs 2013-2020*. (2013, March). Retrieved March 07, 2016, from http://www.who.int/nmh/events/ncd_action_plan/en/
- World Health Organization (WHO). *Obesity and overweight*. (2015, January). Retrieved April 03, 2016, from <http://www.who.int/mediacentre/factsheets/fs311/en/>
- World Health Organization (WHO). *Obesity*. (n.d.). Retrieved April 03, 2016, from <http://www.who.int/topics/obesity/en/>
- World Health Organization (WHO). Obesity: preventing and managing the global epidemic. Report of a WHO consultation. (2000). *World Health Organization Technical Report Series, 894*, i–xii, 1–253.

World Health Organization: Maternal and perinatal health. (n.d.). Retrieved June 10, 2016, from http://www.who.int/maternal_child_adolescent/topics/maternal/maternal_perinatal/en/

World Health Organization (WHO) . 10 facts on obesity. (2014, May). Retrieved April 03, 2016, from <http://www.who.int/features/factfiles/obesity/facts/en/>

2011/12 National Survey of Children's Health. Child and Adolescent Health Measurement Initiative (CAHMI), "2011- 2012 NSCH: Child Health Indicator and Subgroups SAS Codebook, Version 1.0" 2013, Data Resource Center for Child and Adolescent Health, sponsored by the Maternal and Child Health Bureau. www.childhealthdata.org.

2011/12 National Survey of Children's Health. Maternal and Child Health Bureau in collaboration with the National Center for Health Statistics. 2011/12 NSCH [SAS] Indicator Data Set prepared by the Data Resource Center for Child and Adolescent Health, Child and Adolescent Health Measurement Initiative. www.childhealthdata.org