Relationships between Complex Pediatric Health Conditions and the Built, Physical and Social Environments

Brittany Corley
University of Nebraska Medical Center

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Relationships between Complex Pediatric Health Conditions and the Built, Physical and Social Environments

By:

Brittany N. Corley

A DISSERTATION

Presented to the Faculty of the University of Nebraska Graduate College in Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy

Epidemiology Graduate Program

Under the Supervision of Professor Lorena Baccaglini

University of Nebraska Medical Center Omaha, Nebraska

November 2017

Supervisory Committee:

Alan Kolok, Ph. D.  Veenu Minhas, Ph. D.

Eleanor Rogan, Ph. D.
I would like to thank my PhD. advisor and supervisory committee chair, Dr. Lorena Baccaglini, for her mentorship, guidance and teaching during the period of my graduate studies. I greatly appreciate her encouragement to think independently. She gave me the freedom to explore scientific ideas and ensured I never lost focus of my main research goals. I thank her wholeheartedly for her advice, ideas and criticisms throughout the journey of my graduate program of 4 years.

I extend my special thanks to my supervisory committee Dr. Veenu Minhas, Dr. Alan Kolok, and Dr. Eleanor Rogan for all their guidance and encouragement. I thank them sincerely for his advice and discussions, which helped me in completing my thesis work. I also thank Dr. Alan Kolok and Dr, Eleanor Rogan for their immense help and support when I would become overwhelmed, worn-down, and discouraged during the process of completing this dissertation.

I would sincerely like to thank my family Noah Corley, Vanessa Corley, Kristina Corley, Dorothy McAllister, and Peggy Woods, without whom none of my education would have been achievable. I thank them for their continual support, their confidence, and their understanding over the last 4 years.

I thank my fellow graduate students in both the epidemiology department and the environmental health department for their continuous support and friendship over the last 4 years.
Finally, I would like to thank my friends for their support and understanding as I completed my dissertation. With special thanks to Laura Merlin, Katie Mitchell, and Kaeli Samson, for their encouraging conversations, text messages and willingness to celebrate the minor victories.

This research would not have been possible without those listed above, and they have my heartfelt thanks.
RELATIONSHIPS BETWEEN COMPLEX PEDIATRIC HEALTH CONDITIONS AND THE BUILT, PHYSICAL AND SOCIAL ENVIRONMENTS

Brittany N. Corley, Ph. D.

University of Nebraska, 2017

Supervisor: Lorena Baccaglini DDS Ph. D.

The relationship between human health and the environment is complex and overall poorly understood. The environment has a lasting impact on health, and the processes appear to start at conception. Thus, this dissertation examines the potential relationship between pediatric health and the environment.

The first relationship examined was between the built environment and comorbid Attention Deficient Hyperactivity Disorder and Autism Spectrum Disorder. Many publications have reported the relationship between socioeconomic disadvantage, Attention Deficient Hyperactivity Disorder, and Autism Spectrum Disorder. However, the link between the built environment and Attention Deficient Hyperactivity Disorder and Autism Spectrum Disorder symptom severity is poorly understood. An analysis was conducted using the 2011/2012 National Children’s Health Survey accounting for the complex survey
design. For children with insurance and comorbid Attention Deficient Hyperactivity Disorder and Autism Spectrum Disorder, there was a limited association between symptom severity and neighborhood factors.

The second relationship examined was the potential relationship between the physical environment and pediatric health. This was completed by examining if there was an advantage in conducting geospatial analysis relative to congenital abnormalities and pediatric cancer, using watersheds, rather than anthropogenic census tracts, particularly concerning agrichemical runoff.

The last relationship examined in this dissertation was between the social environment and childhood health. Participation in extracurricular activities has been linked to higher educational motivation, achievement and high school graduation. However, the process by which this improvement occurs is poorly understood and has not been well examined in children who have developmental differences. The analysis was conducted using the 2011/2012 National Children’s Health Survey. Extracurricular activities and comorbid Attention Deficient Hyperactivity Disorder and Autism Spectrum Disorder diagnosis affected the odds of educational engagement. The findings suggest that the extracurricular activities have a positive effect on educational engagement regardless of comorbid Attention Deficient Hyperactivity Disorder and Autism Spectrum Disorder.

Overall, this dissertation found that the built, physical, and social environments appear to influence pediatric health.
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<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADHD</td>
<td>Attention Deficit/ Hyperactivity Disorder</td>
</tr>
<tr>
<td>ADHD/ASD</td>
<td>Comorbid ADHD and ASD</td>
</tr>
<tr>
<td>AML</td>
<td>Acute myeloid leukemia</td>
</tr>
<tr>
<td>ASD</td>
<td>Autism Spectrum Disorder</td>
</tr>
<tr>
<td>BCG</td>
<td>Bacillus Calmette-Guérin</td>
</tr>
<tr>
<td>CA</td>
<td>Congenital Abnormalities</td>
</tr>
<tr>
<td>CBC</td>
<td>Complete blood count</td>
</tr>
<tr>
<td>CDC</td>
<td>Centers for Disease Control and Prevention</td>
</tr>
<tr>
<td>CT</td>
<td>Computer tomography</td>
</tr>
<tr>
<td>DD</td>
<td>Developmental Disabilities</td>
</tr>
<tr>
<td>DSM-V</td>
<td>Diagnostic and Statistical Manual of Mental Disorders Version 5</td>
</tr>
<tr>
<td>HBM</td>
<td>Health Belief Model</td>
</tr>
<tr>
<td>HU</td>
<td>Hydrological Unit Code</td>
</tr>
<tr>
<td>IQ</td>
<td>Intelligence Quotient</td>
</tr>
<tr>
<td>IRB</td>
<td>Institutional Review Board</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Full Form</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>NDD</td>
<td>Neurodevelopmental Disorders</td>
</tr>
<tr>
<td>NSCH</td>
<td>National Survey of Children's Health</td>
</tr>
<tr>
<td>PC</td>
<td>Pediatric Cancer</td>
</tr>
<tr>
<td>PET</td>
<td>Positron emission tomography</td>
</tr>
<tr>
<td>SCT</td>
<td>Social Cognitive Theory</td>
</tr>
<tr>
<td>SDH</td>
<td>Social Determinants of Health</td>
</tr>
<tr>
<td>SES</td>
<td>Socioeconomic Status</td>
</tr>
<tr>
<td>TPB</td>
<td>Theory of Planned Behavior</td>
</tr>
<tr>
<td>TRA</td>
<td>Theory of Reasoned Action</td>
</tr>
<tr>
<td>TTM</td>
<td>Transtheoretical Model</td>
</tr>
<tr>
<td>UNMC</td>
<td>University of Nebraska Medical Center</td>
</tr>
<tr>
<td>US</td>
<td>The United States of America</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
<tr>
<td>SAS</td>
<td>SAS version 9.4</td>
</tr>
<tr>
<td>FPL</td>
<td>Federal Poverty Line</td>
</tr>
<tr>
<td>GIS</td>
<td>Geographical Information Systems</td>
</tr>
<tr>
<td>USGS</td>
<td>United States Geological Society</td>
</tr>
<tr>
<td>HU</td>
<td>Hydrological Unit</td>
</tr>
</tbody>
</table>
USCS  United States Cancer Statistics Working Group

DRC  Data Resource Center

IEP  Individualized Education Plan
In traditional infectious disease epidemiology, the research approach typically follows the epidemiologic triangle, of agent, host, and vector/environment (Figure 1) (Davidson, 2015). The Agent, rather the first infectious microorganism or pathogen referred to, or the expanded chemical or physical cause of disease must be present for disease to happen. However, this presence is not always sufficient to cause the disease. A plethora of external and internal factors influences the path from exposure to disease. These can include the agent’s pathogenicity and the dose of exposure. The host refers to the target of the agent. The host itself affects susceptibility to the agent; these factors of influence are often called risk factors, and may include the type of exposure, the host’s genetic and physical sensitivity, or the health behaviors of the host. Lastly, the vector/environment refers to how the exposure of the host to the agent happened. These can be physical factors like geology, climate, and ecology, or social factors like socioeconomic status (SES), sanitation or availability of health services.
While this traditional model works for infectious disease epidemiology, it does not apply easily to chronic conditions, like cardiovascular disease, developmental disorders (DD), cancers, and congenital abnormalities (CA). This is due to the often multifaceted and long-term development of these disorders. These chronic conditions also highlight the health disparities within the human population, where one group of the population appears to have a higher burden of disease morbidity and mortality than another does.

In 2008 the World Health Organization (WHO), published a report from the Commission on Social Determinants of Health (CSDH, 2008). This report created a consensus that there are some major underlying causes of health disparities in the human population. These causes of human health disparities have many names in the literature; for simplicity in this dissertation they are referred to as social determinants of health (SDH).
There are several available lists of SDH, the one employed in this manuscript is from Healthy People 2020 (Figure 2) (Office of Disease Prevention and Health Promotion, 2016).

Figure 2: Examples of Social Determinants of Health (Office of Disease Prevention and Health Promotion, 2016)

The use of SDH allows the shift of the epidemiology perspective to transfer from individual health risks to population based health risks. There are three sub-disciplines of Epidemiology that may be appropriate to examine SDH under, clinical, social, and environmental epidemiology (Figure 3). Due to the population-based methodology used with
SDH, it would be inappropriate to examine SDH under the lens of clinical epidemiology.

Overall, clinical epidemiology focuses on the individual. Social epidemiology may provide a lens for analysis, however social epidemiology only looks at the social aspects of health, and this would ignore the physical aspects of SDH (Davidson, 2015). Lastly, there is environmental epidemiology, which examines both the physical and social environments of health (R. M. Merrill, 2008). For this reason, SDH will be considered through the lens of environmental epidemiology.

**The Environment**

The environment is that which all living and non-living things occur naturally on earth (R. M. Merrill, 2008). The overall concept of the environment is subdivided into the built environment, the physical environment, and the social environment. The built environment includes all urban designs, land use, the transportation system, and encompasses patterns of human activity within the physical environment (Handy, Boarnet, Ewing, &
Killingsworth, 2002). The physical environment includes the part of the human environment that is purely physical factors, like soil, climate, air, and water supply. The social environment encompasses the immediate physical surroundings, social relationships, and cultural milieus within which defined groups of people function and interact (Barnett & Casper, 2001).

Epidemiologists often use the phrase person, place, and time when discussing descriptive epidemiology, and in the first steps of any research project. These three facets are part of a larger set of 5 descriptives used in descriptive epidemiology, which is: “What is the health issue of concern, Who is the person, Where is the place, When is the time, and Why/how are the causes, risk factors, modes of transmission.” (Center for Disease Control, 2012). For chronic diseases the why/how is not clear, the diseases are often complex. For this reason, in chronic disease research, the place and time components are used to try to explain the why and how of disease development.

Application of the environmental subgroups for the place and time is relatively straightforward. The built and physical environments will mostly encompass the place. To have a clear picture of where the person works and lives both built and physical environments would need to be examined. Does the person have access to clean air and water (physical), is the neighborhood where the person lives and works safe (built) and are there sidewalks and health care (built)? Whereas, the social environment would be most affected by the time element. Human perceptions and social interactions change over time, is the person able to work, do they face rigid gender roles, is the person discriminated
against based on sex, race, ethnicity, religion or other descriptives, is the person subjected to gang violence and high levels of crime. These considerations have a direct impact on not only the person but their health as well. SDH are easily placed into these environmental groups as shown in Figure 4.

![Figure 4: The interplay between Social Determinants of Health and the Environment](image)

Within the following chapters, all three facets of the environment and the various SDH will be examined in their relationship to human health.

**Human Health**

Human health has had an overabundance of definitions. Currently, there are three main definitions in use. The first is that health is the absence of any disease or impairment. The second is that health is a state that allows the individual to adequately cope with all demands of daily life (also implying the absence of disease and impairment). The third
definition states that health is a state of balance, an equilibrium that an individual has established within himself and between himself and his social and physical environment (Sartorius, 2006; D. A. Vallero & Vallero, 2004).

In many industrialized countries, the pattern of morbidity and mortality has changed, where previously the burden of health care was infectious disease there is now a burden from chronic conditions, like cancer, cardiovascular diseases, and diabetes (McKeown, 2009). This transition has already happened in the United States (US), where the second most common cause of death overall is cancer, and the leading cause of death in infants is CA (Melonie Heron, 2016; Xu, Murphy, Kochanek, & Bastian, 2016). A leading cause of morbidity in children is developmental disorders (DD) which affects one in six children in the US (C. A. Boyle et al., 2011). In the following chapters, the focus will be on cancer, congenital abnormalities (CA), and selected DD.

**Neurodevelopmental Disorders**

The rate of overall DD fluctuated between 16.8% in 1988, to 13.2 and 13.87% in 1997-2005, and 1997-2008 respectively (Boulet, Boyle, & Schieve, 2009; C. A. Boyle et al., 2011; Coleen A. Boyle, Decoufle, & Allsopp, 1994). A subset of these disorders, Neurodevelopmental Disorders (NDD) typically have onset in early life, before a child enters school, and are characterized by developmental deficits and impairments of personal, social, academic, or occupational functioning (American Psychiatric Association. DSM-5 Task force, 2013). NDD, including intellectual disabilities, communication disorders, Au-
tism Spectrum Disorder (ASD), Attention Deficit/ Hyperactivity disorder (ADHD), specific learning disabilities, motor disorders, and other NDD. NDD often co-occur as comorbid conditions. For some NDD, the clinical presentation includes symptoms of excess as well as deficits and delays in achieving expected milestones.

Persistent deficits characterize ASD in social communication and social interaction across multiple contexts, including deficits in social reciprocity, nonverbal communicative behaviors used for social interaction, and skills in developing, maintaining, and understanding relationships (American Psychiatric Association. & American Psychiatric Association. DSM-5 Task Force., 2013). In addition to the social communication deficits, the diagnosis of ASD requires the presence of restricted, repetitive patterns of behavior, interests, or activities (American Psychiatric Association. & American Psychiatric Association. DSM-5 Task Force., 2013).

Within the diagnosis of ASD, individual clinical characteristics are noted through the use of specifiers, as well as specifics that describe the autistic symptoms. These specifiers provide clinicians with an opportunity to individualize the diagnosis and communicate a richer clinical description of the affected individuals (American Psychiatric Association. & American Psychiatric Association. DSM-5 Task Force., 2013).

ADHD is a neurodevelopmental disorder defined by impairing levels of inattention, disorganization, and hyperactivity-impulsivity (American Psychiatric Association. & American Psychiatric Association. DSM-5 Task Force., 2013). In childhood, ADHD frequently overlaps with disorders considered to be “externalizing disorders,”
such as oppositional defiant disorder and conduct disorder (American Psychiatric Association & American Psychiatric Association. DSM-5 Task Force., 2013). ADHD often persists into adulthood, with resultant impairments of social, academic and occupational functioning.

**Attention Deficit/ Hyperactivity Disorder**

From 1997-1999 to 2006-2008 the prevalence of ADHD in the US has been reported to have risen 33%, to 6.69% of all children (C. A. Boyle et al., 2011). Population surveys suggest that ADHD occurs in about 5% of children (Faraone, Sergeant, Gillberg, & Biederman, 2003) and about 2.5% of adults (Simon, Czobor, Balint, Meszaros, & Bitter, 2009) worldwide.

**Diagnostic Criteria and Definition**

The diagnostic and statistical manual of mental disorders version 5 (DSM-V) has precise diagnostic criteria for ADHD which has been in use since 2013 (Table 1).

<table>
<thead>
<tr>
<th>Table 1: DSM-V Diagnostic Criteria for ADHD from DSM-V</th>
</tr>
</thead>
<tbody>
<tr>
<td>A persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development, as characterized by (1) and/or (2):</td>
</tr>
<tr>
<td><strong>Inattention:</strong> Six (or more) of the following symptoms have persisted for at least</td>
</tr>
<tr>
<td>Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities</td>
</tr>
</tbody>
</table>
6 months to the degree that is inconsistent with developmental level, and that negatively impacts directly on social and academic/occupational activities:

**Note:** The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

<table>
<thead>
<tr>
<th>Hyperactivity and impulsivity: Six (or more) of the following symptoms have persisted for at least 6 months to the</th>
<th>Often has difficulty sustaining attention in tasks or play activities</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Often does not seem to listen when spoken to directly</td>
</tr>
<tr>
<td></td>
<td>Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace</td>
</tr>
<tr>
<td></td>
<td>Often has difficulty organizing tasks and activities</td>
</tr>
<tr>
<td></td>
<td>Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort</td>
</tr>
<tr>
<td></td>
<td>Often loses things necessary for tasks or activities</td>
</tr>
<tr>
<td></td>
<td>Is often easily distracted by extraneous stimuli</td>
</tr>
<tr>
<td></td>
<td>Is often forgetful in daily activities</td>
</tr>
<tr>
<td></td>
<td>Often fidgets with, taps hands or feet, or squirms in seat.</td>
</tr>
<tr>
<td></td>
<td>Often leaves seat in situations when remaining seated is expected</td>
</tr>
</tbody>
</table>
degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

**Note:** The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or a failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Often runs about or climbs in situations where it is inappropriate.</td>
<td></td>
</tr>
<tr>
<td>Often unable to play or engage in leisure activities quietly.</td>
<td></td>
</tr>
<tr>
<td>Is often “on the go,” acting as if “driven by a motor</td>
<td></td>
</tr>
<tr>
<td>Often talks excessively.</td>
<td></td>
</tr>
<tr>
<td>Often blurts out an answer before a question has been completed</td>
<td></td>
</tr>
<tr>
<td>Often has difficulty waiting for his or her turn</td>
<td></td>
</tr>
<tr>
<td>Often interrupts or intrudes on others</td>
<td></td>
</tr>
</tbody>
</table>

Several inattentive or hyperactive-impulsive symptoms were present prior to age 12 years.

Several inattentive or hyperactive-impulsive symptoms are present in two or more settings.

There is clear evidence that the symptoms interfere with, or reduce the quality of, social, academic, or occupational functioning.

The symptoms do not occur exclusively during schizophrenia or another psychotic disorder and are not better explained by another mental disorder.


Diagnostic Features

One of the defining features of ADHD is that it must begin in childhood; several symptoms must be present before age 12. Another key feature is that the symptoms must be present in more than one situation. For this reason, multiple informants are typically consulted to ascertain symptom presence. The symptoms may vary by environment, signs of the disorder may be minimal or absent when the individual is receiving frequent rewards for appropriate behavior, is under close supervision, is engaged in especially interesting activities, has consistent external stimulation, or is interacting in one-on-one situations (American Psychiatric Association. DSM-5 Task force, 2013).

Risk Factors and Diagnostic Difficulties

ADHD is associated with reduced behavioral inhibition, effortful control, or constraint, negative emotionality, and high novelty seeking. Children with these traits may have a predisposition to ADHD.

Environmental

Several factors related to pregnancy and birth are linked to ADHD or have been shown to be causal. Weighing less than 1,500 grams has a two- to threefold increase in the
risk of ADHD. Smoking during pregnancy is correlated with ADHD (Thapar et al., 2009). Factors during early life are also related to ADHD, removing food coloring from the child’s diet lowers ADHD symptomology, however, the potential causality of this relationship has not been established (Nigg, Lewis, Edinger, & Falk, 2012; L. J. Stevens, Kuczek, Burgess, Hurt, & Arnold, 2011). Other associations include child abuse, neglect, multiple foster placements, neurotoxin exposure, infections, alcohol exposure in utero, and various environmental toxicants.

**Genetic and physiological**

There are arguments for a genetic component for ADHD, those children with a first-degree relative with ADHD are at an elevated risk (Stawicki, Nigg, & von Eye, 2006). While some genes have been specified in research, none are sufficient to be causal (Gizer, Ficks, & Waldman, 2009). While no specific physical features have been found to cause ADHD, there are some, which are related and elevated. These include visual and hearing impairments, metabolic abnormalities, sleep disorders, nutritional deficiencies, epilepsy, hypertelorism, highly arched palate, and low-set ears. Subtle motor delays and other neurological soft signs may occur (American Psychiatric Association. DSM-5 Task force, 2013).

**Culture-Related Diagnostic Issues**

In general ADHD prevalence rates across regions are attributed to different diagnostic practices (Faraone et al., 2003). However, there may be cultural variation in attitude towards children’s behaviors which is seen in the lower rates observed for African
Americans and Latinos versus the white population in the US (Froehlich et al., 2007; Kessler et al., 2006; Miller, Nigg, & Miller, 2009).

Gender-Related Diagnostic Issues

ADHD is most frequently diagnosed in males, with a ratio of 2:1 between males and females, this may be due to the fact females typically present as primarily inattentive (Faraone et al., 2003; Kessler et al., 2006).

Functional Consequences of Attention-Deficit/Hyperactivity Disorder

ADHD affects the entire spectrum of human life. Children with ADHD are likely to have reduced school performance, reduced academic attainment, social rejection, have conduct disorders, and antisocial personality disorder (Frazier, Youngstrom, Glutting, & Watkins, 2007; Kessler et al., 2006; Mannuzza, Klein, Bessler, Malloy, & Lapadula, 1998). Adults with ADHD are likely to have poorer occupational performance, attainment and attendance, and overall a higher probability of unemployment, there is also an increased risk of substance use disorders and incarceration, conduct disorders and antisocial personality disorder, injury, traffic incidents, and obesity (Cortese et al., 2008; Fuemmeler, Østbye, Yang, McClernon, & Kollins, 2011; Kessler et al., 2006; Klein et al., 2012; Mannuzza et al., 1998; R. Merrill, Lyon, Baker, & Gren, 2009; Pastor & Reuben, 2006).

Overall those with ADHD, typically have negative family interactions and peer relationships. ADHD is also related to less school completion and achievement. Academic
deficits, school-related problems, and peer neglect tend to be most associated with elevated symptoms of inattention, whereas peer rejection and, to a lesser extent, accidental injury is most salient with marked symptoms of hyperactivity or impulsivity (Willcutt et al., 2012).

**Autism Spectrum Disorder**

The prevalence of ASD in both children and adult populations, in the US and other countries is around 1% (Brugha et al., 2011).

**Diagnostic Criteria**

Due to the varied conditions under the ASD umbrella, several diagnostic criteria must be met (Table 2).

<table>
<thead>
<tr>
<th>Persistent deficits in social communication and social interaction across multiple contexts, as manifested</th>
<th>Deficits in social-emotional reciprocity, ranging, for example, from abnormal social approach and failure of normal back-and-forth conversation; to reduced sharing of interests, emotions, or affect; to failure to initiate or respond to social interactions.</th>
<th><strong>Specify current severity</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tr>
<tr>
<td>by all the following, currently or by history</td>
<td>Deficits in nonverbal communicative behaviors used for social interaction, ranging, for example, from poorly integrated verbal and nonverbal communication; to abnormalities in eye contact and body language or deficits in understanding and use of gestures; to a total lack of facial expressions and nonverbal communication.</td>
<td></td>
</tr>
<tr>
<td>Deficits in developing, maintaining, and understanding relationships, ranging, for example, from difficulties adjusting behavior to suit various social contexts; to difficulties in sharing imaginative play or in making friends; to the absence of interest in peers.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Restricted, repetitive patterns of behavior, interests, or activities, as manifested by at</td>
<td>Stereotyped or repetitive motor movements, use of objects, or speech</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior</td>
<td></td>
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<td></td>
<td>Specify current severity:</td>
<td></td>
</tr>
</tbody>
</table>
least two of the following, currently or by history

<table>
<thead>
<tr>
<th>Highly restricted, fixated interests that are abnormal in intensity or focus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of the environment</td>
</tr>
</tbody>
</table>

Symptoms must be present in the early developmental period

Symptoms cause clinically significant impairment in social, occupational, or other important areas of current functioning.

These disturbances are not better explained by intellectual disability or global developmental delay. Intellectual disability and autism spectrum disorder frequently co-occur; to make comorbid diagnoses of autism spectrum disorder and intellectual disability, social communication should be below that expected for the general developmental level.

**Note:** Individuals with a well-established DSM-IV diagnosis of autistic disorder, Asperger’s disorder, or pervasive developmental disorder not otherwise specified should be given the diagnosis of autism spectrum disorder. Individuals who have marked deficits in social communication, but whose symptoms do not otherwise meet criteria for autism spectrum disorder, should be evaluated for a social (pragmatic) communication disorder.

For both the social components and the motor/behavioral components, the doctor typically diagnoses a severity label (Table 3), this allows the symptomology to be explained.

**Table 3: ASD Clinical Symptom Severity**

<table>
<thead>
<tr>
<th>Severity level</th>
<th>Social communication</th>
<th>Restricted, repetitive behaviors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 3</td>
<td>Severe deficits in verbal and non-verbal social communication skills cause severe impairments in functioning, very limited initiation of social interactions, and minimal response to social overtures from others.</td>
<td>The inflexibility of behavior, extreme difficulty coping with change, or other restricted/repetitive behaviors markedly interfere with functioning in all spheres. Great distress/difficulty changing focus or action.</td>
</tr>
<tr>
<td>&quot;Requiring very substantial support.&quot;</td>
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<tr>
<td>Level 2</td>
<td>Marked deficits in verbal and non-verbal social communication skills; social impairments apparent even with supports in place; limited initiation of social interactions; and reduced or abnormal responses to social overtures from others.</td>
<td>The inflexibility of behavior, difficulty coping with change or other restricted/repetitive behaviors appear frequently enough to be obvious to the casual observer and interfere with functioning in a variety of contexts. Distress and/or</td>
</tr>
<tr>
<td>&quot;Requiring substantial support.&quot;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Level 1</td>
<td>Without supports in place, deficits in social communication cause noticeable impairments. Difficulty initiating social interactions, and clear examples of atypical or unsuccessful responses to social overtures of others. May appear to have decreased interest in social interactions.</td>
<td>The inflexibility of behavior causes significant interference with functioning in one or more contexts. Difficulty switching between activities. Problems of organization and planning hamper independence.</td>
</tr>
</tbody>
</table>


In addition to the severity modifier’s it is also recommended that the doctor specify if the ASD is accompanied by any of the following: intellectual impairment, language impairment, a known medical or genetic condition or environmental factor, another neurodevelopmental, mental, or behavioral disorder, or catatonia (American Psychiatric Association. DSM-5 Task force, 2013).
Diagnostic Features

While ASD must have both impairments in social communication and interaction and restricted, repetitive patterns of behavior, interests, or activities, which must be present from early childhood, these symptoms may be masked in some contexts due to interventions, compensations of the child, and current support. Due to the differences in the manifestation of the disorder, the spectrum was created. This spectrum now includes the previously defined conditions of early infantile autism, childhood autism, Kanner’s autism, high-functioning autism, atypical autism, pervasive developmental disorder not otherwise specified, childhood disintegrative disorder, and Asperger’s disorder (American Psychiatric Association. DSM-5 Task force, 2013).

An example of potential masking behaviors in ASD is when adults with ASD without intellectual or language disabilities learn to suppress repetitive behaviors when in public because it is expected. They are focused, and special interests may provide motivation for education and employment in their adult life. For this reason, when diagnosing adults, the criteria was typically met when the masked behaviors were present in childhood, even if they are now no longer present.

Due to the difficulty in diagnosing ASD, there are standardized behavioral diagnostic instruments, with interviews, questionnaires, and clinical observation measurements.
Risk and Prognostic Factors

The longest established prognostic factors for ASD include the presence of intellectual disability, and language impairment, and additional mental health problems.

Environmental

Environmental risk factors have not been well established, however a few nonspecific risk factors include advanced parental age, extremely/very/low birth weight, environmentally released mercury, and fetal exposure to valproate (Christensen et al., 2013; Croen, Najjar, Fireman, & Grether, 2007; Durkin et al., 2008; Hack et al., 2009; Palmer, Blanchard, Stein, Mandell, & Miller, 2006; Schendel & Bhasin, 2008).

Genetic and Physiological

About 10-20% of all ASD cases can be linked to gene expression (Geschwind, 2011). It has been shown that siblings of an ASD child are more likely to develop social and communication disorders, despite the overall low risk of ASD (Parihar & Ganesh, 2016). This similarly inheritance of disorders may be due to a genetic link between ASD, and ASD traits that do not reach the threshold for diagnosis, or that exist independently of the other traits of ASD (Bralten et al., 2017).

Culture-Related Diagnostic Issues

ASD is highly related to social interactions and in communities where there are different norms for social interactions, nonverbal communication, and relationships; ASD may be underdiagnosed or diagnosed late. This under-diagnosis is believed to be the
cause of the disparity in diagnosis seen in Medicaid populations (Mandell, Listerud, Levy, & Pinto-Martin, 2002).

Gender-Related Diagnostic Issues

ASD is diagnosed four times more often in males than in females; it has been suggested that this is due to the subtle manifestations of social and communication difficulties (Mandy et al., 2012; Rivet & Matson, 2011).

Functional Consequences of Autism Spectrum Disorder

In young children ASD is typically associated with lack of social and communication abilities which may hamper learning, especially learning through social interaction or in settings with peers, insistence on routines and aversion to change, as well as sensory sensitivities, may interfere with eating and sleeping and make routine care extremely difficult. Adaptive skills are typically below measured intelligence quotient (IQ). Extreme difficulties in planning, organization, and coping with change negatively affect academic achievement, even for students with above-average intelligence.

In young adults, there is limited independence, typically low socio-economic status (SES), and persisting behavioral problems (Eaves & Ho, 2008). Adults overall, typically remain very dependent on family and support systems, few live alone, have close friends, or permanent employment, and have poor reading and spelling skills (Howlin, Goode, Hutton, & Rutter, 2004)
Comorbidity

Overall there is a high level of comorbidity in ASD populations, about 70% of individuals have at least one comorbid mental disorder, and 40% have two or more (Simonoff et al., 2008). When criteria for both ADHD and ASD are met, both diagnoses should be given. This principle applies to concurrent diagnoses of ASD and developmental coordination disorder, anxiety disorders, depressive disorders, and other comorbid diagnoses. Among individuals who are nonverbal or have language deficits, observable signs such as changes in sleep or eating and increases in challenging behavior should trigger an evaluation for anxiety or depression. Specific learning difficulties are common, as are developmental coordination disorder (Baird, Douglas, Director, & Murphy, 2011).

Attention Deficit/Hyperactivity Disorder

Abnormalities of attention are common in individuals with ASD, as is hyperactivity. A diagnosis of ADHD should be considered when attentional difficulties or hyperactivity exceeds that typically seen in individuals of comparable mental age.

Comorbid ADHD and ASD

Studies set in both US and European populations found that 29-85% of all children with ASD also had a diagnosis of ADHD (Kenneth D. Gadow, DeVincet, & Pomeroy, 2006; Lee & Ousley, 2006; Rao & Landa, 2014). Despite the research showing that ADHD and ASD were commonly seen together, the comorbid diagnosis of ADHD and ASD was
not recognized until DSM-5 in 2013 (American Psychiatric Association. DSM-5 Task force, 2013). The definition of comorbid ADHD and ASD is future explained in Figure 5.


Figure 5: Concurrent ADHD/ASD description

Causes and Symptoms

There has been a suggestion in both observational and genetic studies that ADHD and ASD might share a familial transmission (Musser et al., 2014; Nijmeijer et al., 2014; Pinto, Rijsdijk, Ronald, Asherson, & Kuntsi, 2016). Genetic linkage studies suggested regions of chromosomes 16p3 2q24, 16p1, 17p11, 5p13 and 15q to be involved in both ADHD and ASD (Antshel, Zhang-James, & Faraone, 2013). A number of known rare mutations that result in syndromes of developmental delay are often associated with symptoms of
ASD and ADHD, such as permutations of FMRI in Fragile X syndrome, 22q11 deletion syndrome and microdeletions/duplications at 15q13.2q13.3 spanning gene CHRNA7 (Antshel et al., 2013). Increased ADHD and ASD symptoms have been found in patients which tuberous sclerosis and neurofibromatosis, two genetic diseases resulted from mutations of mTor inhibitor genes, TSC1 or TSC2 and NF-1 (Antshel, Zhang-James, & Faraone, 2013).

Children with ADHD/ASD have higher comorbid symptoms and higher overall severity, these symptoms include tantrum behaviors, conduct problems, oppositional aggressive symptoms, oppositional defiant disorder, conduct disorder, anxiety, and worry/depression (Kenneth D. Gadow et al., 2006; Guttmann-Steinmetz, Gadow, & Devincent, 2009; Jang et al., 2013; Mulligan et al., 2009). This concurrent ADHD/ASD phenotype differs from ADHD or ASD phenotypes within the IQ levels and in Autistic symptoms severity. However, this phenotypemaintains some clinical aspects that characterize ASD or ADHD phenotypes (Craig et al., 2015).

The concurrent ADHD/ASD diagnosis children typically have lower socialization and greater discrepancy between cognitive and adaptive abilities compared to the ADHD-only group. These children also have reduced adaptive functioning and autism symptoms, but not ADHD symptoms, and exacerbated impairments in adaptive functioning relative to children with ADHD, associated with ASD symptoms (Ashwood et al., 2015).
**Congenital Abnormalities**

CA also known as birth defects, congenital anomaly, congenital defect, and congenital disorders are leading causes of infant morbidity and mortality. In both Europe and the US, 2-3% of all births have a major CA (Helen Dolk, Loane, & Garne, 2010; Rynn, Cragan, & Correa, 2008).

**Definition**

Either CA can be defined as anomalies, structural or functional that occur during gestation, and can be identified prenatally, at birth or later in infancy.

**Causes and risk factors**

Approximately 50% of CA cannot be linked to any specific genetic cause. Nevertheless, there are some known genetic and environmental etiological and risk factors that are known for CA (Quintana, 2015).

**Genetic factors**

Both inherited genes and random mutations play a major role in different CA (ACMG, 2002; Ramalho-Santos, Melton, & McMahon, 2000; Song & Yosypiv, 2011). The inherited genes can cause either high rates of disease such as Hemophilia C in the Ashkenazi Jew population, or explain a significant number of disease even in low prevalence areas like Cystic Fibrosis in the Finnish population (Choi, Kim, Lee, Choi, & Yoo, 2014; Kere et al., 1994).
Socioeconomic and Demographic Factors

An estimated 94% of all severe CA occurs in low and middle-income countries (Sitkin, Ozgediz, Donkor, & Farmer, 2015). This high rate has been attributed to many factors including, increased nutritional deficiency, the prevalence of intrauterine infection, exposure to teratogens, and unsupervised self-medication (Christianson, Howson, & Modell, 2006; Penchasazdeh, 2002). Maternal age is another long established risk factor, for CA (HOOK, 1981).

Environmental factors

Maternal exposure to agrichemicals has been hypothesized to have an etiological role in CA development (Garry, Schreinemachers, Harkins, & Griffith, 1996; Rappazzo et al., 2016; Rocheleau et al., 2011; Winchester, Huskins, & Ying, 2009). Exposure to waste sites, metal smelting or mines, may also play an etiologic role, this role may be increased if nutritional deficiencies are also present (Ahern, Mullett, MacKay, & Hamilton, 2011; Al-Sabbak et al., 2012; Croen, Shaw, Sanbonmatsu, Selvin, & Buffler, n.d.; H Dolk et al., 1998).

Detection

Screening for CA can be divided into three classes, preconception screening, postconception screening, and neonatal screening (Hollinshead, Vigliani, Walsh, LeClair, & Zelano, 2000). During preconception screening, genetic risks for specific CA can be identified. In postconception screening, maternal risk factors are evaluated, also ultra-
sound, and placental markers are utilized to determine structural abnormalities and chromosomal abnormalities. Neonatal screening is what is completed when the infant is born, and checks for disorders of the blood, metabolism and hormone production, screening for deafness and heart defects, as well as early detection of CA is also completed.

*Treatment and care*

Treatment for CA varies with the conditions; however, many structural CA can be corrected surgically, and treatment can typically be administered for functional problems.

**Pediatric Cancer**

While cancer might overall be uncommon in children with only 1 in 285 children developing cancer, it is the second most common cause of death among children in the US (Siegel, Miller, & Jemal, 2015; E. Ward, DeSantis, Robbins, Kohler, & Jemal, 2014).

In general Pediatric Cancer (PC) includes children aged birth to 14, however, in this document children aged to 18 years will be included since, teens and young adult are typically treated in a pediatric setting, in spite of arguments against doing so (Bleyer, Barr, Ries, Whelan, & Ferrari, 2017).

Cancer is when otherwise healthy cells within the body mutate and start to grow out of control. This mass is called a tumor and can be benign (meaning it will not spread throughout the body) or malignant (where it can spread throughout the body). Tumors can form anywhere in the body.
Most Common Types

Leukemia accounts for 30% of all childhood cancers, cancers of the brain and other parts of the nervous system are the second most common cancer types (26%) (Siegel, Miller, & Jemal, 2016). Other common types of cancer include Neuroblastoma (6%), Wilms tumor (5%), Non-Hodgkin’s and Hodgkin’s Lymphoma (5% and 3%), Rhabdomyosarcoma (3%), Retinoblastoma (2%), Osteosarcoma and Ewing Sarcoma (2% and 1%) (Society, 2017).

Those children aged 15-19 have a different risk profile, their most common types of cancer include Hodgkin and Non-Hodgkin Lymphoma (15% and 8%), germ cell tumors (including testicular (8%) and ovarian (2%)), central nervous system tumors (10%), thyroid cancer (11%), melanoma (6%), acute lymphoblastic leukemia (8%), soft tissue sarcoma (7%), bone tumors (7%), and acute myeloid leukemia (4%) (Society, 2017).

Causes and Risk Factors

The etiology of childhood cancer is mostly unknown, but some cases can be linked to genetic causes (Johnson, Zoellner, & Gutmann, 2016; Vega-García et al., 2016). Despite the unknown etiology of most childhood cancers, recent research has linked certain cancers to environmental factors. Gas production has been associated with hematological malignancies, industrial and pesticide pollution has been related to renal cancer, and reti-
noblastomas, whereas pesticides, and crop production proximity have been related to leukemia, neuroblastoma, and hepatic tumors (Garcia-Perez et al., 2016; Gomez-Barroso et al., 2016; McKenzie et al., 2017; Omidakhsh et al., 2017).

**Detection and Diagnosis**

The process to diagnosis cancer can be long and complex. In addition to the typical collection of person and family history, typically lab tests, scans or other test procedures of required.

**Treatment**

Children of all ages are typically treated at a pediatric oncology center. Typically, a team of doctors will be used to ensure the newest treatments and technologies is used with each patient. While some patients will only receive one type of therapy, it is not uncommon to receive multiple types of treatment.

**Human Health and the Environment**

As explained previously environmental epidemiology is the study of the effect on human health of the physical biological and chemical factors in the external environment (Council, 1997). Previous studies have been conducted linking the built (Hynes, Brugge, Watts, & Lally, 2000; Stafford, Cummins, Macintyre, Ellaway, & Marmot, 2005; Weich et al., 2002), physical (Landrigan, Kimmel, Correa, & Eskenazi, 2004; Mitchell & Popham,
2008), and social (Barrington-Trimis et al., 2016; Turecki & Meaney, 2016) environments and human health.

**ADHD/ASD and the Built Environment**

It has been shown that nature and outdoor activities can improve childhood health (Aries, Aarts, & Hoof, 2013; McCurdy, Winterbottom, Mehta, & Roberts, 2010). Research into the built environment and child health is a recent area of study. Urbanization while typically bringing several SES benefits can also bring damages, including but not limited to poor housing conditions, violence, and poor social support. Overall most studies have focused on street connectivity or walkability, food environment and deserts, green spaces, outdoor air pollution, noise, and extreme heat. In terms of overall child health, few other, these foci have sufficient studies conducted. The most well-researched areas have included outdoor air pollution and fetal growth restriction, and respiratory and immune effects (Gascon, Vrijheid, & Nieuwenhuijsen, 2016).

Studies that have been conducted in the built environment and child health have shown that parent perception of neighborhood safety was associated with children’s social-emotional development and overall general health (Christian et al., 2015). It has also been shown that extremely deprived environments override individual family factors in relation to conduct problems (Schonberg & Shaw, 2007). High levels of behavioral problems were associated with socially disadvantaged neighborhoods, and lower SES (Singh & Ghandour, 2012). Overall, antisocial behavior, which can be a symptom of both ADHD
and ASD, was shown to be related to neighborhood disadvantage (Dubow, Edwards, & Ippolito, 1997).

For children with ADHD, it has been demonstrated that natural areas provide consistent positive environments (Van Den Berg & Van Den Berg, 2010). Children were likely to show more non-social, aggressive, inattentive, impulsive and hyperactive behavior when in town versus in nature (Van Den Berg & Van Den Berg, 2010). In a national study neighborhood, social support was shown to be associated with higher odds of ADHD diagnosis and severity; neighborhood amenities were not associated in this study (Razani et al., 2015a).

Cluster reports for ASD have shown that local environmental or social dynamic play a role in ASD risk but not the etiological processes (Mazumdar, King, Liu, Zerubavel, & Bearman, 2010). In a national study, it was shown that poor neighborhood social capital was associated with children having ASD, whereas physical, and social environmental factors along with individual factors were related to parent reported ASD severity (Hock & Ahmedani, 2012). For children with ASD when examining symptom severity, it has been shown that environment has an effect of reported comorbidity conditions and severity (Kanne, Abbacchi, & Constantino, 2009).

While few studies have examined ADHD and ASD in relation to build environments separately, no studies were found that have looked at concurrent ADHD/ASD. Even the way built environment was investigated was poorly defined and not consistent. Some studies only looked at green space, where others only examined social capital.
Congenital Abnormalities, Pediatric Cancer and the Physical Environment

Overall, the risk factors for most chronic conditions are not well known; this is the case for CA, PC. Due to this unknown etiology, the three will be examined using the physical environment.

Congenital Abnormalities

The risk factors for CA are unknown and vary depending on the type. The most notable risk factors include individual maternal exposures (Corlin et al., 2016).

There have been studies conducted that have shown that several physical environmental exposures may be risk factors for CA. These physical environment exposures include agrichemicals, soil type distributions, occupational exposures, and air pollution.

Agrichemicals

Pesticides are known to be both reproductive and neurotoxic agents and have been shown to be teratogenic in animal studies (Shepard & Lemire, 2004). Several studies support the hypothesis that agrichemicals play a role in the etiology of certain CA (Garry et al., 1996; Rappazzo et al., 2016; Rocheleau et al., 2011; Winchester et al., 2009). Pesticides, including atrazine, Alachlor, and Chlorpyrifos, are classified as endocrine disruptors, whereas bifenthrin and diuron are developmental toxicants (Rappazzo et al., 2016). Stud-
ies have shown a potential association between pesticide exposure before or during pregnancy, and various types of CA (A. Jack Agopian, Cai, Langlois, Canfield, & Lupo, 2013; Bove et al., 1995; Ma et al., 2014; Ochoa-Acuna & Carbajo, 2009; Rull, Ritz, & Shaw, 2006).

Other Environmental Exposures

In China, it has been shown that neural tube defects have been related to soil type distributions (Li, Wang, & Wu, 2012). It is hypothesized that this is due to a difference in chemical or biological contaminants transport within these different soils.

Maternal exposure to high levels of colorants, pigments and solvents were related to an increased risk of CA (Torfs, Katz, Bateson, Lam, & Curry, 1996).

Carbon monoxide exposure in the second-month has been linked in a dose-response fashion to cardiac ventricular septal defects, whereas second-month ozone exposure is related to the aortic artery and valve defects, pulmonary artery, and valve anomalies, and conotruncal defects (Ritz et al., 2002).

Pediatric Cancer

There have been studies conducted that have shown that several physical environmental exposures may be risk factors for PC. These physical environment exposures include agrichemicals, industrial pollution, paternal occupational exposure, air pollution, ionizing radiation, and UV radiation.
Agrichemicals

Pesticide exposure and crop production proximity have been related to renal cancers (Garcia-Perez et al., 2016), retinoblastomas (Omidakhsh et al., 2017), leukemia, neuroblastoma, and hepatic tumors (Gomez-Barroso et al., 2016), AML (Robison, Buckley, & Bunin, 1995), Wilms Tumor, soft tissue sarcoma, Ewing’s Sarcoma, Non-Hodgkin’s Lymphoma and cancers of the brain, colorectal and testes (Zahm & Ward, 1998).

Industrial pollution

Overall industrial pollution has been linked to renal cancers (Garcia-Perez et al., 2016), whereas hematological malignancies have been associated with oil and gas production (McKenzie et al., 2017).

Paternal occupational exposure

Paternal occupational exposure or close proximity to a nuclear facility has been linked to AML (Belson, Kingsley, & Holmes, 2007). Paternal exposure to metals was associated with retinoblastoma (Robison et al., 1995). Parental exposure to solvents and petroleum products is linked to AML (Robison et al., 1995). Parental employment as an auto mechanic or welder has been associated with Wilms tumor, whereas exposure to solvents, hydrocarbons, and lead was not confirmed (Robison et al., 1995). Hepatoblastoma has been related to maternal exposure to metals, hydrocarbons, and paints/pigments and paternal occupational exposure to metals and petroleum products (Robison et al., 1995).
Other Exposures

Acute leukemia has been linked to exposure of tobacco smoke, whereas benzene was related to AML (Belson et al., 2007; Buffler, Kwan, Reynolds, & Urayama, 2005). Ionizing radiation has a proven causal relationship with AML (Belson et al., 2007). There has recently been an increased prevalence in pediatric melanoma; this is related to an increased exposure to ambient UV radiation (Strouse, Fears, Tucker, & Wayne, 2005).

ADHD/ASD and the Social Environment

Two ways to access, the social environment of children is to look at their educational engagement and participation in any extracurricular activity.

Educational Engagement

Education engagement includes behavioral engagement, emotional engagement, and cognitive engagement (Moore & Lippman, 2006). Behavioral engagement includes involvement in academic tasks, positive conduct, and the absence of disruptive behaviors. Emotional engagement includes caring about doing well. Cognitive engagement involves curiosity and an investment of time and energy in learning, and a willingness to go beyond the basic requirements to master difficult skills.

ADHD

Overall, poor high school engagement has been reported for students with ADHD (Zendarski, Sciberras, Mensah, & Hiscock, 2017). ADHD is associated with poor grades,
reading and math standardized test scores, and increased grade retention (Loe & Feldman, 2007). Children with core ADHD symptoms but not a diagnosis show the same poor academic and educational outcomes (Loe & Feldman, 2007). While ADHD treatment did show a relationship with increased academic productivity, this did not lead to better test scores (Loe & Feldman, 2007). Children with ADHD exhibit significantly lower rates of academic engagement and high levels of off-task behaviors, low levels of passive academic engagements, and low levels of actively engaged time when compared to their peers (Vile Junod, DuPaul, Jitendra, Volpe, & Cleary, 2006).

**ASD**

There has been a debate on the inclusion of ASD students in general education classes. However, this is the recommended practice. In spite of the legal reasons for this inclusion, it is recommended due to the resulting gains in social development (Schreibman, 2005). Research has shown that those students placed in restrictive settings, tend to only interact with the teachers, whereas those placed in normal classrooms, improve much more in social competence (Donnellan, Mesaros, & Anderson, 1984; Fisher & Meyer, 2002). Studies have been mixed, but overall this inclusion typically leads to increased academic gains, particularly with those students who demonstrate greater intellectual abilities (Goodman & Williams, 2007; Harrower & Dunlap, 2001; Schreibman, 2005).

Overall children with ASD typically exhibit limited classroom engagement (Bryan & Gast, 2000; Nicholson, Kehle, Bray, & Heest, 2011; Sparapani, Morgan, Reinhardt,
Schatschneider, & Wetherby, 2016). There has also been a call for additional research conducted on academic achievement and its relationship to ASD (Keen, Webster, & Ridley, 2015).

**ADHD/ASD**

This comorbidity has been shown to cause a delay in diagnosis age, putting it close to school age (T. Stevens, Peng, & Barnard-Brak, 2016). The nature of delayed diagnoses often serves as a limiting factor in a child’s overall ability to succeed in school, having an impact on their educational, behavioral, emotional, and cognitive engagement compared to their peers. Due to the recent release of DSM-V and the relatively new ability for practitioners to easily diagnosis both disorders there has been little research beyond descriptive characteristics of these children.

**Extracurricular Activities**

Research has shown that extracurricular activities typically have a positive effect on educational engagement (Badura et al., 2016; Hughes, Cao, & Kwok, 2016; Morris, 2016). Research has also shown that specific extracurricular activities are associated with negative developmental outcomes; such as links between sports and alcohol consumption, and full classroom participation and decreased educational outcomes (Morris, 2016).

It has been shown for middle school students that participation in sports specifically, increases levels of prosocial norms, which accounts for the increase in academic competency beliefs, sense of school belonging, course grades, and classroom engagement.
Participation in organized leisure time activities was linked with higher school engagement, lower levels of school related stress, and better academic achievement regardless of age or gender (Badura et al., 2016). In 7th and 8th graders, it has been shown that sports predicted competence beliefs and educational values, whereas performing arts and clubs predicted competence beliefs, teacher rated classroom engagement and teacher awarded grades (Im, Hughes, Cao, & Kwok, 2016).

This tentative relationship between extracurricular activities and educational outcomes is poorly understood. However, there have been some mediating factors that have been found these include; educational expectations, noncognitive skills, accumulation of social capital (Morris, 2016). However, it has been shown that family income does not appear to affect this relationship (Morris, 2016).

**ADHD**

Children with ADHD typically have low levels of motor proficiency, cardiovascular health, and behavior. It has been shown that participating in physical activity programs, either within or outside school can increase all of these (Cuypers, De Ridder, & Strandheim, 2011; Verret, Guay, Berthiaume, Gardiner, & Beliveau, 2012a).

**ASD**

Children with ASD typically have poor social skills and are therefore excluded from social activities, and rarely see their friends outside of school, this is often correlated with low SES, and low cognitive, social skills (Schreibman, 2005).
Those children whose parents have them participate in afterschool programs typically have poor experiences (Haney, 2012). Many of these children are asked to leave due to often vague reasons, or due to the organizers being unable to provide appropriate care for the child (Haney, 2012). Despite, these poor experiences a majority of parents still expressed the desire for these programs for their child, expressing the desire for more than just babysitting, programs that help with social skills, academic support and social protection (Haney, 2012). The need for these programs is even more necessary for those students in the middle or high schools where very few programs exist (Haney, 2012).

ADHD/ASD

Many of the studies discussed above were small scale investigative studies that only looked at children with one disorder. The high level of comorbidity discussed previously shows that a significant portion of children is being ignored by these studies.

Specific Aims and Hypothesis

The environment built, physical, and social, has a lasting impact on health throughout life. The goal of this dissertation is to investigate this relationship in relation to chronic conditions that happen across the spectrum of life.

Specific Aims: ADHD/ASD and the Built Environment

The overall goal of this part of the dissertation is to investigate the potential relationship between ADHD/ASD symptom severity, and the built environment.
Aim 1

Identify the relationship between ADHD and ASD symptom severity, and find the appropriate way to combine into one severity

Aim 2

Examine the relationship between combined ADHD/ASD symptom severity and Built Environment

Aim 2.1

Examine ADHD/ASD symptom severity and Neighborhood Amenities

Aim 2.2

Examine ADHD/ASD symptom severity and Neighborhood Detracting Elements

Aim 2.3

Examine ADHD/ASD symptom severity and Neighborhood Social Support
Specific Aims: CA, PC and the Physical Environment

The goal of this portion of the dissertation is to create a methodology to analyze chronic health conditions, using physical environment relationships, instead of anthropogenic or politically assigned relationships.

*Aim 3*

Identify and explain the difference between hydrological unit code (HU) geographies and anthropogenic or politically assigned geographies.

*Aim 3.1*

Define and identify limitations of HU geographies

*Aim 3.2*

Define and identify limitations of anthropogenic or politically assigned geographies

*Aim 4*

Choose the appropriate HU for both (CA, PC) chronic conditions.

*Aim 5*

Identify and calculate the correct population at risk for all three chronic conditions.
Aim 5.1

Assign population at risk to the HU for each chronic condition

Aim 6

Calculate the incidence rate for each chronic condition within the appropriate HU

Specific Aims: ADHD/ASD and the Social Environment

The overall goal of this part of the dissertation is to investigate the potential relationship between scholarly engagement and extracurricular activity, in the general population and in the ADHD/ASD population.

Aim 7

Examine the relationship between educational engagement and extracurricular activities

Aim 7.1

Examine educational engagement and the relationship between ADHD/ASD diagnosis and participation in extracurricular activities
CHAPTER 1: IT IS MORE THAN INCOME: NEIGHBORHOOD STATUS AND COMBINED ADHD AND ASD SYMPTOM SEVERITY

Abstract

BACKGROUND: Many publications have reported the relationship between socioeconomic disadvantage ADHD, and ASD. However, the link between neighborhood attributes, demographic and socioeconomic factors, and combined Attention Deficient Hyperactivity Disorder and Autism Spectrum Disorder symptom severity is poorly understood.

AIM: This study aimed to examine the link between neighborhood attributes, demographic and socioeconomic factors, and combined Attention Deficient Hyperactivity Disorder and Autism Spectrum Disorder. METHODS AND PROCEDURES: Analysis was conducted using the 2011/2012 National Children’s Health Survey, accounting for the complex survey design. OUTCOMES AND RESULTS: For children with insurance and comorbid ADHD and ASD, there was a limited association between symptom severity and neighborhood factors, and income. Demographic and factors including extracurricular activities, educational engagement, total combined conditions, and parental health all had an association with symptom severity. CONCLUSIONS AND IMPLICATIONS: This
study shows that even after accounting for income, there appear to be residual effects
from the neighborhood status and built environment on ADHD/ASD symptom severity.
**Introduction**

From 1997-1999 to 2006-2008 the prevalence of ADHD in the US has risen 33%, to 6.69% of all children (C. A. Boyle et al., 2011). Population surveys suggest that ADHD occurs in about 5% of children (Faraone et al., 2003) and about 2.5% of adults (Simon et al., 2009) worldwide. In contrast, the prevalence of ASD, in both children and adult populations, in the US and other countries is around 1% (Brugha et al., 2011). The proportion of ASD with an ADHD comorbidity in children in US and European populations ranges from 16-50% (Gjevik, Eldevik, Fjærangranum, & Sponheim, 2011; Hanson et al., 2013; Reiersen, Constantino, Volk, & Todd, 2007; Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2010). Children with ADHD/ASD have more severe symptoms, and substantial symptom overlap (Sinzig, Walter, & Doepfner, 2009). Both observational and genetic studies suggest that ADHD and ASD might share familial transmission (Musser et al., 2014; Nijmeijer et al., 2014; Pinto et al., 2016). Children with ADHD/ASD have higher comorbid symptoms and higher overall severity (K. D. Gadow, DeVincent, & Schneider, 2009; Guttmann-Steinmetz et al., 2009; Jang et al., 2013; Mulligan et al., 2009).

For children with ADHD/ASD, the built environment can affect reported comorbid conditions and severity (Kanne et al., 2009). One facet of the built environment is urbanization, which is typically a benefit to human health. Urbanization can cause increases in SES, street connectivity or walkability, and green space; however, urbanization can also cause poor housing conditions, violence, food deserts, high outdoor air pollution, noise, extreme heat and poor social support.
Studies have suggested an association between parental perception of neighborhood safety and childhood social-emotional development and general health (Christian et al., 2015). High levels of behavioral problems have been associated with socially disadvantaged neighborhoods, and lower SES (Singh & Ghandour, 2012). Whereas, antisocial behavior, which can be a symptom of both ADHD and ASD, has been related to neighborhood disadvantage (Dubow et al., 1997).

For children with ADHD, natural areas and green space provide consistent positive environments, where children were likely to show less non-social, aggressive, inattentive, impulsive and hyperactive behavior (Van Den Berg & Van Den Berg, 2010). In a national study of neighborhoods, social support was associated with higher odds of ADHD diagnosis and severity (Razani et al., 2015b).

Cluster reports for ASD have shown that local environmental or social dynamics play a role in ASD risk but not the etiological processes (Mazumdar et al., 2010). In a national study, poor neighborhood social capital was associated with children having ASD, whereas physical and social environmental factors along with individual factors related to parent-reported ASD severity (Hock & Ahmedani, 2012). For children with ASD, environmental context affecting individuals, which may account for the variance in parent reported versus teacher reported severity (Kanne et al., 2009).
While a few studies have examined ADHD and ASD concerning built environments separately, no studies have looked at concurrent ADHD/ASD. Even the investigation of built environment was poorly defined and not consistent. Some studies only looked at green space, where others only examined social capital.

Thus, this study aimed to investigate the potential relationship between ADHD/ASD symptom severity, and the social and built environment.

**Methods**

**Data Source**

The NSCH 2011-2012 was used in this study (2011/12 National Survey of Children’s Health. Maternal and Child Health Bureau in collaboration with the National Center for Health Statistics, n.d.) The NSCH consisted of 95,677 phone interviews, using random digit dialing, to obtain a dual-frame random sample of households with non-institutionalized children aged 0–17 years. A random child from each family was selected to be the sample child and the parent or adult that had the most knowledge of the sampled child’s health was interviewed. Interviews were conducted between February 28, 2011, and June 25, 2012 (Centers for Disease Control and Prevention, 2013). The University of Nebraska Medical Center Institutional Review Board approved this research as non-human subject research.
Study Participants Eligibility Criteria

Five hundred and sixteen surveys met the criteria for this study, which represented 309,295 children. The eligibility criteria for this study were medically insured children, over age five years with a current parental-reported medical diagnosis of both ADHD and ASD including a reported symptom severity. Children with mild ADHD and severe ASD or mild ASD and severe ADHD were excluded from analysis. No missing values (e.g., do not know, refused, missing, partial survey) were included in this analysis (Figure 6).
Non-Institutionalized US Children Respondents aged 0-17 years
n=95,677

Excluded Due to Lack of ADHD or ASD Diagnosis n=95,004
No ADHD or ASD n=94,785
Unknown n=201
Refused n=4
Missing n=14

Excluded due to age (under 6) n=52

Excluded due to Combined Symptom Severity
(Mild ADHD/Severe ASD, or Mild ASD/Severe ADHD, or missing) n=66

Excluded due to incomplete Predictor Values (missing data) n=39

Total Analyzed n=516

Mild Combined Severity n=286
Moderate/Severe Combined Severity n=230

Figure 6: Flowchart of Survey Participant Selection for Study 1
Survey Instrument

ADHD/ASD

Adults were asked if a doctor or health care provider had ever told them that the survey child had “Attention Deficit Disorder or Attention-Deficit/Hyperactivity Disorder, that is, ADD or ADHD” and “Autism, Asperger’s Disorder, pervasive developmental disorder, or other autism spectrum disorder” (Center of Health Statistics, 2012), the adults were then asked if the diagnosis was current. Screener help was available for adults who were not sure what the conditions might be by name.

Combined Symptom Severity

For each condition, adults were asked: “Would you describe [his/her condition] as mild, moderate, or severe?” (Center of Health Statistics, 2012). Combined severity included either mild (at least one mild no severe) or moderate/ severe (no mild).

Neighborhood Amenities

Neighborhood amenities included “Sidewalks or walking paths? A park or playground area? A recreation center, community center, or boys or girls’ club? A library or bookmobile?” (Center of Health Statistics, 2012). Neighborhood amenities were defined as follows: One to two of the above, two to three of the above, or all four.
Neighborhood Detracting Elements

Neighborhood detracting elements included “litter or garbage on the street or sidewalk? Poorly kept or rundown housing? Vandalism such as broken windows or graffiti?” (Center of Health Statistics, 2012). Neighborhood detracting elements were grouped as follows: zero of the above, one of the above, two to three of the above.

Neighborhood Social Support

The measurement of neighborhood social support used the survey participant’s agreement (Yes/No) with the following statements:

“People in this neighborhood help each other out. We watch out for each other’s children in this neighborhood. There are people I can count on in this neighborhood. If my child was outside playing and got hurt or scared, there are adults nearby whom I trust to help my child.” (Center of Health Statistics, 2012).

Chronic Conditions

The chronic conditions variable measured the total number of additional chronic conditions (0-2, 3-5, or 6+ disorders) the survey child was diagnosed by a doctor with: depression, anxiety, behavioral problems, any developmental delay, intellectual disability, cerebral palsy, speech problems, Tourette syndrome, asthma, diabetes, epilepsy or seizure disorder, hearing problems, uncorrectable vision problems, bone, joint, or muscle problems, or a brain injury or concussion (Center of Health Statistics, 2012).
**Income Measure**

The income variable used in this analysis was an imputed variable created by the Data Resource Center (DRC). The original income variable from the survey was missing for 9.3% (8,856) of the sample. The DRC used single imputation to substitute the missing cases in the NSCH from the National Center for Health Statistics (NCHS).

**Statistical Analysis**

Descriptive analyses (frequencies and percentages) were conducted for individual variables. Relative standard error (RSE) was calculated for each covariate at each level, using the methods of Cai and Shimizu, and any RSE above 30% was reported (Cai & Shimizu, 2010). Rao-Scott chi-squared tests were conducted to access associations between covariates and disease severity. Logistic regression analyses were carried out to calculate crude and adjusted odds ratios and 95% confidence intervals (Institute, 2012). Models included the variables of interest (neighborhood amenities, detracting elements, and support), demographic variables were entered into the model; other variables were included if the estimate of any variable of interest changed by more than 10%.

Survey-specific procedures were used to account for the complex study design, observations were weighted using complex sampling specifications provided by the DRC data manual including stratification by state and sample type (landline or cell-phone) (2011/2012 National Survey of Children’s Health, 2013). Resulting estimates are representative of all non-institutionalized children aged 6–17 years in the US.
Results

Most of the children were aged 9-11 years (35%), male (88%), White Non-Hispanic (64%), and from families with incomes between 200-399% of the federal poverty line (FPL) (37%) (Table 4).

Table 4: Descriptive Characteristics of Children, Parents and Environment (National Survey of Children’s Health; NSCH; 2011-2012)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total</th>
<th>Mild severity</th>
<th>Severe severity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n^1</td>
<td>N^2</td>
<td>Percent (S.E.)</td>
</tr>
<tr>
<td>Child’s Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-8</td>
<td>86</td>
<td>40</td>
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<tr>
<td>9-11</td>
<td>158</td>
<td>109</td>
<td>35.3 (5.3)</td>
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<tr>
<td>12-14</td>
<td>133</td>
<td>85</td>
<td>27.5 (1.4)</td>
</tr>
<tr>
<td>15-17</td>
<td>139</td>
<td>75</td>
<td>24.3 (1.2)</td>
</tr>
<tr>
<td>Child’s Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>441</td>
<td>272</td>
<td>87.9 (1.1)</td>
</tr>
<tr>
<td>Female</td>
<td>75</td>
<td>38</td>
<td>12.1 (1.3)</td>
</tr>
<tr>
<td>Child’s Race</td>
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<tr>
<td>White non-Hispanic</td>
<td>382</td>
<td>197</td>
<td>63.8 (1.1)</td>
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<tr>
<td>Characteristics</td>
<td>Total</td>
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<td>Severe severity</td>
</tr>
<tr>
<td>-------------------------------</td>
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<td>-----------------</td>
</tr>
<tr>
<td></td>
<td>n(^1)</td>
<td>N(^2)</td>
<td>Percent (S.E.(^3))</td>
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<tr>
<td>Other(^4)</td>
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<td>112</td>
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<td>213</td>
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<td>No</td>
<td>136</td>
<td>96</td>
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<td>Household income (FPL)(^5)</td>
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<tr>
<td>&lt;100%</td>
<td>101</td>
<td>56</td>
<td>18.2 (1.0)</td>
</tr>
<tr>
<td>100-199%</td>
<td>106</td>
<td>61</td>
<td>19.9 (1.5)</td>
</tr>
<tr>
<td>200-399%</td>
<td>163</td>
<td>115</td>
<td>37.3 (5.2)</td>
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<td>≥400%</td>
<td>146</td>
<td>76</td>
<td>24.7 (1.2)</td>
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<td>Child’s Chronic conditions(^6)</td>
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<td></td>
</tr>
<tr>
<td>0-2</td>
<td>171</td>
<td>81</td>
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<td>243</td>
<td>174</td>
<td>56.3 (3.8)</td>
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<tr>
<td>6 or more</td>
<td>102</td>
<td>54</td>
<td>17.4 (1.3)</td>
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<tr>
<td>Characteristics</td>
<td>Total</td>
<td>Mild severity</td>
<td>Severe severity</td>
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<tr>
<td>---------------------------------------</td>
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<tr>
<td></td>
<td>n(^1)</td>
<td>N(^2)</td>
<td>Percent (S.E.(^3))</td>
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<td>Child’s Extracurricular activities</td>
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<td>One or more (^7)</td>
<td>366</td>
<td>196</td>
<td>63.2 (0.8)</td>
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<td>Child’s Educational engagement(^8)</td>
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<td>Never/sometimes</td>
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<td>Usually/always</td>
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<td>Neighborhood amenities(^9)</td>
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<td>0-1</td>
<td>58</td>
<td>20</td>
<td>6.5 (0.4)</td>
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<tr>
<td>2-3</td>
<td>188</td>
<td>98</td>
<td>31.8 (0.8)</td>
</tr>
<tr>
<td>4</td>
<td>270</td>
<td>191</td>
<td>61.7 (3.4)</td>
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<td>Neighborhood detracting elements(^10)</td>
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<tr>
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<td>346</td>
<td>194</td>
<td>62.8 (0.6)</td>
</tr>
<tr>
<td>Characteristics</td>
<td>Total</td>
<td>Mild severity</td>
<td>Severe severity</td>
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<tr>
<td>-----------------------------------------</td>
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<td>---------------</td>
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</tr>
<tr>
<td></td>
<td>n¹  N² Percent (S.E.)</td>
<td>Percent (S.E.)</td>
<td>Percent (S.E.)</td>
</tr>
<tr>
<td>1</td>
<td>98  57 18.5 (2.7)</td>
<td>16.5 (2.6)</td>
<td>20.8 (5.7)*</td>
</tr>
<tr>
<td>2-3</td>
<td>72  58 18.6 (5.8)</td>
<td>10.5 (1.8)</td>
<td>27.9 (11.2)*</td>
</tr>
<tr>
<td>Neighborhood Support</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>119  76 24.9 (4.6)</td>
<td>9.7 (2.2)</td>
<td>15.2 (4.1)</td>
</tr>
<tr>
<td>Yes</td>
<td>397  232 75.1 (4.6)</td>
<td>43.4 (4.9)</td>
<td>31.7 (6.1)</td>
</tr>
<tr>
<td>Parental Stress¹¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>264  145 46.9 (5.5)</td>
<td>29.9 (4.2)</td>
<td>17.1 (4.2)</td>
</tr>
<tr>
<td>Yes</td>
<td>252  164 53.1 (5.5)</td>
<td>23.2 (3.5)</td>
<td>29.9 (6.1)</td>
</tr>
<tr>
<td>Parental health</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>No parents in home</td>
<td>36  15 4.8 (0.8)</td>
<td>5.5 (1.7)*</td>
<td>3.9 (0.6)*</td>
</tr>
<tr>
<td>One of two parents in home in poor health</td>
<td>82  42 13.6 (1.4)</td>
<td>17.5 (2.4)</td>
<td>9.2 (2.5)*</td>
</tr>
<tr>
<td>All parent(s) in home in poor health¹²</td>
<td>249  141 45.7 (0.1)</td>
<td>50.2 (2.9)</td>
<td>40.6 (1.6)</td>
</tr>
<tr>
<td>Characteristics</td>
<td>Total</td>
<td>Mild severity</td>
<td>Severe severity</td>
</tr>
<tr>
<td>------------------------------------------</td>
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<td>---------------</td>
<td>-----------------</td>
</tr>
<tr>
<td></td>
<td>n¹</td>
<td>N²</td>
<td>Percent (S.E.³)</td>
</tr>
<tr>
<td>All parent(s) in home in good health¹²</td>
<td>149</td>
<td>111</td>
<td>35.9 (5.4)</td>
</tr>
<tr>
<td>Total</td>
<td>516</td>
<td>309</td>
<td>100.0 (0.0)</td>
</tr>
</tbody>
</table>

Percentages adjusted for complex survey design

* RSE is greater than 30%

1. Number of children in survey sample
2. Population sample of survey, in 1,000’s
3. Standard error
4. Includes Hispanic, Black Non-Hispanic and multiracial categories
5. Reported as Federal Poverty Line (FPL) percentage
6. Number of chronic conditions of child in addition to ADHD and ASD, includes: depression, anxiety problems, behavioral or conduct problems, such as oppositional defiant disorder or conduct disorder, any developmental delay, intellectual disability or mental retardation, cerebral palsy, speech or other language problems, Tourette syndrome, asthma, diabetes, epilepsy or seizure disorder, hearing problems, vision problems that cannot be corrected with standard glasses or contact lenses, bone, joint, or muscle problems, and a brain injury or concussion
7. Includes organized activities outside of school, such as sports teams or lessons, clubs, organizations, music, dance, language or other arts
8. Children usually or always cared about doing well in school and did all required homework during the previous month
9. Includes sidewalks or walking paths, a park or playground area, a recreation center, community center, or boys’ or girls’ club, and a library or bookmobile
10. Includes litter or garbage on the street or sidewalk, poorly kept or rundown housing, and vandalism such as broken windows or graffiti
11. “Usually” or “Always” to one or more of three questions about how they felt during the past 30 days: child was much harder to care for than other children; were often bothered a lot by their child’s behavior; and/or angry with child.
12. Homes with both parents, single fathers, and single mothers

These children also had three or more chronic conditions in addition to their ADHD/ASD diagnosis (73%). Most children lived in neighborhoods with all four amenities (62%), no detracting elements (63%), and that were socially supportive (75%).
Children with 3 or more additional chronic conditions had 5-17 times the adjusted odds of moderate/severe reported ADHD/ASD symptom severity compared to those children with 0-2 additional chronic conditions (Table 5).

Table 5: ADHD/ASD Symptom Severity by Characteristics of Children, Parents, Environment, and ADHD/ASD Symptom Severity (National Survey of Children’s Health; NSCH; 2011-2012)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Odds Ratio and 95% Confidence Intervals for Moderate/ Severe vs Mild Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unadjusted</td>
</tr>
<tr>
<td>Childs Age</td>
<td></td>
</tr>
<tr>
<td>6-8</td>
<td>0.99 (0.39, 2.52)</td>
</tr>
<tr>
<td>9-11</td>
<td>2.36 (0.82, 6.82)</td>
</tr>
<tr>
<td>12-14</td>
<td>1.64 (0.68, 3.92)</td>
</tr>
<tr>
<td>15-17</td>
<td>1.00</td>
</tr>
<tr>
<td>Childs Sex</td>
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<tr>
<td>Male</td>
<td>0.74 (0.29, 1.89)</td>
</tr>
<tr>
<td>Female</td>
<td>1.00</td>
</tr>
<tr>
<td>Childs Race</td>
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<tr>
<td>White non-Hispanic</td>
<td>1.00</td>
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</table>
### Odds Ratio and 95% Confidence Intervals for Moderate/Severe vs Mild Severity

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Unadjusted</th>
<th>Adjusted(^1)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Other(^2)</strong></td>
<td>2.49 (0.99, 6.27)</td>
<td>1.27 (0.59, 2.77)</td>
</tr>
<tr>
<td><strong>Household income (FPL)(^3)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;100%</td>
<td>3.13 (1.24, 7.90)</td>
<td>1.60 (0.53, 4.78)</td>
</tr>
<tr>
<td>100-199%</td>
<td>1.95 (0.72, 5.29)</td>
<td>1.32 (0.44, 3.94)</td>
</tr>
<tr>
<td>200-399%</td>
<td>3.21 (1.04, 9.90)</td>
<td>1.40 (0.55, 3.57)</td>
</tr>
<tr>
<td>≥400%</td>
<td>1.00</td>
<td>1.00</td>
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<tr>
<td><strong>Childs Chronic conditions(^4)</strong></td>
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</tr>
<tr>
<td>0-2</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>3-5</td>
<td>5.21 (2.23, 12.15)</td>
<td>4.95 (2.19, 11.20)</td>
</tr>
<tr>
<td>6 or more</td>
<td>12.93 (4.81, 34.77)</td>
<td>16.78 (5.62, 50.07)</td>
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<tr>
<td><strong>Childs Extracurricular activities</strong></td>
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<tr>
<td>None</td>
<td>3.61 (1.50, 8.69)</td>
<td>2.05 (1.06, 3.96)</td>
</tr>
<tr>
<td>One or more(^5)</td>
<td>1.00</td>
<td>1.00</td>
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<tr>
<td><strong>Childs Educational engagement(^6)</strong></td>
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<td></td>
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<tr>
<td>Never/sometimes</td>
<td>2.62 (1.05, 6.55)</td>
<td>2.35 (1.20, 4.60)</td>
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### Odds Ratio and 95% Confidence Intervals for Moderate/ Severe vs Mild Severity

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<th>Characteristics</th>
<th>Unadjusted</th>
<th>Adjusted&lt;sup&gt;1&lt;/sup&gt;</th>
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<tbody>
<tr>
<td>Usually/always</td>
<td>1.00</td>
<td>1.00</td>
</tr>
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<td>Neighborhood amenities&lt;sup&gt;7&lt;/sup&gt;</td>
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<tr>
<td>0-1</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>2-3</td>
<td>0.84 (0.36, 1.94)</td>
<td>2.06 (0.74, 5.67)</td>
</tr>
<tr>
<td>4</td>
<td>1.15 (0.46, 2.84)</td>
<td>2.35 (0.94, 5.86)</td>
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<tr>
<td>Neighborhood detracting elements&lt;sup&gt;8&lt;/sup&gt;</td>
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</tr>
<tr>
<td>0</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>1</td>
<td>1.80 (0.63, 5.14)</td>
<td>0.96 (0.38, 2.40)</td>
</tr>
<tr>
<td>2-3</td>
<td>3.77 (1.00, 14.22)</td>
<td>2.29 (0.77, 6.74)</td>
</tr>
<tr>
<td>Neighborhood Support</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>2.15 (0.84, 5.49)</td>
<td>1.79 (0.72, 4.46)</td>
</tr>
<tr>
<td>Yes</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Parental Stress&lt;sup&gt;9&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>2.25 (0.94, 5.41)</td>
<td></td>
</tr>
</tbody>
</table>
### Odds Ratio and 95% Confidence Intervals for Moderate/Severe vs Mild Severity

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Unadjusted</th>
<th>Adjusted¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>No parents in home</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>One of two parents in home in poor health</td>
<td>0.75 (0.20, 2.90)</td>
<td>1.05 (0.23, 4.78)</td>
</tr>
<tr>
<td>All parent(s) in home in poor health¹⁰</td>
<td>2.46 (0.67, 9.06)</td>
<td>2.05 (0.54, 7.76)</td>
</tr>
<tr>
<td>All parent(s) in home in good health¹⁰</td>
<td>1.15 (0.40, 3.39)</td>
<td>5.42 (1.34, 21.96)</td>
</tr>
</tbody>
</table>

Odds Ratios and 95% confidence intervals are adjusted for complex survey design

1. Model adjusted for all variables in column
2. Includes Hispanic, Black Non-Hispanic and multiracial categories
3. Reported as Federal Poverty Line (FPL) percentage
4. Number of chronic conditions of child in addition to ADHD and ASD, includes: depression, anxiety problems, behavioral or conduct problems, such as oppositional defiant disorder or conduct disorder, any developmental delay, intellectual disability or mental retardation, cerebral palsy, speech or other language problems, Tourette syndrome, asthma, diabetes, epilepsy or seizure disorder, hearing problems, vision problems that cannot be corrected with standard glasses or contact lenses, bone, joint, or muscle problems, and a brain injury or concussion
5. Includes organized activities outside of school, such as sports teams or lessons, clubs, organizations, music, dance, language or other arts
6. Children usually or always cared about doing well in school and did all required homework during the previous month
7. Includes sidewalks or walking paths, a park or playground area, a recreation center, community center, or boys’ or girls’ club, and a library or bookmobile
8. Includes litter or garbage on the street or sidewalk, poorly kept or rundown housing, and vandalism such as broken windows or graffiti
9. "Usually” or “Always” to one or more of three questions about how they felt during the past 30 days: child was much harder to care for than other children; were often bothered a lot by their child’s behavior; and/or angry with child.
10. Homes with both parents, single fathers, and single mothers
Children who did not participate in extracurricular activities had two times the adjusted odds of reported moderate/severe ADHD/ASD symptom severity compared to those children who did participate. Those children who were not educationally engaged had two times higher odds of moderate/severe reported ADHD/ASD symptom severity compared to those children who were educationally engaged.

Children with two or more neighborhood amenities had two times the adjusted odds of moderate/severe reported ADHD/ASD symptom severity compared to those children who had no amenities. Children in neighborhoods with all three detracting elements had adjusted odds of 2.29 (0.77, 6.74) of having more severe reported ADHD/ASD symptoms compared to children who lived in neighborhoods with no detracting elements. Children in neighborhoods with no social support had adjusted odds of 1.79 (0.72, 4.46) of having more severe reported ADHD/ASD symptoms compared to children who lived in neighborhoods with no social support.

Discussion

This study analyzed non-institutionalized US children aged 6-17 years old with medical insurance, who were diagnosed with both ADHD and ASD. The reason only children with insurance were included is by 2012, 33 states had mandated ASD coverage. As a result, only 0.01% of children did not have insurance. Those children aged below 6 years were excluded due to the delay in diagnosis caused by the comorbid diagnosis of ADHD/ASD.
Those children living in neighborhoods with two or more amenities, two to three detracting elements, and no social support had higher odds of moderate/severe reported ADHD/ASD symptom severity compared to those children without these neighborhood characteristics. Those children with high levels of additional chronic conditions, or not participating in extracurricular activities or not educationally engaged also had higher odds of reported symptom severity when compared to children without these descriptors.

Children with higher number of neighborhood amenities had higher odds of moderate/severe reported ADHD/ASD symptom severity, than those without. A hypothesized reason why these amenities may increase symptom severity may be due to children diagnosed with ASD have difficulty participating in unstructured social activities (Tobin, Drager, & Richardson, 2014). A correlation was also found between unstructured socialization and later internalizing symptoms in a teenage ASD population (Lounds Taylor, Adams, & Bishop, 2017). Having access to these amenities may exacerbate the ASD symptoms in this population causing higher parental reported symptoms. These findings differ from those in previous studies, which showed that amenities (green space) has a positive effect on ADHD symptoms, the disagreement in this study may due to the difference in definition of amenities (Van Den Berg & Van Den Berg, 2010).

In this, study the higher the number of detracting neighborhood elements, the more severe the reported ADHD/ASD symptoms. This finding is supported by other stud-
ies which found behavioral problems, anti-social behaviors, and increased ASD symptoms to be associated with neighborhood disadvantage (Dubow et al., 1997; Hock & Ahmedani, 2012; Kanne et al., 2009; Singh & Ghandour, 2012).

In this study, not having social support was associated with high symptom severity, which was also found in previous studies. Social support has been shown to be associated with higher odds of ADHD diagnosis and severity (Razani et al., 2015b), whereas poor neighborhood social capital (e.g. having people to count on in community) was associated with ASD diagnosis (Hock & Ahmedani, 2012).

The high number of comorbid conditions reported in this study are consistent with other studies. The higher odds associated with the increase in number of comorbid conditions agrees with these studies which linked higher comorbidity to increase in levels of overall dysfunction (Joshi et al., 2010; Simonoff et al., 2008).

The relationship found with extracurricular activity participation and lower combined symptom severity was expected, as this relationship has been reported before in ADHD populations (Verret, Guay, Berthiaume, Gardiner, & Beliveau, 2012b). However, in the ASD population, participation has been researched more than the effects participation may have on the child so this potential relationship, is a potential area for future research (Shattuck, Ormond, Wagner, & Cooper, 2011; Solish, Perry, & Minnes, 2010).

Previous studies have shown that when compared to the normal population children with ADHD or ASD typically have lower levels of educational engagement. This study adds to this area of research by showing even among children with both disorders,
those who are educationally engaged have lower odds of moderate/severe combined ADHD/ASD symptoms, compared to those who are not (Bryan & Gast, 2000; Keen et al., 2015; Nicholson et al., 2011; Zendarski et al., 2017).

Strengths and Limitations

A limitation of this study is that it was cross-sectional and therefore does not allow for the inference of causality. Another limitation of this study was the possibility of misclassification of ADHD/ASD diagnosis and severity grouping, due to the use of parental-reported data. For ADHD/ASD diagnosis, the potential misclassification was minimized by asking about current medical diagnosis specifically. In ADHD populations, it has been theorized that parents may over-report the severity of the condition in children, especially in the youngest age groups (Narad et al., 2015).

Another limitation of this study was the use of an imputed income variable, due to the imputed nature; children were assigned household incomes that may be incorrect. Income was initially missing for 9.7% of the data or 8,856 cases in the overall dataset, in this sample, 33 cases (22 mild, 11 moderate/severe severity) were affected.

The overall strength of this study was the ability to examine both ADHD and ASD together. Overall, 16-50% of all ASD cases will have comorbid ADHD, so examining the two together is necessary (Gjevik et al., 2011; Hanson et al., 2013; Reiersen et al., 2007; Rommelse et al., 2010). Another strength of this study included the large sample size, which is uncommon for studies of childhood developmental disorders. The availability of
the demographic, social, parental and environmental data, for the same sample population, was also a strength. Another strength was the low numbers of missing data; overall few children were excluded from the study for this reason.

Conclusions and Implications

Children living in neighborhoods with all four amenities, two or more detracting elements, and no social support had higher odds of reported symptom severity. Those children with three or more additional chronic conditions, and not participating in extracurricular activities and not being educationally engaged also had higher odds of reported symptom severity. This study shows that even after accounting for income, there appear to be residual effects from the neighborhood status and built environment features on ADHD/ASD symptom severity.
CHAPTER 2: USING WATERSHED BOUNDARIES TO MAP ADVERSE HEALTH OUTCOMES: EXAMPLES FROM NEBRASKA, USA

Abstract:

In 2009, a paper was published suggesting that watersheds provide a geospatial platform for establishing linkages between aquatic contaminants, the health of the environment and human health. This paper is a follow-up to that original article. From an environmental perspective, watersheds segregate landscapes into geospatial units that may be relevant to human health outcomes. From an epidemiological perspective, the watershed concept places anthropogenic health data into a geospatial framework that has environmental relevance. Research discussed in this manuscript includes information gathered from the literature, as well as recent data collected and analyzed by this research group. It is our contention that the use of watersheds to stratify geospatial information may be both environmentally and epidemiologically valuable.
Introduction

Contaminants in water can cause adverse health effects. A poignant example of this is the contamination of Flint, Michigan, drinking water with lead and the subsequent elevated blood levels in children. In that community, lead contamination is a post-treatment issue, as the corrosive water from the Pontiac River solubilized lead from the distribution system that delivers water to individual households (Mona Hanna-Attisha, LaChance, Sadler, Schnepp, & Champney Schnepp, 2016). The geospatial distribution of lead exposure in Flint is relatively easy to map as the source of the exposure (the drinking water) is easy to identify and the latency period between the initial exposure and elevated blood lead levels is short (M Hanna-Attisha, LaChance, Sadler, & Champney Schnepp, 2016).

Unlike the relationship between lead in drinking water and elevated blood lead levels, there are other examples where the water contamination is diffuse and the latency period for adverse health effects may be years or decades. One such example would be the development of cancers or birth defects when individuals are exposed to water contaminated with agrichemicals (Jones et al., 2016). The agrichemicals can include pharmaceuticals, such as steroids or antibiotics used on livestock, nutrients, such as nitrates and phosphates, as well as herbicides and insecticides. Agrichemical residues have been found in food, water, and juices (Hills & Welford, 2005; McGill & Robinson, 1968; Zambonin, Quinto, De Vietro, & Palmisano, 2004). While the concentrations found are within set safe limits the true health risk at these levels are not well understood, and could be subjected to synergistic effects (Kortenkamp, 2007). Pesticides in particular have been detected in human breast milk,
which has led to concerns about prenatal exposure and various health effects in children (Pirsaheb, Limoe, Namdari, & Khamutian, 2015). In this case, the chemical source can be agricultural fields, and efforts to map the geospatial organization of the exposure must invariably involve mapping large areas upstream from the communities whose water is affected.

Efforts to map the geospatial distribution of diseases are often conducted by first compartmentalizing the relevant geography into established geographical census units, such as census blocks and block groups, census tracts, zip codes, counties or states. While this may be appropriate for some environmental exposures, it may not be at all appropriate for waterborne agrichemicals, steroids, and antibiotics, since the pathways by which these contaminants travel do not respect anthropogenic geospatial boundaries (Kolok, Beseler, Chen, & Shea, 2009). Rather, these contaminants become mobile when rain storms induce surface runoff that transports the chemicals from land and deposits them into local waterways. These waters ultimately flow downstream within well-defined watersheds.

A watershed is a topographic area within which surface and shallow groundwater drains to a specific point (Griffith, Omernik, & Woods, 1999; Kolok et al., 2009; Omernik & Bailey, 1997). States or other geographic regions can easily be divided into specific watersheds, as everyone lives within one watershed or another. Furthermore, when it comes to waterborne contaminant exposure, two individuals that live miles apart, but within the same watershed, may experience similar exposures, whereas two individuals that live close to each other but in different watersheds may experience very different exposure profiles.
The central hypothesis of this manuscript is that there is an advantage in conducting geospatial analysis relative to adverse health outcomes using watersheds, rather than anthropogenic census tracts, particularly with respect to agrichemical runoff. We contend that the relationship between watershed geography and contaminant distribution is critical for certain classes of chemical contaminants, and this manuscript illustrates a methodology for investigating that relationship.

**Relationship between watershed boundaries and population geography**

**The Watershed**

From an epidemiological perspective, exposure assessment is more complicated when dealing with environmental health studies than it is in occupational health studies. Exposure assessment is the process of measuring the magnitude, frequency, and duration of exposure to a chemical (D. Vallero, 2004), and occupational health studies typically have excellent assessments of exposure due to the defined geospatial boundaries (i.e., the workplace) and the use of predefined exposure definitions by job title.

In contrast to occupational exposure assessment, two of the largest problems encountered when attempting to define a chemical exposure through natural waters are the lack of defined boundaries and the spatial heterogeneity of exposure. Poorly defined boundaries (also known as fuzzy objects) occur when there is no clear boundary of an object in Geographical Information Systems (GIS), an eventuality that is common when
dealing with highly variable metrics within a geography (Ridland, 1997; Zhan, 1998), such as soil type. Spatial heterogeneity of exposure occurs when there is an uneven distribution of various concentrations of chemicals and exposures within a given spatial area, and has been a problem in studies of exposures to airborne contaminants (Monn, 2001)(Zhan, 1998). When the population was organized by population geography, spatial heterogeneity of exposure has caused problems in studies looking at agricultural exposures and birth defects (A. Jack Agopian et al., 2013; Rappazzo et al., 2016), environmental movement of contaminants and neural tube defects (Li et al., 2012), urban environment exposures and cancer incidence (Jagai et al., 2017), and iodine exposure and thyroid cancer (Zimmermann & Galetti, 2015).

We propose that environmental assessment of contaminant exposure via natural waters can best be dealt with when the watershed is used as the defining geospatial boundary. A watershed is defined as an “area of land where drainage of streams and rainfall meet at a common outlet, such as the outflow of a reservoir, mouth of a bay, or any point along a stream channel”((USGS), 2015). A watershed is also an area of connectivity where any activity that affects the water quality, quantity, or rate of movement at one location, can change the characteristics of a watershed downstream, providing a common level of exposure between contaminants.

The United States Geological Society (USGS) has subdivided the US into successively smaller hydrologic units (HU) which can be classified as: regions (HU 2), sub-regions (HU 4), basins (HU 6), sub-basins (HU 8), watershed HU (10) and sub-watershed
(HU 12), respectively. There are 21 major regions in the US, and these are either major river drainage systems or drainage systems for several rivers, such as the Missouri Region and Texas-Gulf Region respectively ((USGS), 2015). Next there are 222 sub regions in the US, these include drainage areas for a river system (“What is a Watershed?,” 2016). There are 370 basins in the US, which are nested within the sub regions. The cataloging unit, which is what the term watershed most frequently is used to represent, has 2264 units across the US(“What is a Watershed?,” 2016).

**The Population**

While the geospatial distribution of waterways can be represented through hydrological units, the most common way that geospatial distribution of humans is represented in the US is through census entities. These entities include the nation, regions, divisions, states, counties, census tracts, census block groups, and census blocks (Table 6).

*Table 6: Definitions of Census Groupings*(Torreiri, 2005)

<table>
<thead>
<tr>
<th>Nation</th>
<th>Regions</th>
<th>Divisions</th>
<th>States</th>
<th>Counties</th>
<th>Census Tracts</th>
<th>Census Block Groups</th>
<th>Census Blocks</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>4 in the US West</td>
<td>9 in the US</td>
<td>50 in the US</td>
<td>3,007 in the US</td>
<td>small, relatively permanent statistical subdivisions of a county or county equivalent and generally have a population</td>
<td>statistical divisions of census tracts and generally contain between 600 and 3,000 people</td>
<td>consists of statistical areas bounded by visible features, such as streets, roads, streams,</td>
</tr>
<tr>
<td></td>
<td>Northeast</td>
<td>New England</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Middle Atlantic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Relative to human health studies, the most commonly used population geographies are city (Alinejad et al., 2016) and county (A. J. Agopian, Lupo, Canfield, & Langlois, 2013; A. Jack Agopian et al., 2013; Henke & Petropoulos, 2013; Khan et al., 2013; Navoni et al., 2014).

Studies that have looked at water exposure and human health outcomes often have limitations of potential classification error (A. J. Agopian, Langlois, Cai, Canfield, &
Classification error is a type of information bias in which study participants are assigned to an incorrect classification group. For example, if a person is assigned to a county, but the county contains multiple watersheds this can cause misclassification, as the assumed exposure may be very different than the actual one. For studies focusing on exposures to natural surface water, we recommend using the watershed as the primary geography.

Overlap

For watersheds to be used in environmental exposure assessment, it is necessary to overlap the human census data with the hydrological unit (HU) data. Unfortunately, watersheds and population geographies were developed by different groups of professionals for very different reasons, and consequently there is very little overlap between the two. While the watershed geography focuses on natural water flow, population geography focuses on governmental delineations and how the population is organized. For this reason, very few points of overlap are seen within the two (Figure 7). Unfortunately, the lack of overlap between watershed HU and human census tracts is not improved when different levels of organization are used. Due to this geographic mismatch, it is not prudent to assume that the exposure to contaminated water is consistent within a county or any other human census group.
Choosing the appropriate hydrologic unit

To map the incidence of adverse health outcomes by watershed, one of the HUs needs to be selected above the others. In Nebraska, the selection of HU was driven by two different descriptors: the exposure profile (land-use) found within the state and the underlying population density.
In Nebraska, soil and precipitation patterns change from East to West, as well as agricultural land use. The term “land use” is defined as the land’s purpose relative to human activity and is usually, but not always, related to land cover. For lands to be useful for agriculture, certain environmental factors are required, including soil conditions and climate (e.g. soil texture, mineralogy, precipitation patterns) which determine the suitability for crop production (e.g. type of fertilizer/pesticide application, type of crop). Furthermore, since the environmental conditions of Nebraska change geospatially, it is likely that agrichemical exposure also changes accordingly.

Population density is defined as “the number of people living per unit of area (e.g. per square mile); the number of people relative to the space occupied by them.” Parts of Nebraska have a very sparse population density. The population density not only within states but also between states. Some states like California is the most populated of the three states presented, whereas Kentucky (Error! Reference source not found., panel A) is mostly evenly populated, with the central portion of the state with the highest population density.
Nebraska can be divided by using 6 different HU codes (Figure 8). The selection of HU was based on observing how the HU divided the state of Nebraska. If the HU delineation was too large, then watersheds with vastly different agricultural practices would be combined, thereby reducing the effectiveness of the analysis. For example, HU code 2 encompasses the entire Missouri river valley and the entire state of Nebraska. Obviously, this does not allow any discrimination of watersheds within the state, and includes so much terrain that there is a vast amount of geographic variability within it. HU codes 4 and 6, were also too large, and had the same problems as HU code 2.
If the HU code selected is too small, then the population within each watershed designation would be too small to allow any meaningful analysis, as many, perhaps the majority, of these watersheds would not cover a geography where an adverse health impact had occurred. According to the United States Cancer Statistics Working Group (USCS), relative incidence rates containing less than 16 cases are unstable and prone to error (United States Cancer Statistics (USCS), 2014). Based on this proposition the following minimum populations per watersheds are required. For birth defects, a population of 600, on average is necessary, to obtain more than 16 cases. For pediatric cancer, a population of 60,000 is needed for 18 cases and for thyroid cancer; a population of 1,500 is necessary for 17 cases. For this reason, HU codes 10 and 12 were too small due to very low population numbers, particularly in the panhandle (western) section of the state. Based on these observations HU 8 was used for preliminary mapping.

**Case Study Diseases and the Population at Risk**

A geospatial analysis favoring a watershed approach lends itself much more readily to some adverse health outcomes rather than others. For example, waterborne contaminants have been linked to birth defects (Alman et al., 2016; Corlin et al., 2016; Kumar, Sarma, & Mohan, 2016; Winchester et al., 2009), pediatric cancers (Costas, Knorr, & Condon, 2002; Infante-Rivard, Olson, Jacques, & Ayotte, 2001; J Liaw et al., 2008; Zahm & Ward, 1998), and thyroid cancer (Drozd et al., 2016; Korobova, Kolmykova, Ryzhenko, Berezkin, & Saraeva, 2016; Malandrino et al., 2016; Pearce & Braverman, 2017). While all three diseases differ in incidence, etiology, and outcomes, the analysis of potential risk factors may benefit
from the use of a watershed approach. For this reason they will be used as case studies for this manuscript.

**Birth defects**

The leading cause of infant mortality in the United States (US) is birth defects or congenital abnormalities (M Heron, 2016). The cost of birth defect related hospitalizations for all age groups represents 5.2% of total costs for all hospital discharges (Arth, 2017). Not only are birth defects costly, but they also affect one in every thirty-three live births in the US (Hoyert, Mathews, Menacker, Strobino, & Guyer, 2006). In 2011, Nebraska had a higher burden of birth defect related death in relation to the nation, with rates of 1.94 per 100,000 and 1.27 per 100,000, respectively (“Nebraska Title V 2015 Needs Assessment,” n.d.).

The risk factors for birth defects are mostly unknown, and vary depending on the type. The most notable risk factors include: alcohol, illicit drug use in pregnancy, smoking, obesity, diabetes mellitus, phenylketonuria, multiple gestations, advanced maternal age, advanced paternal age, family history, folic acid deficiency, medication exposures, and radiation exposure (Corlin et al., 2016).

Several studies support the hypothesis that agrichemicals play a role in the etiology of certain birth defects (Garry et al., 1996; Rappazzo et al., 2016; Rocheleau et al., 2011; Winchester et al., 2009). Pesticides are known to be both reproductive and neurotoxic agents, and have been shown to be teratogenic in animal studies (Shepard & Lemire, 2004). Pesticides, including atrazine, alachlor, and chlorpyrifos, are classified as endocrine disruptors, whereas bifenthrin and diuron are developmental toxicants (Rappazzo et al.,
Studies have shown a potential association between pesticide exposure before or during pregnancy, and various types of birth defects (A. Jack Agopian et al., 2013; Bove et al., 1995; Ma et al., 2014; Ochoa-Acuna & Carbajo, 2009; Rull et al., 2006).

Applications of spatial assessments for birth defects in relation to agricultural land use have provided further insight regarding the etiology of birth defects. For instance, spatial attributes such as elevation, soil types, lithology, watersheds, fertilizer use, and neighborhood characteristics are associated with specific neurological birth defects (Bai, Ge, Wang, & Lan Liao, 2010; Li et al., 2012).

**Pediatric cancers:**
Cancer is the second most common cause of death among children in the US (Siegel et al., 2015). A child born in the US has 0.35% chance of developing cancer before 20 years of age; this is equivalent to an average of 1 in 285 children being diagnosed with cancer before 20 years of age (E. Ward et al., 2014). The Nebraska rates of pediatric cancer were reported to be above the national average in 2010-2012; however, the trend has regressed back to the national average in recent years (“Pediatric Cancer in Nebraska,” 2010). The etiology of childhood cancer is mostly unknown, but some cases can be linked to genetic causes (Johnson et al., 2016; Vega-García et al., 2016). Despite the unknown etiology for most childhood cancers, recent research has linked certain cancers to environmental factors, such as hematological malignancies to oil and gas production (McKenzie et al., 2017),
renal cancers to industrial and pesticide pollution exposure (Garcia-Perez et al., 2016), retinoblastomas and pesticides (Omidakhsh et al., 2017), and crop production proximity and leukemia, neuroblastoma, and hepatic tumors (Gomez-Barroso et al., 2016).

Population at risk

A population at risk is defined as the population that has a chance of developing a disease or condition of interest. This paper features three different adverse health outcomes: birth defects, pediatric cancer, and thyroid cancer, and each of the three has a different population at risk.

Defining the population at risk

Finding a population at risk for an adverse health outcome implies that the researcher understands who in the population is at risk. One way to define this population is to use your case definition, i.e., how the cases are determined to truly be a case. For this article, the following definitions were used, and the cases were gathered from the Nebraska birth defects registry, and the Nebraska cancer registry at the Nebraska Department of Health and Human Services.

Birth defects

The definition used for birth defects was any congenital anomaly from a baby resulting from a live birth, that was recorded in the Nebraska birth defect registry from 1995-2014 (“Nebraska Birth Defects Registry,” n.d.). Based on this definition, the population at risk was infants born alive in Nebraska from 1995-2014.
Pediatric cancer

The definition used for pediatric cancer was any malignancy occurring in someone aged 19 and under, which was recorded in the Nebraska cancer registry from 1987-2014 ("Nebraska DHHS: Cancer Registry: The Latest Nebraska Cancer Data," n.d.). Based on this definition, the population at risk was any child living in Nebraska aged 19 and under from 1987-2014.
Classifying population data based on watershed delineations

Converting the geography of populations into the geography of watersheds may result in misplacing individual cases in a geographically incorrect watershed. Clearly, the smaller the population geography unit, the lower the probability of misclassification error. An example of this is shown in Figure 9. The initial watershed map (Figure 9, Panel A) shows Nebraska with the HU 8 watersheds overlaid on the state. As mentioned above, counties (Figure 9, Panel B) do not overlap well with watersheds. The mismatch is exacerbated by the method used to assign counties to watersheds in GIS, which is by county

Figure 9: Nebraska HU 8 Profile by County (B), Zip Code (C), and Census Block (D)
center. Zip codes (Figure 9, Panel C) and Census Blocks (Figure 9, Panel D) overlap better with watersheds. For this paper, census blocks were chosen to categorize the population at risk.

**Which Watershed to use for these case studies**

According to the United States Cancer Statistics Working Group (USCS), relative incidence rates containing less than 16 cases are unstable and prone to error (United States Cancer Statistics (USCS), 2014). Based on this proposition the following minimum populations per watersheds are required. For example for birth defects, a population of 600, on average is necessary, to obtain more than 16 cases. For this reason, HU codes 10 and 12 were too small due to very low population numbers, particularly in the panhandle (western) section of the state. Based on these observations HU 8 was used for preliminary mapping.

**Incidence rate calculations**

To determine which watersheds to include when mapping statewide adverse health impacts, it was first necessary to determine watershed incidence rate. The true incidence rate ($I_T$) is the number of cases that occur over a given time divided by the current population at risk, and is typically reported per 100,000 (Equation 1).

**Equation 1 General Incidence Rate Calculation for Each Watershed**

$$ \frac{\text{New Cases}_{\text{Watershed}}}{\text{Population at Risk}_{\text{Watershed}} \times Time_{of\ Analysis}} \times 100,000 = \text{Incidence rate (I_T)} $$
Due to the different time periods examined and the different sources of at risk populations, there were three different incidence rates used in this analysis that were based off the general crude incidence rate calculation for each watershed. For example, thyroid cancer cases were aggregated over 28 years and featured a population at risk that was equivalent to the entire population (all ages); therefore Census 2000 population counts were multiplied by 28 years to calculate the total population at risk (Bureau, 2000). In contrast, birth defects were aggregated over 19 years and featured a population at risk equivalent to all live births from 1995 to 2014 (“Nebraska DHHS: Birth Certificate Request,” n.d.). Regardless of the slight differences in how incidence rates were calculated, the final datasets that were developed could then be mapped by watershed.

While the three adverse health impacts that this paper reports can all be considered rare, pediatric and thyroid cancers are rarer than birth defects. The probability of a birth defect is approximately 3 percent, and the incidence rate is likely be mostly stable in Nebraska (Hoyert et al., 2006). For pediatric and thyroid cancers, the probability is much lower (0.3 percent for pediatric cancers and thyroid cancers between 0.6% for men and 1.7% for women). What this means functionally is that that maps developed for pediatric and thyroid cancers are, by necessity, more variable than those developed for birth defects.

**Determining which watersheds to include**

The main way of excluding watersheds from analysis is based on a percent error calculation. In this approach, one calculates the difference adding 1 additional case per
watershed would introduce to the incidence calculation. The first step was to complete the percent error calculation (Equation 3), which includes two parts: the true incidence (Equation 1, \(I_T\)) and the “error” incidence (Equation 2, \(I_E\)), which is the incidence if one additional case was present in the watershed.

**Equation 2 Generalized “Error” Incidence Rate Calculation for Each Watershed**

\[
\frac{\text{Cases}_{\text{Watershed}} + 1}{\text{Population at Risk}_{\text{Watershed}} \times \text{Time of Analysis}} \times 100,000 = \text{Incidence Error (I}_E\text{)}
\]

**Equation 3 General Percent Error Calculation for Each Watershed**

\[
\frac{I_E - I_T}{I_T} \times 100 = \text{Percent Error}
\]
For birth defects, and pediatric cancer, the percent error cut off chosen was 20. The USCS reported a high rate of error to be 25% and that is the cut off they have used; however, due to the small population sizes in rural Nebraska, this was relaxed to 20% to limit the number of excluded watersheds (United States Cancer Statistics (USCS), 2014). Based on this cut off for birth defects 22 out of 72 watersheds were removed from analysis while for pediatric cancer 35 out of 72 watersheds were excluded from analysis.

For birth defects, watershed exclusion based upon percent error was conducted maps were created (Figure 10).

For pediatric cancers, unfortunately, over 40% of the available HU 8 watersheds were excluded. Due to the overall low population values, and high percent error the decision was made to recreate the pediatric cancer graph using HU 6, to allow to more data to be usable within the state. Thus, pediatric cancers are reported using both HU 6 and 8 (Figure 11).

Figure 10: Nebraska HU 8 Profile: (A) Birth Defect Incidence 1995-2014
Results

In Figures 12 and 13 the unadjusted incidence rates for Nebraska, for birth defects, pediatric cancer and thyroid cancer are mapped. For birth defects (Figure 10 panel A) the incidences range from 0-7,692 per 100,000, for pediatric cancers (Figure 11 panel B) the incidence ranges from 0-177 per 100,000 and lastly for thyroid cancers the incidence ranges from 3.25-16.96 per 100,000.

When incidence rates across the three different adverse health outcomes were compared to each other, there were no significant intercorrelations (Table 7). Intercorrelations might have occurred if one or more of the watersheds were contaminated with a key aquatic compound that is known to be associated with one of more adverse health outcomes. When the watersheds were viewed in composite, shed some light on this lack of intercorrelation. For birth defects the watersheds of interest include the Loup River (28), the north fork of the Elkhorn River (30), the lower Platte River (50), the lower Elkhorn (52), the upper Republican (58) and the south fork of the big Nemaha River (68). For pediatric
cancers, the watersheds of interest include the Turkey River (3), the Cedar River (29), and the Upper Elkhorn River (43). For thyroid cancer, the watersheds of interest include the Turkey River (3), the upper Middle Loup River (20), and the lower North Loup River (26). Clearly, the distribution of each adverse health outcome across the watersheds is a different pattern, and these patterns may have to do with the underlying etiology of the disease.

Table 7: Correlation between Birth Defect, Pediatric Cancer, and Thyroid Cancer Incidence in Nebraska by HU 8

<table>
<thead>
<tr>
<th></th>
<th>Birth Defect Incidence</th>
<th>Pediatric Cancer Incidence</th>
<th>Thyroid Cancer Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth Defect Incidence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pediatric Cancer Incidence</td>
<td>-0.07</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Thyroid Cancer Incidence</td>
<td>0.12</td>
<td>0.07</td>
<td>1</td>
</tr>
</tbody>
</table>

For example, thyroid cancer has been linked to higher average levels of nitrate in water supplies (exceeding 5 mg/L) (M. H. Ward et al., 2010). There was also a suggestion for the potential of a link between volcanic elements in water and papillary thyroid cancer, although this potential link needs to be investigated further (Pellegriti et al., 2009). Whereas, overall, pediatric cancer has been linked to pesticides in water (Zahm & Ward, 1998), liver cancer has been specifically linked to arsenic in water supplies (Jane Liaw et al., 2008). Similarly, specific birth defects have been linked with various waterborne exposures, including: central nervous system defects linked with trihalomethanes, carbon tetrachloride, trichloroethylene, and dichloroethylenes (Bove et al., 1995). Oral cleft defects
were linked with trihalomethanes, Carbon tetrachloride, trichloroethylene, tetrachloroethylene, and dichloroethylenes (Bove et al., 1995). Major cardiac defects were linked with trihalomethanes, benzene, and 1,2-dichloroethane (Bove et al., 1995). In addition, congenital cardiac disease was linked with trichloroethylene, dichloroethylene and chromium in ground water (Goldberg, Lebowitz, Graver, & Hicks, 1990). Neural tube defects were linked with carbon tetrachloride, trichloroethylene, and benzene (Bove et al., 1995).

**Conclusions**

The central hypothesis of this manuscript was that there is an advantage in conducting geospatial analysis relative to adverse health outcomes using watersheds, rather than by anthropogenic census tracts, particularly with respect to agrichemical runoff. We contend that the relationship between watershed geography and contaminant distribution is critical for certain classes of chemical contaminants, and this manuscript illustrates a methodology for investigating that relationship.

This use of HUs as geographic spatial polygons for systems that are not necessarily strictly hydrologic in nature has been documented previously (Omernik, Griffith, Hughes, Glover, & Weber, 2017; Zank, Bagstad, Voigt, & Villa, 2016). HU’s have previously been used in ecological modeling (Affuso & Duzy, 2013; Daggupati et al., 2016; Ghimire & Johnston, 2013; Gurung, Githinji, & Ankumah, 2012; Pai, Saraswat, & Daniels, 2011), which is commonly applied to human health behavior research (Sallis, 2010).
It has recently been noted that watersheds seldom circumscribe regions of similarity in that influence water quality (Omernik et al., 2017). Omernik, et al., correctly point out that HUs are not only composed of watersheds but also parts of watersheds (Omernik et al., 2017). Consequently, from a strict hydrological point of view HUs may not represent watersheds. Nevertheless from an epidemiological point of view HU delineation brings a natural, rather than an anthropogenic, focus to the process of geospatial mapping of adverse health impacts. While the delineation of the three adverse health impacts featured in this manuscript did not result in strong intercorrelations, we still think that the use of HUs is a novel and dramatic improvement.

Due to the preliminary nature of this methodology two important factors were not previously discussed these include, the inclusion of sociodemographic information, as well as the potential effects of upstream processes on water quality within the watershed. Due to the nature of the case studies, used social demographic data was not included. This was done to avoid the potential of introducing an ecological fallacy within the data. However, if one was to conduct a cohort study, a case-control study or a cross-sectional study correction for sociodemographic data would easily be included, and is necessary to have corrected and usable incidence rates.

There is potential for upstream human pressure or agricultural activities influence water quality downstream taking into account water routing, evapotranspiration, precipitation and other climate variables. This was not discussed previously in this manuscript due
to the preliminary nature of the study. However, in future refinements of this methodology this will be included.

**Future work**

In the future, we plan to refine this methodology and incorporate water quality data into the approach. Environmental datasets on water quality will include the water quality data from the EPA STORET dataset, as it is comprehensive and includes data from several sources. This process may prove to be complicated as was suggested by Omernik et al, for some HU delineations may experience contaminant input from multiple sources (Omernik et al., 2017). A method to average water quality over the spatial HU scale used in the analysis will need to be developed. A first step of this may be to compare the HU’s used above to the land use maps for Nebraska to quantify the variation within each HU area. Future work also includes applying this process to other states within the Midwest to observe if they show similar profiles.

Overall, the methodology demonstrated in this paper is a way to identify areas of interest with respect to watersheds and human health. As demonstrated by this study, there appears to be link between specific watersheds and the incidence of birth defects, pediatric cancer, and thyroid cancers in the state of Nebraska.
CHAPTER 3: EDUCATIONAL ENGAGEMENT IN CHILDREN WITH OR WITHOUT COMORBID ASD AND ADHD: THE IMPACT OF EXTRACURRICULAR ACTIVITIES

Abstract

BACKGROUND: Participation in extracurricular activities has been linked to higher educational motivation, achievement and high school graduation. However, the process by which this improvement occurs is poorly understood, and has not been well examined children who have developmental differences. AIM: The aim of this study is to examine the link between extracurricular activities and educational engagement for both the typical child, and children with both Attention Deficient Hyperactivity Disorder and Autism Spectrum Disorder. METHODS AND PROCEDURES: Analysis was conducted using the 2011/2012 National Children’s Health Survey. Appropriate modeling was conducted to account for the complex survey design of the dataset. OUTCOMES AND RESULTS: Extracurricular activities and ADHD/ASD diagnosis, physical activity, smoking exposure, parental stress, IEP, age, and sex had the largest effects on the odds of educational engagement. CONCLUSIONS AND IMPLICATIONS: The findings suggest that the extracurricular activities have a positive effect on educational engagement regardless of Attention Deficient Hyperactivity Disorder and Autism Spectrum Disorder. This finding
suggests that future research is needed to identify the optimum types of extracurricular activities to utilize this relationship so that an intervention can be implemented.

**Introduction**

From 1997-1999 to 2006-2008 the prevalence of Attention Deficit Hyperactivity Disorder (ADHD) in the US has risen by 33%, and now affects 6.69% of all children (C. A. Boyle et al., 2011). Population surveys suggest that ADHD occurs in about 5% of children (Faraone et al., 2003) and about 2.5% of adults (Simon et al., 2009) worldwide. In contrast, prevalence of the Autism Spectrum Disorder (ASD) in both children and adult populations, in the US and European countries, is approximately 1% (Brugha et al., 2011). Studies from both the United States and European populations reported that 35-85% of all children with ASD also exhibited symptoms of ADHD (Rao & Landa, 2014). Children with ADHD/ASD have higher numbers of comorbid symptoms and overall severity and these symptoms include tantrum behaviors, conduct problems, oppositional aggressive symptoms, oppositional defiant disorder, conduct disorder, anxiety, and worry/depression (Kenneth D. Gadow et al., 2006; Guttmann-Steinmetz et al., 2009; Jang et al., 2013; Mulligan et al., 2009).

ADHD/ASD comorbidity has been shown to cause a delay in age at diagnosis (T. Stevens et al., 2016). The nature of delayed diagnoses often serves as a limiting factor in a child’s overall ability to succeed in school, having a negative impact on their educational, behavioral, emotional, and cognitive engagement compared to their peers.
A way to measure this impact is in the examination of educational engagement, which includes behavioral, emotional, and cognitive engagement (Moore & Lippman, 2006). Behavioral engagement includes involvement in academic tasks, positive conduct, and the absence of disruptive behaviors. Emotional engagement includes caring about doing well. Cognitive engagement involves curiosity and an investment of time and energy in learning, and a willingness to go beyond the basic requirements to master difficult skills.

Limited classroom engagement has been reported in students with ASD (Bryan & Gast, 2000; Nicholson et al., 2011; Sparapani et al., 2016). Overall, poor high school engagement has been reported in students with ADHD (Zendarski et al., 2017). There has also been a call for additional research conducted on academic achievement and its relationship to ASD (Keen et al., 2015).

Research has shown that extracurricular activities typically have a positive effect on educational engagement in the general population (Badura et al., 2016; Hughes et al., 2016; Morris, 2016). Participation in organized leisure activities was linked with higher school engagement, lower levels of school related stress, and better academic achievement regardless of age or gender (Badura et al., 2016). In 7th and 8th graders, it has been shown that sports predicted competence beliefs and educational values, whereas performing arts and clubs predicted competence beliefs, teacher rated classroom engagement and teacher awarded grades (Im et al., 2016). The biological and social process as behind this tentative
relationship between extracurricular activities and educational outcomes are poorly un-
derstood.

Many of the previously reported studies discussed above were small scale inves-
tigative studies that only investigated children with normal development or one neuro-
developmental disorder. The high level of comorbidity discussed previously shows that
significant portions of children are often excluded from these studies.

Therefore, the aim of this study is to examine the relationship between extracur-
ricular activities and educational engagement to investigate if the relationship is different
based on ADHD/ASD diagnosis.

**Methods**

**Data Source**

Data collected as part of the National Survey of Children’s Health (NSCH) 2011-
2012 was used in this study (2011/12 National Survey of Children’s Health. Maternal and
Child Health Bureau in collaboration with the National Center for Health Statistics, n.d.).
Briefly, the survey consisted of 95,677 phone interviews, using random digit dialing, to
obtain a dual-frame random sample of households with non-institutionalized children
aged 0–17 years. A random child from each household was selected to be the sample child
and the parent or adult that had the most knowledge of the sampled child’s health was
interviewed. Interviews were conducted between February 28, 2011 and June 25, 2012
(Centers for Disease Control and Prevention, 2013). Additional details of the NSCH have
been reported previously (Centers for Disease Control and Prevention, 2013). The University of Nebraska Medical Center Institutional Review Board approved this research as non-human subject research.

**Participants and Eligibility Criteria**

In total 52,835 of the 95,677 surveys collected were analyzed in this study representing 39,309,216 children when accounting for sampling design (Figure 12). The eligibility criteria for this study were, children over 5 years of age with a current parental-reported medical diagnosis of both ADHD and ASD or no parental reported ADHD and ASD. No missing values (e.g. do not know, refused, missing, partial survey) were used in this analysis.
Non-Institutionalized US Children Respondents aged 0-17 years
n=95,677

Excluded Due to Diagnosis
n=19,058
ADHD Only n=152
ASD Only n=130
Unknown n=22
Refused n=0
Missing n=8
Skipped n=18,746

Excluded due to age (under 6)
n=19,430

Excluded due to incomplete Predictor Values
(missing data)
n=4,337

Excluded due to Extracurricular Activity n=17

Total Analyzed
n=52,835

Usually/ Always Educationally Engaged
n=44,960

Never Educational Engaged
n=7,875

Figure 12: Flowchart of Survey Participant Selection for Study 3
**Variables**

**Outcomes**

Educational engagement was determined asking the interviewed adult to indicate if the survey child in question “never, rarely, sometimes, usually, or always” cared about doing well in school, and if he or she completed all required homework (Center of Health Statistics, 2012). These questions were dichotomized into either those who were usually, always engaged in school or were never, rarely, sometimes engaged in school. Those never, rarely, sometimes engaged in school were deemed to be not educationally engaged.

**Exposures**

Interviewed adults were asked to indicate if a doctor or other health care professional had ever diagnosed the survey child with “Attention Deficit Disorder or Attention-Deficit/Hyperactivity Disorder, that is, ADD or ADHD” and “Autism, Asperger's Disorder, pervasive developmental disorder, or other autism spectrum disorder”; the adult then indicated if this diagnosis was current (Center of Health Statistics, 2012). Screener help was available for adults who were not sure what the conditions may be by name.

Extracurricular Activity included participation in any of the following in the last 12 months: “sports team or sports lessons, any clubs or organizations, other organized activities or lessons, such as music, dance, language, or other arts after school or on weekends?” (Center of Health Statistics, 2012), these answers were combined and dichotomized (Yes/No).
There were four options for the intervention domain analyzed in this manuscript: No ADHD/ASD and No Extracurricular Activity, No ADHD/ASD and Yes Extracurricular Activity, Yes ADHD/ASD and No Extracurricular Activity, and Yes ADHD/ASD and Yes Extracurricular.

Covariates

Other variables utilized include child’s age, child’s race, income, parental health, parental education, parental stress, child’s insurance status, child’s individual education plan (IEP) status, childhood physical activity level, child’s smoking exposure, and parental involvement (parent has met all of child’s friends). The income variable used in this analysis was an imputed variable for income as percentage of the federal poverty line (FPL) the imputation has been explained elsewhere (2011/2012 National Survey of Children’s Health, 2013).

Statistical Analysis

Descriptive analyses (frequencies and percentages) were conducted for individual variables. Relative standard error (RSE) was calculated for each covariate at each level, using the methods of Cai and Shimizu, and any RSE above 30% was reported (Cai & Shimizu, 2010). Rao-Scott chi-squared tests were conducted to access associations between covariates and disease severity. Logistic regression analyses were carried out to calculate crude and adjusted odds ratios and 95% confidence intervals (Institute, 2012). Models in-
cluded the variables of interest (neighborhood amenities, detracting elements, and support), demographic variables were forced into the model other variables were included if the estimate of any variable of interest changed by more than 10%.

Survey-specific procedures were used to account for the complex study design. Observations were weighted using complex sampling specifications provided by the Data Resource Center (DRC) data manual including stratification by state and sample type (landline or cell-phone) (Centers for Disease Control and Prevention, 2013). Resulting estimates are representative of all non-institutionalized children aged 6–17 years in the US.

**Results**

Descriptive characteristics of the children in the study population have been summarized in Table 8.

Table 8: Descriptive Characteristics of Children, Parents and Environment (National Survey of Children’s Health; NSCH; 2011-2012)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total</th>
<th>Engaged</th>
<th>Not Engaged</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n¹</td>
<td>N²</td>
<td>Percent (S.E.³)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-8</td>
<td>12,572</td>
<td>9,710</td>
<td>24.7 (0.3)</td>
</tr>
<tr>
<td>9-11</td>
<td>12,786</td>
<td>9,580</td>
<td>24.4 (0.3)</td>
</tr>
<tr>
<td>Characteristics</td>
<td>Total</td>
<td>Engaged&lt;sup&gt;4&lt;/sup&gt;</td>
<td>Not Engaged&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>----------------</td>
<td>-------</td>
<td>---------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td></td>
<td>n&lt;sup&gt;1&lt;/sup&gt;</td>
<td>N&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Percent (S.E.&lt;sup&gt;3&lt;/sup&gt;)</td>
</tr>
<tr>
<td>12-14</td>
<td>14,559</td>
<td>9,940</td>
<td>25.3 (0.3)</td>
</tr>
<tr>
<td>15-17</td>
<td>12,572</td>
<td>9,710</td>
<td>24.7 (0.3)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>25,930</td>
<td>19,000</td>
<td>48.3 (0.2)</td>
</tr>
<tr>
<td>Female</td>
<td>26,905</td>
<td>20,310</td>
<td>51.7 (0.2)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>35,584</td>
<td>20,850</td>
<td>53.0 (0.1)</td>
</tr>
<tr>
<td>Non-Hispanic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>5,068</td>
<td>5,600</td>
<td>14.3 (0.3)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>6,657</td>
<td>8,890</td>
<td>22.6 (0.4)</td>
</tr>
<tr>
<td>Multi-racial&lt;sup&gt;6&lt;/sup&gt;</td>
<td>5,526</td>
<td>3,970</td>
<td>10.1 (0.2)</td>
</tr>
<tr>
<td>Household</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>income (FPL) &lt;sup&gt;7&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;100%</td>
<td>6,691</td>
<td>7,610</td>
<td>19.4 (0.3)</td>
</tr>
<tr>
<td>Characteristics</td>
<td>Total</td>
<td>Engaged$^4$</td>
<td>Not Engaged$^4$</td>
</tr>
<tr>
<td>----------------------------</td>
<td>-------------</td>
<td>-------------</td>
<td>-----------------</td>
</tr>
<tr>
<td></td>
<td>n$^1$</td>
<td>N$^2$</td>
<td>Percent (S.E.$^3$)</td>
</tr>
<tr>
<td>100-199%</td>
<td>8,841</td>
<td>8,320</td>
<td>21.2 (0.3)</td>
</tr>
<tr>
<td>200-399%</td>
<td>16,657</td>
<td>11,610</td>
<td>29.5 (0.3)</td>
</tr>
<tr>
<td>≥400%</td>
<td>20,646</td>
<td>11,770</td>
<td>29.9 (0.2)</td>
</tr>
<tr>
<td>Insurance status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>50,397</td>
<td>36,940</td>
<td>94.0 (0.0)</td>
</tr>
<tr>
<td>No</td>
<td>2,438</td>
<td>2,370</td>
<td>6.0 (0.2)</td>
</tr>
<tr>
<td>Extracurricular activities and ADHD/ASD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>neither</td>
<td>6,620</td>
<td>6,720</td>
<td>17.1 (0.3)</td>
</tr>
<tr>
<td>One or more</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>activity neither</td>
<td>45,630</td>
<td>32,200</td>
<td>81.9 (0.1)</td>
</tr>
<tr>
<td>Characteristics</td>
<td>Total</td>
<td>Engaged&lt;sup&gt;4&lt;/sup&gt;</td>
<td>Not Engaged&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>-------------------------------------</td>
<td>-------</td>
<td>--------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td></td>
<td>n&lt;sup&gt;1&lt;/sup&gt;</td>
<td>N&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Percent (S.E.&lt;sup&gt;3&lt;/sup&gt;)</td>
</tr>
<tr>
<td>No activity both diagnoses</td>
<td>171</td>
<td>140</td>
<td>0.3 (0.1)</td>
</tr>
<tr>
<td>One or more activity&lt;sup&gt;a&lt;/sup&gt; both diagnoses</td>
<td>414</td>
<td>250</td>
<td>0.6 (0.1)</td>
</tr>
<tr>
<td>Individualized education plan</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>49,013</td>
<td>36,660</td>
<td>93.3 (0.0)</td>
</tr>
<tr>
<td>No</td>
<td>3,822</td>
<td>2,640</td>
<td>6.7 (0.2)</td>
</tr>
<tr>
<td>Physical activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 days</td>
<td>3,644</td>
<td>3,250</td>
<td>8.3 (0.2)</td>
</tr>
<tr>
<td>1-3 days</td>
<td>12,852</td>
<td>9,920</td>
<td>25.2 (0.3)</td>
</tr>
<tr>
<td>4-6 days</td>
<td>21,865</td>
<td>15,220</td>
<td>38.7 (0.2)</td>
</tr>
<tr>
<td>Everyday</td>
<td>14,474</td>
<td>10,920</td>
<td>27.8 (0.3)</td>
</tr>
<tr>
<td>Smoking exposure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>at home</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Characteristics</td>
<td>Total</td>
<td>Engaged(s)</td>
<td>Not Engaged(s)</td>
</tr>
<tr>
<td>---------------------------------------------</td>
<td>-----------</td>
<td>------------</td>
<td>----------------</td>
</tr>
<tr>
<td></td>
<td>n(^1)</td>
<td>N(^2)</td>
<td>Percent (S.E.(^3))</td>
</tr>
<tr>
<td>No exposure</td>
<td>41,297</td>
<td>30,200</td>
<td>76.8 (0.1)</td>
</tr>
<tr>
<td>Exposure to smoke outside home</td>
<td>8,769</td>
<td>6,940</td>
<td>17.7 (0.3)</td>
</tr>
<tr>
<td>Exposure to smoke inside home</td>
<td>2,769</td>
<td>2,170</td>
<td>5.5 (0.2)</td>
</tr>
<tr>
<td>Parental health</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No parents in home</td>
<td>1,808</td>
<td>1,160</td>
<td>2.9 (0.1)</td>
</tr>
<tr>
<td>One of two parents in home in poor health</td>
<td>15,111</td>
<td>13,310</td>
<td>33.9 (0.3)</td>
</tr>
<tr>
<td>All parent(s) in home in poor health</td>
<td>9,365</td>
<td>6,220</td>
<td>15.8 (0.2)</td>
</tr>
<tr>
<td>Characteristics</td>
<td>Total</td>
<td>Engaged&lt;sup&gt;4&lt;/sup&gt;</td>
<td>Not Engaged&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>-----------------------------------------------------</td>
<td>----------------</td>
<td>---------------------</td>
<td>-------------------------</td>
</tr>
<tr>
<td></td>
<td>n&lt;sup&gt;1&lt;/sup&gt;</td>
<td>N&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Percent (S.E.&lt;sup&gt;3&lt;/sup&gt;)</td>
</tr>
<tr>
<td>All parent(s) in home in good health&lt;sup&gt;9&lt;/sup&gt;</td>
<td>26,551</td>
<td>18,620</td>
<td>47.4 (0.2)</td>
</tr>
<tr>
<td>Parental education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No parents in home</td>
<td>1,808</td>
<td>1,160</td>
<td>2.9 (0.1)</td>
</tr>
<tr>
<td>One of two parents in home less than high school</td>
<td>2,368</td>
<td>2,390</td>
<td>6.1 (0.2)</td>
</tr>
<tr>
<td>All parent(s) in home less than high school&lt;sup&gt;8&lt;/sup&gt;</td>
<td>2,669</td>
<td>4,300</td>
<td>10.9 (0.3)</td>
</tr>
<tr>
<td>All parent(s) in home at least high school&lt;sup&gt;9&lt;/sup&gt;</td>
<td>45,990</td>
<td>31,450</td>
<td>80.0 (0.1)</td>
</tr>
<tr>
<td>Parental stress</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Characteristics</td>
<td>Total</td>
<td>Engaged&lt;sup&gt;4&lt;/sup&gt;</td>
<td>Not Engaged&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>--------------------------</td>
<td>-------------</td>
<td>----------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td></td>
<td>n&lt;sup&gt;1&lt;/sup&gt;</td>
<td>N&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Percent (S.E.&lt;sup&gt;3&lt;/sup&gt;)</td>
</tr>
<tr>
<td>Yes&lt;sup&gt;10&lt;/sup&gt;</td>
<td>3,925</td>
<td>3,730</td>
<td>9.5 (0.2)</td>
</tr>
<tr>
<td>No&lt;sup&gt;11&lt;/sup&gt;</td>
<td>48,910</td>
<td>35,580</td>
<td>90.5 (0.1)</td>
</tr>
<tr>
<td>Parents met child’s</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>friends</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>16,716</td>
<td>11,100</td>
<td>28.2 (0.2)</td>
</tr>
<tr>
<td>Most</td>
<td>27,365</td>
<td>19,500</td>
<td>49.6 (0.2)</td>
</tr>
<tr>
<td>Some</td>
<td>8,198</td>
<td>7,880</td>
<td>20.0 (0.3)</td>
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<tr>
<td>None</td>
<td>479</td>
<td>750</td>
<td>1.9 (0.2)</td>
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<tr>
<td>Child has no</td>
<td>77</td>
<td>80</td>
<td>0.2 (0.0)</td>
</tr>
<tr>
<td>friends</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>52,835</td>
<td>39,310</td>
<td>100 (0.0)</td>
</tr>
</tbody>
</table>

Percentages adjusted for complex survey design
1. Number of children in survey sample
2. Population sample of survey, in 1,000’s
3. Standard error
4. Educational Engagement Status
5. Rao-Scott Chi-Square p-value for educational engagement status
6. Includes other non-Hispanic
7. Reported as federal poverty line (FPL) percentage
8. Includes organized activities outside of school, such as sports teams or lessons, clubs, organizations, music, dance, language or other arts
<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total</th>
<th>Engaged (^4)</th>
<th>Not Engaged (^4)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n(^1)</td>
<td>N(^2)</td>
<td>Percent (S.E.(^3))</td>
</tr>
</tbody>
</table>

9. Homes with both parents, single fathers, and single mothers
10. Parents responded "usually" or "always" to at least one of three questions: child was much harder to care for than other children were; often bothered a lot by their child's behavior; and/or angry with child.
11. Parents responded "sometimes" or "never" to all three questions: child was much harder to care for than other children were; often bothered a lot by their child's behavior; and/or angry with child.

Children were predominantly White Non-Hispanic (53%), in households with incomes above 200% the FPL (59%), and insured (94%). ADHD/ASD diagnosis was rare, affecting only 1% of the children. Children participated in afterschool activities (82%), and lived in homes with at least one parent (97%).

The association between a range of child and parent characteristics and educational engagement was investigated (Table 9).
<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Educational engagement&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Crude odds ratios &lt;sup&gt;(95% CI)&lt;/sup&gt;</th>
<th>Adjusted odds ratios&lt;sup&gt;2&lt;/sup&gt; &lt;sup&gt;(95% CI)&lt;/sup&gt;</th>
<th>Adjusted odds ratios&lt;sup&gt;3&lt;/sup&gt; &lt;sup&gt;(95% CI)&lt;/sup&gt;</th>
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<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>6-8</td>
<td>1.80 (1.55, 2.10)</td>
<td>2.16 (1.82, 2.57)</td>
<td>2.14 (1.80, 2.54)</td>
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<tr>
<td>9-11</td>
<td>2.23 (1.90, 2.61)</td>
<td>1.74 (1.48, 2.05)</td>
<td>1.73 (1.47, 2.03)</td>
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</tr>
<tr>
<td>12-14</td>
<td>1.35 (1.18, 1.55)</td>
<td>1.29 (1.11, 1.49)</td>
<td>1.28 (1.10, 1.48)</td>
<td></td>
</tr>
<tr>
<td>15-17</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>2.16 (1.94, 2.41)</td>
<td>2.26 (2.02, 2.53)</td>
<td>2.27 (2.02, 2.54)</td>
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<tr>
<td>Race</td>
<td></td>
<td></td>
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<tr>
<td>White non-Hispanic</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td></td>
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<tr>
<td>Black non-Hispanic</td>
<td>0.62 (0.54, 0.72)</td>
<td>0.77 (0.66, 0.90)</td>
<td>0.77 (0.66, 0.90)</td>
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<tr>
<td>Hispanic</td>
<td>0.93 (0.79, 1.10)</td>
<td>1.09 (0.90, 1.32)</td>
<td>1.21 (1.00, 1.46)</td>
<td></td>
</tr>
<tr>
<td>Characteristics</td>
<td>Educational engagement&lt;sup&gt;1&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------------------</td>
<td>-----------------------------------</td>
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<tr>
<td></td>
<td>Crude odds ratios</td>
<td>Adjusted odds ratios&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Adjusted odds ratios&lt;sup&gt;3&lt;/sup&gt;</td>
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<tr>
<td></td>
<td>(95% CI)</td>
<td>(95% CI)</td>
<td>(95% CI)</td>
<td></td>
</tr>
<tr>
<td>Multi-racial&lt;sup&gt;4&lt;/sup&gt;</td>
<td>1.15 (0.95, 1.39)</td>
<td>1.26 (1.04, 1.52)</td>
<td>1.29 (1.07, 1.56)</td>
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<td>Household income</td>
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<td>(FPL)&lt;sup&gt;5&lt;/sup&gt;</td>
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<tr>
<td>&lt;100%</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>100-199%</td>
<td>1.25 (1.06, 1.48)</td>
<td>1.10 (0.91, 1.33)</td>
<td>1.02 (0.85, 1.23)</td>
<td></td>
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<tr>
<td>200-399%</td>
<td>1.49 (1.27, 1.74)</td>
<td>1.14 (0.94, 1.38)</td>
<td>0.98 (0.82, 1.18)</td>
<td></td>
</tr>
<tr>
<td>≥400%</td>
<td>2.06 (1.76, 2.41)</td>
<td>1.43 (1.17, 1.75)</td>
<td>1.20 (0.99, 1.46)</td>
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</tr>
<tr>
<td>Insurance status</td>
<td></td>
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<td>Yes</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
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<tr>
<td>No</td>
<td>0.84 (0.65, 1.09)</td>
<td>1.04 (0.79, 1.37)</td>
<td>1.09 (0.83, 1.43)</td>
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<tr>
<td>Extracurricular activities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>and ADHD/ASD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No activity, neither</td>
<td>7.74 (3.35, 17.89)</td>
<td>2.55 (1.07, 6.09)</td>
<td>2.72 (1.09, 6.78)</td>
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</tr>
<tr>
<td>Characteristics</td>
<td>Educational engagement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------</td>
<td>------------------------</td>
<td>---</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Crude odds ratios (95% CI)</td>
<td>Adjusted odds ratios (95% CI)</td>
<td>Adjusted odds ratios (95% CI)</td>
<td></td>
</tr>
<tr>
<td>One or more activities, neither diagnoses</td>
<td>14.25 (3.20, 32.77)</td>
<td>3.64 (1.53, 8.69)</td>
<td>3.81 (1.53, 9.48)</td>
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<tr>
<td>No activity both diagnoses</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
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<tr>
<td>One or more activities, both diagnoses</td>
<td>1.65 (0.66, 4.13)</td>
<td>1.46 (0.57, 3.73)</td>
<td>1.57 (0.59, 4.17)</td>
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<td>Individualized education plan</td>
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<td>1.00</td>
<td>1.00</td>
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<tr>
<td>No</td>
<td>3.16 (2.70, 3.71)</td>
<td>2.27 (1.90, 2.71)</td>
<td>2.26 (1.90, 2.69)</td>
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<tr>
<td>Physical activity</td>
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<td>0 days</td>
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<td>1.00</td>
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</tr>
<tr>
<td>1-3 days</td>
<td>1.92 (1.59, 2.33)</td>
<td>1.48 (1.20, 1.83)</td>
<td>1.48 (1.20, 1.82)</td>
<td></td>
</tr>
<tr>
<td>Characteristics</td>
<td>Educational engagement¹</td>
<td>Crude odds ratios</td>
<td>Adjusted odds ratios²</td>
<td>Adjusted odds ratios²</td>
</tr>
<tr>
<td>---------------------------------------</td>
<td>-------------------------</td>
<td>------------------</td>
<td>-----------------------</td>
<td>-----------------------</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(95% CI)</td>
<td>(95% CI)</td>
<td>(95% CI)</td>
</tr>
<tr>
<td>4-6 days</td>
<td></td>
<td>2.37 (1.97, 2.86)</td>
<td>1.70 (1.38, 2.10)</td>
<td>1.67 (1.35, 2.06)</td>
</tr>
<tr>
<td>Everyday</td>
<td></td>
<td>2.05 (1.68, 2.50)</td>
<td>1.54 (1.23, 1.93)</td>
<td>1.51 (1.21, 1.89)</td>
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<td>Smoking exposure at home</td>
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<td></td>
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<td>No exposure</td>
<td></td>
<td>2.59 (2.19, 3.07)</td>
<td>1.95 (1.62, 2.35)</td>
<td>1.85 (1.54, 2.22)</td>
</tr>
<tr>
<td>Exposure to smoke mostly outside home</td>
<td></td>
<td>1.76 (1.45, 2.12)</td>
<td>1.38 (1.13, 1.70)</td>
<td>1.35 (1.10, 1.65)</td>
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<tr>
<td>Exposure to smoke mostly inside home</td>
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<td>1.00</td>
<td>1.00</td>
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<td>Parental health</td>
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<td></td>
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<td>No parents in home</td>
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<td>1.00</td>
<td>1.00</td>
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<tr>
<td>One of two parents in home in poor health</td>
<td></td>
<td>1.96 (1.49, 2.59)</td>
<td></td>
<td>1.42 (1.03, 1.96)</td>
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<tr>
<td>All parent(s) in home in poor health</td>
<td></td>
<td>1.25 (0.96, 1.63)</td>
<td></td>
<td>1.15 (0.85, 1.56)</td>
</tr>
<tr>
<td>Characteristics</td>
<td>Educational engagement¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------------------------------------------------</td>
<td>--------------------------</td>
<td>------------------</td>
<td>------------------</td>
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<tr>
<td></td>
<td>Crude odds ratios</td>
<td>Adjusted odds ratios²</td>
<td>Adjusted odds ratios²</td>
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</tr>
<tr>
<td></td>
<td>(95% CI)</td>
<td>(95% CI)</td>
<td>(95% CI)</td>
<td></td>
</tr>
<tr>
<td>All parent(s) in home in good health²</td>
<td>2.35 (1.81, 3.06)</td>
<td>1.59 (1.17, 2.16)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parental education</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No parents in home</td>
<td>1.00</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>One of two parents in home less than high school</td>
<td>1.57 (1.15, 2.15)</td>
<td>0.78 (0.53, 1.15)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All parent(s) in home less than high school²</td>
<td>1.31 (0.93, 1.84)</td>
<td>1.36 (0.97, 1.91)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All parent(s) in home at least high school²</td>
<td>1.86 (1.44, 2.40)</td>
<td>0.98 (0.75, 1.28)</td>
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<td></td>
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<tr>
<td>Parental stress</td>
<td></td>
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</tr>
<tr>
<td>Yes⁸</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>No⁹</td>
<td>3.15 (2.71, 3.66)</td>
<td>2.35 (2.01, 2.76)</td>
<td>2.23 (1.90, 2.61)</td>
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</tr>
</tbody>
</table>
### Educational engagement

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Crude odds ratios</th>
<th>Adjusted odds ratios(^3)</th>
<th>Adjusted odds ratios(^3) (95% CI)</th>
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<tbody>
<tr>
<td>Parents met child’s friends</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>5.12 (2.28, 11.51)</td>
<td>1.65 (0.74, 3.65)</td>
<td>1.53 (0.70, 3.39)</td>
</tr>
<tr>
<td>Most</td>
<td>3.98 (1.78, 8.91)</td>
<td>1.29 (0.58, 2.86)</td>
<td>1.21 (0.55, 2.67)</td>
</tr>
<tr>
<td>Some</td>
<td>2.36 (1.05, 5.31)</td>
<td>0.95 (0.43, 2.10)</td>
<td>0.91 (0.41, 2.01)</td>
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<tr>
<td>None</td>
<td>1.71 (0.70, 4.21)</td>
<td>0.83 (0.34, 2.04)</td>
<td>0.82 (0.34, 2.01)</td>
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<tr>
<td>Child has no friends</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Odds ratios adjusted for complex survey design
1. Modeling for positive educational engagement
2. Confidence interval
3. Model includes all characteristics with odds ratios in the column
4. Includes other non-Hispanic
5. Reported as federal poverty line (FPL) percentage
6. Includes organized activities outside of school, such as sports teams or lessons, clubs, organizations, music, dance, language or other arts
7. Homes with both parents, single fathers and single mothers
8. Parents responded “usually” or “always” to at least one of three questions: child was much harder to care for than other children were; often bothered a lot by their child's behavior; and/or angry with child
9. Parents responded “sometimes” or “never” to all three questions: child was much harder to care for than other children were; often bothered a lot by their child’s behavior; and/or angry with child
Females had twice the odds of positive educational engagement compared to males in both crude and adjusted models. Those children that participated in extracurricular activities and did not have ADHD/ASD had adjusted odds 3.6 to 3.8 times higher for being educationally engaged when compared to those children who had ADHD/ASD and did not participate in extracurricular activities. Whereas, those children who had ADHD/ASD and participated in extracurricular activities has about 1.5 the adjusted odds of being educationally engaged compared to those children who had ADHD/ASD and did not participate in extracurricular activities. Children with all parents in home in good health had 1.6 times the odds of educational engagement than those children with no parents in home in the adjusted model. Those children who had parents that were not stressed had about 2.3 times higher adjusted odds of being educationally engaged compared to those children who had parents that were stressed.

**Discussion and implications**

Extracurricular activities and ADHD/ASD diagnosis, physical activity, smoking exposure, parental stress, IEP, age, and sex had the largest effects on the odds of educational engagement. Overall, the findings in this study are consistent with findings from similar studies, which have reported that sex, and age have both been related to educational engagement, in previous studies (Wilcox, Mcquay, Blackstaffe, Perry, & Hawe, n.d.; Wilcox, McQuay, Blackstaffe, Perry, & Hawe, 2016).

Extracurricular activities as a whole have been linked to educational engagement (Sauerwein, Theis, & Fischer, 2016). The positive effect on educational engagement from
extracurricular activities finding agrees with previously published studies (Badura et al., 2016; Hughes et al., 2016; Morris, 2016).

This study found that children with ADHD/ASD had lower educational engagement than the general population. This finding agrees with previously published studies, that children with ADHD and ASD separately have lower educational engagement and attainment (Bryan & Gast, 2000; Loe & Feldman, 2007; Nicholson et al., 2011; Sparapani et al., 2016; Vile Junod et al., 2006; Zendarski et al., 2017). Participation in extracurricular activities attenuated this negative effect. This agrees with studies that show children with ADHD, who participate in physical activities typically have better behavior which would likely lead to higher educational engagement (Cuypers et al., 2011; Verret et al., 2012a).

Wilcox et al (2016) stated that only 20% of the variance in educational engagement was explained by extracurricular activities, while in the crude model participation in extracurricular activities more than doubled the odds of educational engagement in the general population, in the adjusted models this effect was greatly lessened. Parental stress, smoking exposure, parental health, and individualized educational plans, all had the same effect on overall odds ratio in the adjusted model as the extracurricular activity participation had within the normal population. These factors may help explain the additional 80% of variance, in Wilcox et al’s study.
Strengths and Limitations

The major strength of this study was the large sample size, which allowed sufficient power to examine the relationship of interest. A limitation in this study is the cross-sectional nature of the study, which does not allow the measurement of causality. Another limitation in this study is the possibility of misclassification of ADHD/ASD grouping, due to the parental reported aspect of the survey; this was minimized by the availability of screeners that described the conditions to the adults participating, and asked about diagnosis from a medical professional. Another limitation in this study is the use of an imputed income variable, by using an imputed variable you are assigning children in to an income classification that may not be correct.

Conclusions and Implications

The findings suggest that extracurricular activities have a positive effect on educational engagement, regardless of ADHD/ASD. While this study did show the overall decrease in educational engagement for ADHD/ASD, there was an increase in odds when extracurricular activities was added. Though these findings were not significant, they offer an intervention for investigation into ways to increase overall educational engagement in this population. It is also suggested that physical activity, smoking exposure, parental stress, individualized education plan, age, and sex all influence the educational engagement of children. Future research is necessary to understand the relationships found in this study.
DISCUSSION

The previous chapters have shown that there is an impact on health from the environment throughout childhood. This relationship was found in relation to all chronic conditions examined.

ADHD/ASD and the Built Environment

Overall all three facets of the built environment examined (neighborhood amenities, detracting elements, and support) were found to be related to ADHD/ASD symptom severity, even if this relationship was not significant.

Aim 1

The creation of the ADHD/ASD symptom severity variable was not as straightforward as originally planned. It was decided that those children with vastly different symptomology between the two disorders would be excluded from analysis. The most common diagnosis group was Mild ASD, and Moderate ADHD followed closely by those children with Moderate for both disorders. The least common diagnosis group was those children with Severe ASD and Moderate ADHD.

The somewhat similar definitions and disease, presentation between ADHD and ASD is cause for concern. It is likely that the two diseases feed off each other, and make it difficult for the parents to ascertain which condition is actually driving the more severe symptoms their child is exhibiting.
The overall, symptom severity was lower than was expected for ADHD/ASD, previous literature has shown that children with ADHD/ASD have high comorbid symptoms and higher overall severity (Kenneth D. Gadow et al., 2006; Guttmann-Steinmetz et al., 2009; Jang et al., 2013; Mulligan et al., 2009). This discrepancy might be because there was no difference in ADHD medication between the two severity groups, which may show an overlapping severity profile. This study also had no way of accounting for treatment profiles for the children when accessing severity due to the survey nature of the data.

**Aim 2**

The three built environment variables examined in this study included neighborhood amenities, detracting elements, and support. There was no statistical difference in the level of the three variables between the two severity groups. Neighborhood amenities had the highest odds when related to symptom severity, followed by neighborhood detracting elements, and lastly neighborhood social support.

Previous research has shown that deprived environments can lead to conduct problems, which agrees with these findings for neighborhood detracting elements, however, disagrees with the findings for neighborhood amenities (Schonberg & Shaw, 2007). In this study, it was found that more amenities was related to higher symptom severity. This disagreement may be a product of the specific amenities examined, which included sidewalks or walking paths, parks or playground areas, a recreation center, community center, or boys’ or girls’ club, and library or bookmobile. While research for
ADHD has shown that green space should lower severity, other neighborhood amenities have not been well researched (Van Den Berg & Van Den Berg, 2010).

The findings for neighborhood detracting elements agrees with previously published findings. Neighborhood detracting elements included litter or garbage on the street or sidewalk, poorly kept or run-down housing, and vandalism such as broken windows or graffiti. All of which are commonly found in socially disadvantaged neighborhoods with lower SES. The higher the levels of detracting elements, the more severe the ADHD/ASD symptoms. This finding is supported by other studies which found behavioral problems in these areas, anti-social behaviors, and increased ASD symptomology (Dubow et al., 1997; Hock & Ahmedani, 2012; Kanne et al., 2009; Singh & Ghandour, 2012).

The national social support findings of this study also agreed with previously published studies. In this study not having social support was associated with high symptom severity, which was also found in previous studies. Social support has been found to be associated with higher odds of ADHD diagnosis and severity (Razani et al., 2015a). There is poor neighborhood social capital was associated with ASD diagnosis (Hock & Ahmedani, 2012).

**Specific Aims: Congenital Abnormalities, Pediatric Cancers and the Physical Environment**

From an epidemiological point of view, HU delineation brings a natural, rather than an anthropogenic; focus on the process of geospatial mapping of chronic health conditions.
While the delineation of the three adverse health impacts featured in this chapter did not result in strong intercorrelations, the use of HUs is a novel and dramatic improvement. As seen in the maps for CA and PC the HU where someone lives appear to be related to the incidence of all three chronic conditions.

**Aim 3**

Human health is typically explored using census block or other anthropogenic groupings. However as shown in chapter 2 this is not an effective way to examine physical environmental exposures. The relationship between geography and contaminant distribution is critical for certain classes of chemical contaminants. For this reason, HU was used in chapter 2. However, it should be noted that it has recently been noted that watersheds seldom circumscribe regions of similarity in that influence water quality (Omernik et al., 2017). Consequently, from a strict hydrological point of view, HUs may not represent watersheds.

In rural areas like Nebraska, most of pollution will be water-soluble due to being an agrichemical pollutant. For this reason, using a geographical shape that is related in some fashion to the water flow in the state is a starting point for identifying the exposures at play.

**Aim 4**

As previously stated the HU system is not foolproof. As pointed out by Omernik et al., HUs are not only composed of watersheds but also parts of watersheds (Omernik et al., 2017). This will affect how the HU is used and which one is ultimately used. Due to the
change in soil and precipitation in Nebraska, which HU that is used here may not be appropriate in a state where this change does not occur. Another big factor in choosing the HU here was the sparse population. As shown in chapter 2 Nebraska had very low population density, which proved to be a problem with the PC maps.

**Aim 5**

Identifying the population at risk for each condition was straightforward and did not offer any surprises.

**Aim 6**

The incidence for CA, and PC, were mapped using HU 8, 8 and 6, and 8 respectively. The incidence rates did not act as expected. As stated, there were no significant intercorrelations. There are several reasons this may be the case. As listed, the risk factors previously for CA and PC are not well known. Compounds that may have been estimated in these maps may include agrichemicals, soil types, air pollution, industrial pollution, and heavy metals.

Agrichemicals specifically pesticides have been linked to all three conditions studied. However, the same agrichemicals do not cause the risk of the three conditions. While overall non-specific pesticide exposure has been linked PC, CA has had specific pesticides linked; the only overlapping pesticide name is Atrazine, which is used almost uniformly over the eastern part of Nebraska in the corn belt (Lerro et al., 2016; Rappazzo et al., 2016). Soil type distributions have only been linked to CA (Li et al., 2012).
Air pollution has been linked most with PC. However different air pollutants have been linked with different outcomes (Belson et al., 2007; Buffler et al., 2005; Ritz et al., 2002).

Various industrial pollutants have been linked to PC of the renal system, whereas hematological malignancies have been linked to oil and gas (Garcia-Perez et al., 2016; McKenzie et al., 2017).

While Nebraska does have two nuclear power stations, the most commonly found source of radiation within the state is the naturally occurring radon in the ground, which has not been directly linked to any of the conditions researched here.

The only heavy metal in Nebraska commonly found above EPA levels in soil is Arsenic, which is most common along the Nebraska, Iowa border, and the Elkhorn water basin. Arsenic is most commonly causes developmental toxicity and has not been found to cause PC or TC.

**Specific Aims: ADHD/ASD and the Social Environment**

Overall, participation in any extracurricular activity lead to higher educational engagement, this relationship held true for the ADHD/ASD population as well.

**Aim 7**

The findings from this study about the relationship between educational engagement and extracurricular activities agree with previously published studies (Badura et al.,
This study found that participating in any extracurricular activity was associated with higher odds of educational engagement. This study also agreed with previously published work, that family income does not appear to be related to this relationship, with no significant relationship being found (Morris, 2016).

**Aim 7.1**

This study found that children with ADHD/ASD overall had lower educational engagement than the general population. This finding agrees with previously published studies that state children with ADHD and ASD separately have lower educational engagement and attainment (Bryan & Gast, 2000; Loe & Feldman, 2007; Nicholson et al., 2011; Sparapani et al., 2016; Vile Junod et al., 2006; Zendarski et al., 2017).

If ADHD/ASD children participated in extracurricular activities, this effect was attenuated even if not to a significant effect. This agrees with studies that show children with ADHD, who participate in physical activities typically have better behavior which would likely lead to higher educational engagement (Cuypers et al., 2011; Verret et al., 2012a).

**Overall relationship between health and environment**

In the previous research, the relationship between overall environment and human health was explored. There is evidence to support the overall hypothesis that the environment has a lifelong effect on chronic health.
This overall finding is supported by previous literature (Barrington-Trimis et al., 2016; Council, 1997; Hynes et al., 2000; Landrigan et al., 2004; Mitchell & Popham, 2008; Stafford et al., 2005; Turecki & Meaney, 2016; Weich et al., 2002).

**Lifelong relationships**

This research shows that the effect the environment has on human health starts at conception. This finding agrees with previous research (Burris, Baccarelli, Wright, & Wright, 2016; Heindel et al., 2015; Zambrana, Scrimshaw, Collins, & Dunkel-Schetter, 1997). The relationship between environment and health continues into childhood, adolescences, and teenage years. In the previous research, mental health and cancer were the topics of research. While these findings have already been discussed other areas of childhood health have been researched in relation to the environment, these findings were mixed outcomes but showed the relationship between environment and health (G. Evans, 2004; Nelson, Gordon-Larsen, Song, & Popkin, 2006; Viner et al., 2012). The relationship between the environment and health in adulthood is best researched. While this research focused on cancer, both mental health and other chronic conditions have been researched (Hammersen, Niemann, & Hoebel, 2016; Mitchell & Popham, 2008; Yan, Bastian, & Griffin, 2015). Research has also been conducted on the elderly specifically (Deierlein, Morland, Scanlin, Wong, & Spark, 2014; Moran et al., 2014; Park, Smith, Dunkle, Ingersoll-Dayton, & Antonucci, 2017)


A compounded effect

The research showed that all three facets of the environment affect human health, for the entirety of human life. This effect may be compounded by a few factors including SES (locations specific accumulation), lack of economic mobility (longitudinal accumulation), and health behaviors (mitigating/ additive factors).

Location Specific Accumulation

Most studies looking at human health will examine SES status, rather it should medically influence the outcome or not. This is especially common in research done in the US where income is directly related to health insurance status. There has been research done recently showing that SES might also be an indicator or substitute for stress on the body (Adler & Newman, 2002; Finkelstein, Kubzansky, Capitman, & Goodman, 2007; Matthews & Gallo, 2011).

I hypothesize that the tenuous relationship between SES and health is also related the accumulation of positive or negative environmental factors. Typically, poorer neighborhoods will have poor built, physical, and social environments, which contribute to the stress factors that have been researched previously. This would also explain why a gradient effect is rarely found between health and SES. In theory, if SES is playing a direct role in the etiology of health, it should show a gradient effect with the effect getting progressively worse as SES gets worse. Typically, the only substantial difference seen is between the highest and lowest levels of SES. Those people in middle SES levels might
live in transition neighborhood where the built environment is improved or might have the money to attend social functions outside their neighborhood. This shows that there may not be an equal contribution between the three facets of environment and human health. Evans et al. also commented on the possibility of multiple environment risks (G. W. Evans, 2003).

Longitudinal Accumulation

In the US, specifically, economic mobility is very difficult. Compared to other developed countries, the US is more class bound than Denmark, for example, where the chance for economic mobility is half in the US (Ewing, Hamidi, Grace, & Wei, 2016). In the US, specifically, one's family is a determinant of individual success, 42% of children born with the lowest income levels will stay there (Isaacs, Sawhill, & Haskins, 2008). It has also been shown that intergenerational mobility is lower in the US than in other comparable countries, the argument has also been made that higher level of income inequality limits the economic mobility that will be seen in future generations (Corak, 2013; Isaacs et al., 2008).

I hypothesize that due to this overall lack of economic mobility there is the possibility for the longitudinal accumulation of environmental risk factors for health. Research into the effect of income alone on health has shown a consistent, graded association be-
between sustained economic hardship and physical, psychological, and cognitive functioning, this relationship was not attenuated by risk factors or prevalent diseases (Glymour, Avendano, & Kawachi, 2014; Lynch, Kaplan, & Shema, 1997).

As discussed in the previous section geographical areas tend to have similar levels of built, social, and physical environments. Therefore, you might have a triple poor area or a triple good area. If as the research suggests people in the US have a hard time with economic mobility, it is likely even if they move, they are moving to an area like the one they previously lived in. This could mean living their whole life in a triple poor area. The effects of this lifelong exposure might be additive or multiplicative for various disorders. Exposure in childhood might be more important for certain disorders whereas exposure as an adult might be important for other diseases (Gilbert et al., 2015). This has also been looked at as a life-course approach or across the lifespan (Braveman, 2014; Russ, Larson, Tullis, & Halfon, 2014; Taylor, Repetti, & Seeman, 1997).

**Mitigating/ Additive Factors**

When discussing risk factors and mitigating or additive factors for disease risk, the most commonly discussed are health behaviors. Health behaviors are directly linked to the environment; social, cultural, and physical environments influence them. Positive health behaviors are mitigating factors, whereas negative health behaviors would be additive factors.
The major problem with health behaviors is that there are several models to identify them. These models include Health Belief Model (HBM), Theory of Reasoned Action (TRA), Theory of Planned Behavior (TPB), Social Cognitive Theory (SCT), and the Trans-theoretical Model (TTM) (Ajzen & Fishbein, 1980; Azen & Madden, 1986; Bandura, 1986; Maiman & Becker, 1974; Prochaska & Diclemente, 1983). In addition to these prescribed models, there are often specialized models illness and behaviors like safe sex and exercise. While HBM, TRA, TPB, SCT, and TTM are most commonly used in the literature, there has been no quantification of which is best in which instance (Glanz, Rimer, & Viswanath, 2008; Noar & Zimmerman, 2005).

The easiest way to quantify the effect health behaviors have on chronic health is the delay in immigrants on matching the US averages for illnesses, and health behaviors. In Mexican-American women, it was seen that the level of  acculturation was directly related to prenatal health behaviors, despite the limited effect outcomes (Zambrana et al., 1997). It has also been shown that in immigrant women as a whole, acculturation is often related to poorer health behaviors, this outcome is not seen in men (Zambrana et al., 1997). Even within the same ethnic group there is a difference in the effect acculturation has on health, associations differed by gender, country of origin, and measurement (Zambrana et al., 1997).

I hypothesize that based on these differences seen in health behavior due to acculturation, the environment will affect what the health behaviors are prevalent in a certain population. Research has been conducted into the relationship between health behavior
and various social environment factors. These include religion, rural area, family status, SES, and overall risk between similar SES and different areas.

Religion was found to have a differing impact on health, based on race, but affects several chronic health conditions (Ellison, 1995; Ellison & Levin, 1998). In relation to the urban/rural status, it was found that there was distinct regional difference among risky health behaviors and that rural populations were more likely to have risky behavior. This shows that there may be social, environmental factors in these areas that make this “normal” (Hartley, 2004). It has been shown that age-adjusted mortality rates are higher for unmarried nonparent adults, it has been hypothesized that material and parental status modifies health behaviors, through social, environmental factors (Umberson, 1987). Within lower SES regions it has been shown that health behaviors have a lower overall impact, but do account for some of the mortality difference (Lantz et al., 1998; Stringhini et al., 2010). It has also been shown that within populations of similar lower SES status, there appeared to be an environmental context which accounted for differing rates of adolescent pregnancy (Brahmbhatt et al., 2014). These studies show that there appears to be an effect between social environment and health behaviors.

**Future Work**

**Specific Aim 1**

Future work is needed to examine the effect treatment modality might have on parental perceived combined symptom severity. This is required because there was no
difference in ADHD treatment between the mild and severe groups. It is unknown if this is because for some groups treatment did not lower severity, or if the ASD symptoms drive up the perceived ADHD severity.

**Specific Aim 2**

Future work is needed to determine which specific factors in the built environment should be accessed. There are several methods for analyzing built environment, with little standardization between them. These measurements need to be standardized so research conclusions can be drawn. For this specific study, the specific parts of the amenities and detracting elements should be examined to see if all parts contribute the same amount. There also needs to be research into if the built environment elements affect each other, while no interaction was found in the sample this might be due to the overall small sample size. The relationship between these strictly built environment factors and the overall social support structure also need to be investigated; they may influence each other. Having social support might attenuate some of the effects of living in a poor built environment.

**Specific Aims 4-6**

The future work for this study is to connect exposure databases and to repeat this analysis in other states to see if the same patterns show up. There is also need to investigate specific disorders, there are disorders that are almost purely genetic in the BD data, and this could be muddying the relationship. Another step would be to attempt to identify a lab test that can be used on children and adults living in “hot-spots” to test their urine or blood for chemical factors that might be the trigger for these areas.
Specific Aim 7

The future work for this study is to examine the effect ADHD/ASD severity and treatment might have on the relationship between extracurricular activities and educational engagement. This project should also be reported for other common combinations of school-aged disorders that occur in this population; just because the effect is comparable in the ADHD/ASD population does not mean all populations will be the same.

Overall

Overall, this dissertation highlights some key issues present in environmental epidemiology research. The first issue is the lack of standardized measures. This is especially prevalent in built and social environments since most physical environment measures can be taken from toxicology or environmental health research. For the built environment, specifically, there are several tools in existence for measurement purposes. Some focus on walkability, bike-ability, and disability ease of use, green space, food deserts and more. Do to this plethora of measurement criteria; it is exceedingly difficult to compare findings across studies. For this reason, no conclusive findings are available.

There also needs to be more research conducted considering the overall environment and its effect on human health. While it is easier to examine only certain parts of the environment as was done in these studies, this is not a valid way of examining the environment. As discussed previously, these environments likely work either synergistically or antagonistically on human health. Therefore all 3 should be considered whenever possible, this would be easier to do once measurements have been standardized.
Conclusions

This dissertation investigated the claims that the environment affects human health. It was found that in terms of chronic conditions across childhood, the built, physical, and social environments appear to influence human health. This dissertation also identified several areas for improvement within environmental when it comes to accessing environmental factors.
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APPENDIX A: IRB APPROVALS

Corley, Brittany N

From: DeHaai, Kristi A
Sent: Friday, April 15, 2016 10:27 AM
To: Corley, Brittany N
Cc: Kotulak, Gail D
Subject: RE:

Based on the information you provided, the UNMC IRB has determined that this project does not constitute human subject research as defined at 45CFR46.102. Therefore, it is not subject to the federal regulations. No further action is required.

Please be advised that, should anything change which would result in the project meeting the definition of human subject research, the IRB must be notified before any further research activity continues.

Should you have any questions please do not hesitate to contact the Office of Regulatory Affairs at 402-559-6463.

Kristi A. DeHaai, MS
IRB Administrator
Office of Regulatory Affairs
University of Nebraska Medical Center
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402.559.8561 | fax 402.559.3300
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The information in this e-mail may be privileged and confidential, intended only for the use of the addressee(s) above. Any unauthorized use or disclosure of this information is prohibited. If you have received this e-mail by mistake, please delete it and immediately contact the sender.
July 19, 2016

Alan Kolok, PhD
COPH Environ, Agri & Occ Health
UNMC - 68182

IRB # 414-16-EP

TITLE OF PROPOSAL: Midwesten watersheds and adverse health outcomes

DATE OF EXPEDITED REVIEW: 06/17/2016

DATE OF FINAL APPROVAL: 07/19/2016 VALID UNTIL: 06/17/2017

CLASSIFICATION OF RISK: Minimal

EXPEDITED CATEGORY OF REVIEW: 45 CFR 46.110, Category 5

SUBPART D CATEGORY OF REVIEW: 45 CFR 46.404--research not involving greater than minimal risk

The UNMC IRB has completed its review of the above-titled protocol, including any revised material submitted in response to the IRB’s review. The Board has expressed it as their opinion that you are in compliance with HHS Regulations (45 CFR 46), applicable FDA Regulations (21 CFR 50, 56), and HRPP policies and you have provided adequate safeguards for protecting the rights and welfare of the subjects to be involved in this study. The IRB has, therefore, granted unconditional approval of your research project. This letter constitutes official notification of the final approval and release of your project by the UNMC IRB, and you are authorized to implement this study as of the above date of final approval.

Please be advised that the IRB accepted the justification presented in Addendum J for a waiver or alteration of consent and has therefore granted a waiver of consent under the provisions of 45 CFR 46.116(d) and HIPAA Privacy Rule.

Finally, under the provisions of this institution’s Federal Wide Assurance (FWA00002093), the Principal Investigator (PI) is directly responsible for submitting to the IRB any proposed change in the research. In addition, any unanticipated adverse events or other problems related to the research which involve risk to the subject or others must be promptly reported to the IRB. This project is subject to review and monitoring by the IRB and, as part of their monitoring, the IRB may request reports of progress and results. For projects which continue beyond one year, it is the responsibility of the Principal Investigator to initiate a request to the IRB for continuing review and re-approval of the research project.

On Behalf of the IRB,

Signed on: 2016-07-19 14:00:00.000

Gail Paulsen, RN, BSN, CIP
IRB Administrator III
Office of Regulatory Affairs
APPENDIX B: NSCH DATA REQUEST FORM

**Request for Data Resource Center Indicator Data Set**

**Date:** 04/15/206  
**Name:** Brittany Corley  
**E-mail address:** Brittany.Corley@unmc.edu  
**Daytime Phone #** 502-645-3110  
**Organization Name:** University of Nebraska Medical Center  
**Location (State, Country):** Nebraska, United States of America  
**Type:**  
- [x] Academic Research  
- [ ] Non-Academic policy research  
- [ ] Government – Federal  
- [ ] Government – State:  
- [ ] Health care provider (HCP)  
- [ ] Health plan  
- [x] Advocacy  
- [ ] Student (SAS)  
- [ ] Private Vendor  
- [ ] Other  

Select the data set(s) you are requesting:  

**National Survey of Children’s Health (NSCH)**  
- [ ] 2003  
- [ ] 2007  
- [x] 2011-2012  
- [ ] 03 & 07 merged—Please note that only variables that were identical between 2003 and 2007 are included in this dataset  

**Survey of Pathways to Diagnosis and Services (Pathways)**  
- [ ] 2011  

**National Survey of Children with Special Health Care Needs (NS-CSHCN)**  
- [ ] 2001 (Merged Interview File)  
- [ ] 2005/06 (Merged Interview File)  
- [ ] 2009/10 (Merged Interview File)  

**National Health Interview Survey (NHIS)**  
- [ ] 2012 (Child Complementary and Alternative Medicine Supplement)  

**Format preference:**  
- [ ] SPSS  
- [x] SAS  
- [ ] STATA  

Please tell us how you plan to use the data: data will be used in dissertation.  

Submit request to: Child and Adolescent Health Measurement Initiative (CAHMI)  
Attention: Kathleen Powers  
E-Mail: info@cahmi.org  

Do you have a question?  
http://childhealthdata.org/help/dataset or  
http://childhealthdata.org/help/askus

---

Data Resource Center Indicator data sets contain constructed measures that were developed by CAHMI in collaboration with a national technical advisory panel for the Data Resource Center for Child and Adolescent Health. The purpose of this project is to provide support and technical assistance to states for interpretation and utilization of results of the National Survey of Children’s Health and National Survey of Children with Special Health Care Needs.
libname test 'C:\Users\brittany.corley\Desktop\drc_nsch_2011-12_sas_64bit\2011-2012 NSCH - SAS Dataset for 64-bit SAS';

*** make sure to run in SAS unicode version;

proc format lib = test ; /* code to make formats */
options fmtsearch = (test);
run ;

**************************************************************

Creating sub populations

**************************************************************,

data version1;

    set test.Nsch2011_12_drc_formatted;

    *variable age_6_17 will denote those children who are of the correct age to be in anaylasis;

    age_6_17=;
if age3_11=1 then age_6_17=0; *those children who will not be in analysis;

else age_6_17=1; *those children who will be in analysis;

*variable ADHD_ASD will denote diagnosis groups;

ADHD_ASD=.;

if K2Q31B in (1) and K2Q35B in (1) then ADHD_ASD=1; *those children with concurrent ADHD/ASD;

if K2Q31A in (0) and K2Q35A in (0) then ADHD_ASD=0; *those children with no ADHD/ASD;

*predictor variables;

Sev=.M;*combined ADHD/ASD Symptom Severity;

if K2Q31C=1 and K2Q35C=1 then sev=0;

ELSE if K2Q31C=1 and K2Q35C=2 then sev=0;

ELSE if K2Q31C=2 and K2Q35C=1 then sev=0;

Else if K2Q31C=2 and K2Q35C=2 then sev=1;

Else if K2Q31C=2 and K2Q35C=3 then sev=1;

Else if K2Q31C=3 and K2Q35C=2 then sev=1;

Else if K2Q31C=3 and K2Q35C=3 then sev=1;
else Sev=.M;

*New Age Categories;

Age_Cat_New=.M;

if AGYEYR_CHILD in (6,7,8) then Age_Cat_New=1;
else if AGYEYR_CHILD in (9,10,11) then Age_Cat_New=2;
else if AGYEYR_CHILD in (12,13,14) then Age_Cat_New=3;
else if AGYEYR_CHILD in (15,16,17) then Age_Cat_New=4;
else Age_Cat_New=.M;

*Parent Health Interactions;

PH=.M;

if ind6_3_11=.L and ind6_3a_11=.L then PH=0;
if ind6_3_11=.L and ind6_3a_11=1 then PH=1;
if ind6_3_11=1 and ind6_3a_11=.L then PH=2;
if ind6_3_11=.L and ind6_3a_11=2 then PH=3;
if ind6_3_11=2 and ind6_3a_11=.L then PH=4;
if ind6_3_11=1 and ind6_3a_11=1 then PH=5;
if ind6_3_11=1 and ind6_3a_11=2 then PH=6;
if ind6_3_11=2 and ind6_3a_11=1 then PH=6;
if ind6_3_11=2 and ind6_3a_11=2 then PH=7;

PH1=;

if PH in (0) then PH1=0;

if PH in (1,2,5) then PH1=1;

if PH in (6) then PH1=2;

if PH in (3,4,7) then PH1=3;

*Parent Education Interactions;

PE=.M;

if EDUC_MomR=.L and EDUC_DadR=.L then PE=0;

if EDUC_MomR=.L and EDUC_DadR=1 then PE=1;

if EDUC_MomR=1 and EDUC_DadR=.L then PE=2;

if EDUC_MomR=.L and EDUC_DadR=2 then PE=3;

if EDUC_MomR=.L and EDUC_DadR=3 then PE=3;

if EDUC_MomR=2 and EDUC_DadR=.L then PE=4;

if EDUC_MomR=3 and EDUC_DadR=.L then PE=4;

if EDUC_MomR=1 and EDUC_DadR=1 then PE=5;

if EDUC_MomR=1 and EDUC_DadR=2 then PE=6;

if EDUC_MomR=1 and EDUC_DadR=3 then PE=6;
if EDUC_MomR=2 and EDUC_DadR=1 then PE=6;
if EDUC_MomR=3 and EDUC_DadR=1 then PE=6;
if EDUC_MomR=2 and EDUC_DadR=2 then PE=7;
if EDUC_MomR=2 and EDUC_DadR=3 then PE=7;
if EDUC_MomR=3 and EDUC_DadR=2 then PE=7;
if EDUC_MomR=3 and EDUC_DadR=3 then PE=7;

PE1=;

if PE in (0) then PE1=0;
if PE in (1,2,5) then PE1=1;
if PE in (6) then PE1=2;
if PE in (3,4,7) then PE1=3;

*variable dom2 will denote initial domain for chapter1;

dom2=1;

if age_cat_new=.M then dom2=2;
if sex in (6,7,.M) then dom2=2;
if race=.M then dom2=2;
if povlev4_11 in (6,7,.M) then dom2=2;
if ind3_1_11 in (0,6,7,.M) then dom2=2;
if chronic=.M then dom2=2;

if ind5_1b_11=.M then dom2=2;

if ind1_5_11=.M then dom2=2;

if ind2_7_11 in (.M,.L) then dom2=2;

if ind4_6_11=.M then dom2=2;

if ind6_12_11=.M then dom2=2;

if ind6_11_11=.M then dom2=2;

if ind7_1_11=.M then dom2=2;

if ind7_2_11=.M then dom2=2;

if amen=.M then dom2=2;

if det=.M then dom2=2;

if ADHD_ASD in (0,.) then dom2=2;

if sev=.M then dom2=2;

if PH=.M then dom2=2;

race=.M;

if race4_11=2 then race=1;

if race4_11 in (1,3,4) then race=0;

Amen=.M;
if ind7_4_11 in (0,1) then amen=0;
if ind7_4_11 in (2,3) then amen=1;
if ind7_4_11 in (4) then Amen=2;

Det=.M;

if ind7_5_11 in (0) then det=0;
if ind7_5_11 in (1) then det=1;
if ind7_5_11 in (2,3) then det=2;

Det1=.M;

if ind7_5_11 in (0) then det1=0;
if ind7_5_11 in (1,2,3) then det1=1;

Chronic=.M;

if anycondition in (2,3,4) then chronic=1;
if anycondition in (5,6,7) then chronic=2;
if anycondition in (8,9,10,11,12,13,14,15,16,17,18) then chronic=3;

Ed_Eng=.M;

if ind5_2a_11=1 then Ed_eng=0;
else if ind5_2a_11=2 or ind5_2a_11=3 then Ed_eng=1;
else Ed_Eng=.M;
run;

proc format; *formats for domains;

value domtwo

   2 = 'Not Analyzed'

   1 = 'Concurrent ADHD/ASD';

value adhdasd

   1 = 'Concurrent ADHD/ASD'

   0 = 'No ADHD or ASD';

value sev

   0 = 'Mild Severity'

   1 = 'Severe Severity'

   .M = 'Missing';

value eng

   0 = 'Never Engaged'

   1 = 'Usually/Always Engaged'

   .M = 'Missing';

value age

   .M = 'Age not Analyzed'
1 = '6-8'
2 = '9-11'
3 = '12-14'
4 = '15-17';

Value Chronic

1 = '2-4'
2 = '5-7'
3 = '8+'
. = 'Not Analyzed';

value race

0 = 'Other'
1 = 'WNH'
.M = 'Missing';

value amen

.M = 'Missing'
0 = '0-1 Amenities'
1 = '2-3 Amenities'
2 = '4 Amenities';
Value det

.M = 'Missing'

0 = '0 Detracting elements'

1 = '1 Detracting elements'

2 = '2-3 Detracting elements';

value PH

.M = 'Missing'

0 = 'No Parents in Home'

1 = 'Father only Parent Poor health'

2 = 'Mother only Parent Poor health'

3 = 'Father only Parent Good health'

4 = 'Mother only Parent Good health'

5 = 'Both Parents Poor health'

6 = '1 Parent Poor health'

7 = 'Both Parents Good health';

Value PE

.M = 'Missing'

0 = 'No Parents in Home'
1 = 'Father only Parent Less than High School'

2 = 'Mother only Parent Less than High School'

3 = 'Father only Parent At Least High School Completed'

4 = 'Mother only Parent At Least High School Completed'

5 = 'Both Parents Less than High School'

6 = '1 Parent Less than High School'

7 = 'Both Parents At Least High School Completed';

value PHI

.M = 'Missing'

0 = 'No Parents in Home'

1 = 'All Parents in Home Poor Health'

2 = '1 Parent in Home Poor Health'

3 = 'All Parents in Home Good Health';

value PEI

.M = 'Missing'

0 = 'No Parents in Home'

1 = 'All Parents in Home Less than High School'

2 = '1 Parent in Home Less than High School'
3 = 'All Parents in Home At Least High School';

run;

*Creating Population Selection Flow Chart;

proc freq data=version1;
   tables k2q31B/missing;
   tables k2q35B/missing;
   tables adhd_asd*k2q31B*k2q35B/nocol norow nopercent missing;
   tables adhd_asd/missing;
run;

proc freq data=version1;
   where ADHD_ASD=1;
   tables age_6_17;
run;

proc freq data=version1;
   where ADHD_ASD=1 and age_6_17=1;
   tables sev;
run;

proc freq data=version1;
where ADHD_ASD=1 and Age_6_17=1;

tables sev*ind7_1_11*amen*det;

run;

*Checking how many children were effected by povlev being imputated;

proc freq data=version1;

where dom2=1;

tables sev*POVLEVEL_F;

run;

***Running Frequencies for table 1;

proc surveyfreq data=version1;

strata state;

cluster idnumr;

weight NSCHWT;

tables dom2*age_cat_new;

tables dom2*sex;

tables dom2*race;

tables dom2*ind5_3_11;
tables dom2*amen;
tables dom2*ed_eng;
tables dom2*PH1;
tables dom2*chronic;
tables dom2*det_pov;
tables dom2*amen;
tables dom2*povlev4_11;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.
Age_Cat_New age.
chronic chronic. race race. amen amen. det det. PH1 PHl. PE1 PEl.
run;

proc surveyfreq data=version1;
strata state;
cluster idnumr;
weight NSCHWT;
tables dom2*sev*age_cat_new/chisq1;
tables dom2*sev*sex/chisq1;
tables dom2*sev*race/chisq1;

tables dom2*sev*ind5_3_11/chisq1;

tables dom2*sev*amen/chisq1;

tables dom2*sev*ed_eng/chisq1;

tables dom2*sev*PH1/chisq1;

tables dom2*sev*chronic/chisq1;

Tables dom2*sev*ind2_7_11/chisq1;

Tables dom2*sev*amen/chisq1;

Tables dom2*sev*povlev4_11/chisq1;

Tables dom2*sev*det/chisq1;

Tables dom2*sev*ind7_1_11/chisq1;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PH1. PE1 PE1

PE1. ;

run;

proc surveyfreq data=version1;

strata state;
cluster idnumr;

weight NSCHWT;

tables dom2*sev*ind6_12_11/chisq1;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.
Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PHl. PE1
PEl. ;

run;

proc surveyfreq data=version1;

strata state;

cluster idnumr;

weight NSCHWT;

tables dom2*PH*PH1;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.
Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PHl. PE1
PEl. ;

run;

proc surveyfreq data=version1;
strata state;

cluster idnumr;

weight NSCHWT;

tables dom2*sev*k2q31c*k2q35c;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PHl. PE1

PEl. ;

run;

****Creating bivariate Analysis;

Proc surveylogistic data=version1;*paper 2;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11
chronic(ref='2-4') amen(descending)

povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;
model sev (ref='Mild Severity') =age_cat_new /clparm clodds corrb;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age.

PH PH. PE PE. chronic chronic race race. smoke smoke. amen amen. det det.

rank rank. ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age.

PH1 PHl. PE1 PEl. chronic_med chronic_med. amen_income amen_income.;

run;

Proc surveylogistic data=version1;"paper 2;"

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)

 povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;
model sev (ref='Mild Severity') = ind5_3_11 / clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PHl. PE1 PEl. ;

run;

Proc surveylogistic data=version1; paper 2;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)

povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11 / param=glm;

model sev (ref='Mild Severity') = sex / clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.
chronic chronic. race race. amen amen. det det. PH1 PHl. PE1

PEI. ;

run;

**Proc surveylogistic** data=version1:*paper 2;*

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11
chronic(ref='2-4') amen(descending)

povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity') =race /clparm clodds corrb;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng
eng. Age_Cat_New age.

PH PH. PE PE. chronic chronic. race race. smoke smoke. amen
amen. det det.

rank rank. ADHD_ASD adhdasd. dom1 domone. dom2 domtwo.

sev sev. ed_eng eng. Age_Cat_New age.
Proc surveylogistic data=version1; *paper 2;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)

povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity') =ed_eng /clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PHl. PE1

run;

Proc surveylogistic data=version1; *paper 2;
strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)

    povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity') = PH1/clparm clodds corrb;

    format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age.

    chronic chronic. race race. amen amen. det det. PH1 PHl. PE1 PEl. ;

run;

Proc surveylogistic data=version1;*paper 2;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;
class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11
chronic(ref='2-4') amen(descending)

povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity') = povlev4_11 / clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

run;
model sev (ref='Mild Severity') =det /clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PH1. PE1

PE1. ;

run;

Proc surveylogistic data=version1;*paper 2;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)

povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity') =chronic /clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.
chronic chronic. race race. amen amen. det det. PH1 PH1. PE1

PEI. ;

run;

Proc surveylogistic data=version1;*paper 2;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11
chronic(ref='2-4') amen(descending)

povlev4_11(ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity') =amen /clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PH1. PE1

PEI. ;

run;

Proc surveylogistic data=version1;*paper 2;
strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)
povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity') =ind7_1_11 /clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PH1. PE1 PE1.

run;

Proc surveylogistic data=version1;*paper 2;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;
class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11
chronic(ref='2-4') amen(descending)

povlev4_11(ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity') = ind6_12_11 /clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PHl. PE1

PEl. ;

run;

*creating final model;

**Proc surveylogistic data=version1;*base model;**

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11
chronic(ref='2-4') amen(descending)
povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity')=age_cat_new sex race povlev4_11 det amen ind7_1_11 /clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age.
chronic chronic. race race. amen amen. det det. PHI PH1 PE1 PEl. ;
run;

Proc surveylogistic data=version1; *base model;

strata state;
cluster idnumr;
weight NSCHWT;
domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PHI ind5_3_11 chronic(ref='2-4') amen(descending)
povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;
model sev (ref='Mild Severity') = age_cat_new sex race povlev4_11 det amen ind7_1_11

ind5_3_11/clparm
clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PH1. PE1 PE1.

run;

Proc surveylogistic data=version1;*base model;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)

povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;
model sev (ref='Mild Severity') = age_cat_new sex race povlev4_11 det amen ind7_1_11

ind5_3_11

ed_eng/ clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic race race. amen amen det det. PH1 PH1. PE1 PE1.

run;

Proc surveylogistic data=version1; *base model;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)

povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;
model sev (ref='Mild Severity') = age_cat_new sex race povlev4_11 det amen ind7_1_11 ind5_3_11 ed_eng

chronic/clparm clodds corrb;

format ADHD_ASD adhadas. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PH1. PE1 PE1.

run;

Proc surveylogistic data=version1; *base model;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)

povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;
model sev (ref='Mild Severity') = age_cat_new sex race povlev4_11 det amen ind7_1_11

ind5_3_11 ed_eng chronic ind6_12_11/clparm clodds corrb;

format ADHD_ASD adhdasd. dom2 domtwo. sev sev. ed_eng eng.

Age_Cat_New age.

chronic chronic. race race. amen amen. det det. PH1 PH1. PE1 PE1;

run;

**FINAL MODEL;

Proc surveylogistic data=version1;*base model;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom2;

class sev age_cat_new(ref='15-17') sex race ed_eng PH1 ind5_3_11 chronic(ref='2-4') amen(descending)
povlev4_11 (ref='400% or more FPL') det(ref='0 Detracting elements') ind7_1_11 ind6_12_11/param=glm;

model sev (ref='Mild Severity') = age_cat_new sex race povlev4_11 det amen ind7_1_11

ind5_3_11 ed_eng

chronic/clparm clodds corrb;

format ADHD_ASD adhdasd dom2 domtwo sev sev ed_eng eng.

Age_Cat_New age.

chronic chronic race race amen amen det det PH1 PHl PE1

PEl. ;

run;
libname test 'C:\Users\brittany.corley\Desktop\drc_nsch_2011-12_sas_64bit\2011-2012 NSCH - SAS Dataset for 64-bit SAS';

*** make sure to run in SAS unicode version;

proc format lib = test ; /* code to make formats */

options fmtsearch =(test);

run ;

******************************************************************************

Creating sub populations

******************************************************************************;

data version1;

    set test.Nsch2011_12_drc_formatted;

    *variable age_6_17 will denote those children who are of the correct age to be in analysis;

    age_6_17=;
if age3_11=1 then age_6_17=0; *those children who will not be in analysis;

else age_6_17=1; *those children who will be in analysis;

*variable ADHD_ASD will denote diagnosis groups;

ADHD_ASD=.;

if K2Q31B in (1) and K2Q35B in (1) then ADHD_ASD=1; *those children with concurrent ADHD/ASD;

if K2Q31A in (0) and K2Q35A in (0) then ADHD_ASD=0; *those children with no ADHD/ASD;

*predictor variables;

Ed_Eng=.M;*paper1;

if ind5_2a_11=1 then Ed_eng=0;

else if ind5_2a_11=2 or ind5_2a_11=3 then Ed_eng=1;

else Ed_Eng=.M;

*New Age Categories;

Age_Cat_New=.M;

if AGEYR_CHILD in (6,7,8) then Age_Cat_New=1;

else if AGEYR_CHILD in (9,10,11) then Age_Cat_New=2;
else if AGEYR_CHILD in (12,13,14) then Age_Cat_New=3;
else if AGEYR_CHILD in (15,16,17) then Age_Cat_New=4;
else Age_Cat_New=.M;

*extracurricular and diagnosis Interaction;

diag_Extra=.M;

if ADHD_ASD=0 and ind5_3_11=1 then diag_Extra=1;
if ADHD_ASD=0 and ind5_3_11=2 then diag_Extra=0;
if ADHD_ASD=1 and ind5_3_11=1 then diag_Extra=3;
if ADHD_ASD=1 and ind5_3_11=2 then diag_Extra=2;

*Parent Health Interactions;

PH=.M;

if ind6_3_11=.L and ind6_3a_11=.L then PH=0;
if ind6_3_11=.L and ind6_3a_11=1 then PH=1;
if ind6_3_11=1 and ind6_3a_11=.L then PH=2;
if ind6_3_11=.L and ind6_3a_11=2 then PH=3;
if ind6_3_11=2 and ind6_3a_11=.L then PH=4;
if ind6_3_11=1 and ind6_3a_11=1 then PH=5;
if ind6_3_11=1 and ind6_3a_11=2 then PH=6;
if ind6_3_11=2 and ind6_3a_11=1 then PH=6;

if ind6_3_11=2 and ind6_3a_11=2 then PH=7;

PH1=;

if PH in (0) then PH1=0;

if PH in (1,2,5) then PH1=1;

if PH in (6) then PH1=2;

if PH in (3,4,7) then PH1=3;

PH2=.M;

if PH1 in (0) then PH2=0;

if PH1 in (1,2) then PH2=1;

if PH1 in (3) then PH2=2;

*Parent Education Interactions;

PE=.M;

if EDUC_MomR=\.L and EDUC_DadR=\.L then PE=0;

if EDUC_MomR=\.L and EDUC_DadR=1 then PE=1;

if EDUC_MomR=1 and EDUC_DadR=\.L then PE=2;

if EDUC_MomR=\.L and EDUC_DadR=2 then PE=3;

if EDUC_MomR=\.L and EDUC_DadR=3 then PE=3;
if EDUC_MomR=2 and EDUC_DadR=.L then PE=4;

if EDUC_MomR=3 and EDUC_DadR=.L then PE=4;

if EDUC_MomR=1 and EDUC_DadR=1 then PE=5;

if EDUC_MomR=1 and EDUC_DadR=2 then PE=6;

if EDUC_MomR=1 and EDUC_DadR=3 then PE=6;

if EDUC_MomR=2 and EDUC_DadR=1 then PE=6;

if EDUC_MomR=3 and EDUC_DadR=1 then PE=6;

if EDUC_MomR=2 and EDUC_DadR=2 then PE=7;

if EDUC_MomR=2 and EDUC_DadR=3 then PE=7;

if EDUC_MomR=3 and EDUC_DadR=2 then PE=7;

if EDUC_MomR=3 and EDUC_DadR=3 then PE=7;

PE1=.;

if PE in (0) then PE1=0;

if PE in (1,2,5) then PE1=1;

if PE in (6) then PE1=2;

if PE in (3,4,7) then PE1=3;

*variable dom1 will denote initial domain for paper 1;

**PAPER 1;
do

    dom1=1;

    if age_cat_new = .M then dom1=2; *those who will not be analyzed;

    if ed_eng = .M then dom1=2; *those who will not be analyzed;

    if ind5_3_11 =.M then dom1=2; *those who will not be analyzed;

    if ind1_5_11 =.M then dom1=2; *those who will not be analyzed;

    if ind3_1_11 =.M then dom1=2; *those who will not be analyzed;

    if ind5_1b_11 =.M then dom1=2; *those who will not be analyzed;

    if ind6_4a_11 =.M then dom1=2; *those who will not be analyzed;

    if ind6_12_11 =.M then dom1=2; *those who will not be analyzed;

    if ind7_4_11 =.M then dom1=2; *those who will not be analyzed;

    if ind4_7_11 in (6,7,.M) then dom1=2; *those who will not be analyzed;

    if sex in (6,7,.M) then dom1=2; *those who will not be analyzed;

    if ADHD_ASD=. then dom1=2; *those who will not be analyzed;

    if race4_11 in (6,7,.M) then dom1=2; *those who will not be analyzed;

    if povlev4_11 in (6,7,.M) then dom1=2; *those who will not be analyzed;
IF PH = .M then dom1=2; *those who will not be analyzed;

IF PE = .M then dom1=2; *those who will not be analyzed;

if mediamon_11 in (6,7,.M) then dom1=2; *those who will not be analyzed;

if medialimit_11 in (6,7,.M) then dom1=2; *those who will not be analyzed;

if K7Q34 in (6,7,.M) then dom1=2; *those who will not be analyzed;

run;

proc format; *formats for domains;

value domone

2 = 'Not Analyzed'
1 = 'Analyzed';

value adhdasd

1 = 'Concurrent ADHD/ASD'
0 = 'No ADHD or ASD';

value eng

0 = 'Never Engaged'
1 = 'Usually/Always Engaged'

.M = 'Missing';

value age

.M = 'Age not Analyzed'

1 = '6-8'

2 = '9-11'

3 = '12-14'

4 = '15-17';

value extra

.M = 'Missing'

0 = 'No ADHD/ASD or ExtraCurricular'

1 = 'No ADHD/ASD Yes ExtraCurricular'

2 = 'YES ADHD/ASD NO ExtraCurricular'

3 = 'YES ADHD/ASD And ExtraCurricular';

value PH

.M = 'Missing'

0 = 'No Parents in Home'

1 = 'Father only Parent Poor health'
2 = 'Mother only Parent Poor health'

3 = 'Father only Parent Good health'

4 = 'Mother only Parent Good health'

5 = 'Both Parents Poor health'

6 = '1 Parent Poor health'

7 = 'Both Parents Good health';

Value PE

.M = 'Missing'

0 = 'No Parents in Home'

1 = 'Father only Parent Less than High School'

2 = 'Mother only Parent Less than High School'

3 = 'Father only Parent At Least High School Completed'

4 = 'Mother only Parent At Least High School Completed'

5 = 'Both Parents Less than High School'

6 = '1 Parent Less than High School'

7 = 'Both Parents At Least High School Completed';

value PHI

.M = 'Missing’
0 = 'No Parents in Home'

1 = 'All Parents in Home Poor Health'

2 = '1 Parent in Home Poor Health'

3 = 'All Parents in Home Good Health';

value PEI.

.M = 'Missing'

0 = 'No Parents in Home'

1 = 'All Parents in Home Less than High School'

2 = '1 Parent in Home Less than High School'

3 = 'All Parents in Home At Least High School';

Value PHz.

.M = 'Missing'

0 = 'No Parents in Home'

1 = 'At least 1 parent poor health'

2 = 'All parents in home good health';

run;

*Creating Population Selection Flow Chart;

proc freq data=version1;
tables k2q31B/missing;

tables k2q35B/missing;

tables adhd_asd*k2q31B*k2q35B/nocol norow nopercent missing;

tables adhd_asd/missing;

run;

proc freq data=version1;

tables age_6_17*adhd_asd;

run;

proc freq data=version1;

where dom1=1;

tables ind5_3_11/missing;

run;

proc freq data=version1;

where ADHD_ASD in (0,1) and Age_6_17=1;

tables ind5_3_11/missing;

run;

*creating table 1;
Proc SURVEYFREQ data=version1 missing;

strata state;

cluster idnumr;

weight NSCHWT;

TABLES DOM1* age_cat_new/cv deff cl clwt cvwt expected;

TABLES DOM1* ed_eng /cv deff cl clwt cvwt expected;

TABLES DOM1* ind5_3_11 /cv deff cl clwt cvwt expected;

TABLES DOM1* ind1_5_11 /cv deff cl clwt cvwt expected;

TABLES DOM1* ind3_1_11 /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* ind5_1b_11 /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* ind6_4a_11 /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* ind6_12_11 /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* ind7_4_11 /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* ind4_7_11 /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* sex /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* ADHD_ASD /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* race4_11 /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* povlev4_11 /cv deff cl clwt cvwt expected chisq1;
TABLES DOM1* PH /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* PE /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* mediamon_11/cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* medialimit_11 /cv deff cl clwt cvwt expected chisq1;

TABLES DOM1* K7Q34 /cv deff cl clwt cvwt expected chisq1;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev.
ed_eng eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

Proc SURVEYFreq data=version1;

strata state;

cluster idnumr;

weight NSCHWT;

TABLES dom1*adhd_asd* age_cat_new/cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* ed_eng /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* ind5_3_11 /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* ind1_5_11 /cv deff cl clwt cvwt expected chisq1;
TABLES dom1*adhd_asd* ind3_1_11 /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* ind5_1b_11 /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* ind6_4a_11 /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* ind6_12_11 /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* ind7_4_11 /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* ind4_7_11 /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* sex /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* race4_11 /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* povlev4_11 /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* PH /cv deff cl clwt cvwt expected chisq1;

TABLES dom1*adhd_asd* PE /cv deff cl clwt cvwt expected chisq1;
PROC SURVEYFREQ data=version1;

   strata state;

   cluster idnumr;

   weight NSCHWT;

   TABLES dom1*ed_eng* age_cat_new /chisq1;

   TABLES dom1*ed_eng* diag_extra /chisq1;

   TABLES dom1*ed_eng* ind1_5_11 /chisq1;

   TABLES dom1*ed_eng* ind3_1_11 /chisq1;

   TABLES dom1*ed_eng* ind5_1b_11 /chisq1;

   TABLES dom1*ed_eng* ind6_4a_11 /chisq1;

   TABLES dom1*ed_eng* ind6_12_11 /chisq1;

run;
TABLES dom1*ed_eng* ind7_4_11 /chisq1;
TABLES dom1*ed_eng* ind4_7_11 /chisq1;
TABLES dom1*ed_eng* sex /chisq1;
TABLES dom1*ed_eng* race4_11 /chisq1;
TABLES dom1*ed_eng* povlev4_11 /chisq1;
TABLES dom1*ed_eng* PH /chisq1;
TABLES dom1*ed_eng* PE /chisq1;
TABLES dom1*ed_eng* mediamon_11 /chisq1;
TABLES dom1*ed_eng* medialimit_11 /chisq1;
TABLES dom1*ed_eng* K7Q34 /chisq1;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev.
ed_eng eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;
run;

Proc SURVEYFreq data=version1;

strata state;

cluster idnumr;

weight NSCHWT;

tables dom1*ed_eng*ph1;
tables dom1*ed_eng*PE1;

TABLES dom1*ed_eng* K7Q34 ;

format dom1 domone. dom2 domtwo. ed_eng eng. PH1 PHl. PE1 PEl.;

run;

proc surveyfreq data=version1;

   strata state;

   cluster idnumr;

   weight NSCHWT;

   tables dom1*PH*PH1;

   tables dom1*PE*PE1;

   format dom1 domone. dom2 domtwo. ed_eng eng. PH1 PHl. PE1 PEl.;

run;

**********************************************************************

Creating Univariate Models

**********************************************************************,

Proc surveylogistic data=version1,*paper 1;

   strata state;

   cluster idnumr;
weight NSCHWT;
domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')
K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')
Age_Cat_New(ref='15-17') race4_11
ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)
ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')
povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') = ind1_5_11 /clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng.
Age_Cat_New age. diag_extra extra. PH1 PHl. PE1 PEl. PH2 PHz.;

run;

Proc surveylogistic data=version1;*paper 1;

strata state;

cluster idnumr;

weight NSCHWT;
domain dom1;
class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17')race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') = ind5_1b_11 /clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

Proc surveylogistic data=version1;*paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')
K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17')race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') = ind6_4a_11/cparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng

eng. Age_Cat_New age. diag_extra extra. PH1 PHl. PE1 PEI. PH2 PHz.;

run;

Proc surveylogistic data=version1; paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17')race4_11
ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') = ind6_12_11/clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age. diag_extra extra. PH1 PH1. PE1 PE1. PH2 PH2.;

run;

**Proc surveylogistic data=version1; paper 1;**

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng ind7_4_11 ;

model Ed_Eng (ref='Never Engaged') = ind7_4_11/clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age. diag_extra extra. PH1 PH1. PE1 PE1. PH2 PH2.;

run;

**Proc surveylogistic data=version1; paper 1;**

strata state;
cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17') race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') = Age_Cat_New/clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age. diag_extra extra. PH1 PH1. PE1 PEI. PH2 PHz.;

run;

Proc surveylogistic data=version1;*paper 1;

strata state;

cluster idnumr;

weight NSCHWT;
domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17') race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') = sex/clparm clodds corrbs rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng

eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

Proc surveylogistic data=version1;*paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;
class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17')race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') =K7Q34/clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng

eng. Age_Cat_New age. diag_extra extra. PH1 PH1. PE1 PE1. PH2 PHz.;

run;

**Proc surveylogistic** data=version1;*paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')
K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17') race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') = MediaLimit_11/clparm clodds corrb
r square;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng
eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

Proc surveylogistic data=version1; *paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17') race4_11
ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') =MediaMon_11/ clparm clodds corrb

rsquare;

    format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng
    eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

Proc surveylogistic data=version1;*paper 1;

    strata state;

    cluster idnumr;

    weight NSCHWT;

    domain dom1;

    class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')
        K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')
        Age_Cat_New(ref='15-17')race4_11
ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') =povlev4_11/clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

Proc surveylogistic data=version1;*paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17')race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;
model Ed_Eng (ref='Never Engaged') =race4_11/clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng
eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

Proc surveylogistic data=version1; *paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17')race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') =diag_extra/clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng
eng. Age_Cat_New age. diag_extra extra. PH PH1. PE1 PE1. PH2 PH2.;
run;

**Proc surveylogistic** data=version1;*paper 1;*

    strata state;

    cluster idnumr;

    weight NSCHWT;

    domain dom1;

    class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

    K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

    Age_Cat_New(ref='15-17')race4_11

    ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

    ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

    povlev4_11 ind3_1_11 ind5_3_11;

    model Ed_Eng (ref='Never Engaged') =PH/clparm clodds corrb rsquare;

    format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng

    eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

**Proc surveylogistic** data=version1;*paper 1;*

    strata state;
cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17') race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') =PE/clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng

eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

**Proc surveylogistic** data=version1; paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;
class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17') race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') =ind3_1_11 /clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng
eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

Proc surveylogistic data=version1;*paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')
K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17') race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Some uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') = ind4_7_11 / clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age. diag_extra extra. PH PH. PE PE.;

run;

*****Updating PH and PE in table 1;

Proc surveylogistic data=version1; *paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17') race4_11
ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

model Ed_Eng (ref='Never Engaged') =PH1 /clparm clodds corrb rsquare;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age. diag_extra extra. PH1 PHl. PE1 PEl.;

run;

Proc surveylogistic data=version1;*paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17')race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;
**model** Ed_Eng (ref='Never Engaged') =PE1 /clparm clodds corrb rsquare;

**format** ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age. diag_extra extra. PH1 PHl. PE1 PEl.;

**run**;

****Creating final model final model;

**Proc surveylogistic data=version1;**paper 1;

strata state;

cluster idnumr;

weight NSCHWT;

domain dom1;

class ed_eng PE1 (descending) PH1(ref='No Parents in Home') diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')

K7Q34 (ref='5 - CHILD HAS NO FRIENDS') sex(ref='1 - MALE')

Age_Cat_New(ref='15-17')race4_11

ind6_12_11(Descending) ind5_1b_11 ind1_5_11(descending)

ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')

povlev4_11 ind3_1_11 ind5_3_11;

**model** Ed_Eng (ref='Never Engaged') = sex Age_Cat_New race4_11

ind6_12_11 ind5_1b_11 ind1_5_11 ind6_4a_11 ind3_1_11
PE1 K7Q34 povlev4_11 diag_extra/clparm clodds corrb rsquare ;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng.
Age_Cat_New age. diag_extra extra. PH1 PHl. PE1 PEI. PH2 PHz.;
run;

****Current final model;

Proc surveylogistic data=version1;*paper 1;
    strata state;
    cluster idnumr;
    weight NSCHWT;
    domain dom1;
    class ed_eng PE1 (descending) PH1(ref='No Parents in Home')
        diag_extra(ref='YES ADHD/ASD NO ExtraCurricular')
        K7Q34 (ref='5 - CHILD HAS NO FRIENDS')
        sex(ref='1 - MALE')
        Age_Cat_New(ref='15-17') race4_11
        ind6_12_11(Descending) ind5_1b_11 ind1_5_11(ind_11(descending))
        ind6_4a_11(ref='Someone uses tobacco - smokes inside the house')
        povlev4_11 ind3_1_11 ind5_3_11;
    model Ed_Eng (ref='Never Engaged') = sex Age_Cat_New race4_11
        ind6_12_11 ind5_1b_11 ind1_5_11 ind6_4a_11 ind3_1_11
PH1 K7Q34 povlev4_11 diag_extra/clparm clodds corrb rsquare ;

format ADHD_ASD adhdasd. dom1 domone. dom2 domtwo. sev sev. ed_eng eng. Age_Cat_New age. diag_extra extra. PH1 PHl. PE1 PEl. PH2 PHz.;

run;