2018

Childhood Lead Exposure and Adult Verbal Comprehension and Perceptual Reasoning

Carrie A. Wonderly
Walden University

Walden University

College of Social and Behavioral Sciences

This is to certify that the doctoral dissertation by

Carrie Wonderly

has been found to be complete and satisfactory in all respects,
and that any and all revisions required by
the review committee have been made.

Review Committee
Dr. Anthony Perry, Committee Chairperson, Psychology Faculty
Dr. Michael Durnam, Committee Member, Psychology Faculty
Dr. Virginia Salzer, University Reviewer, Psychology Faculty

Chief Academic Officer
Eric Riedel, Ph.D.

Walden University
2018

Abstract

Childhood Lead Exposure and Adult Verbal Comprehension and Perceptual Reasoning

by

Carrie Wonderly

B.A., State University of New York Oneonta, 2002

M.S., University of Phoenix, 2009

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Psychology

Walden University

July 2018

Abstract

Lead neurotoxicity is considered a problem in young children and the long-term effects of lead exposure on them have yet to be determined. Studies have been completed to determine how lead exposure has affected children through their most important developmental stages. However, there is a lack of research to uncover any long-term effects lead may have as children enter adulthood. The purpose of this study was to determine long-term effects of lead exposure on IQ. The theoretical foundation for this study is the Cattell Horn Carroll theory of intelligence, which is based on fluid (novel experiences) and crystallized (previous experiences) intelligence. This study focused on individuals who were exposed to lead as children and are now young adults (ages 18-25). These individuals were part of a class action lawsuit and referred to a clinical psychologist from a private clinic who conducted IQ testing. In this non-experimental quantitative study, a multiple regression analysis was conducted on secondary data. Independent variables were first and last blood lead and Appearance, Pulse, Grimace, Activity, and Respiration scores as indicated from medical records. The dependent variables were the perceptual reasoning index, and verbal comprehension index of the Wechsler Adult Intelligence Scale (IV) administered by a licensed clinical psychologist. The results of this study showed that childhood lead toxicity did not predict IQ scores in young adulthood. More research needs to be completed so that governmental agencies will have more information to be proactive in creating and changing policies around the use of lead in products that people use daily. This could lead to positive social change by placing an emphasis on early identification and treatment of lead exposure.

Childhood Lead Exposure and Adult Verbal Comprehension and Perceptual Reasoning

by

Carrie Wonderly

B.A., State University of New York Oneonta, 2002

M.S., University of Phoenix, 2009

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Psychology

Walden University

July 2018

Dedication

I dedicate this dissertation to my husband. He supported me every step of the way by cheering me on and being my sound board every time I needed one. He always made sure I kept a balanced life through the toughest struggles. He is my best friend, my rock, and my love. With his support I have been able to complete my studies and make my dreams come true.

Love You Much

Acknowledgements

I would like to express my sincere gratitude and appreciation to all those who were instrumental in helping me along the way, either academically or otherwise. This includes my family and friends (especially Tricia) who has shown their support over the years, even when I was absent for gatherings. Dr. Anthony Perry for taking over as my dissertation chair when my first one resigned from teaching. His support and patience with me has been invaluable.

Table of Contents

List of Tables

Chapter 1: Introduction to the Study

People are exposed to environmental toxins on a daily basis. Environmental toxins can be attributed to plastics used in baby bottles or everyday plastic food storage containers; industrial and man-made chemicals used in cleaning supplies; burning of coal, gas, oil, and garbage; and natural gas such as propane, butane, and liquefied petroleum gas (Jurewicz, Polanska, & Hanke, 2013). All of these pollutants are dispersed into the atmosphere and inhaled or ingested into the body and could harm human development. This research focuses on just one specific type of environmental toxin: lead. Research has shown that lead neurotoxin impacts IQ in childhood. However, there is a lack of research on the effects of lead on IQ in adulthood (Mazumdar et al., 2011). In this study, I examined the impact of long-term effects of lead neurotoxin. As a result of this study, showing any long-term effects of lead toxicity, governmental agencies will have more information to be proactive in creating and changing policies around the use of lead in products that people use on a daily basis. This could lead to positive social change by placing an emphasis on early identification and treatment of lead exposure, resulting in people being more successful in their educational, occupational, and economic goals.

Chapter 1 will include the background, problem statement, and purpose of the study. The research questions are also listed. This chapter will conclude with assumptions, scope and delimitations, as well as limitations and significance of the study.

Background

In 2007, the U.S. Department of Health and Human Services (HHS) wrote a report on the toxicology of lead (Abadin et al., 2007). Lead is a heavy metal that is

naturally developed in the Earth's crust. Lead melts at low heating temperatures, does not erode, and is often used in combination with other metals to form alloys which are used in pipes or batteries. Other uses for lead compounds are in paints and dyes. In 1980, the United States started to phase out the use of lead and in 1996, the use of lead in gasoline for motor vehicles was banned; however, it may still be used for off-road vehicles and airplanes. Even though the Earth creates lead naturally, lead toxicity occurs mainly due to human actions. The HHS stated that between 1950 and 2000 the use of leaded gasoline increased worldwide (Abadin et al., 2007). This increase released more lead into the atmosphere through vehicle exhaust. Another way lead gets into the atmosphere is through mining. Coal, oil, and its waste are burned and lead is discharged into the air (Abadin et al., 2007). When lead is disbursed into the atmosphere, it is removed during rainfall and pollutes the soil. Other ways lead contaminates the ground is from lead-based paint flaking away from old structures (buildings or bridges) or through landfills where mining and other manufacturing industries put their waste (Abadin et al., 2007).

The Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO) conducted independent research on the impact of lead exposure on children. Both organizations acknowledged that exposure to lead can be detrimental to children's development (CDC, 2016; WHO, 2014); however, the CDC has specific guidelines for what is considered a "reference value" (CDC, 2014). The CDC has lowered its reference value from 10 micrograms per deciliter (µg/dl) to 5 µg/dl. This change has allowed for early interventions in the attempt to decrease any further exposure to lead. However, there was no change in the CDC's recommendation for medical

treatment. Medical treatment should occur when blood lead levels are greater than or equal to the value of 45 µg/dl. Though the CDC has more specific guidelines, the WHO states that even concentrations of lead in blood at 5 µg/dl may have an impact on development during childhood and that there is no safe level (WHO, 2014). They further stated that lead toxicity could be prevented by phasing out the use of lead in everyday products, such as gasoline.

Humans can ingest lead in two main ways: by inhalation and orally. Individuals who work in the mining industry will breathe lead into their lungs or by items placed in a person's mouth that are contaminated with lead, and can become toxic (Abadin et al., 2007). Lead can also be transferred in utero, from mother to child, if the mother has lead in her system (Miranda, Edwards, Swamy, Paul, & Neelon, 2010). Lead is stored in the body, either in soft tissue or bone, because the body cannot differentiate between lead and calcium. When a woman is pregnant, stored lead is activated because there is a demand for calcium from the fetus. Calcium is extracted from the mother's body to support the fetus. If lead is stored in the body it will be taken from the mother and given to the baby as if it were calcium. Regardless of how lead is introduced into the body, humans will still suffer from the effects of lead contamination. The HHS (Abadin et al., 2007) discussed in depth the impact that lead could have on humans. For the scope of this paper, the focus is on long-term effects of lead on an individual's neurodevelopment, specifically IQ.

The HHS (Abadin et al., 2007) reviewed multiple studies from around the world where intellectual functioning was assessed during childhood due to elevated blood lead

levels. The CDC recognizes limitations to all the studies; however, there is an agreement that lead toxicity can affect intellectual development in children. A noted limitation is the lack of information on long-term effects of lead neurotoxicity in early adulthood.

Problem Statement

Research has shown that lead neurotoxin impacts IQ in childhood. However, there is a lack of research on the effects of lead on IQ in adulthood (Mazumdar et al., 2011). In this study, I looked at the impact of lead neurotoxin on two specific areas of IQ: the verbal comprehension index (VCI) and the perceptual reasoning index (PRI). I reviewed the Wechsler Adult Intelligence Scale–IV (WAIS-IV) results and first and last blood lead levels in individuals ages 18-25 who were identified and treated for lead poisoning in their childhood. Each participant was referred for testing by his or her lawyer in a class action lawsuit. Each participant signed a consent form stating that they acknowledged their IQ testing, Appearance, Pulse, Grimace, Activity, and Respiration (APGAR) scores, blood lead levels, and birthdates that were used in this study. IQ testing was administered and completed by a licensed clinical psychologist at a private agency, ADHD and Autism Psychological Services and Advocacy in Central New York. I was provided with a practicum and internship site at the private agency whereupon I learned about work with lead assessments.

After completing a literature review on lead studies, a gap in the research for long term effects on IQ among the age population of 18- to 25-year-olds was noted. There were two attempted follow up studies on lead exposure. In 2009, Mazumdar et al. (2011) sought out individuals whose last IQ assessment was at age 10. These same individuals,

at approximately 29 years old, were asked to fill out a questionnaire. The questionnaire gathered information on demographics, education, employment, history of arrests, current medications, and alcohol and tobacco use. A second group was sought out and tested using the Wechsler Abbreviated Scale of Intelligence (WASI). This group was demographically similar to the original group: white and college educated. Socioeconomic status, blood lead history, and IQ scores from childhood were also collected. In 2008-2009, Searle et al. (2014) conducted a follow up study that originated in the 1970s in South Australia. They attempted to locate the participants 15 years later when the subjects were approximately 29 years old. Again, these individuals only filled out questionnaires (the Adult Self Report, Community Assessment of Psychic Experiences, Alcohol Use Disorders Identification Test, Adverse Childhood Experiences Survey, and Composite International Diagnostic Interview, as well as sociodemographics, including educational attainment, employment, and household structure) and there was no follow up with IQ testing, further explanation is provided in Chapter 2.

Jakubowski (2011), Ferrie et al. (2012), and Liu et al. (2013) have indicated that more research needs to be completed because the majority of the studies have been done with children and infants because children are the most vulnerable population. Also, the number of subjects in the studies has not been large enough to draw a more formal conclusion that lead may have negative consequence no matter what age the person is. This study was an attempt to determine if there was a relationship between increased blood lead levels and a decrease in perceptual reasoning and/or verbal comprehension IQ scores.

Given the results of this study combined with previous research, and given how far reaching the consequences could be, emphasis should be placed on early identification and treatment of lead exposure. By examining the long-term effects of lead toxicity, governmental agencies will have more information so they can be more proactive in creating and changing policies around the use of lead in products that people use on a daily basis. In the interest of positive social change, a person without any complications from lead exposure would be more successful regarding educational, occupational, and economic goals.

Purpose of the Study

The purpose of this quantitative, nonexperimental study was to discern if lead toxicity affects individuals' IQ in early adulthood. Lead is a neurotoxin that has always been a concern because of its impact on the developing brain. Beginning in the 1980s, the impact of lead on a person's intellectual ability has been studied. However, the focus was on children and how exposure to lead affects the developing brain. Conversely, very little attention has been paid to the developing brains of young adults who were exposed and treated for lead poisoning. For this study, I collected secondary data from a private agency in Central New York where lead testing was completed. The WAIS-IV was used to measure IQ. However, only PRI and VCI scores were used. Demographic data such as gender and birth date (to determine age) were collected. First and last blood lead levels and the APGAR scores were collected from the medical records that were provided by each participant's lawyer. First blood lead level is the first time the person was tested for lead. Last blood lead level is the last time the person was tested for lead.

Nature of the Study

Participants had been referred to the private agency in Central New York for lead assessments by their lawyers due to a class action lawsuit. The lawyers provided the medical records to the agency for each participant who was assessed. The medical records indicated the first and last blood lead levels and APGAR scores for each participant, which was the only information used from the medical records. Blood was drawn and tested for lead by medical doctors. A licensed clinical psychologist from Central New York administered all IQ testing. Each participant signed a consent form acknowledging his or her IQ testing, blood lead levels, APGAR scores, gender, and birth dates to determine which ages would be included in the study. By consenting, they agreed to allow their data to be included in a study with the possibility of being studied at a later time by a third party. The private agency provided secondary data to me. I did not have access to any other identifying information, such as names, and I had no contact with any of the participants, nor will I have any in the future. I will not benefit from this study in any way and there are no ties to the class action lawsuit.

Variables

This study was nonexperimental and quantitative. A multiple regression analysis was completed on secondary data. The independent variables were the first and last blood lead levels and APGAR scores as indicated from medical records. APGAR scores are given as soon as an infant is born. These scores are used as a measure of the overall wellbeing of an infant. An average APGAR score is 7 to 10. This means the infant is healthy and does not need any medical intervention. APGAR scores were gathered for

this study to be used as a baseline of the overall health of each participant upon birth. The dependent variables were from two indexes of IQ testing, the PRI and VCI, including the subtests of each index: block design, matrix reasoning, visual puzzles, vocabulary, information, and similarities. The purpose was to determine if childhood exposure to lead and subsequent blood lead levels impact reasoning and comprehension.

Research Questions and Hypotheses

RQ1: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of PRI scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their childhood?

$H_0$1: Early childhood lead exposure is not a significant predictor of PRI scores in early adulthood.

H_a1: Early childhood lead exposure is a significant predictor of PRI scores in early adulthood.

RQ2: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of block design scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$2: Early childhood lead exposure is not a significant predictor of block design scores in early adulthood.

H_a2: Early childhood lead exposure is a significant predictor of block design scores in early adulthood.

RQ3: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of matrix reasoning scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$3: Early childhood lead exposure is not a significant predictor of matrix reasoning scores in early childhood.

H_a3: Early childhood lead exposure is a significant predictor of matrix reasoning scores in early adulthood.

RQ4: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of visual puzzles scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$4: Early childhood lead exposure is not a significant predictor of visual puzzles scores in early childhood.

H_a4: Early childhood lead exposure is a significant predictor of visual puzzles scores in early adulthood.

RQ5: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of VCI scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their childhood?

$H_0$5: Early childhood lead exposure is not a significant predictor of VCI scores in early adulthood.

H_a5: Early childhood lead exposure is a significant predictor of VCI scores in early adulthood.

RQ6: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of vocabulary scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$6: Early childhood lead exposure is not a significant predictor of vocabulary scores in early childhood.

H_a6: Early childhood lead exposure is a significant predictor of vocabulary scores in early adulthood.

RQ7: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of information scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$7: Early childhood lead exposure is not a significant predictor of information scores in early childhood.

H_a7: Early childhood lead exposure is a significant predictor of information scores in early adulthood.

RQ8: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of similarities scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$8: Early childhood lead exposure is not a significant predictor of similarities scores in early childhood.

H_a8: Early childhood lead exposure is a significant predictor of similarities scores in early adulthood.

Theoretical Framework for the Study

The theoretical framework for this study was based on Cattell-Horn-Carroll's (CHC) theory; Cattell's three-stratum and Horn-Carroll's Gf–Gc theory of fluid and crystalized intelligence. The CHC theory has been used in psychometric development of human intelligence (Reynolds, Keith, Flanagan, & Alfonso, 2013). The five components of the CHC theory are verbal comprehension/knowledge (Gc), visual-spatial ability (Gv), fluid/novel reasoning (Gf), associative memory (MA), and short-term memory (Gsm; Reynolds et al., 2013). The WAIS-IV is the current version of intelligence testing used based on the CHC theory (Wechsler, Coalson, & Raiford, 2008). I chose the CHC theory for this study due to its relation to IQ testing and there being a known link between lead exposure and a decrease in IQ.

For the purposes of this study, the areas of interest from the WAIS-IV IQ testing are the VCI and PRI. Ward, Bergman, & Hebert (2011) discussed the link between the WAIS-IV indexes and how they relate to CHC theory. The VCI subtests (similarities, vocabulary, and information) correlate with verbal comprehension/knowledge (Gc), and the PRI subtests (block design and visual puzzles) correlate with visual-spatial ability, and (Gv) and (matrix reasoning) with fluid/novel reasoning (Gf; Ward et al., 2011). The CHC theory is the theoretical basis for intelligence testing. Further explanation is provided in Chapter 2

Definitions

Verbal Comprehension Index (VCI): Measures cognitive abilities by retrieving verbal information from long-term memory and reasoning into verbal communication (Wechsler et al., 2008).

Perceptual Reasoning Index (PRI): Measures nonverbal cognitive abilities through the manipulation of stimuli (Wechsler et al., 2008).

Blood lead levels: The amount of lead measured in deciliters in the blood upon testing.

First blood lead level: The first time a person's blood was tested for lead.

Last blood lead level: The last time a person's blood was tested for lead.

Centers for Disease Control and Prevention (CDC): A governmental agency in the United States that conducts critical science investigations, provides health information that protects our nation against expensive and dangerous health threats, and responds when these arise.

World Health Organization (WHO): An organization whose primary role is to direct and coordinate international health within the United Nations' system.

Lead (Pb): A chemical element that when absorbed or ingested causes poisoning, which affects the brain, nervous and digestive systems, and blood [http://medical-dictionary.thefreedictionary.com/Lead+(element)].

Neurotoxin (neurotoxicity): A substance that is poisonous or destructive to nerve tissue.

APGAR scores: Scores assigned to newborns based on evaluation of *Appearance* (skin color), *Pulse* (heart rate), *Grimace response* (reflexes), *Activity* (muscle tone), *Respiration* (breathing rate and effort). In 1952, Virginia Apgar, an anesthesiologist, developed the APGAR score to be given to infants when they are born. The first test is given at 1 minute and then again at 5 minutes. There may be a third evaluation at 10 minutes. The APGAR score is used to evaluate whether any emergency or additional medical care is needed (*http://kidshealth.org/en/parents/apgar.html).*

Assumptions

This study was based on the following assumptions. These assumptions were necessary based on the use of secondary data, medical information provided, and the confines of IQ testing. Secondary data are data collected by someone other than the user. The secondary data were provided by a private clinic in Central New York. First, I assumed that the IQ testing was completed by a licensed clinical psychologist. Another assumption was that the WAIS-IV accurately measured the participants' current level of functioning and was sensitive to the individuals' level of lead toxicity. Blood was used to test for lead levels. I assumed that the blood was drawn and accurately tested for lead by a medical professional. These assumptions were important for this study because of the use of secondary information.

Scope and Delimitations

While conducting a literature review on lead toxicity, a gap in the research was noted. The majority of research had been focused on infants and children. Research on long-term effects of lead toxicity had been limited, and only a couple follow up studies

had been attempted (Mazumdar et al., 2011; Searle et al., 2014). This study was chosen because the age population for this research had not been considered before. Thus, it could be helpful for society to see the effects of lead poisoning on adults who were originally diagnosed with lead toxicity when they were children. Individuals included in this study were 18- to 25-year-olds who were identified in childhood with lead toxicity. Individuals who were excluded were children, teens, and adults over the age of 25. VCI and PRI from IQ testing, as well as blood lead levels and APGAR scores, were evaluated.

Limitations

Issues of Internal Validity

In this cross-sectional study, I looked at a specific age population at a single point in time. Each individual was given a battery of assessments. However, based on the areas of the brain that had been studied, only the VCI and PRI were being considered for this study. These subtests of IQ were specifically chosen due to the areas of the brain that had been studied. The Brain Development Group (2012) and Burgaleta et al. (2014) conducted separate studies indicating that, as a person ages, gray matter decreases while white matter increases, which shows changes in brain structure over the years. Parental educational background and socioeconomic status were not considered for this study. How a person was exposed to lead was also not considered for this study. The length of time a person was exposed to lead was not considered for this study. A list of medications was provided by each participant, and participants were asked if they took them as prescribed. Each participant was asked upon arrival if any drugs or alcohol were taken the day of testing. All participants were given a break for lunch during the day of their

testing. How well the individuals slept the night before was not considered for this study. Each participant traveled and stayed in a hotel the night before their testing session. Each participant was referred for lead assessment by his or her lawyer who was conducting a class action lawsuit.

Issues of External Validity

External validity is being able to generalize results of a study across people, places, and/or times. One way to ensure that a study can be generalized is through random selection. Random selection is when a sample of the population is drawn for a study. Random selection was not used for this study because it was based on secondary data collected from one source, a private clinic in Central New York that conducted all IQ testing.

Significance

Lead toxicity is considered a problem with children and their developing brains (Liu et al., 2013). Children are more often the subjects of lead studies because they are considered more vulnerable (Jakubowski, 2011). However, there is a lack of research on the effects of lead toxicity in adulthood. Two follow up studies, both with their own limitations, had been conducted on long-term effects of lead toxicity (Mazumdar et al., 2011; Searle et al., 2014). Mazumdar et al. (2011) and Searle et al. (2014) conducted follow up studies of adults (age 29) who were identified as newborns and in young childhood with lead toxicity. Both studies had a smaller sample size than the original studies. Mazumdar et al. (2011) used the WASI to measure IQ. Searle et al (2014) did not conduct IQ assessments. This project was unique because it addressed an age population

that had not been considered before (ages 18-25), had access to a larger sample size, and used first and last blood lead levels and a fully completed WAIS-IV test battery instead of the abbreviated WASI test battery. This study was focused on young adulthood lead toxicity and its impact on the PRI and VCI from the WAIS-IV. This study was an attempt to determine if there was a relationship between increased blood lead levels and a decrease in perceptual reasoning and/or verbal comprehension IQ scores.

Given the results of this study combined with previous research, emphasis should be placed on early identification and treatment of lead exposure given how far reaching the consequences could be. In the interest of positive social change by showing long-term effects of lead toxicity, governmental agencies will have more information so they can be more proactive in creating and changing policies around the use of lead in products that people use on a daily basis. A person without lead exposure complications would be more successful with educational, occupational, and economic goals.

Summary

Environmental insults such as lead are known to cause problems to human functioning. The CDC and WHO conducted their own research linking lead contamination to developmental and neurological problems. Research on the effects of lead insult has come a long way to help determine how detrimental lead can be to human development and overall health. However, due to the lack of follow-up research on lead insult into adulthood, this study should help bridge the gap of information. In Chapter 2, the theoretical foundation and previous research will be discussed.

Chapter 2: Literature Review

Lead is a known neurotoxin and can have a negative impact on overall health (Bakhireva et al., 2013; Miranda et al., 2010; Sanders et al., 2012). Lead is a heavy metal that is naturally developed in the earth's crust (Abadin et al., 2007). Lead melts at low heating temperatures, does not erode, and is often used in combination with other metals to form alloys used in pipes, batteries, paints, and dyes. Even though environmental exposure to lead has decreased over the decades, there is still concern over lead exposure and its effects (Canfield et al., 2003). The United States banned leaded gasoline, but there has been an increase in its use worldwide, and it can be used in off road vehicles. Lead is also distributed into the air during the mining process, and then when it rains, the rain picks up lead particles and carries them to the ground, contaminating the soil. Lead will also pollute the ground when lead-based paint chips away from a building and falls to the ground. All of these pollutants are dispersed into the atmosphere and inhaled or ingested into the body and could harm human development (Jurewicz et al., 2013).

The majority of the research conducted over the decades has focused on the impact lead toxicity has on IQ during childhood, on the environment, and on overall physical health. There is a lack of research, however, on the long-term impact on adults who suffered from lead toxicity in their childhood. In this chapter, I will discuss the strategy used for the literature search, the theoretical foundations for this study, as well as the biological and environmental exposures to lead and its effects on the brain and intelligence.

Literature Search Strategy

I conducted the literature review through Walden University Library using the search engines MEDLINE with FULL TEXT, Health & Medical Complete, PsychINFO, PsycARTICLES, PsycBOOKS, and PsycEXTRA. I sought out peer-reviewed articles using key search terms such as *lead insult, lead contamination, lead toxicity, IQ and lead, lead insult and IQ, environmental impact of lead toxicity, health impact of lead toxicity, impact of lead exposure, adults with lead exposure, blood lead levels and neurotoxicity, executive functioning and lead toxicity, lead toxicity and Intelligence, cognitive development and lead exposure/ toxicity, cognition and lead toxicity, brain stem function, synaptic genesis, neurochemical, lead exposure,* and *lead poisoning.* Two governmental websites, those of the CDC and WHO, were also used to gather information.

Theoretical Foundation

The theoretical framework for this study was based on the CHC theory, Carroll's three-stratum and Horn-Cattell's G*f*–G*c* theory of fluid and crystalized intelligence. Carroll's theory was based on Spearman's G-theory (Alfonso, Flanagan, & Radwan, 2005; Cattell, 1963; Kan, Kievit, Dolan, & van der Mass, 2011; McGrew, 2009). Carroll expanded on the G-theory (stratum III) to incorporate two additional strata: broad abilities (stratum II) and narrow abilities (stratum I). In the 1940's, Cattell developed the fluid (G*f*) and crystallized (G*c*) intelligence theory. When testing fluid abilities, activities are novel and do not require previous experience. Testing crystallized abilities is for activities that have been done before and require previous experience. In the 1960s, Horn expanded on Cattell's theory adding four more areas of intelligence: (a) visual perception

and processing, (b) short-term memory, (c) long-term storage and retrieval, and (d) speed of processing and auditory processing abilities (Alfonso et al., 2005; Horn, 1968). In 1968, Horn wrote about the reasons behind the development of the CHC theory. He stated that the CHC theory focuses on the development of capability and the difference between the broad design of abilities (Horn, 1968). In the 1990's Horn carried out more research and ended up adding four more areas of intelligence: reaction time, decision speed, quantitative ability, and broad reading/ writing abilities.

In 1993, Carroll completed a review on all the world's writings regarding human cognition (Alfonso et al., 2005; McGrew, 2009). Carroll's work was to help show similarities and differences between his theory and Cattell and Horn's theory. There is only one similarity: there are multiple broad abilities (stratum II). There are four differences: (a) Carroll used the G-factor to measure the broadest and most general levels of ability while Cattell and Horn dropped G; (b) Carroll put quantitative reasoning as a narrow ability while Cattell and Horn added reasoning and put it as a broad ability; (c) Carroll has reading and writing as a narrow ability while Cattell and Horn have it as a broad ability; and (d) Carroll lumps short term, associative, meaningful, and free recall memory into one category whereas Cattell and Horn put short term memory and long-term retrieval into separate categories under broad abilities and associative, meaningful, and free recall under narrow abilities.

In the late 1990's, McGrew wanted to resolve the differences and incorporate both Carroll's and Cattell and Horn's theories together to create what is now known as the CHC theory. He kept both broad and narrow abilities while expanding on both to make

10 broad and 76 narrow abilities (Alfonso et al., 2005). He eliminated "G" based on how functional the broad and narrow abilities are. The CHC theory is used today to help explain abilities and constructs of cognition in the development of intelligence testing. The five components of the CHC theory are verbal comprehension/knowledge (*Gc*), visual-spatial ability (*Gv*), fluid/novel reasoning (*Gf*), associative memory (*MA*), and short-term memory (*Gsm*). The WAIS-IV is the current version of intelligence testing used based on CHC's theory. Ward et al. (2012) discussed the link between the CHC theory and WAIS-IV indexes. Crystalized intelligence (*Gc*) measures acquired knowledge of language, information, and concepts of a specific culture. Fluid intelligence (*Gf*) measures the ability to solve novel problems, draw inferences, and inductive and deductive reasoning.

During the infant stages of development, distinctions between fluid and crystalized intelligence cannot be drawn (Horn, 1968). However, fluid and crystalized intelligence can be seen in children as young as 4 years of age (Tusing & Ford, 2004). As a person ages into their teen years, fluid intelligence diminishes while crystalized intelligence increases until around 30 years of age or beyond (Cattell, 1963). Horn (1968) and Cattell (1963) agreed that acculturation plays a role in developing the building blocks needed to support crystalized intelligence. Horn discussed how through physiological functioning, neurons work together in a specific pattern to create networks. These networks work together through fluid intelligence (or acculturation) to create crystalized intelligence.

The Hazards of Lead Toxicity

Exposure to Lead: Biological Factors

It is known that lead enters the body in various ways (living in older housing units, parental employment, food, etc.); however, females who have been contaminated with lead also pass it onto their fetus and corrupt the vital development needed for a healthy baby (Bakhireva et al., 2013; Forns et al., 2014; Miranda et al., 2010; Sanders et al., 2012). Miranda et al. (2010) measured lead levels of pregnant women to find out where lead is stored in the mother's body and how lead is transferred to the fetus and baby. They learned that lead is stored in different areas of the body: the periosteum, which is a thick layer of vascular connective tissue that surrounds bones, excluding joints, and is most common in infants. This releases lead into the blood stream. Another area is the trabecular bones (pelvis, ribs, and skull) where lead can be stored 3 to 5 years. The last place is in the cortical bones (midtibia and midfemur) where lead can be stored up to 30 years.

Lead is transferred from mother to child during pregnancy (Bakhireva et al., 2013; Forns et al., 2014; Miranda et al., 2010; Sanders et al., 2012). When a woman is pregnant, there is a high demand put on her body for calcium to support the developing baby. If the mother has lead in her body from historical exposure, the risk of transferring lead to the fetus is high because the body cannot decipher between lead and calcium. The lead is extracted from the mother's bones, like calcium, and transmitted to the fetus through the placenta barrier. When lead is introduced into the developing fetus, damage can be done to the central nervous system (CNS) and the brain (Lu et al., 2013; Lucchini et al., 2012;

Martinez et al., 2013; van der Kuijp, Hunag, & Cherry, 2013). When a woman has multiple pregnancies, there is less lead in the mother's system to pass on (Miranda et al., 2010).

Lead exposure from the environment and household products are dangerous to the mother and fetus (Bakhireva et al., 2013; Sanders et al., 2012). Bakhireva et al. (2013) studied the pervasiveness of elevated blood lead levels in pregnant women with low socio-economic status to pinpoint behaviors this population exhibits that link to possible causes of lead exposure. After blood lead levels were measured, it was found that Mexican-American immigrant women had the highest blood lead levels. The processing of traditional candy wrappers may have lead in them causing the candies to be contaminated with lead. Chile that is used in cooking may have been grown in soil that was polluted with lead. Clay pots that used as food storage containers made with lead based glaze and used to store the candies and chile would taint the food. However, through a change in environment within two months their lead levels decreased. This may be due to the change in where traditional Mexican candies, cooking seasonings, and food storage containers are produced.

Several different metals have been studied, including lead, in North Carolina (Sanders et al., 2012). Through biomonitoring it was learned what population is at more risk for exposure to metals in an attempt to reduce future exposure and any unfavorable developmental effects on future generations. Biomonitoring is when blood is drawn and levels of toxic metals are determined. Evidence showed that race and location of their home were associated with elevated blood lead levels. Through more maternal education

on lead and biomonitoring, exposures to toxic metals would be diminished. Sander et al. (2012) supports biomonitoring programs on toxic metals to help care for the health of future generations. In 2012, Minnesota and New York participated in biomonitoring. Currently, there are there are nine more states (California, Massachusetts, New Hampshire, New Jersey, Virginia, Utah, Arizona, Colorado, and New Mexico) that participate (CDC, 2014). Measuring lead can be done in many ways. Once lead has contaminated the body it can be found in hair, fingernails, and teeth (Barton, 2011; Deshommes, Tardif, Edwards, Sauve, & Prevost, 2012; Kim & Kim, 2011). Biomonitoring seems to be the best way to measure lead toxicity. However, blood is the most direct indicator of the exact amount of lead in the body (Forns et al., 2014; Sander et al., 2012; Yoshinaga et al., 2012;).

Forns et al. (2014) conducted a longitudinal study with pregnant women to see if there were any deficits in child cognition due to exposure to lead. During the first and third trimester of pregnancy, the women provided urine samples to measure lead levels. When the infant was born, they were enrolled into the study. Histories on maternal employment, physical and mental health, diet, education, and breastfeeding practices were obtained. Women were given a perceptive-performance using Factor "G" by Cattell and Cattell and verbal IQ using the third edition Wechsler Adult Intelligence assessment. When the children turned 4 years of age, neuropsychological assessments were conducted using the McCarthy Scales of Children's Abilities. Forns et al (2014) found the results to be non-significant between lead toxicity and developmental deficits. They stated that using urine samples from the mother's may not have been the best way to measure lead.

Blood would have been the best choice because blood reveals recent intake and stability from tissue and bones.

Exposure to Lead: Environmental Factors

Environmental lead insults can be found in the air due to industrial and man-made chemicals used in batteries, cleaning supplies, plastics used in baby bottles, every day plastic food storage containers, burning of coal, gas, oil, and garbage, and natural gas such as propane, butane, and liquefied petroleum gas (Jurewicz et al., 2013; van der Kuijp et al., 2013). All of these pollutants are dispersed into the atmosphere and inhaled or ingested into the body and could harm human development. Lead can also be found in some plastic candy wrappers, water, soil, housing renovations (Spanier, Wilson, Ho, Hornung, & Lanphere, 2013), and paint that was produced before 1970s (Blando et al., 2013).

Housing built before the 1970s has shown risks for increased blood lead levels (Blando Antoine, & Lefkowitz, 2013; Spanier et al., 2013). In addition, renovations being done on homes or buildings that were constructed in the same time frame have the same potential risk for increase blood lead levels. The older the building structure the more likelihood lead based paint was used, which is the most significant source of lead exposure in the United States (Blando et al., 2013; Pourmand, Al-tiae, & Mazer-Amirshahi, 2012). When renovations occur on older buildings, dust from the paint enters the air. Construction workers may breathe in the dust or it gets on their skin and clothes thus taking lead contaminated dust home to their families. The Environmental Protection Agency (EPA) has regulations on homes build before 1978. They require all individuals

who work in construction to be educated and trained on proper ways to handle possible lead contaminated buildings. This is to help reduce further possible contamination from lead.

Studies have been conducted all over the world on lead contamination and toxicity. In Rochester, New York, home renovations were linked to an increase in blood lead levels compared to homes with no renovations (Spanier et al., 2013). In Iran, the potential for exposure to lead is very high. Air, soil, water, and food are all at risk for lead contamination. Iran still uses leaded gasoline that causes lead to be disbursed into the air, their mining and smelting facilities do not have regulations on proper ways to discard waste leading to soil and water contamination, and fish, rice, as well as canned goods have shown elevated lead levels (Pourmand et al., 2012). Iran has not created a national organization for development, oversight, and enforcement of contamination. Iran has not set clear guidelines on investigating or implementation of regulations on lead exposures. Iran has left it up to local small town or rural governments to monitor industrial and residential lead exposures. In Brazil, four risk factors of lead exposure were identified as age, living near an industrial site, tobacco smoking, and burning of domestic waste (Cho et al., 2010; Menezes-Filho, de Sousa Viana, & Paes 2012).

China has a rising industry on batteries. From 1998 to 2011, production of lead-acid batteries rose from about 2000 batteries to over 14000 a year (van der Kujip et al., 2013). Through mining, smelting, assembly of batteries, and recycling, lead is disbursed into the atmosphere. The waste is either sent to a treatment facility or sent directly back into the community water supply. Families living in industrial communities have a higher

risk of exposure to lead. These children are at extremely high risk of toxicity because 70% of ingested lead is absorbed into their bodies while 30% is discharged in body waste. Adults are at less risk of toxicity because 99% of exposure is discharged in body waste. However, millions of children are at a very high risk for lead poisoning. If a change is not made soon in China regarding policies, procedures, and over sight, there will be a huge increase in childhood lead toxicity that would demand change at an increased cost.

Japan has minimal risk of lead exposure compared to other countries. One of the main indicators of exposure to lead worldwide has been due to lead based paint and lead based gasoline (Blando et al., 2013; Spanier et al., 2013; Yoshinaga, 2012). Since the 1980s there has been a decrease in exposure to lead due to gasoline and in the Japanese culture household paint is not used. However, there is still daily exposure to lead, even a low amount, due to diet (dried foods and fish), soil (vegetables), commercial bottled water, toys (due to paint), and household dust. Smoking can also be a risk for lead exposure, but there is a minimal risk.

Dooyema et al. (2012) traveled to northwestern Nigeria to conduct a study to learn reasons behind an upsurge in childhood mortality and a possible link between an increase of gold mining and exposure to lead. They traveled to 119 family compounds to test community well water, soil, children's blood, and asked about maternal employment. Two-thirds of the family compounds was where gold ore was handled. Of the 463 children assessed, 25% of them died due to lead toxicity. From the children who were living, 97% started Chelation therapy due to extremely high levels of lead in their system.

Chelation therapy is the process of two molecules that combine with a heavy metal (lead) and then secreted from the body (CDC, 1991). Due to the very high levels of exposure and negative impact on individuals, public health education and environmental remediation were brought to the family compounds (Dooyema et al., 2012). Due to public health education family mining activities was removed from the family compounds.

Lead contaminated soil puts children as risk for exposure. Both urban and rural areas place children at risk, however, urban areas cause the greatest risk for exposure due to an increase of potential sources of lead; such as road ways and industry leading to higher concentrations of lead in soil (Aelion et al., 2013; Taylor & Schniering, 2010). Studies in South Carolina and Australia examined why such a high number of children were being diagnosed with intellectual disabilities, behavior problems, and diminished school performance without a known cause (Aelion et al., 2013; Taylor & Schniering, 2010). The results found that the children had elevated blood lead levels. Upon further investigation, there was a minimization from governmental agencies of how lead effects children (Taylor & Schniering, 2010). The minimization may have been because even at low levels children do not show extreme physical symptoms of lead poisoning or toxicity. As a result, parental education and early interventions were increased to help minimize the risk of lead toxicity (Aelion et al., 2013; Taylor & Schniering, 2010).

Ferrie, Rolf, and Troesken (2012) conducted a study to ascertain a possible link between exposure to lead through household plumbing called plumbosolvency and its impact on IQ. Plumbosolvency is the immersion of lead into the water supply. To limit the amount of lead absorbed into the water a pH balance is necessary. A solid measure of

pH is a 7. Starting at 6.5 or blow means water is more acidic and 8.5 or above means water is more alkaline. If water is 6.5 or below the water will absorb lead do to the lack of pH balance. Lead absorption can also occur if the water is 8.5 or above it can absorb lead. If there is not a balance of pH in the water lead is absorbed. Ferrie et al. (2012) reviewed archival data from the military on individuals who were enlisting during World War II. Scores from the Army General Classification Test (AGCT) were used to measure IQ. The scores from the AGCT were lumped together by where the individuals lived during the 1930s. They found that individuals living in areas where if pH was balanced around 7 AGCT scores were higher than if pH was too high or too low. This indicates that a pH balance is influential in lead levels to the water supply.

The Potential of Lead Toxicity on Intelligence

Impact of Lead Exposure to the Brain

Healthy brains change structurally throughout childhood development (Brain Development Cooperative Group, 2011; Brant et al., 2013; Burgaleta et al., 2014;). Magnetic Resonance Imaging (MRI) was used to view the regional brain volumes (Brain Development Cooperation Group, 2012). They were looking at white matter, gray matter, and total brain volumes. MRIs were reviewed on 325 participants aged 4.6 to 18.3 years and represented all socioeconomic backgrounds across the United States. They found that the biggest increase in total brain volume occurs between age 5 and early school age and as people age gray matter decreases while white matter increases.

Burgaleta et al. (2014) examined the relationship between cortical thickness and IQ. MRI scans were used to measure cortical thickness and the WASI was used to

measure IQ. Burgaleta et al. (2014) found that if a person showed an increase in their full-scale IQ (FSIQ) there was no significant change in their cortical thickness. However, some individuals who showed the largest increase in IQ showed some increase in cortical thickness. If there were no change in the individuals FSIQ there was a modest decrease in cortical thickness and if there were declines in FSIQ there was the steepest decrease in cortical thickness.

There is no answer as to why cortical thickness and gray matter decrease as people age and white matter increases in healthy brains (Houston, Herting, & Sowell, 2014). However, there is a theory that synaptic pruning is a factor. Synaptic pruning is when extra or damaged neurons are purged to increase effectiveness of the remaining neurons. There is also no answer as to why there is a decrease in cortical thickness as we age, even though alterations in cortical thickness happens at different times during infancy and early childhood development.

To find out what areas of the brain were affected by lead, brain scans (magnetic resonance spectroscopy, MRS, or magnetic resonance imaging, MRI) were taken (Cecil et al., 2011). The areas of the brain that were scanned were three gray matter sections (left basal ganglia, left cerebellar hemisphere, and cerebellar vermis) and two white matter sections (left frontal and left parietal) on adults who were diagnosed with lead poisoning as children. When there is an increase in blood lead levels there is a decrease in the brain structure areas. The gray matter sections of the cerebellar hemisphere and basal ganglia showed a decrease in *N*-acetyl aspartate (NAA). The cerebellar vermis and parietal white matter showed a decrease in glutamate and glutamine (GLX). In the white

matter frontal lobe, parietal lobe, and cerebellar hemisphere there was a decrease in glycerolphosphocholine and phosphocholine (Cho). Thus, as the blood lead levels increased so did the risk for greater neurological injury.

Cortical thickening takes place in early childhood and as the child develops into late childhood, early adolescence cortical thinning occurs until it levels out as the person enters into young adulthood (Brant et al., 2013). If cortical thinning is delayed this may lead to higher IQ. Shared family experiences are influential in how long the cortical thickening lasts. In a replication study, the focus was on the how much influence twins, biological siblings and adoptive siblings had on each other. They found that higher IQs are associated with extended sensitive period due to environmental influences. Extended-sensitivity-period is when cortical thickening lasts longer in childhood resulting in a higher IQ. When a child is allowed to grow and change through age appropriate developmental stages, cortical thickness lasts longer. However, if a child has to step into adult like roles during childhood cortical thickness thins earlier which could lead to lower IQ.

The populations at greatest risk of lead toxicity are infants, children, and pregnant women (Finkelstein, Markowitz, & Rosen, 1998). The blood-brain barrier has a higher absorption rate of lead which might cause initial structural damage to astrocytes with additional injury to endothelial cells. After lead exposure has penetrated the blood-brain barrier, the cerebral cortex (prefrontal cortex), hippocampus, and cerebellum are areas of the brain that may results in injury. Injury to the prefrontal cortex results in difficulty with distractibility, improper behavior reactions, and perseverations. Injury to the

hippocampus results in impairment to memory and learning and injury to the cerebellum results in diminished fine motor skills (Finkelstein et al., 1998; Lu et al., 2013).

Once lead has crossed the blood-brain barrier the central nervous system has been compromised starting with the synapse (first messenger) through the neuron (second messenger) and into the nucleus (third messenger), affecting the messenger systems (Finkelstein et al., 1998; Lu et al., 2013; Lucchini et al., 2012; Martinez et al., 2013; van der Kuijp et al., 2013). In the first messengers (neurotransmitters), lead plays a role of a chemical stressor on the septohippocampus and acetylcholine. According to Finkelstein et al. (1998), both play an essential role in emotion, memory, and learning. In the second messenger system (the neuron), lead passes through the membrane and interrupts what should be a calcium enriched process and can causes faulty information transferring from neuron to neuron. In the third messenger system (the nucleus), lead may disrupt DNA repair. Lead interferes with the actions needing to take place in the restoration of DNA.

Mazumdar et al. (2012) investigated a possible link between lead exposure, changes in gene expression, and Alzheimer's disease. They wanted to see is if childhood exposure to lead changes the genetic material that is connected to Alzheimer's disease. Mazumdar et al. (2012) evaluated plasma concentrations from two different amyloid β proteins $A\beta_{40}$ and $A\beta_{42}$. The individuals were selected due to participating in a study when they were infants and children who were identified having increased blood lead levels. They found that individuals who had been exposed to lead toxicity before birth had decreased gene expression found to be linked to Alzheimer's disease.

Animal studies have also been completed on how lead toxicity has affected different areas of the brain. Guinea pigs, mice, and rats have been exposed to lead at various stages of development to see how lead has affected the brains. Studies have been completed on the hippocampus, the cortex, thalamus, blood, and right femur as well as undeveloped neurons in the second doublecortin (DCX) layer, the subventricular (SVZ) and subgranular (SGZ) zones and memory (Huang et al., 2012; Li et al., 2013; Lu et al., 2013; Rao Barkur, Rao, & Bairy, 2011; Sanchez-Martin, Fan, Lindquist, Xia, & Puga, 2013).

The cortex and thalamus showed gene expression damage. However, the hippocampus has shown greater damage over all (Sanchez-Martin et al., 2013). The hippocampus demonstrated impairment to the mitochondria, microfilaments, and the microtubules (Li et al., 2013). The hippocampus showed alterations to the Golgi complex expansion, apoptotic cells, and there were abnormal dense bodies in the cytoplasm. However, there was no major change in the overall synaptic structure. Even when rats showed evident brain damage, the spatial learning tests were not statistically significant.

The hippocampus has been the most studied area of the brain because of the effects on learning and memory due to lead exposure (Finkelstein et al., 1998; Li et al., 2013; Lu et al., 2013; Sanchez-Martin et al., 2013). Treatments have been developed to help reverse lead toxicity that have proven to work. The treatment is not used unless a child's blood lead levels are 45 µg/dl or higher (CDC, 2014). A study was done with rats to find out if a natural medication is just as effective. Mangiferin is a very strong antioxidant that was discovered in fruits, leaves, roots, and bark. They compared

Mangiferin to Dimercaptosuccinic (DMSA) (Li et al., 2013). DMSA is used to treat individuals diagnosed with lead poisoning. The rats were divided into six groups. The groups were based on the amount of time (8 to 12 weeks) the rats were exposed to lead and the treatment of Mangiferin or DMSA. Ten rats from each group were given the Morris water maze test. The Morris water maze test is a spatial test to see how long it takes for each participant to reach the escape platform in a pool of water. The rats were placed in the water from predetermined locations of the pool and timed to see how long it will take for each rat to arrive at the platform. The results demonstrated that Mangiferin not only improved spatial learning, but it also reduced the amount of lead in the blood and bones. The body has a natural ability to detoxify cells using antioxidants and the natural ability to determine what cells should be eliminated due to DNA damage. By exposing rats to lead and then dissected the brains, a significant decrease in neural activity within the hippocampus was found due to oxidative stress and apoptosis within the hippocampus. The younger the rats the more apoptotic cells that were found. This interrupted the natural process of purging damaged cells.

Rao Barkur et al. (2011) also found that memory can be affected due to lead toxicity. Adult rats were exposed to lead during late pregnancy through the first few weeks after birth. The rats were then tested using a passive-avoidance task. The task was putting the rats into a box with two compartments. On one side of the box there was light and the other side there was no light. In the beginning the rats were given time to explore the two sides. After a few test trials, the room with no light was rigged to shock the rats upon entrance. In their observations the rats that were exposed to lead would enter the no

light room more often than the rats that were not exposed to lead showing impaired memory retention.

Impact of Lead Exposure on Intelligence

The CDC has a reference value of lead of 10µg/dl and the WHO states that 5µg/dl may impact development but there is no safe lead level (CDC, 2014; WHO, 2014). Neuropsychological assessments are used when there is brain injury (Lidsky & Schneider, 2006). These assessments help to determine what areas on the brain have been affected by the injury so interventions may be used. The WAIS-IV is a tool used to measure IQ. When conducting neuropsychological evaluations, IQ is an important piece of information. Lead is known to cause brain damage and lower IQ in children, but very little is still known about the long-term effects in young adulthood (Mazumdar et al., 2011).

Neuropsychological testing is very important, but doing just one assessment may not give you all the answers (Lidsky & Schneider, 2006). Giving multiple assessments to measure numerous areas of functioning is best. This way the opportunity to look at the whole picture will help determine diagnosis and treatment. A majority of the studies that have been completed focus on multiple areas of the subject's life. However, a combination of the subject's life and full neuropsychological assessments has not been attempted.

Lidsky and Schneider (2006) conducted a case study with a girl who was diagnosed with lead toxicity. She was observed and assessed to see where her deficiencies lye. A larger study was conducted to see if there were any correlations

between the girl, the larger study, and any impairments. They found that impairments can differ from one child to another. It depends on when lead was introduced to the developing child that will depend on what area of the brain will be most affected. The brain changes and develops at different times and rates as well as neurons. This indicates that children with lead toxicity have brain damage.

According to Jakubowski (2011), several different governmental agencies have established connections between exposure to lead from the environment and the overall health risks, including brain development and IQ. When reviewing the history of blood lead levels, there was a discrepancy between agencies regarding blood lead levels and the level considered acceptable for low levels of exposure. Even though the governmental agencies lowered the blood lead levels to 100µg/dl, an inverse relationship between IQ and blood lead levels was uncovered. Higher the blood lead levels were associated with lower the IQ scores. Data collected on 1333 children who participated in seven international studies on the effects of lead insult on IQ found an estimated reduction of 6.9 IQ points when blood lead levels were measured between 24 and 300µg/dl. Though this was a large study, there is very little research on long-term effects on individuals who were exposed to high levels of lead in childhood and how lead insult affects IQ in early adulthood.

Ramsden et al. (2011) tested 33 healthy adolescents were tested to see if brain plasticity changes occurred during brain development in teen years. IQ testing was administered at ages 12 to 16 years and again between 15 and 20 years. Magnetic resonance imaging (MRI) of the teens brains were also taken. They showed that young

brains change and develop rapidly. Any toxin, like lead that is introduced to the brain at an early age could possibly alter a person's IQ, even in young adulthood.

China has a rising industry on lead -acid batteries causing a potential risk for an increase in lead exposure and toxicity (van der Kujip et al., 2013). Testing individuals with low levels of lead toxicity is a focus to help show that any level of lead impacts development. A correlation between low blood lead concentrations and children's IQ with school performance in China and northern Italy has been reported (Liu et al., 2013; Lucchini et al., 2012). In China, blood lead levels that were between 6µg/dl and 10µg/dl in children at 6 years old were assessed using Wechsler Preschool and Primary Scale of Intelligence – Revised (WPPSI-R). The perceptual (visual- spatial skills), verbal (verbal skills), and full IQ scores were reviewed. At ages 8 – 10 years old, three major school subjects were evaluated (Chinese, Math, and English as a second language). The scores showed that blood lead levels impacted perceptual IQ and school performance more than full scale or verbal IQ. Their findings support low levels of lead in the blood still have a significant association with cognitive development. In Italy, adolescents aged 11-14 who have been exposed to low levels of lead found that there was a ratio between lead levels and IQ. For every 2.4 decrease in IQ, blood lead levels are doubled.

When blood lead levels are elevated in children ages 2-5, their behavior, mental development and IQ can be compromised (Canfield et al., 2003; Hou et al., 2013). To understand how compromised the children's functioning may be, assessments are given. Due to the young ages the Gesell Development Scale, Achenbach Child Behavior Checklist, and Stanford-Binet Intelligence Scale was used. A negative correlation was

found between lead poisoning and the development of the children's gross and fine motor functioning, language growth, social behavior and IQ.

Two follow-up studies on the long-term effects of lead have been attempted. The first study originally took place in the late 1970s to 1980s in the Boston area. It involved individuals who were sought out at the age of 10 for testing do to being diagnosed with lead poisoning (Mazumdar et al., 2011). Approximately 19 years later, attempts were made to find the same individuals for a follow up assessment. Separate subjects were chosen to complete tested using the WASI. These individuals were similar demographically to the original cohort. They were white, college educated, same socio-economic status, had a blood lead history, and IQ scores from their childhood were collected. Even though the same participants were not used in the follow up study there was support of long term negative and irreversible consequences to early childhood lead exposure. IQ scores were lower even in adulthood compared to individuals without lead poisoning.

Searle et al. (2014) conducted a second study follow up study was originally completed in the 1970s in South Australia. Port Pirie was chosen due to the location of the world's largest lead smelter, which was the town's main industry. Again, do to time constraints and the location of where the participants were living, these individuals only filled out questionnaires and there was no follow up with IQ testing. Given the inability to administer IQ testing in adulthood to the same individuals, there is still a question on how lead insult affects said individuals due to early childhood exposure. However, low-level lead exposure can have a negative impact on individuals. This is more evident at a

population level compared to an individual level. Another finding was that lead affected females showing greater developmental deficits than males. The slightest decrease in IQ scores could necessitate childhood interventions and possibly adulthood interventions as well.

Summary and Conclusions

Lead is a biological and environmental toxin. Humans are exposed to lead toxins daily that can have harmful effects to the brain and body. Humans can store lead in their bones and women can transfer lead to their fetus through blood exchange. Lead toxicity is known to cause brain damage that will later impact a person's learning and memory abilities. A majority of studies that have been completed are on infants, children, and youth. The brain develops and changes so much in these young years that any damage that is incurred can be permanent. Longitudinal studies on adults who were identified with lead toxicity in their childhood are limited. This current study will help fill the gap on long term effects of lead toxicity on IQ. In Chapter 3 research methods and the use of secondary data will be discussed.

Chapter 3: Research Methods

The impact of lead, a neurotoxin, on the developing brain has been a concern since the 1970s (Bakhireva et al., 2013; Blando et al., 2013; Miranda et al., 2010; Sanders et al., 2012; Spanier et al., 2013). Research has centered on children and how exposure to lead affects their intellect, but little attention has been paid to young adults who were exposed and treated for lead neurotoxicity. For this study, I accessed and analyzed secondary data from a private agency, ADHD and Autism Psychological Services and Advocacy, in Central New York where lead testing had been completed. In Chapter 3, I will discuss the data set, population, instrumentation, design, and analysis for this study.

Research Design

This was a nonexperimental, quantitative study in which multiple regression analysis was completed on secondary data. The predictor variables were first and last blood lead levels and APGAR scores upon birth as indicated from medical records. The APGAR scores were gathered for this study to be used as a baseline of the overall health of each participant upon birth. The criterion variables are from two areas of IQ testing, the PRI and VCI including the subtests block design, matrix reasoning, visual puzzles, vocabulary, information, and similarities. The purpose of this study was to determine if blood lead levels impact WAIS subscale scores (PRI, block design, matrix reasoning, visual puzzles, VCI, vocabulary, information, and similarities).

Procedures for Recruitment, Participation, and Data Collection

Participants for this study were part of a class action lawsuit. The lawyers sought them out due to a history of childhood lead poisoning and are currently young adults. I do

not know how each individual was exposed to lead, and this was not relevant to this study. Their lawyers referred each participant to a clinic in Central New York where IQ testing was completed. The lawyers provided all medical documentation for each participant. The medical records indicated first and last blood lead levels and APGAR scores. Blood was drawn and tested for lead by medical doctors and APGAR scores had been given at birth to help determine if medical intervention was necessary to help support the new born. Blood lead levels and APGAR scores was the only information taken from the medical records. Each participant signed a consent form acknowledging that their participation was voluntary, and how their information would be used in the future, their awareness of confidentiality, and that they had the right to refuse or withdraw at any time from the study.

Access to the data set was granted by a clinic in Central New York where I was provided an internship and learned about the clinic's work with lead testing. All IQ testing was completed by a Licensed Clinical Psychologist. Secondary data was used for this study. I did not have access to any identifying information, such as names, and did not have any contact with any of the participants. Corresponding birthdates to determine age, gender, first and last blood lead levels, APGAR scores, and IQ scores were provided. I will not benefit from this study in any way and there are no ties to the class action lawsuit.

Data Analysis Plan

This quantitative study was designed to determine if there are long-term effects of lead neurotoxicity on IQ. The data were analyzed using the SPSS 21.0 software package.

Research questions were developed to test the possible relationship between blood lead levels and IQ, specifically the VCI, vocabulary, information, similarities and PRI, block design, matrix reasoning, and visual puzzles. These subtests of IQ were specifically chosen due to the areas of the brain that were studied. Studies have indicated that the brain changes and develops throughout life. The Brain Development Group (2012) and Burgaleta et al., (2014) explained that, as a person ages, gray matter decreases while white matter increases. Multiple regression was used to analyze these variables and determine whether they predict subscale scores on the WAIS-IV IQ.

Research Questions and Hypotheses

RQ1: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of PRI scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their childhood?

$H_0$1: Early childhood lead exposure is not a significant predictor of PRI scores in early adulthood.

H_a1: Early childhood lead exposure is a significant predictor of PRI scores in early adulthood.

RQ2: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of block design scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$2: Early childhood lead exposure is not a significant predictor of block design scores in early adulthood.

H_a2: Early childhood lead exposure is a significant predictor of block design scores in early adulthood.

RQ3: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of matrix reasoning scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

H_03: Early childhood lead exposure is not a significant predictor of matrix reasoning scores in early childhood.

H_a3: Early childhood lead exposure is a significant predictor of matrix reasoning scores in early adulthood.

RQ4: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of visual puzzles scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

H_04: Early childhood lead exposure is not a significant predictor of visual puzzles scores in early childhood.

H_a4: Early childhood lead exposure is a significant predictor of visual puzzles scores in early adulthood.

RQ5: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of VCI scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their childhood?

H_05: Early childhood lead exposure is not a significant predictor of VCI scores in early adulthood.

H_a5: Early childhood lead exposure is a significant predictor of VCI scores in early adulthood.

RQ6: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of vocabulary scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

H_06: Early childhood lead exposure is not a significant predictor of vocabulary scores in early childhood.

H_a6: Early childhood lead exposure is a significant predictor of vocabulary scores in early adulthood.

RQ7: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of information scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

H_07: Early childhood lead exposure is not a significant predictor of information scores in early childhood.

H_a7: Early childhood lead exposure is a significant predictor of information scores in early adulthood.

RQ8: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of similarities scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

H_08: Early childhood lead exposure is not a significant predictor of similarities scores in early childhood.

H_a8: Early childhood lead exposure is a significant predictor of similarities scores in early adulthood.

Population

This study included an age population that had not been considered in previous research (ages 18-25). Lead poisoning is considered a serious problem with children and their developing brains (Liu et al., 2013). Children are more often the subjects of lead studies because they are considered more vulnerable (Jakubowski, 2011). Prior research had shown lead insult having a negative impact on brain development (Mazumdar et al., 2011). Due to the focus on children, there is a lack of research on the effects of lead toxicity in adulthood.

This study was an attempt to determine if there was a relationship between blood lead levels and a decrease in perceptual reasoning and/or verbal comprehension IQ scores. Each participant was referred for testing by their lawyer in a class action lawsuit. Each participant signed a consent form stating they acknowledged their IQ testing, blood lead levels, APGAR scores and birthdates (to determine age) were used in this study. IQ testing was administered and completed by a licensed clinical psychologist in Central New York.

I conducted an a priori power analysis in order to determine the sample size required for the original proposed design using the statistical program, G*Power 3 (Faul, Erdfelder, Lang, & Buchner, 2007). For the power analyses, the alpha level was set at .01 with a power of .95 and a medium effect size of 0.15 will be used for this study as this has been determined to be the largest effect size appropriate for studies in the behavioral

sciences (Cohen, 1988). The alpha level was set at .01 to reduce the likelihood of a Type 1 error. The recommended total sample size needed to determine a significant difference with three predictors (first and last blood lead levels and APGAR scores) for multiple regression was n =157 young adults. This study involved n =170 participants. I used multiple regression to evaluate the long-term effects lead has on comprehension and reasoning skills.

Instrumentation and Operationalization of Constructs

The WAIS-IV is a multidimensional assessment used to calculate the Intelligence Quotient (IQ) on individuals ages 16 years and 0 months through 90 years and 11 months (Wechsler et al., 2008). The WAIS-IV measures multiple areas of intelligence. It is divided into four sections (VCI, PRI, Working Memory Index, and Processing Speed Index) to generate an overall score that represents a person's intellectual ability (full scale IQ). Based on the areas of the brain that have been studied, this study focused only two indexes, the VCI and PRI.

The VCI measures crystallized knowledge. This knowledge comes from a life time of learning, through personal and educational environments, words, definitions, and how to communicate thoughts and ideas through the use of vocabulary. There are three subtests to measure the VCI; Vocabulary, Similarities, and Information. All three subtests measure long term memory and retrieval. Vocabulary and similarities measure verbal concept formation while vocabulary also measures language development, information measures verbal comprehension, and similarities measure abstract verbal reasoning,

categorical thinking, and the ability to distinguish between non-essential and essential features of words.

The PRI measures fluid reasoning with some perceptual organization. There are three subtests to measure the PRI; Block Design, Matrix Reasoning, and Visual Puzzles. All three subtests measure visual perceptual reasoning and organization. Block design and visual puzzles measure non-verbal reasoning and analysis and synthesis of visual material. Visual puzzles and matrix reasoning measure visuospatial abilities and simultaneous processing.

Scores that are obtained do to IQ testing are explained in the level of current functioning. Majority of people score in the average IQ category, some people are above and others are below. No matter what IQ score a person obtains, IQ is supposed to remain stable over a person's life time (Burgaleta et al., 2014), however a person could have an illness or a traumatic brain injury of some kind that can alter a person IQ score. The results come in three sets of scores, the subtests, index scores, and full-scale IQ (FSIQ). The scores are in the form of sum of scaled scores, the subtest scores have a mean of 10 and a standard deviation of 3 while the index and FSIQ scores have a mean of 100 and a standard deviation of 15.

Reliability on the WAIS-IV is strong (Lichtenberger & Kaufman, 2009). Average split-half reliability coefficients for FSIQ is .98, VCI is .96 and PRI is .95. For the subtests, block design and similarities is .87, matrix reasoning is .90, vocabulary is .94, visual puzzles is .89 and information is .93.

Test-retest reliability was determined by having the subjects tested again after 3 weeks (Lichtenberger & Kaufman, 2009). Test-retest reliability coefficients for FSIQ and VCI is .96, PRI and similarities is .87, block design is .80, matrix reasoning and visual puzzles is .74, vocabulary is .89, and information is .90.

Construct validity shows strong support for the WAIS-IV when compared to the CHC theory five-factor model. VCI and PRI showed a factor loading of .08 and .43, respectively. The subtests similarities, vocabulary, and information show a factor loading between .80 and .89. Block design, matrix reasoning, and visual puzzles show a factor loading between .72 and .76.

While multiple studies have been conducted confirming validity of the WAIS-IV, Nelson, Canivez, and Watkins (2013) were the first to use a clinical sample versus a standardization sample. They had three objectives, the first was to find the best fitting structural model (one-factor, two-factor, three-factor, or four-factor), second was to find the best fitting hierarchical model (direct or indirect), and third if the four index scores as just a good at providing incremental predictive validity in academic skills beyond the FSIQ compared to the Woodcock-Johnson III (WJ-III) and the Nelson-Denny Reading Test (NDRT). They found the correlated four-factor model was the best fit of all the models tested and the WAIS-IV is statistically significant at predicting academic skills when compared to the WJ-III and NDRT.

Ethical Considerations

This study was conducted on secondary data provided through a private clinic in Central New York. Participants were referred to the private agency for lead assessments

by their lawyers due to a class action lawsuit. The lawyers provided the medical records for each participant who was assessed. The medical records indicate the first and last blood lead levels, APGAR scores, and birthdates (to determine age) for each participant, which was the only information used from the medical records. Blood was drawn and tested for lead by medical doctors. A Licensed Clinical Psychologist from the clinic administered all IQ testing. Each participant signed a consent form acknowledging his or her IQ testing, blood lead levels, APGAR scores, gender, and birthdates (to determine which ages) were included in the study. Each participant signed a consent stating that their data will be part of a study, potentially conducted by a third party at later time. This researcher did not have contact with any of the participants, nor was there any, and does not have access to any other identifying information, such as names. This researcher will not benefit from this study in any way and there are no ties to the class action lawsuit.

Summary

Chapter 3 reviewed the research design and approach to this study. The rationale for choosing a quantitative study and research questions were discussed. Criteria for the sample, sample size, and recruitment procedures were addressed, as well as informed consent. The instrument that was used during the testing process was described in detail, including reliability and validity of the WAIS-IV.

Chapter 4: Results

The purpose of this quantitative study was to discover if lead neurotoxicity affects IQ in young adults. More specifically, it sought to determine if there is a relationship between exposure to lead in young childhood and any possible effects on IQ in young adulthood (between ages 18 and 25). The following research questions and hypotheses were tested:

RQ1: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of PRI scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their childhood?

$H_0$1: Early childhood lead exposure is not a significant predictor of PRI scores in early adulthood.

H_a1: Early childhood lead exposure is a significant predictor of PRI scores in early adulthood.

RQ2: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of block design scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$2: Early childhood lead exposure is not a significant predictor of block design scores in early adulthood.

H_a2: Early childhood lead exposure is a significant predictor of block design scores in early adulthood.

RQ3: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of matrix reasoning scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$3: Early childhood lead exposure is not a significant predictor of matrix reasoning scores in early childhood.

H_a3: Early childhood lead exposure is a significant predictor of matrix reasoning scores in early adulthood.

RQ4: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of visual puzzles scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$4: Early childhood lead exposure is not a significant predictor of visual puzzles scores in early childhood.

H_a4: Early childhood lead exposure is a significant predictor of visual puzzles scores in early adulthood.

RQ5: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of VCI scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their childhood?

$H_0$5: Early childhood lead exposure is not a significant predictor of VCI scores in early adulthood.

H_a5: Early childhood lead exposure is a significant predictor of VCI scores in early adulthood.

RQ6: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of vocabulary scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$6: Early childhood lead exposure is not a significant predictor of vocabulary scores in early childhood.

H_a6: Early childhood lead exposure is a significant predictor of vocabulary scores in early adulthood.

RQ7: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of information scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$7: Early childhood lead exposure is not a significant predictor of information scores in early childhood.

H_a7: Early childhood lead exposure is a significant predictor of information scores in early adulthood.

RQ8: Is lead neurotoxicity (first and last blood lead levels) a significant predictor of similarities scores, as determined by the WAIS-IV, of individuals between the ages of 18 and 25, who were exposed to lead in their early childhood?

$H_0$8: Early childhood lead exposure is not a significant predictor of similarities scores in early childhood.

H_a8: Early childhood lead exposure is a significant predictor of similarities scores in early adulthood.

In Chapter 4, I will discuss the use of the secondary data set, how and when the data were initially collected, and screening of the data. A discussion of how all the variables are normally distributed, if there are any outliers, and any homoscedasticity and mulitcollinearity issues will follow. Finally, the results from the multiple regression analyses will be presented and the research questions will be answered.

Data Collection

Following the approval of the Internal Review Board (IRB; approval number 04-21-17-0184878), the gathering of secondary data began. During the data screening, I noted that there would not be enough participants due to the number of people who were tested using the WAIS-III instead of the WAIS-IV. A revised application was submitted to the IRB for approval to gather the WAIS-III data. Once I received the approval, the WAIS-III data were also gathered.

There were 242 possible participants. After screening the data, 25 participants were excluded due to their age, either above or below the age range for the study. Twenty-three participants were excluded due to a lack of information provided, 15 participants were not included due to the WAIS not being used for IQ assessment, and nine participants were not included due to missing blood lead levels. This resulted in a total of 170 participants included in the analyses.

The WAIS-III and WAIS-IV testing was completed between 2010 and 2017. Of the 170 testing scores, 22 participants were tested using the WAIS-III, and 148 were tested using the WAIS-IV. The ages of the participants at the time of WAIS testing were 18 to 25, with the average age being 22. First and last blood lead levels were tested at

different ages. First blood lead levels were tested when the participants were infants to young childhood with an average age of 1 year. Last blood lead levels were tested when the participants were infants to teen years with an average age of 6. APGAR scores were gathered; however, more than half of the scores were missing so this data could not be used as a predictor.

Descriptive Data

Table 1 provides a summary of the demographic characteristics of the sample in frequency and percentage. The sample included an equal number of women (50%) to men (50%). Age represents how old the participants were at the time the WAIS-III or WAIS-IV was given.

Table 1

Demographic Data for Participants

Character		Frequency	Percent
Gender	Female	85	50
	Male	85	50
Age	18	2	1.2
	19	10	5.9
	20	14	8.2
	21	20	11.8
	22	41	24.1
	23	33	19.4
	24	44	25.9
	25	6	3.5

Table 2 shows the means and standard deviations for all the variables. The APGAR scores were used to measure overall health at birth. As previously stated, APGAR scores of 7-10 are average and no medical intervention is needed. All the scores

that were collected were in the average range; however, 51.8% of the APGAR scores were missing. Because so many scores were missing, they could not be used as a predictor.

Table 2

Means and Standard Deviations for all Variables

Scale	*N*	Min	Max	*M*	*SD*
Perceptual reasoning index	170	60	131	84.81	12.00
Block design	170	3	17	6.98	2.24
Matrix reasoning	170	1	14	7.67	2.96
Visual puzzles	170	4	16	7.62	2.38
Verbal comprehension index	170	56	114	79.04	10.01
Vocabulary	170	1	12	6.09	2.16
Similarities	170	1	13	6.18	2.20
Information	170	4	14	6.52	1.93
First blood lead level	170	-4.0	76	21.68	11.48
Last blood lead level	170	-4.0	38	11.70	6.36
APGAR scores	82	7	10	8.50	.653

Statistical Assumptions

Skewness and kurtosis were analyzed on the numerical scale variables to check for normal distribution. PRI was approximately normally distributed, with a skewness of .49 (*SE* = .19) and a kurtosis of .61 (*SE* = .37). Block Design (BD) was positively

skewed, with a skewness of 1.16 (*SE* = .19) and a kurtosis of 1.93 (*SE* = .37). Matrix Reasoning (MR) was approximately normally distributed with a skewness of -.18 (*SE* = .19) and a kurtosis of -.77 (*SE* = .37). Visual Puzzles (VP) was positively skewed with a skewness of .93 (*SE* = .19) and a kurtosis of 1.08 (*SE* = .37). VCI was approximately normally distributed with a skewness of .52 (*SE* = .19) and a kurtosis of .25 (*SE* = .37). Vocabulary was approximately normally distributed with a skewness of .46 (*SE* = .19) and a kurtosis of -.23 (*SE* =.37). Similarities was approximately normally distributed with a skewness of .73 (*SE* = .19) and a kurtosis of .78 (*SE* =.37). Information was positively skewed, with a skewness of 1.14 (*SE* = .19) and a kurtosis of 1.20 (*SE* =.37). First Blood Lead Level was positively skewed, with a skewness of 1.30 (*SE* = .19) and a kurtosis of 1.71 (*SE* =.37). Last Blood Lead Level was positively skewed, with a skewness of 1.33 (*SE* = .19) and a kurtosis of 1.37 (*SE* =.37). Skewness and kurtosis show that all variables were normally distributed.

Multiple regression assumes the relationship between the independent variables and dependent variables are linear. A visual inspection of the boxplots indicated there are no outliers between variables (see Appendix B).

Homoscedasticity is the assumption that residuals at each level of the independent variable have similar variances at each level of the dependent variable. The scatterplots of standardized predicted values show that the numerical data met the assumptions of homogeneity of variance and linearity (see Appendix C).

Mulitcollinearity assumes there are two or more variables closely linearly related. The test to determine if the data met the assumption of collinearity indicated that

multicollinearity was not present for the predictor variables (PRI, *Tolerance = .992, VIF = 1.008*; Block Design, *Tolerance = .992, VIF = 1.008*; Matrix Reasoning, *Tolerance = .992, VIF = 1.008*; Visual Puzzles, *Tolerance = .992, VIF = 1.008*; VCI, *Tolerance = .992, VIF = 1.008*; Vocabulary, *Tolerance = .992, VIF = 1.008*; Similarities, *Tolerance = .992, VIF = 1.008*; Information, *Tolerance = .992, VIF = 1.008;* APGAR Scores, *tolerance = .992, VIF = 1.008*).

Study Results

This study attempted to determine whether individuals who were diagnosed with lead toxicity in their childhood affected IQ scores in their young adulthood. For this study, a standard multiple regression was used. The SPSS program was used to run the standard multiple regression. Eight separate analyses were run. The predictor variables were first and last blood lead levels. More than half of the APGAR scores were missing so these could not be used at predictor variables. The criterion variables were IQ scores (PRI, block design, matrix reasoning, visual puzzles, VCI, vocabulary, information, and similarities).

RQ 1: Perceptual Reasoning Index

The multiple linear regression results showed that blood lead levels were not significant predictors of PRI scores: first blood lead level ($t = 1.71, p = .089, \beta = .132$), and last blood lead level ($t = -.459, p = .647, \beta = -.035$). These results indicated that first and last blood lead levels were not significant predictors of PRI scores. Therefore, the null hypothesis was not rejected.

RQ 2: Block Design

The multiple linear regression results showed that blood lead levels were not significant predictors of Block Design Scores: first blood lead level (*t =1.51, p = .134, β = .116*), and last blood lead level (*t = -1.10, p = .273, β = -.085)*. These results indicated that first and last blood lead levels were not significant predictors of Block Design Scores. Therefore, the null hypothesis was not rejected.

RQ 3: Matrix Reasoning

The multiple linear regression results showed that blood lead levels were not significant predictors of Matrix Reasoning Scores: first blood lead level (*t =1.59, p = .113, β = .123*), and last blood lead level (*t = -.086, p = .931, β = .007)*. These results indicated that first and last blood lead levels were not significant predictors of Matrix Reasoning Scores. Therefore, the null hypothesis was not rejected.

RQ 4: Visual Puzzles

The multiple linear regression results showed that blood lead levels were not significant predictors of Visual Puzzles Scores: first blood lead level (*t =1.27, p = .205, β = .099*), and last blood lead level (*t = -.167, p = .867, β = -.031)*. These results indicated that first and last blood lead levels were not significant predictors of Visual Puzzles Scores. Therefore, the null hypothesis was not rejected.

RQ 5: Verbal Comprehension Index

The multiple linear regression results showed that blood lead levels were not significant predictors of VCI Scores: first blood lead level (*t = -.358, p = .721, β = -.028*), and last blood lead level (*t = .183, p = .855, β = .014)*. These results indicated that

first and last blood lead levels were not significant predictors of VCI Scores. Therefore, the null hypothesis was not rejected.

RQ 6: Vocabulary

The multiple linear regression results showed that blood lead levels were not significant predictors of Vocabulary Scores: first blood lead level (t =-.119, p = .905, β = -.009), and last blood lead level (t = .126, p = .900, β = .010). These results indicated that first and last blood lead levels were not significant predictors of Vocabulary Scores. Therefore, the null hypothesis was not rejected.

RQ 7: Similarities

The multiple linear regression results showed that blood lead levels were not significant predictors of Similarities Scores: first blood lead level (t = -.651, p = .506, β = -.050), and last blood lead level (t = .931, p = .363, β = .071). These results indicated that first and last blood lead levels were not significant predictors of Similarities Scores. Therefore, the null hypothesis was not rejected.

RQ 8: Information

The multiple linear regression results showed that blood lead levels were not significant predictors of Information Scores: first blood lead level (t = -.015, p = .988, β = -.001), and last blood lead level (t = -.837, p = .404, β = -.065). These results indicated that first and last blood lead levels were not significant predictors of Information Scores. Therefore, the null hypothesis was not rejected.

Summary

It was hypothesized that lead neurotoxicity (first and last blood lead levels) decreases IQ scores in individuals between the ages of 18 and 25, who were exposed to lead in their early childhood. Specifically, it was expected that elevated blood lead levels would result in lower IQ scores (PRI, Block Design, Matrix Reasoning, Visual Puzzles, VCI, Vocabulary, Similarities, and Information). Eight separate multiple regression analyses were run and indicated that blood lead levels were not significant predictors of IQ scores. In Chapter 5, I discuss the findings, limitations of the study, and recommendations for future research.

Chapter 5: Discussion, Conclusions, and Recommendations

The purpose of this study was to discern if lead toxicity affects individuals' IQ in early adulthood. More specifically, I sought to determine if there is a relationship between lead toxicity from early childhood and IQ in young adulthood. Secondary data were gathered from a private agency in Central New York where lead testing was completed on individuals who were plaintiffs in a class action lawsuit. The WAIS-IV was used to measure IQ. While I was gathering data, I learned that the WAIS-III was also used to measure IQ. After approval from the IRB, I also gathered the data from the WAIS-III. Only PRI, VCI, and the corresponding subtest scores were gathered.

The nature of this study was quantitative. Research questions were evaluated by reviewing the relationships between first and last blood lead levels and IQ scores (PRI, Block Design, matrix reasoning, visual puzzles, VCI, vocabulary, information, and similarities). The multiple regression analyses determined that blood lead levels in early childhood were not-significant predictors of IQ scores in young adulthood.

Interpretation of Findings

Mazumdar et al. (2011) noted the lack of research on the effects of lead on IQ in adulthood. Ferrier et al. (2012), Jakubowski (2011), and Liu et al. (2013) indicated that more research needed to be completed because the majority of studies had been done with children and infants. Mazumdar et al. (2011) and Searle et al. (2014) attempted to conduct follow-up studies on long-term effects of lead on IQ. In both studies, researchers attempted to locate individuals who were tested in their childhood for lead toxicity. In their follow-up study, Searle et al. (2014) was not able to conduct any IQ testing due to

time constraints. Mazumdar et al. (2011) conducted IQ testing using the WASI on a group of individuals that were similar demographically to the original group. Even though the same participants were not used in the follow-up study, there was support of long-term negative and irreversible consequences to early childhood lead exposure. IQ scores were lower even in adulthood compared to those individuals without lead poisoning. In this study, it was expected that elevated blood lead levels would be a predictor of lower IQ scores (PRI and VCI; including the subtests block design, matrix reasoning, visual puzzles, vocabulary, information, and similarities). However, the results showed that childhood lead toxicity did not predict IQ scores in young adulthood.

Results of this study regarding the blood lead levels and IQ scores may be limited partially due to the lack of demographics gathered. Gender and age were the only demographics gathered; however, race, the home, and the environment people grew up in have been associated with elevated blood lead levels (Sanders et al., 2012). Environmental lead insults can be found in the air, on the ground, and in products used daily, such as food storage containers and products to heat a home or drive a motor vehicle (Jurewicz et al., 2013; van der Kuijp et al., 2013). Lead can also be found in some plastic candy wrappers, water, soil, housing renovations (Spanier et al., 2013), and paint products before the 1970s (Blando et al., 2013). Other pertinent information would be having blood lead levels measured at the time IQ testing was completed. Testing blood levels is the most direct indicator of the exact amount of lead in the body (Forms et al., 2014; Sander et al., 2012; Yoshinaga et al., 2012;). This would give a current and more

accurate assessment of blood lead levels for each participant at the time IQ testing was completed.

The theoretical foundation for this study is based on the CHC theory, which supports the use of WAIS assessments to measure IQ (Wechsler et al., 2008). The WAIS assessments, developed using the CHC theory as the foundation, measure crystalized and fluid intelligence. Crystalized intelligence (*Gc*) measures acquired knowledge of language, information, and concepts of a specific culture. The VCI measures Gc. Fluid intelligence (*Gf*) measures the ability to solve novel problems, draw inferences, and inductive and deductive reasoning. The PRI measures Gf. As a person ages, fluid intelligence diminishes while crystalized intelligence increases (Cattell, 1963). Through physiological functioning, neurons work together in a specific pattern to create networks (Horn, 1968). These networks work together through fluid intelligence (or acculturation) to create crystalized intelligence.

Neuropsychological assessments, such as IQ testing, are used when there is brain injury (Lidsky & Schneider, 2006). These assessments help to determine what areas of the brain have been injured. Lead is known to cause brain damage and lower IQ in children, but very little is known about the long-term effects in young adulthood (Mazumdar et al., 2011). In this study, I reviewed IQ scores from a secondary data set of individuals ages 18-25 who were treated for elevated blood lead levels in their childhood. This study was unable to show a relationship between an increased blood lead levels in childhood and lower IQ scores in young adulthood.

Limitations

There were several notable limitations of this study. This analysis was a cross-sectional study of a specific age population at a single point in time. Each participant was given a battery of assessments, but IQ was the only assessment reviewed for this study. The focus on the VCI and PRI and their subtests was based on the areas of the brain that had been previously researched. The Brain Development Group (2012) and Burgaleta et al. (2014) conducted separate studies showing that, as a person ages, gray matter decreases while white matter increases, which shows changes in brain structure. As a person grows into their teen years, fluid intelligence diminishes, while crystalized intelligence increases to around 30 years of age or beyond (Cattell, 1963). Horn (1968) and Cattell (1963) agreed that acculturation plays a role in developing the building blocks needed to support crystalized intelligence.

While some studies presented in Chapter 2 indicated that lead toxicity impacted IQ, the present study was unable to show the same relationship. Liu et al. (2013) completed a study with children ages 3-5 where the average blood lead levels were 6.43 µg/dl, and a significant difference was noted when blood lead levels reached $\geq$10 µg/dl and IQ scores decreased by 2 points. Lucchini et al. (2012) conducted another study with young adolescents, ages 11-14, where the average blood lead levels were 1.71 µg/dl and was associated with a decrease of 2.4 IQ points. In the current study, the average blood lead level for the sample at first measurement was 21.68 µg/dl and the average blood lead level for the sample at last measurement was 11.70 µg/dl. Those first and last blood lead level averages were higher than previous studies; however, they did not show an impact

on IQ scores. In addition, the last blood lead level measurements in the current study were not taken at the time IQ assessments were completed. Thus, the amount of lead that may have remained in the participants at the time of IQ testing was not known.

It should also be noted that while this study did not find a relationship between childhood lead exposure and later IQ scores in young adulthood, one should not conclude that lead exposure is not linked to cognitive deficits. Previous research reviewed in Chapter 2 has demonstrated links between childhood lead exposure and various cognitive deficits (e.g., Canfield et al., 2003; Hou et al., 2013; Jakubowski, 2011; Lidsky & Schneider, 2006; Liu et al., 2013; Mazumdar et al., 2011; Searle et al., 2014; van der Kuijp et al., 2013). This apparent discrepancy with previous research could be explained by thresholds of exposure. It may be the case that for children who are exposed to lead and have compromised cognitive systems, higher levels of exposure do not further compromise them. In addition, the present study was not a test of whether early exposure results in decreases in IQ. In this study, I did not compare exposed and non-exposed individuals. I examined specifically whether there were any relationships between childhood lead exposure and IQ levels in young adulthood among individuals from a private clinic in central New York who were plaintiffs in a class action lawsuit involving lead exposure during their childhood.

Areas that were not considered for this study were educational background, socio-economic status, the type of exposure to lead, as well as how long they were exposed. Blood lead levels were not measured at the time of testing so the amount of lead that may have remained in their bodies at the time of testing was not known. The secondary data

showed that only a few participants took Chelation for treating increased blood lead levels and their lead levels dropped. Chelation therapy is the process of two molecules that combine with a heavy metal (lead) and then secreted from the body (CDC, 1991). However, it is not known if their lead levels remained low. There is debate if chelation therapy has any effect on individuals with blood lead levels between 10 µg/dl and 45 µg/dl (Beaudin et al., 2007). A study was conducted in Flint Michigan, before the change in water supply, to understand fluctuations in blood lead levels in children (Laidlaw et al., 2016). They found that every third quarter (late summer/ early autumn) of the year the lead in the soil gets stirred up due to warm dry weather. Children play outside more and are exposed to lead contaminated soil so blood lead levels increase. Blood lead levels fluctuate throughout the year depending on the risks of exposure. When exposure to lead increases blood lead levels increase. When exposure decreases blood lead levels decrease. They also found that children who live in the inner city, blood lead levels were four times higher than those in the outskirts. Random selection was not used for this study because my research was based on secondary data collected from one source, a private clinic in Central New York that conducted the IQ testing.

Future Recommendations

Further research where individuals who were diagnosed with childhood lead toxicity and have grown into young adults needs to be completed. These studies could include extensive background information. This would include education, socio-economic status, race, and medical history of lead exposure. The medical history would include how they were exposed, age of initial exposure, length of time they were

exposed, how long they were monitored for increased blood lead levels, blood lead levels measured at the time of IQ testing, and if chelation therapy was used. Many studies have looked at a direct link between lead toxicity and IQ (Jakubowski, 2011; Lidsky & Schneider, 2006; Mazumdar et al., 2011). Jakubowski (2011) collected data on 1333 children who participated in seven international studies on the effects of lead insult on IQ and found an estimated reduction of 6.9 IQ points when blood lead levels were measured between 24 and 300µg/dl. Lidsky and Schneider (2006) conducted a case study with a girl who was diagnosed with lead toxicity. Their case study showed that the brain changes and develops at different times and rates. Their conclusions indicate that children with lead toxicity have brain damage. Mazumdar et al. (2011) conducted a follow up study approximately 19 years after the original. They found that IQ scores were lower in adulthood compared to individuals without lead poisoning.

Some studies have included parental background information because the studies involved children (Forns et al., 2014; Miranda et al., 2010). Forns et al. (2014) conducted a study with pregnant women to see if there were any deficits in childhood cognition due to exposure to lead. After taking full background information and urine samples from the mothers, they found the results to be non-significant between lead toxicity and childhood developmental deficits. Forns et al. (2014) stated that urine samples are not the best way to measure lead and that measuring blood levels would have provided a more accurate assessment of lead exposure.

Conclusion

This investigation examined the relationship between lead neurotoxicity and IQ scores in young adults ages 18-25. This study was unable to show a relationship between childhood lead neurotoxicity and IQ scores in young adulthood. Previous research has shown a relationship between childhood lead toxicity and IQ scores. More research needs to be done to determine how much of a problem childhood lead neurotoxicity is in adulthood. By showing any long-term effects of lead toxicity, governmental agencies will have more information to be proactive in creating and changing policies around the use of lead in products that people use on a daily basis. This could lead to positive social change by placing an emphasis on early identification and treatment of lead exposure resulting in people being more successful in their educational, occupational, and economic goals.

References

Abadin, H., Ashizawa, A., Stevens, Y., Llados, F., Diamond, G., Sage, G., . . . & Swarts, S. G. (2007). Toxicological profile for lead. Department of Health and Human Services: Public Health Service, Agency for Toxic Substance and Disease Registry Retrieved from http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf

Aelion, C. M., Davis, H. T., Lawson, A. B., Cai, B., & McDermott, S. (2013). Associations between soil lead concentrations and populations by race/ethnicity and income-to-poverty ration in urban and rural areas. *Environmental Geochemical Health, 35,* 1-12.

Alfonso, V. C., Flanagan, D. P., & Radwan, S. (2005). The impact of the Cattell-Horn-Carroll theory on test development and interpretation of cognitive and academic abilities. In D. P. Flanagan & P. L. Harrison (Eds.), *Contemporary intellectual assessment: Theories, tests, and issues* (pp. 185-202). New York: Guilford Press.

Bakhireva, L. N., Rowland, A. S., Young, B. N., Cano, S., Phelan, S. T., Artyushkova, K., . . . & Lewis, J. (2013). Sources of potential lead exposure among pregnant women in New Mexico. *Maternal and Child Health, 17,* 172-179.

Barton, H. J. (2011). Advantages of the use of deciduous teeth, hair, and blood analysis for lead and cadmium bio-monitoring in children. A study of 6-year-old children from Krakow (Poland). *Biological Trace Element Research, 143,* 637-658. doi:10.1007/s12011-010-8896-6

Bellinger, D. C. (2008). Neurological and behavioral consequences of childhood lead exposure. *PLoS Medicine, 5,* 5.

Benson, N., Hulac, D. M., & Kranzler, J. H. (2010). Independent examination of the Wechsler Adult Intelligence Scale – Fourth Edition (WAIS-IV): What does the WAIS-IV measure? *Psychological Assessment, 22*(1), 121-130.

Blando, J. D., Antoine, N., & Lefkowitz, D. (2013). Lead-based paint awareness, work practices, and compliance during residential construction and renovation. *Journal of Environmental Health, 75,* 20-27.

Brain Development Cooperative Group. (2012). Total and regional brain volumes in a population-based normative sample from 4 to 18 years: The NIH MRI study of normal brain development. *Cerebral Cortex, 22,* 1-12.

Brant, A. M., Munakata, Y., Boomsma, D. I., DeFries, J. C., Haworth C. M. A., Keller, M. C., . . . & Hewitt, J. K. (2013). The nature and nurture of high IQ: An extended sensitive period for intellectual development. *Psychological Science*, *24,* 1487-1495.

Burgaleta, M., Johnson, W., Waber, D. P., Colom, R., & Karama, S. (2014). Cognitive ability changes and dynamics of cortical thickness development in healthy children and adolescents. *NeuroImage, 84,* 810-819.

Canfield, R. L., Henderson, C. R., Cory-Slechta, D. A., Cox, C., Jusko, T. A., & Lanphear, B. P. (2003). Intellectual impairment in children with blood lead concentrations below 10 µg per deciliter. *The New England Journal of Medicine, 348,* 1517-1526.

Cattell, R. B. (1963). Theory of fluid and crystallized intelligence. *Journal of Educational Psychology, 54,* 1-22.

Cecil, K. M., Dietrich, K. N., Altaye, M., Egelhoff, J. C., Lindquist, D. M., Brubaker, C. J., & Lanphear, B. P. (2011). Proton magnetic Resonance Spectroscopy in adults with childhood lead exposure. *Environmental Health Perspectives, 119,* 403.

Centers for Disease Control and Prevention. (2014, August 18). State Biomonitoring Grants. Retrieved from http://www.cdc.gov/biomonitoring/state_grants.html

Centers for Disease Control and Prevention. (2016, March 15). Update on blood lead levels in children. Retrieved from http://www.cdc.gov/nceh/lead/ACCLPP /blood_lead_levels.htm

Cho, S. C., Kim, B. N., Hong, Y. C., Shin, M. S., Yoo, H. J., Kim, J. W., . . . & Kim, H. W. (2010). Effect of environmental exposure to lead and tobacco smoke on inattentive and hyperactive symptoms and neurocognitive performance in children. *The Journal of Child Psychology and Psychiatry, 51,* 1050-1057.

Coalson, D. L., Raiford, S. E., Saklofske, D. H., & Weiss, L. G. (2010). WAIS-IV: Advances in the assessment of intelligence. In L. G. Weiss, D. H. Saklofske, D. L. Coalson and S. Engi Raiford (Eds.), *WAIS-IV Clinical Use and Interpretation* (pp. 3-20). Location: Academic Press.

Cohen, J. (1988). Statistical power analysis for the behavioral sciences (2nd edition). Retrieved from http://www.lrdc.pitt.edu/schneider/p2465/Readings /Cohen,%201988%20%28Statistical%20Power,%20273-406%29.pdf

Deshommes, E., Tardif, R., Edwards, M., Sauve, S., & Prevost, M. (2012). Experimental determination of the oral bioavailability and bioaccessibility of lead particles. *Chemistry Central Journal, 6,* 138.

Dooyema, C. A., Neri, A., Lo, Y. C., Durant, J., Dargan, P. I., Swarthout, T., . . . & Brown M. J. (2012). Outbreak of fetal childhood lead poisoning related to artisanal gold mining in Northwestern Nigeria, 2010. *Environmental Health Perspectives, 120,* 4.

Faul, F., Erdfelder, E., Lang, A.G., & Buchner, A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39,* 175-191.

Ferrie, F. P., Rolf, K., & Troesken, W. (2012). Cognitive disparities, lead plumbing, and water chemistry: prior exposure to water-borne lead and intelligence test scores among World War Two U.S. Army enlistees. *Economics & Human Biology, 10*(1), 98-111.

Finkelstein, Y., Markowitz, M. E., & Rosen, J. F. (1998). Low-level lead-induced neurotoxicity in children: an update on central nervous system effects. *Brain Research Reviews, 27,* 168-176.

Forns, J., Fort, M., Casas, M., Caceres, A., Guxens, M., Gascon, M., . . . & Sunyer, J. (2014). Exposure to metals during pregnancy and neuropsychological development at the age of 4 years. *NeuroToxicology, 40,* 16-22.

Horn, J. L. (1968). Organization of abilities and the development of intelligence. *Psychology Review, 75,* 242-259.

Hou, S., Yuan, L., Jin, P., Ding, B., Qin, N., Li, L., . . . & Deng, Y. (2013). A clinical study of the effects of lead poisoning on the intelligence and neurobehavioral abilities on children. *Theoretical Biology and Medical Modeling, 10,* 1-9.

Houston, S. M., Herting, M. M., & Sowell, E. R. (2014). The neurobiology of childhood structural brain development: concept through adulthood. Andersen, S. L., Pine, D. S. The *Neurobiology of Childhood* (3-17), Dordrecht London: Springer Heidelberg New York.

Huang, J., Huang, K., Shang, L., Wand, H., Zhang, M., Fan, C. L., . . . & Xiong, K. (2012). Chronic lead exposure reduces doublecortin-expressing immature neurons in young adult guinea pig cerebral cortex. *BMC Neuroscience*, *13*, 2-10.

Izenberg, N. (1995-2018). About the Apgar score. Retrieved from *http://kidshealth.org/en/parents/apgar.html*

Jakubowski, M. (2011). Low-Level environmental lead exposure and intellectual impairment in children – the current concepts of risk assessment. *International Journal of Occupational Medicine and Environmental Health*, *24*(1), 1-7.

Jurewicz, J., Polanska, K., & Hanke, W. (2013) Exposure to widespread environmental toxicants and children's cognitive development and behavioral problems. *International Journal of Occupational Medicine and Environmental Health*, *26* (2), 185-204.

Kan, K. J., Kievit, R. A., Dolan, C., & van der Maas, H. (2011). On the interpretation of the CHC factor Gc. *Intelligence*, *39*, 292-302.

Kim, M., & Kim, K. (2011). Biomonitoring of lead and cadmiun in the hair and fingernails of elderly Korean subjects. *Biological Trace Element Research*, *143*, 794-802.

Laidlaw, M.A.S., Filippelli, G.M., Sadler, R.C., Gonzales, C.R., Ball, A.S., & Mielke, H.W. (2016). Children's blood lead seasonlity in Flint, Michigan (USA), and soil-sourced lead hazard risks. International Journal of Environmental Research and Public Health, *13*, 358.

Li, H. W., Deng, J. G., Du, Z. C, Yan, M. S., Long, Z. X., Thi, P. T. P., & Yang, K. D. (2013). Protective effects of Mangiferin in subchronic developmental lead-exposed rats. *Biological Trace Element Research*, *152*, 233-242.

Lichtenberger, E. O., & Kaufman, A. S. (2009). Essentials of WAIS-IV Assessment (pp 29-38). Location: Hoboken, New Jersey.

Lidsky, T. I., & Schneider, J. S. (2006). Adverse effects of childhood lead poisoning: the clinical neuropsychological perspective. *Environmental Research*, *100*, 284-293.

Liu, J., Li, L., Wang, Y., Yan, C., & Liu, X. (2013). Impact of low blood lead concentrations on IQ and school performance in Chinese children. *PLOS ONE*, *8*(5), 1-9.

Lu, X., Jin, C., Yang, J., Liu, Q., Wu, S., Li, D., Guan, Y., & Cai, Y. (2013). Prenatal and lactational lead exposure enhanced oxidative stress and altered apoptosis status in offspring rats' hippocampus. *Biological Trace Element Research*, *151*, 75-84.

Lucchini, R. G., Zoni, S., Guazzetti, S., Bontempi, E., Micheletti, S., Broberg, K., Parrinello, G., & Smith, D. R. (2012). Inverse association of intellectual function with very low blood lead but not with manganese exposure in Italian adolescents. Environmental research, *118*, 65-71.

Mankiewicz, A. F. (2011). Adult Careers: Does childhood IQ predict later life outcome? *Journal of Policy and Practice in Intellectual Disabilities*, *8*, 1, 1-9.

Mazumdar, M., Bellinger, D.C., Gregas, M., Abanilla, K., Bacic, J., &Needleman, H. L. (2011). Low-level environmental lead exposure in childhood and adult intellectual function: a follow-up study. *Environmental Health*, *10*(24), 1-7.

Mazumdar, M., Xia, W., Hofmann, O., Gregas, M., Sui, S. H., Hide, W., Yang, T. ... & Bellinger, D. C. (2012). Prenatal lead levels, plasma amyloid β levels, and gene expression in young adulthood. *Environmental Health Perspectives*, *120*, 702-707.

McGrew K. S. (2009). CHC theory and the human cognitive abilities project: Standing on the shoulders of the giants of psychometric intelligence research. *Intelligence, 37,* 1-10.

Menezes-Filho, J. A., de Sousa Viana, G. F., & Paes, C. R., (2012). Determinants of lead exposure in children on the outskirts of Salvador, Brazil. *Environmental Monitoring and Assessment*, *184*, 2593-2603.

Miranda, M. L., Edwards, S. E., Swamy, G. K., Paul, C. J., & Neelon, B. (2010). Blood Lead Levels Among Pregnant Women: Historical Versus Contemporaneous Exposures. *International Journal of Environmental Research and Public Health*, *7*, 1508-1519.

Nisbett, R. E., Aronson, J., Blair, C., Diskens, W., Flynn, J., Halpern, D. F., & Turkheimer, E. (2012). New findings and theoretical developments. *American Psychologist*, *67*, 2, 130-159.

Pourmand, A., Al-tiae, T. K., & Mazer-Amirshahi, M. (2012). Perspective on lead toxicity, a comparison between the United States and Iran. *Journal of Pharmaceutical Sciences*, *20*, 70.

Ransden, S., Richardson, F. M., Josse, G., Thomas, M. C., Ellis, C., Shakeshaft, C., Seghier, M. L., & Price, C. J. ((2011). Verbal and non-verbal intelligence changes in the teenage brain. *Nature*, *479*, 113-116.

Rao Barkur, R., Rao, M. S., & Bairy, L. K.(2011). Low lead exposure during foetal and early postnatal life impairs passive avoidance learning in adulthood in rats. *Arh Hig Rada Toksikol*, *62*, 147-153.

Reynolds, M.R., Keith, T.Z., Flanagan, D.P., & Alfonso, V.C. (2013). A cross-battery, reference variable, confirmatory factor analytic investigation of the CHC taxonomy. *Journal of School Psychology*, *51*, 535-555.

Roper, W.L., Houk, V. N., Falk, H., Binder, S. (1991, October 1). Preventing Lead Poisoning in Young Children: Chapter 7. Retrieved from http://www.cdc.gov/nceh/lead/publications/books/plpyc/Chapter7.htm

Sanchez-Martin, F. J., Fan, Y., Lindquist, D. M., Xia, Y., & Puga, A. (2013). Lead induces similar gene expression changes in brains of gestationally exposed adult mice and in neurons differentiated from mouse embryonic stem cells. *PLoS ONE*, *8*, 1-13. Retrieved from www.plosone.org.

Sanders, A. P., Flood, K., Chiang, S., Herring, A. H., Wolf, L., & Fry, R. C. (2012). PLoS ONE, *7*,1-7. Retrieved from www.plosone.org.

Searle, A. K., Baghurst, P. A., van Hooff, M., Sawyer, M. G., Sim, M. R., Galletly, C., Clark, L. S., & McFarlan A. C. (2014). Tracing the long-term legacy of childhood lead exposure: A review of three decades of the Port Pirie cohort study. *NeuroToxicology* http://dx.doi.org/10.1016/j.neuro.2014.04.004.

Spanier, A. J., Wilson, S., Ho, M., Hornung, R., & Lanphere, B. P. (2013). The contribution of housing renovation to children's blood lead levels: a cohort study. *Environmental Health*, *12*, 1-8.

Taylor, M. P., & Schniering, C. (2010). The public minimization of the risks associated with environmental lead exposure and elevated blood lead levels in children, Mount Isa, Queensland, Australia. *Archives of Environmental & Occupational Health*, *65*, 45-48.

Tusing M. E. & Ford, L. (2004). Examining Preschool Cognitive Abilities using a CHC framework. *International Journal of Testing*, *4*, 91-114.

van der Kuijp, T. J., Hunag, L., & Cherry, C. R., (2013). Health hazards of China's lead-acid battery industry: a review of its market drivers, production processes, and health impacts. *Environmental Health*, *12*, 1-10.

Ward, L. C., Bergman, M. A., & Hebert K. R. (2011). WAIS-IV Subtest Covariance Structure: Conceptual and Statistical Considerations. *Psychological Assessment*, *24*, 2, 328-340.

Wechsler, D., Coalson, D. L., & Raiford, S. E. (2008). Technical and Interpretive Manual. In Wechsler Adult Intelligence Scale – Fourth Edition (pp. 1-5). Location: Pearson.

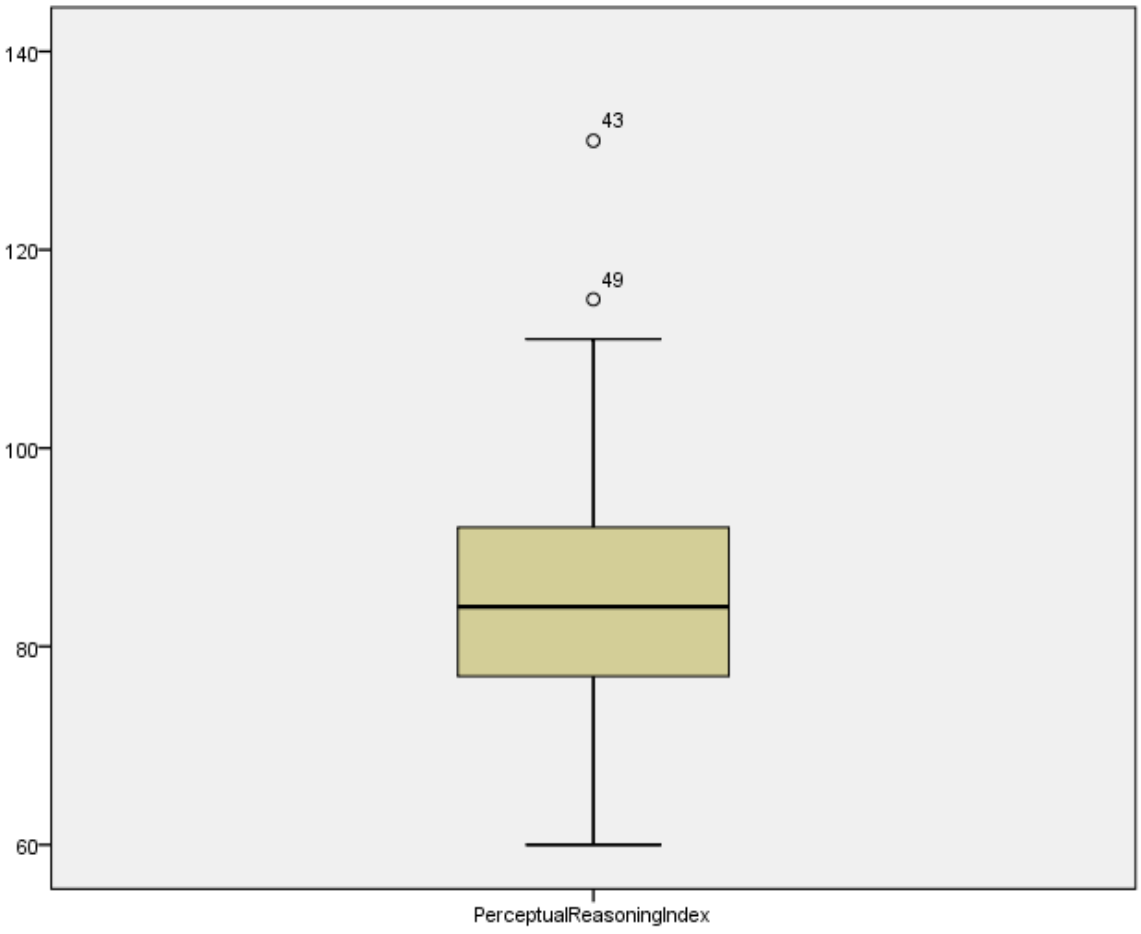
Weiss, L. G., Saklofske, D. H., Coalson, D. L., & Raiford, S. E., (2010). Theoretical, empirical and clinical foundations or the WAIS-IV index scores. WAIS-IV Clinical Use and Interpretation (pp. 61-91). Location: Academic Press.

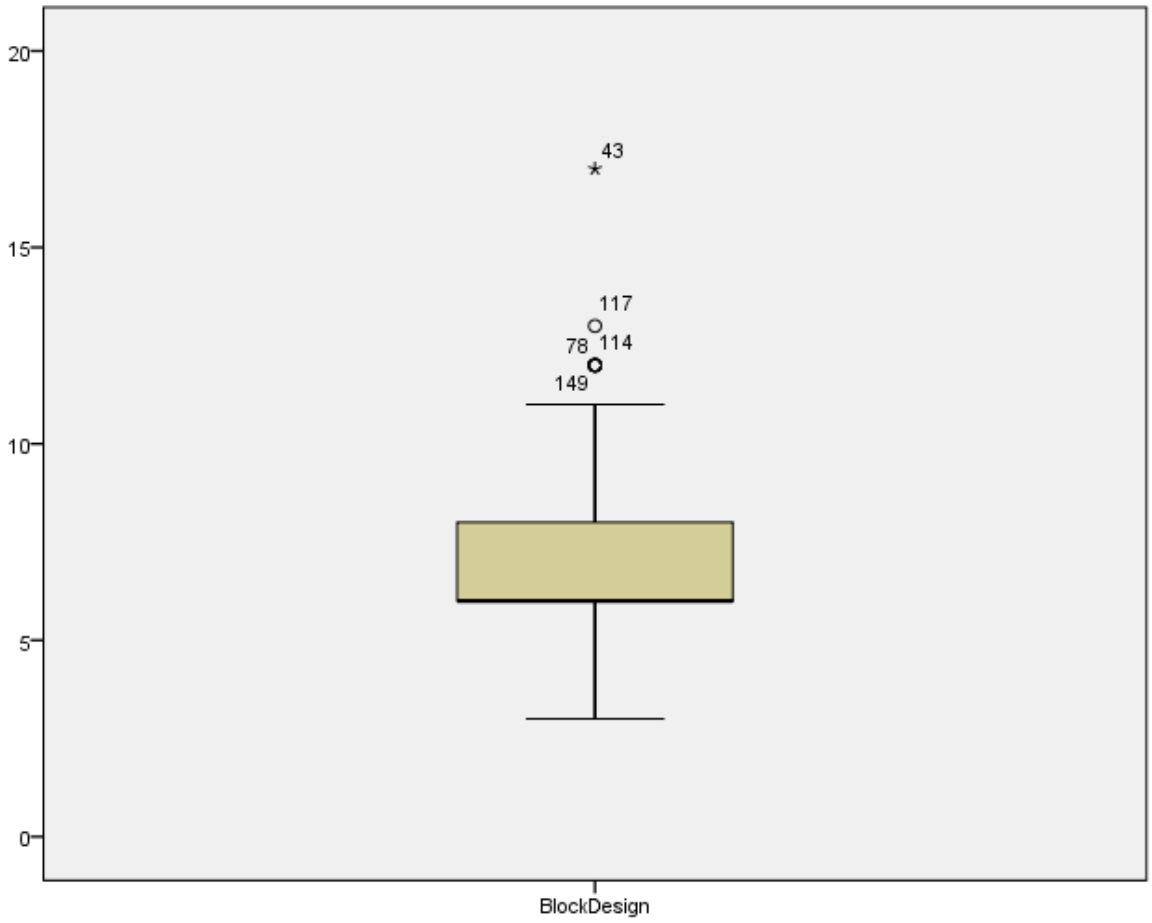
World Health Organization (2014, October). Lead poisoning and health. Retrieved from http://www.who.int/mediacentre/factsheets/fs379/en/.

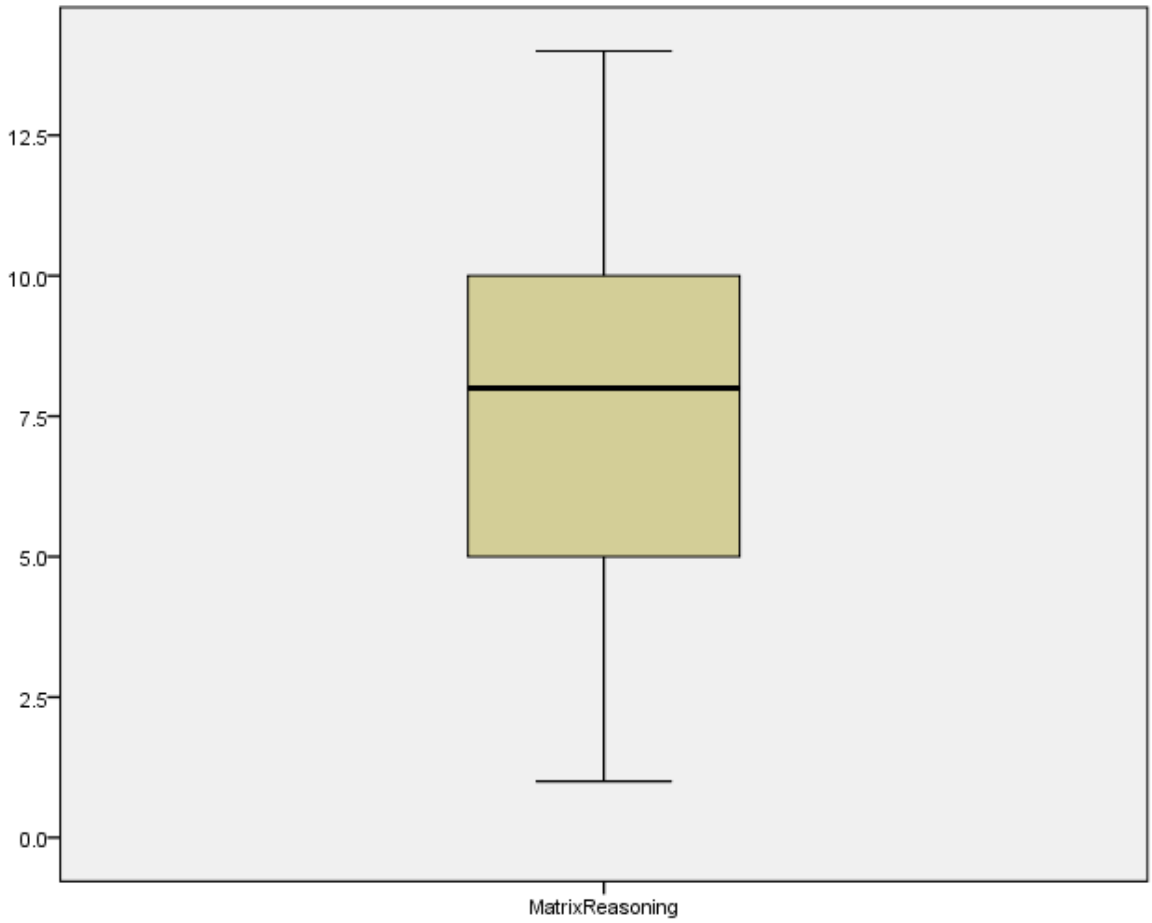
Yoshinaga, J. (2012). Lead in the Japanese living environment. *Environmental Health and Preventive Medicine*. *17*, 433-443.

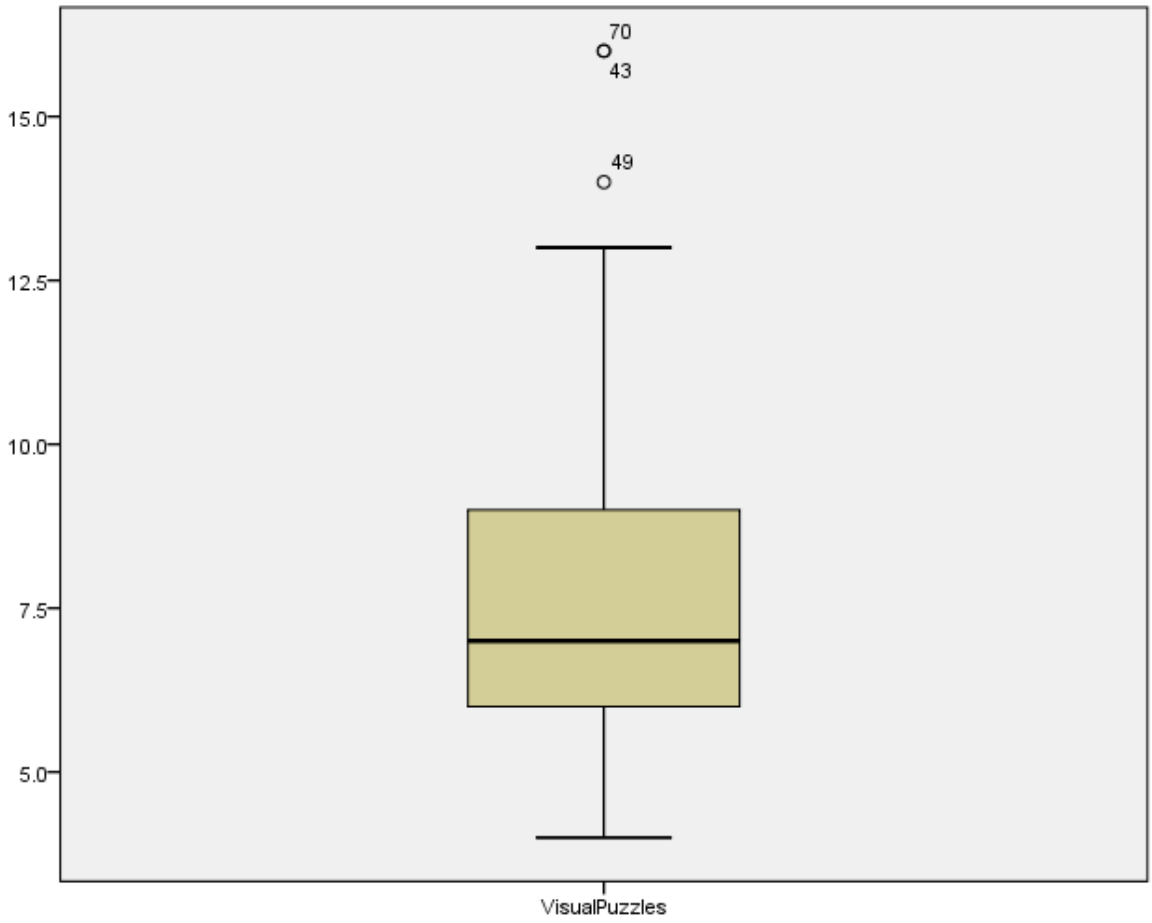
Yoshinaga, J., Takagi, M., Yamasaki, K., Tamiya, S., Watanabe, C., & Kaji, M. (2012). Blood lead levels of contemporary Japanese children. *Environmental Health and Preventive Medicine*. *17*, 27-33.

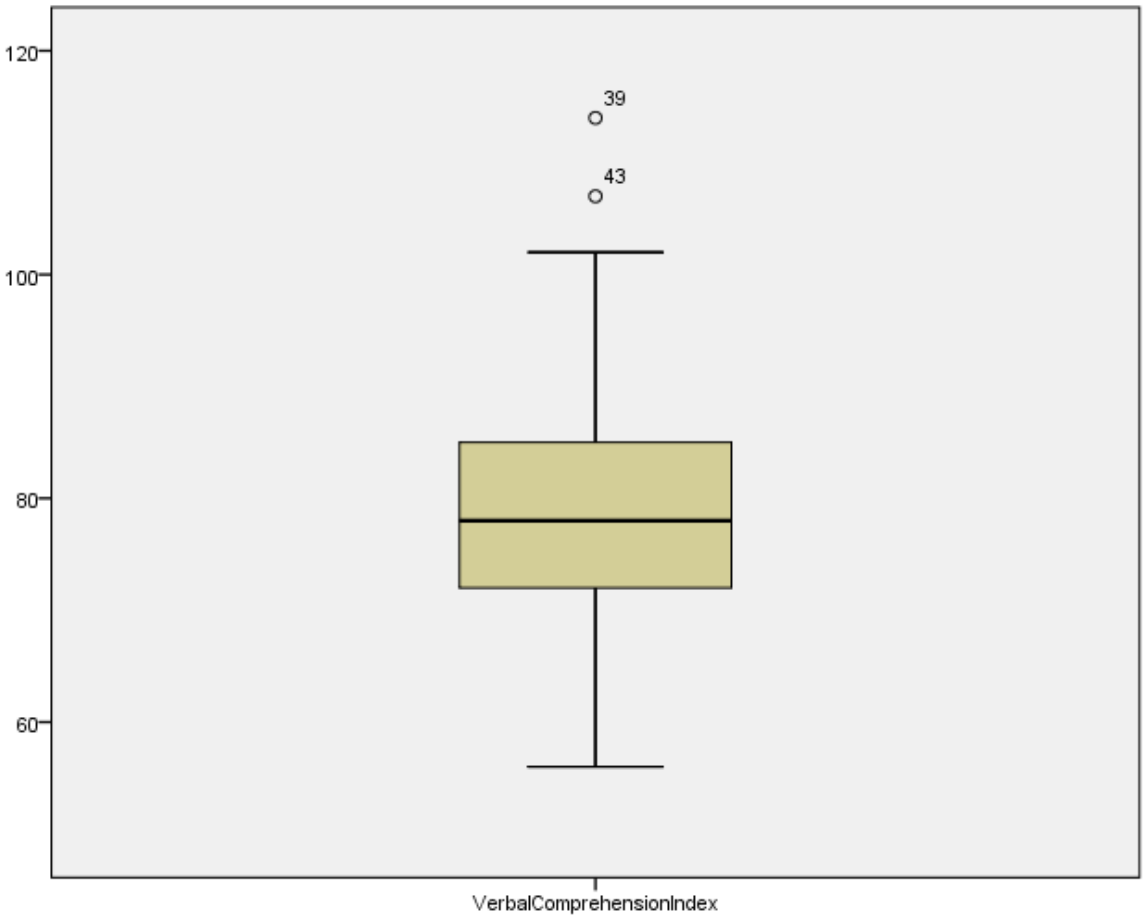
Appendix A: Box Plots

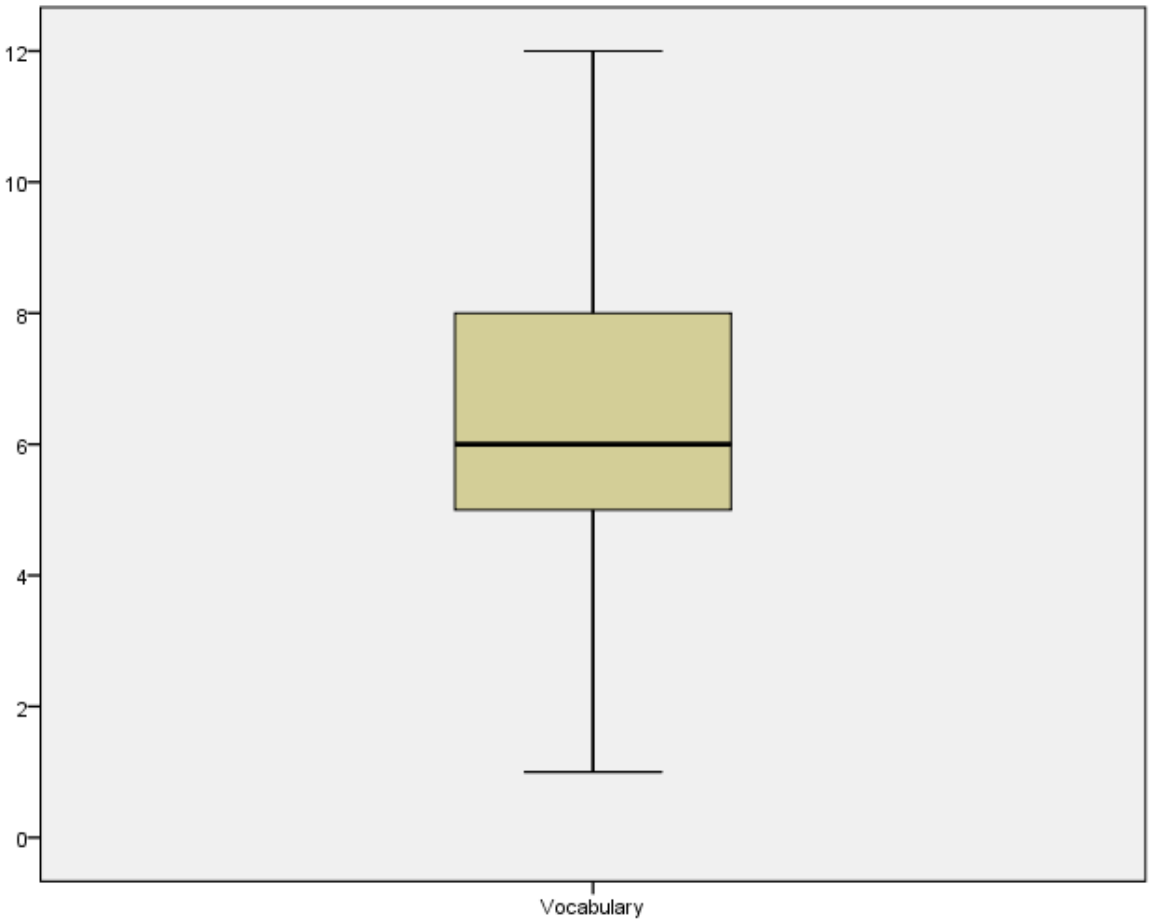


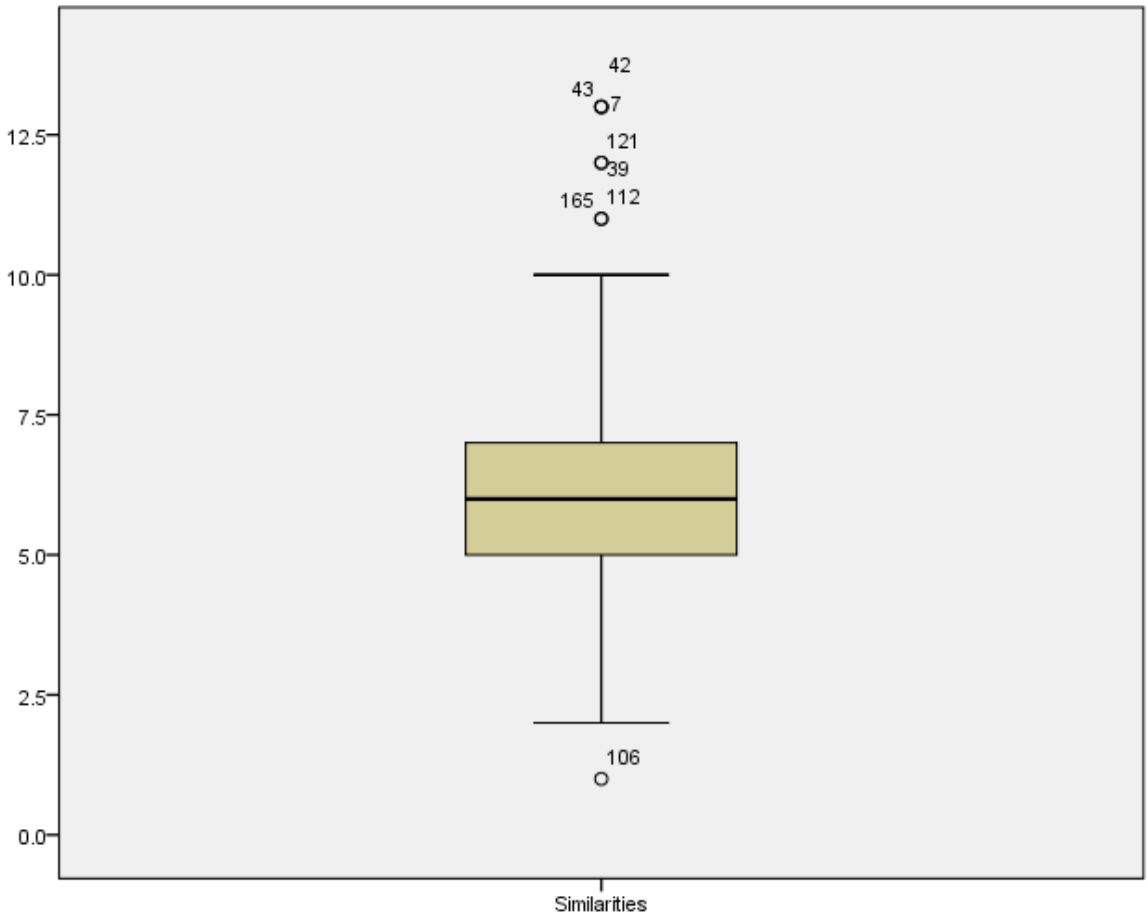

20
15
10
5
0
43
117
78
114
149
BlockDesign

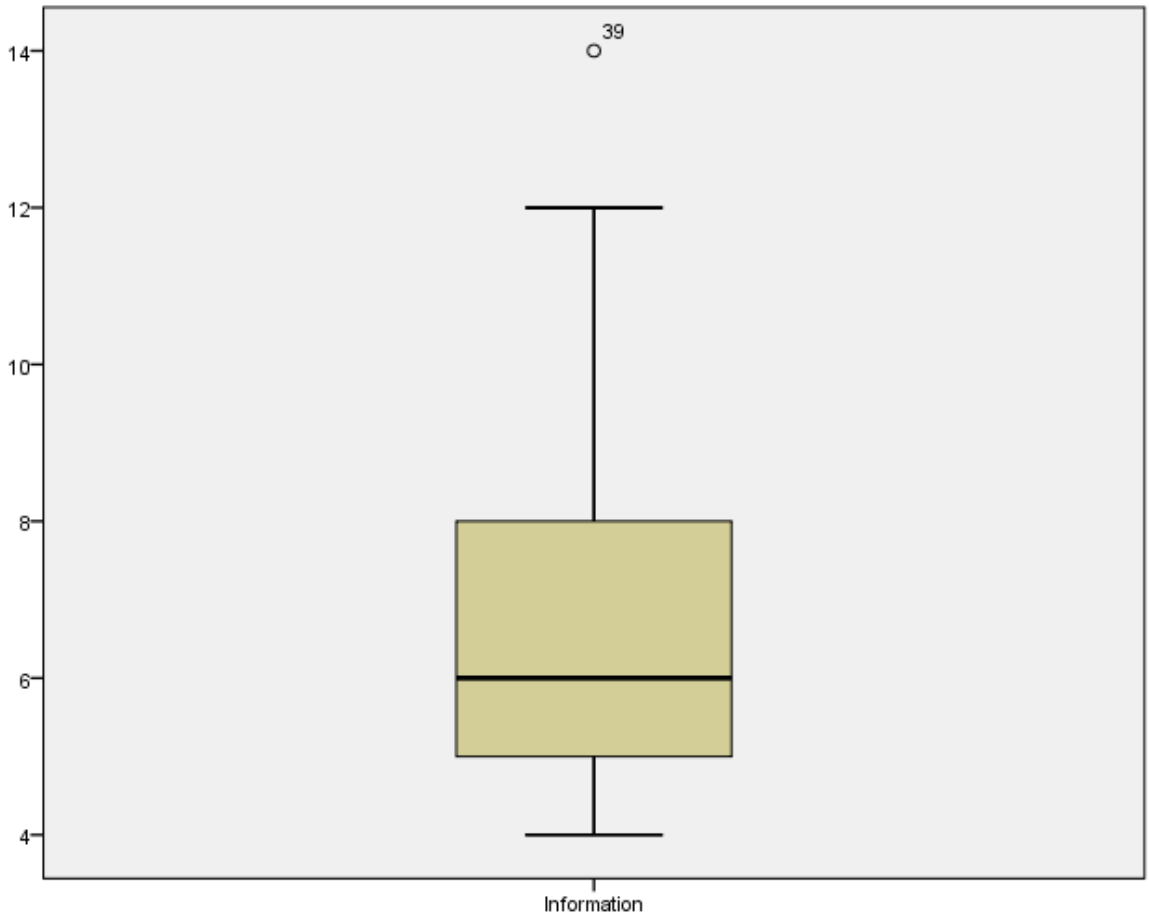

12.5
10.0
7.5
5.0
2.5
0.0
MatrixReasoning


70
43
49
15.0
12.5
10.0
7.5
5.0
VisualPuzzles

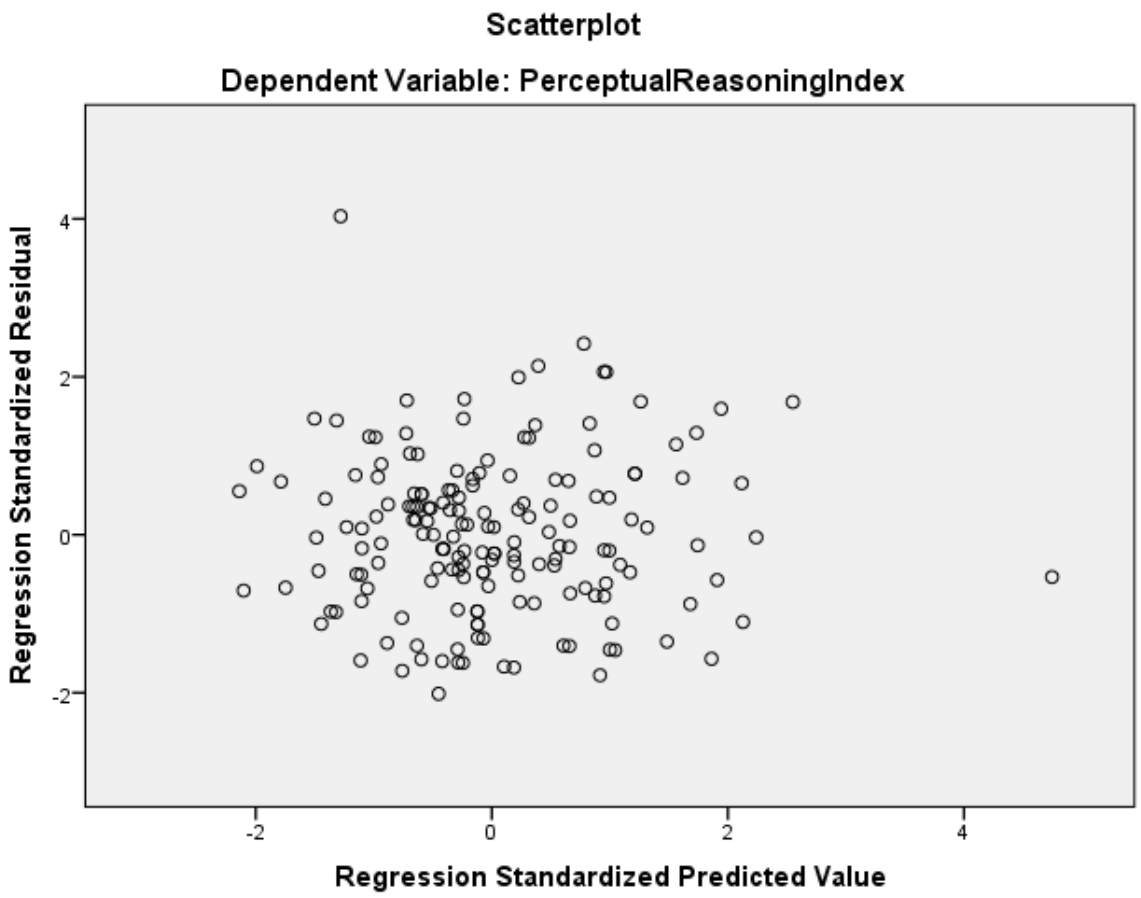

120
100
80
60
39
43
VerbalComprehensionIndex

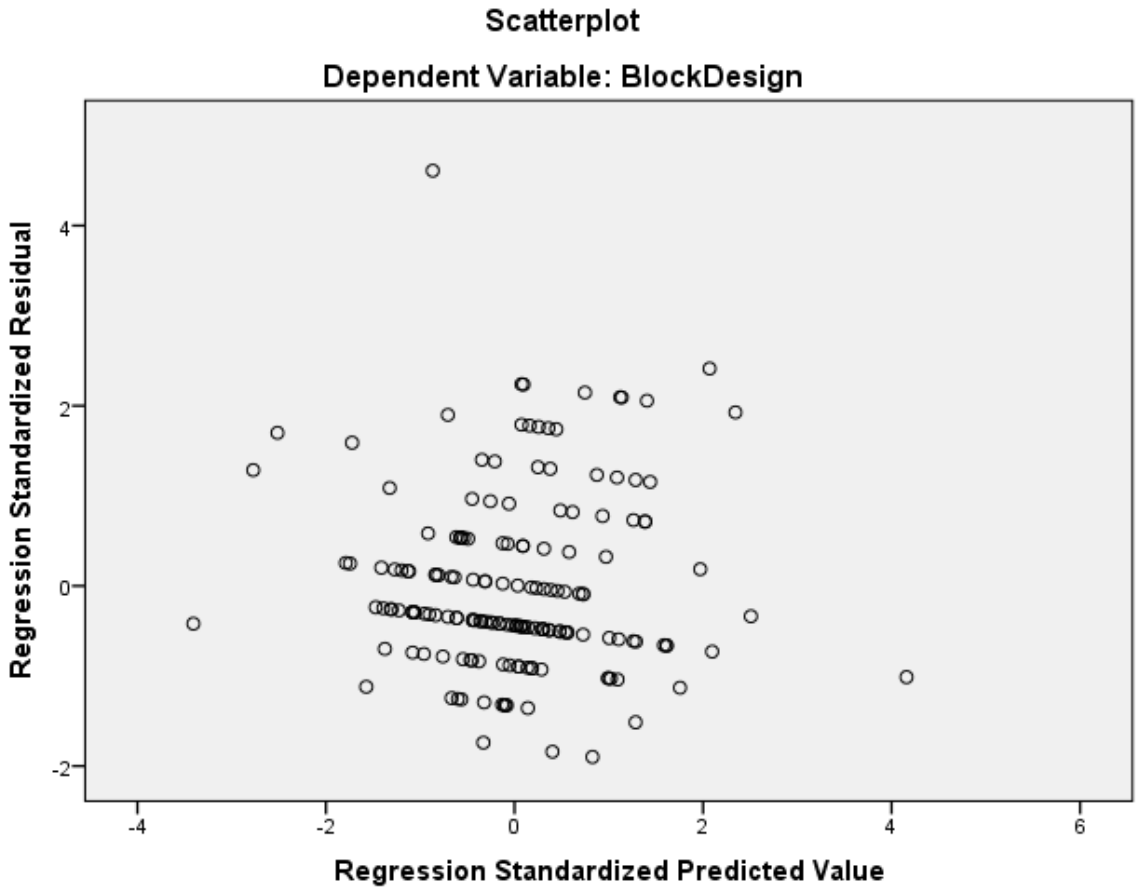

12
10
8
6
4
2
0
Vocabulary

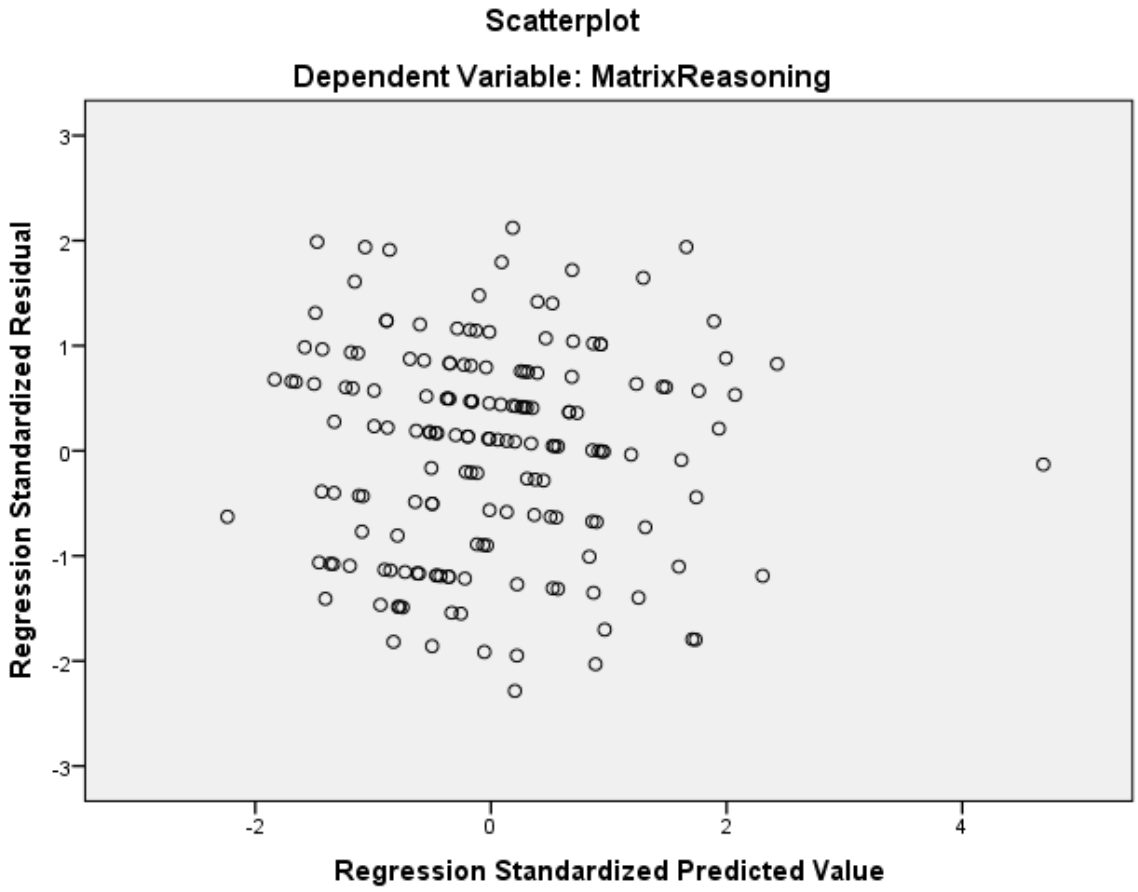

42
43
7
121
39
165
112
106
12.5
10.0
7.5
5.0
2.5
0.0
Similarities

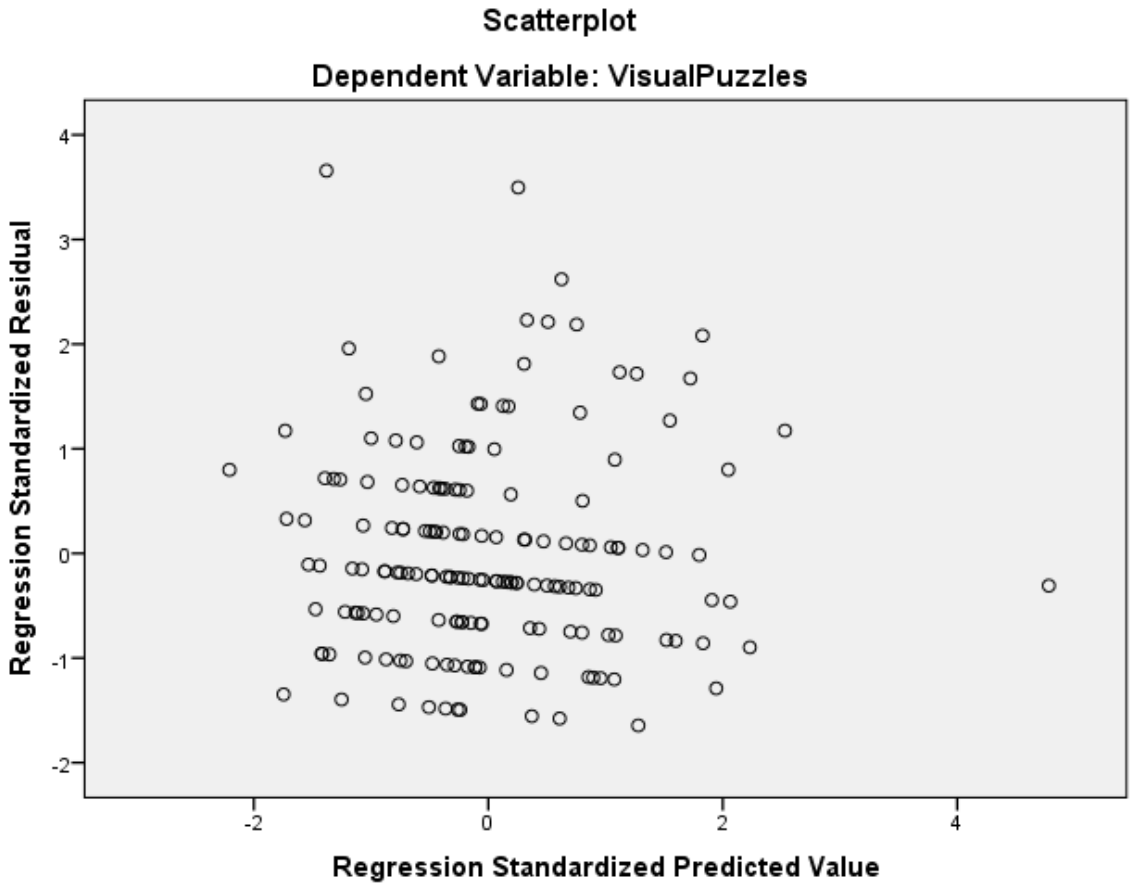

39
14
12
10
8
6
4
Information

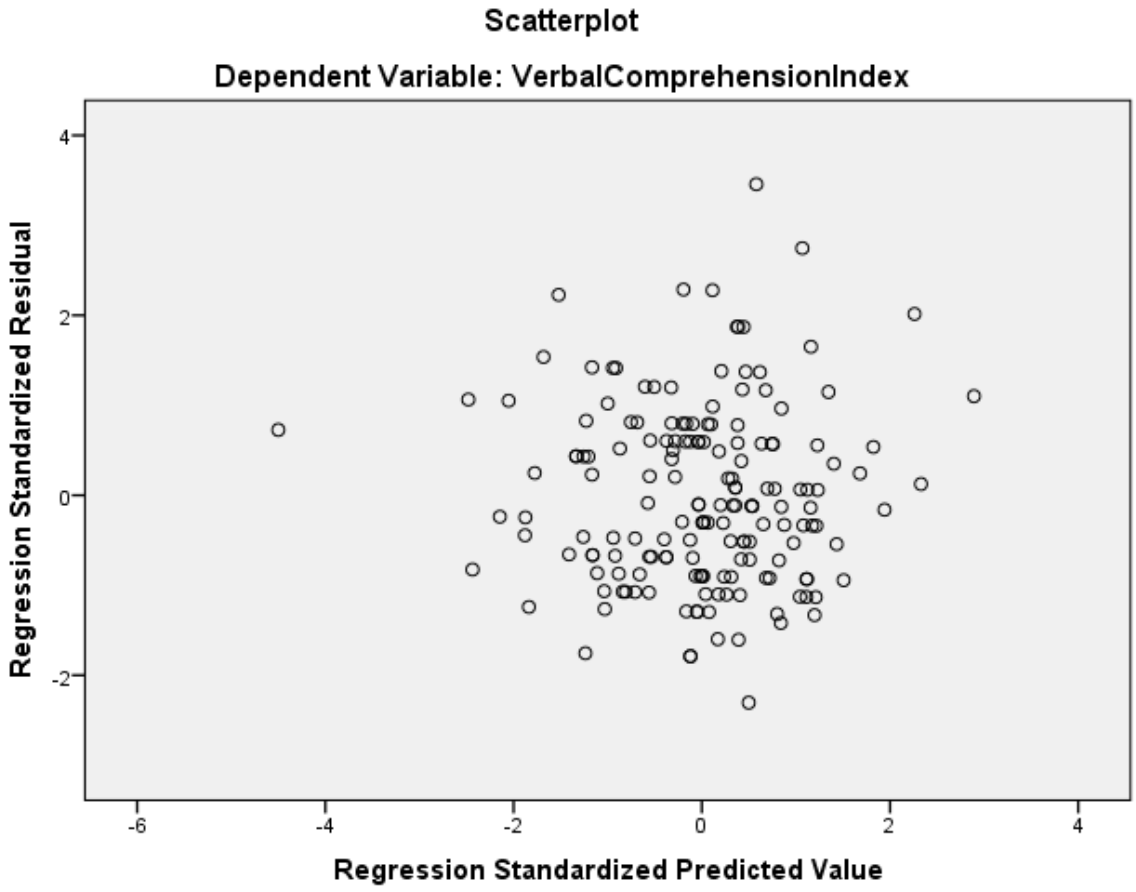
Appendix B: Assumptions of Heteroscedasticity

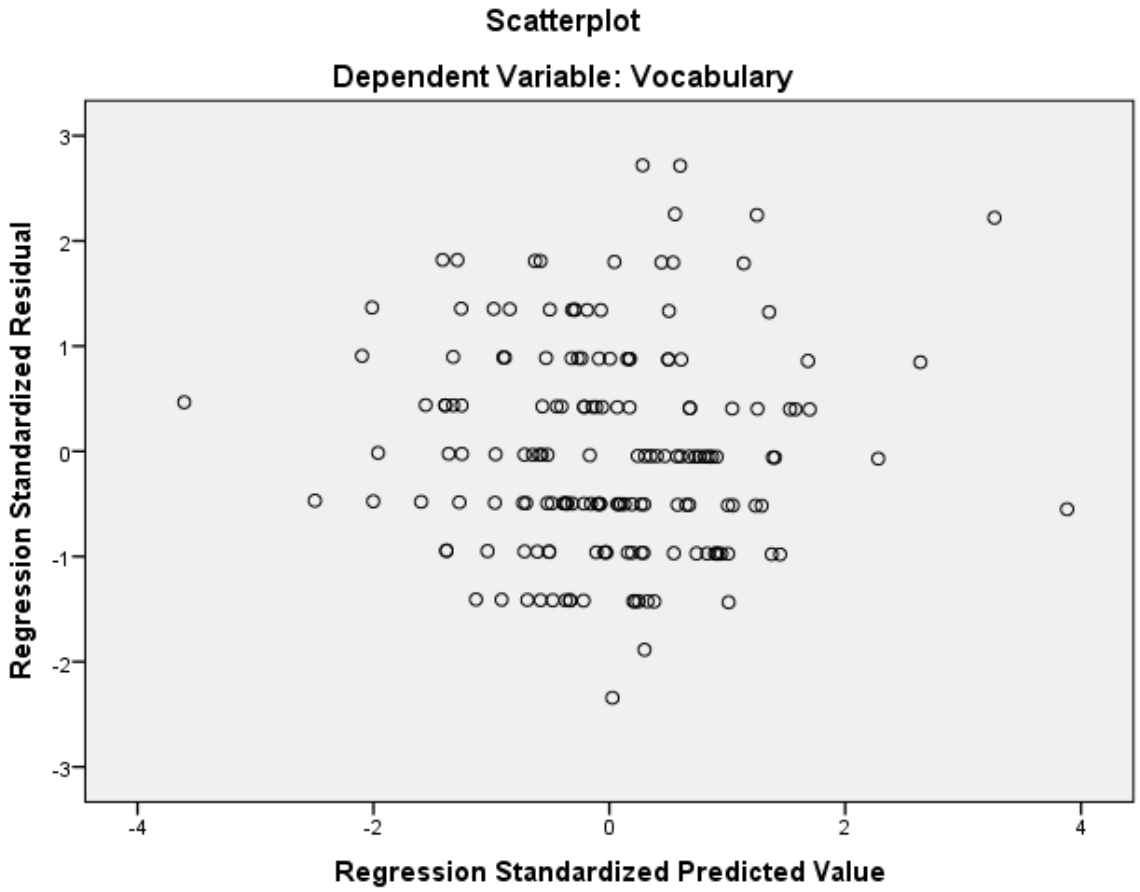


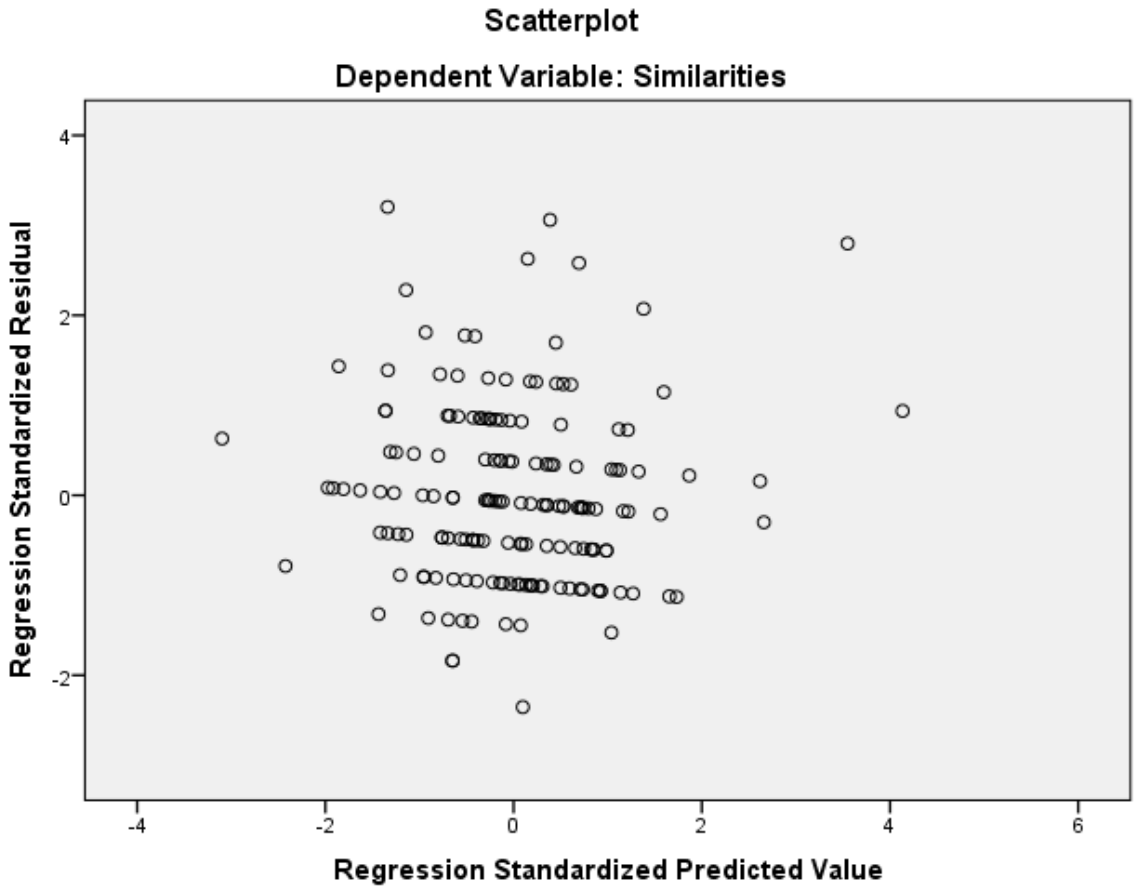

Scatterplot
Dependent Variable: BlockDesign
Regression Standardized Residual
Regression Standardized Predicted Value
4
2
0
-2
-4
-2
0
2
4
6

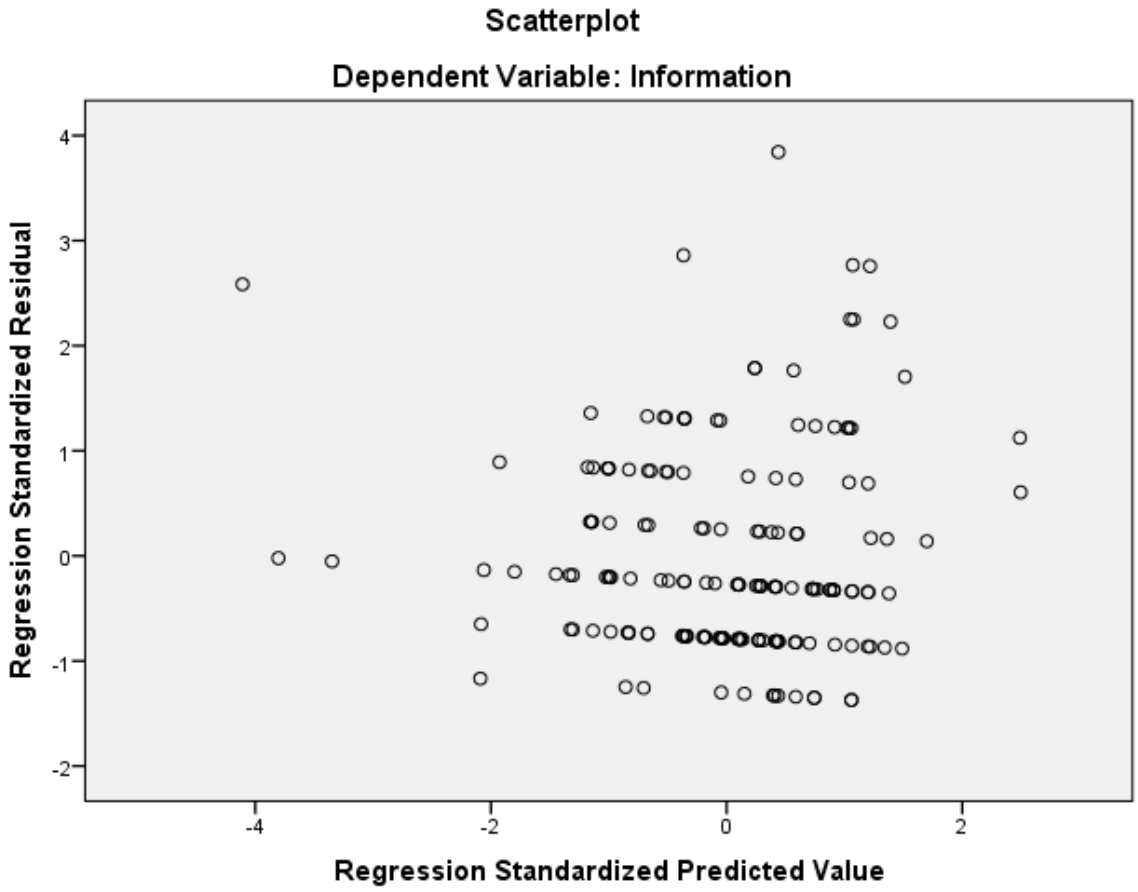

Scatterplot
Dependent Variable: MatrixReasoning
Regression Standardized Residual
Regression Standardized Predicted Value
3
2
1
0
-1
-2
-3
-2
0
2
4


Scatterplot
Dependent Variable: VisualPuzzles
Regression Standardized Residual
Regression Standardized Predicted Value
4
3
2
1
0
-1
-2
-2
0
2
4


Scatterplot
Dependent Variable: VerbalComprehensionIndex
Regression Standardized Residual
Regression Standardized Predicted Value
4
2
0
-2
-6
-4
-2
0
2
4


Scatterplot
Dependent Variable: Vocabulary
Regression Standardized Residual
Regression Standardized Predicted Value
3
2
1
0
-1
-2
-3
-4
-2
0
2
4


Scatterplot
Dependent Variable: Similarities
Regression Standardized Residual
Regression Standardized Predicted Value
4
2
0
-2
-4
-2
0
2
4
6


Scatterplot
Dependent Variable: Information
Regression Standardized Residual
Regression Standardized Predicted Value
4
3
2
1
0
-1
-2
-4
-2
0
2