The Role of Contralateral Movement in Boys with Attention Deficit Hyperactivity Disorder (ADHD)

Dissertation

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Abstract

This ex post facto study examined the role of contralateral movement abilities in boys with and without Attention Deficit Hyperactivity Disorder (ADHD) diagnosis and the subsequent impact of medication within the ADHD group to contralateral movement execution. The Ohio State University Scale of Intra-Gross Motor Assessment (OSU SIGMA) was used to rank contralateral movement ability as operationalized through the scale's skipping portion. Results indicate a significant difference on OSU SIGMA ranks between ADHD-diagnosed and non-ADHD diagnosed boys. Findings also suggest that medication does not elicit a significant impact on overall OSU SIGMA ranks. Survey analysis revealed that boys with ADHD diagnosis tended to crawl for longer durations, walk later, and participate less in sports, gymnastics, or dance. These preliminary findings provide a foundation for future studies into the efficacy of developmental contralateral movement interventions for the treatment of disorders such as ADHD.

This document is dedicated to my parents who always encouraged me to think creatively, and to my husband who has been by my side through its entire course.

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CHAPTER 1: INTRODUCTION

Background and Setting

This interdisciplinary study seeks to bridge conversational camps within the fields of anatomy, education, kinesiology, neuroscience, psychiatry, psychology, and somatics. Each field has a purview relating to how human movement plays a role in the dynamics of the human experience and the expression of the human psyche. This study attempts to integrate these varied breadths of scope in an effort to diversify and make more holistic the understanding of and excepted treatment for disorders wherein developmental movement abilities and cognitive abilities both seem impaired along similar trajectories. Specifically, this goal is applied to Attention Deficit Hyperactivity Disorder (ADHD), which holds functional applications across all of these fields and has been an area of research within each discipline. A broader, more interdisciplinary dialogue could pave the way toward novel approaches for clarifying diagnoses, complementing current treatment strategies, and better understanding the heterogeneous population of individuals suffering with ADHD.

Human movement is necessarily an interdisciplinary area of focus. From medicine to the fine arts and from education to architecture, the human movement

experience permeates nearly all niches in life, as well as academia. Each discipline hones its interest in movement uniquely, and there is often a paucity of cross-communication between academics and bodies of literature in diverse fields. This lack of interdisciplinary dialogue leaves certain aspects of human movement and its impact on life experiences under-acknowledged, under-investigated, and misunderstood.

The two unifying constructs being investigated in this study are attentional ability and contralaterality of movement. Of the two, attention is the more broadly understood. William James, in his 1890 work *Principles of Psychology*, stated:

> "Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, one out of what seem several simultaneously possible objects or trains of thought. Focalization, concentration of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others, and is a condition which has a real opposite in the confused, dazed, scatterbrained state which in French is called distraction, and Zerstreutheit in German."

Contralateral movement is less widely apprehended and differentially defined. In the field of somatics, contralaterality represents the conglomerate of smaller movements that create diagonals across the body as the movement of the upper limb on one side is coordinated with that of the lower limb on the opposite side. Contralateral movements often include crossing the anatomical midline of the body. Examples include crawling, walking, running or marching when swinging opposite arm to leg, and skipping. In somatic studies and the dance/movement and music communities, the importance of developmental movement acquisition and fluid contralaterality has been studied and applied since the 1960s (Bartenieff, 1998; Dennison, 1989; Feldenkrais, 1977; Fitt, 1996; Godfrey, 1969). The somatic perspective states that coordination of contralateral movement requires seamless integration of sensory and motor systems, and

when malfunctions in contralateral fluidity occur they cause serious deterioration to the individual (Dennison, 1998; Hanna, 1988). These suppositions are grounded namely in phenomenological endeavors and case studies.

In neuroscience, anatomy, and kinesiology, contralateral behaviors are viewed in a brain-based manner clarifying movements as being largely coordinated by the contralateral, or opposite, side of the brain. These definitions share only the meaning of contralateral as being "opposite" as in opposite arm to leg or opposite side of the body from the primary motor areas of the brain. The Western medical definition is applied without any interest or premise relating to the developmental or foundational importance of contralaterality as a larger movement phenomenon. In psychology and psychiatry the word "contralateral" and the concepts associated with this type of movement are absent from the clinical scope and literature. In education there have been waves of interest in the somatic conglomerate definition of developmental movement and its relationships to learning. There have been few peer-reviewed efforts to isolate the role of contralaterality in learning potential or any scientific evidence reaching statistical significance indicating that contralateral ability is correlated with attention (Goswami, 2006; Hyatt, 2007).

The interdisciplinary disparity of perceived importance placed upon contralateral abilities is clear. Importance of these differential definitions lies in practical implications to individuals suffering with disorders marked by contralateral movement deficits. Patients with movement disorders do not usually seek out an educator or a dance class for therapy. They go to their Western medical provider for diagnosis and

treatment. If the somatic definition of contralaterality could become more broadly incorporated into Western medical education, research, and practice, patients would likely be offered a more diverse and holistic range of therapeutic interventions or, minimally, community referrals to complement their current treatment regimens.

One of the more frequently used somatic, movement-centered methods implemented in classrooms and by community practitioners is Educational Kinesiology, which was copyrighted as *Brain Gym* by Dennison. The word "education" derives from the Latin "educare," meaning 'to draw out'; the word "kinesiology" derives from the Greek word kinesis or 'motion.' Educational kinesiology is an emerging field grounded in the idea that movement can be used as a tool for drawing out learning potential in individuals of all ages. A core premise of educational kinesiology is 'laterality.' Educational kinesiologists work to enhance efficiency of constructs underlying learning, such as attention, focus, anxiety, and motivation by facilitating communication within the nervous system through contralateral movements that require bilateral and coordinated activation of the brain (Dennison, 1989). The methods applied in educational kinesiology work to enhance contralateral movement abilities with the goal of improving classroom-related skills.

The implication of educational kinesiology is that individuals can make themselves more efficient cognitively by working to gain physical awareness and efficiency. *Brain Gym* is a specific area of educational kinesiology that uses many tailored physical activities to aid in the preparation of and transition between learning situations. These activities are simple, usually fun for children, and intended to be

energizing. Specific activities include the cross-crawl, or marching in place with strong contralateral upper limb movement; Lazy-8s, or drawing large horizontal figure-8 shapes using both hands; Double Doodle, or doodling using both hands simultaneously; Alphabet-8s, or drawing the alphabet, alternating letters within each side of a horizontal figure-8 shape; the Elephant, which looks like a Lazy-8 but the shape is traced with body moving through space instead of drawn; and simple neck rolls (Dennison, 1989). Many educators from the elementary to the high school level are becoming certified as *Brain Gym* educators due to the ease of implementing *Brain Gym* in the classroom (Hyatt, 2007).

Educational kinesiology expands from three premises: 1) learning is a natural activity that continues throughout life, 2) "learning blocks," or any barriers to efficient learning, are the inability to move through stress and uncertainty in a new task, and 3) we are all "learning blocked" to the extent that we have not learned to move. It is this final assumption that begs an interesting question and sets the premise for this investigation. It has not been tested to a degree that it can be stated as fact and thus remains a theoretical and unfounded premise to support educational kinesiology. Globally and pertaining specifically to contralaterality, this correlation between impaired learning ability and developmental movement patterning is weakly documented. Furthermore, the lack of acknowledgment for the somatic definition of contralaterality on the part of Western medical literature makes it moot to directly study methods, such as those used in *Brain Gym*, because no scientific premise for a somatically defined contralateral deficit in individuals with attentional problems currently exists.

In the somatic community, there seems to be an unsubstantiated notion that development of contralateral movement ability and attention are explicitly interwoven. In the Western medical community, there seems to be unsubstantiated notions that motor development begins early and ends early, while cognitive development begins later and progresses on a different trajectory (Diamond, 2000). Research has shown that attentional skills and self-regulation emerge around the end of the first year of life and continue to mature toward the preschool and elementary school years. The first attention system is operational from as early as three months and consists of orienting and exploring behaviors. The second attention system sets in around 18 months and continues to be enhanced for around four years. This establishes that the attentional system is roughly in place by the age of six years (Reebye, 2009).

It can be seen through evaluation of traditional developmental milestones and somatic literature on developmental movement patterning that movement abilities are forming in tandem to support these attentional systems (Anderson, 1998; Brook, 2001; Goddard, 2005; Hartley, 1995; Todd, 1977). To support the orienting and exploring behaviors of the first attentional system, a three-month old child will bring hands to the mouth, reach for and grasp objects, visually follow moving objects, and turn the head to orient to sounds. Hanna (1996) argues that both the attentional and motor systems will begin to deteriorate in function to the extent that movements cannot support the attentional system. By the age of 18 months, children have moved through all of the prone and supine variations of developmental movement patterns and can usually walk without any help and run stiffly. These activities mark the beginning of the

developmental locomotor movement patterns, or ambulatory movement patterns, which continue enhancement for approximately four more years establishing full locomotor acquisition by around the age of six (Frego, 1998; Wellman, 1938).

Drawing from Western medical literature, reviewed by Diamond and Reebye, and somatic literature, summarized by Frego and Wellman, it can be seen that attentional systems and developmental motor acquisition do develop along similar chronological trajectories. This chronological similarity is not enough to conclude that the development of one system necessarily correlates or impacts the development of the other. The supposition that attention and movement are functionally linked comes solely from the somatics literature, which is mostly comprised of case studies, naturalistic observation, and phenomenological inquiry.

The original intent of this investigation was to apply *Brain Gym* techniques to ADHD children and evaluate the impact of these movement-based interventions to traditional ADHD assessment tools and attentional instruments. However, such an investigation would be premature and cannot be launched without tremendous theoretical gaps. As already stated, educational kinesiology makes a key assumption that diminished contralateral coordination is at the core of blocks, or obstacles, to attention and learning. Before studying the methods of educational kinesiology specifically, it is of scientific importance to theoretically back up and evaluate whether or not this assumed contralateral deficit is, in fact, present in children with known learning and attentional problems such as those seen in ADHD.

The exploration into ADHD and developmental movement began in 2001 at a community psychological center housing the private practices of various mental health and somatic-based practitioners. Adults and children were seen at the facility for psychiatric and psychological counseling, and somatic therapies. Developmental movement patterning was one of the somatic modalities offered by the center. During these sessions, an analyst guides an individual through the developmental movement pattern sequence in an effort to realize which patterns can be easily coordinated upon command and which patterns seem difficult to initiate or absent from the individual's movement repertoire. Developmental movement patterns progress from simple navel radiation to mouthing then spinal activation followed by homologous movements (superior - inferior), homolateral movements (right side - left side), and finally to coordination of contralateral movement patterns. Interestingly, those with prior ADHD diagnoses generally displayed a marked deficit in both initiation and ease of the contralateral movements.

To review, contralateral movement refers to any movement coordinating opposite sides of the body and often crossing the anatomical midline. An example of a contralateral movement would be marching in place, and using opposite arm to leg motions while marching. Accordingly, the verbal command to these individuals for the contralateral observation was "march in place." The first movement often observed in the ADHD individuals was a marching in place with the upper limbs statically held at the side. With further prompting of "move both your arms and legs to march in place," this group of patients showed an ipsilateral, or same-sided, movement preference, if not

total dominance, for the activity. Proximity to midline, crossing of midline, contralateral 'hits' to ipsilateral 'misses,' and speed varied within the group, but remained relatively consistent within each individual. Individuals stated the intent to move opposite arm to leg but went on to exhibit asynchronous, delayed, and non-fluid attempts at the contralateral cross-crawl.

The most compelling aspect of this observation is that all individuals should be able to move with contralateral ease by the age of six years. This motor age was established by Wellman in 1938 and substantiated by Frego in 1998. The inability to elicit fluid contralateral movement beyond this documented motor age suggests a developmental motor patterning delay. When questioned about developmental milestones and movement history, the ADHD individuals frequently reported that they bypassed crawling, were generally clumsy, and considered themselves to be uncoordinated movers. In developmental movement patterning terms, they moved directly from homolateral reaching and pulling, demonstrating ipsilateral coordination, to standing alone indicating lack of supportive integration of the developmental movement patterns.

With these informal findings and knowledge of educational kinesiology, it appeared an interesting possibility to the clinicians that *Brain Gym* could serve as a possible nonpharmacological complementary intervention in the treatment of ADHD. In fact, books on the topic of educational kinesiology suggest a direct benefit to ADHD symptomatology with the incorporation of *Brain Gym* (Dennison, 1998; Hannaford, 1995). Once again, the major hypothetical caveat is that conglomerate developmental

contralaterality is not mentioned in the psychological, neuroscientific, or psychiatric literature pertaining to attention. Without a founded premise for a contralateral deficit within the ADHD population, the specific impact of educational kinesiology, or *Brain Gym* in particular, to ADHD symptoms cannot be rationally studied. In order to responsibly investigate modalities such as these, the question must be simplified. Is there a significant observable difference in contralateral movement ability between those with an ADHD diagnosis and those without ADHD? This is the primary research question driving this study.

Altered movement ability and attentional problems could represent two domains of the complex, heterogeneous disorder that ADHD is known to be. As is the case with many neurodevelopmental disorders, the symptoms may share no functional relationship at all. Even if this is of no functional relevance, the interest in better defining movement capabilities of children with ADHD still has important clinical value (Slaats-Willemse, 2005). Movement phenomena are assessed in current clinical practice and research but they are limited to homologous, homolateral, fine motor, oculomotor, and vestibular types of movement abilities and associated qualities.

It is known that contralateral movement chronologically enters the movement repertoire after both homologous and homolateral movement and locomotor patterns. Contralateral movement differs from manual dexterity and hand-eye coordination in the sense that these fine motor skills must be taught over time and require practice to master. Developmental movement patterns emerge on their own without any instruction or coaxing from external sources. Should a contralateral deficit be observed, it would be

fitting with the hypotheses of this study that oculomotor coordination and balance abilities be impacted to a similar degree as contralateral movement abilities. Oculomotor control is developed through similar areas of the brain as developmental movement patterns; both deal extensively with one's ability to coordinate the right and left hemispheres of the brain, both become nearly reflexive in execution once established, and both functionally demand a working relationship with appreciation of anatomical midline.

Educational kinesiology proponents state that other disorders present with contralateral movement abnormalities, but there would have to exist an assessment tool for contralaterality before that statement could be made definitively. ADHD is being used in this study because it is prevalent and neurodevelopmental. Other disorders that seem likely to present with definable contralateral deficits include cerebral palsy, Parkinson's disease, autism, traumatic brain injuries such as stroke, tumors, or head injury, and various apraxias. The somatic literature suggests that movement therapy incorporating developmental movement patterning may also prove beneficial for these disorders, regardless of whether they are developmental or acquired (Brook, 2001; Feldenkrais, 1977; Hanna, 1996; Hannaford, 1995; Hartley, 1998).

With cerebral palsy and certain apraxias being the primary exceptions, a key difference between many of these disorders and ADHD is in the neurodevelopmental nature of ADHD. It is a hypothesis of this study that ADHD children will display less mature developmental locomotor patterns while medicated and while unmedicated. The theory is that ADHD children do not move thoroughly and completely through the

developmental movement patterning process. In turn, these children find themselves neurodevelopmentally delayed in their movement ability. If a child never acquired contralateral movement patterns in their movement repertoire a medication should not improve their ability to execute such movement skills. Conditions with this characteristic hold a different therapeutic hope for facilitating the incorporation of these developmental skills into the individual's system and improving function. Disorders wherein the movement deficit results from injury or disease, such as Parkinson's disease, the damage is definitive and clearly understood. Parkinson's disease would be an interesting disorder in which to study contralateral movement because it shares very similar neuroanatomy with ADHD and can also involve the same executive functioning abilities impacted by ADHD.

The current range of Western medical movement assessments does not include measures to specifically address contralateral movement ability. This study aims to learn if there is a developmentally definable contralateral movement deficit associated specifically with ADHD. If one is observed, future studies can investigate the relationship of the movement phenomena to overall educational performance and to contralateral discrepancies found within other psychiatric conditions. Enhanced understanding of the movement profile associated with ADHD would be another tool in the difficult process leading to diagnosis and could also be beneficial toward unraveling epidemiological understanding.

ADHD is a prevalent, diversified, and varied disorder impacting children, adolescents, and adults. There are no biological tests to confirm a diagnosis of ADHD,

and so clinicians rely on psychometric rating instruments, interviews, parent/teacher participation, and exclusion of other disorders to make an accurate diagnosis of ADHD. The current version of the DSM-IV provides for 5 separate ADHD subtypes: primarily inattentive subtype (ADHD-PI), hyperactive impulsive subtype (ADHD-HI), combined subtype (ADHD-C), not otherwise specified subtype (ADHD-NOS), and ADHD in remission. The DSM-IV focuses primarily on the constructs of attention, impulsivity, and hyperactivity. There is an abundance of research into the pharmacotherapy, neuropsychology, and neuropsychiatry of attention, and many neurobiological models of ADHD exist, but the role and significance of the motor impairment seen with ADHD, and specifically developmental movement patterning, have received far less attention.

It is usually the case that children with motor problems have attentional deficits (Piek, 2004). In addition to the DSM-IV conditional problems of inattention, hyperactivity, and impulsiveness, 50% of children with ADHD will experience problems with motor output, fluency, flexibility, adjustment, inhibition, and preparation (Ben-Pazi, 2003; Slaats-Willemse, 2005; Tseng, 2004). Complex motor control tasks were found to be unstable and inaccurate in children that were later diagnosed with ADHD (Slatts-Willemse, 2005). Children with ADHD-PI have poorer fine motor skills, while ADHD-C children have more problems with gross motor activities. In Sweden, this combination has been termed Deficits in Attention, Motor control and Perception (DAMP), which is defined as a neurodevelopmental dysfunction syndrome with frequent psychological comorbidities. This high level of comorbidity within attentional

and motor disorders suggests that they may share a common neural mechanism (Piek, 2004).

As the behavioral diagnostic processes of ADHD have progressed, so has the knowledge of the psychobiology and pharmacology of the disorder. As the psychobiological picture of attention has presented areas such as the cerebellum and basal ganglia as part of the attentional process, the interest in the motoric implications and the role of gross and fine motor function involved within these brain regions and ADHD has also grown. Educators, clinicians, and researchers working with ADHD are all asking related questions about the relationship between cognitive and motoric efficiency within this disorder, but very few are scientifically researching developmental movement patterning or conglomerate contralaterality.

Conglomerate, contralateral movement is the most mature of the locomotor patterns to develop; it is the last motoric skill adapted by humans without being taught or otherwise facilitated. The ADHD literature currently features tests that measure homology, using upper limbs and/or lower limbs but not necessarily with any coordination between upper and lower limbs, or homolaterality, using right and left sides of the body independently without coordination between the sides. Measures using finger tapping, pegboards, and handwriting are implemented to evaluate fine motor skills. Frequently, balancing skills and sporting skills are evaluated to assess gross motor ability.

Most of the literature supports the premise that ratings on movement tests improve with medication for ADHD. One could justly surmise that if a movement is

available in the individual's repertoire, giving ADHD-diagnosed individuals medication to improve attention would allow them to better focus on execution of the movement, and therefore bring improvement in rated movement ability. This has been substantiated multiple times in the literature (Ben-Pazi, 2003; Biederman, 1998; Hannaford, 1995; Piek, 2004). Contralateral movement might be different though. Contralateral movement behaviors are developmental movements, not learned skills. If the particular developmental movement pattern is not available within the individual's repertoire, then no amount of medication should produce a movement for which the person has never founded the developmental capability. It is, in fact, entirely unclear if medicating individuals with developmental motor delay and ADHD would yield an improvement in the execution of developmental locomotor patterns. There is currently no significant literature looking at contralateral movement ability in ADHD. The research question motivating this study aims to evaluate the contralateral abilities of children with ADHD in both the medicated and nonmedicated states in an effort to broaden the scope of movement analysis and inquiry as it relates to attentional ability.

Statement of the Problem

The most remarkable discrepancy between the somatic and the Western medical approaches to looking at movement rests in the focus on developmental movement patterning that is dominant in the somatic point of view. In this study relating specifically to ADHD, developmental conglomerate contralateral movement ability is

the bridging point between these often disparate bodies of literature. The goal of this study is to integrate the educational, somatic, psychological, and medical literature by looking at the contralateral abilities of ADHD-diagnosed children in both medicated and unmedicated states. The current view of ADHD takes an interest in the movement deficits that are observed in children with ADHD but does not investigate movement tasks from a developmental movement perspective. Specific skills are evaluated in some ADHD assessments such as fine motor skills, visual tracking, hand-eye coordination, or balance, but conglomerate developmental contralateral movement phenomena are not part of the current vocabulary in psychology or psychiatry.

To begin investigating the relationship of cognitive development and contralateral movement development, this study asks the following research question: how will contralateral locomotor movement abilities in boys with ADHD, both while medicated and unmedicated, compare to age-matched boys without an ADHD diagnosis? The variables involved are ADHD diagnosis, medication, and The Ohio State University Scale of Intra Gross Motor Assessment–skipping scale (OSU SIGMA) ratings. The independent variables of diagnosis and medication will be treated as nominal, or categorical; the dependent variable of the OSU SIGMA provides an ordinal measure of contralateral ability. All variables are attribute and will not be manipulated.

ADHD is most prevalent in children, tending to become less symptomatic into adolescence and adulthood. For this reason children will be used for the study. Boys aged seven to nine years will be the target population because earlier research has established the motor age for the contralateral locomotor acquisition of skipping to be at

the age of six in boys. Girls are being excluded from the study because the motor age is earlier in girls, and ADHD often presents differently in female versus male populations. Dependent upon the finding of this study, a follow up study looking at the same variables in a female population would be of interest.

The literature indicates that children with ADHD tend to perform more poorly on movement ratings than non-ADHD children (Diamond, 2000; Findling, 1998; Fliers, 2010; Gillberg, 2003; Piek, 2004; Pitcher, 2002; Slaats-Willemse, 2005). It could be expected then that children with ADHD will have lower OSU SIGMA ratings than their non-ADHD peers. The literature also indicates that children with ADHD perform better on movement assessments when under the influence of their medication. The provocative unknown lies in the developmental specificity of contralateral movement ability and whether the children will show improved contralateral movement when medicated.

If a child can skip and does it more efficiently while medicated, it implies that the child progressed through and acquired the entirety of their normal foundation of developmental movement patterns. The child has available to them the full repertoire of movement patterns but something in the pathology of ADHD impacted the child's ability to display coordinated movement consistently and efficiently. If there is no improvement in OSU SIGMA ratings while the child is medicated, it implies that the child has not yet completely progressed through, or perhaps bypassed, levels of developmental movement acquisition. The child simply cannot skip; if a child did not have the ability to skip in the first place, medicating him should have no effect on a skill

that child cannot perform. It would be expected that the child would execute a movement that confirms itself earlier and perhaps execute that less mature movement with better observable skill on their medication versus when unmedicated.

This latter scenario indicates a delay or abnormality in progression through the developmental motor patterns. If patterning is slow or inhibited, perhaps the neurological repertoire and associated anatomy is also impacted, which would provide a foundation for future interdisciplinary investigations into the developmental trajectories and overlaps between the developmental motor processes and cognitive systems. This could open doors to investigate somatic principles as potential physically therapeutic interventions to complement current treatment strategies for ADHD.

Hypotheses of the Study

The developmental research hypothesis, the answer to the research question, for this study is that ADHD diagnosed children will have lower OSU SIGMA ratings than their non-ADHD peers both while medicated and unmedicated. If the cognitive and developmental locomotor systems develop and function in a coordinated fashion with each other, contralateral movement ability may be diminished in children suffering with some developmental cognitive disorders. Any improvements observed while the child is medicated will be limited to increased efficiency in the most mature developmental ability the child has mastered, or patterned, into their movement repertoire. Medication should only help children better express movement patterns into which they have

already matured and previously added to their available movement inventory. Medication should not promote a child to a more mature developmental pattern that was not previously mastered.

Hypothesis One: Contralateral movement ability, as measured using the OSU SIGMA skipping scale, will be less mature in children diagnosed with ADHD compared to children without ADHD.

Hypothesis Two: Children with ADHD will not show improved OSU SIGMA skipping scale scores in the medicated state.

Additional variables relating to a child's movement experience and medication type were built into the study design via a parent questionnaire. Confounds such as whether or not the child crawled, how long they crawled, at what age they walked, if they participate in sports or other motor skill-building activities, and type and dose of medication will be brought into the statistical analysis to clarify the possible importance of these variables to any results.

Limitations of the Study

This investigation is limited by lack of control due to its ex post facto nature, which leads to concerns of differential selection, common cause, reverse causality, and the possibility of alternative explanations. Due to this diminished control of variables,

strong inference about causal relationships cannot be formed. In the hypothesized presence of contralateral deficits in the ADHD group of children, it will not be known from this study whether ADHD causes the deficit, whether the immature developmental continuum of movements causes ADHD, or whether a completely separate factor influences both ADHD and developmental movement capabilities independently.

Having progressed beyond the established motor age of six, this sample of seven to nine year old boys should theoretically all be developmentally capable of skipping. Any inability to skip only informs that the child is not developmentally on track relative to this motor age of developmental locomotor movement patterning. This may have started in utero, in infancy, or earlier in their childhood, but this study will not be able to control for the possibility of reverse causality because it is not possible to know whether the presence of ADHD or the presence of an abnormal developmental movement pattern trajectory came first. This study investigates the presence of a complex functional relationship by asking if a change in one variable, in this case conglomerate contralateral ability operationalized by skipping ability, is accompanied by a change in the other, ADHD diagnosis and subsequent medication. This study does not attempt to explore a direct causal relationship between the variables of contralateral ability, ADHD diagnosis, and medication.

Building variables into the design can, in part, control alternative explanations for a difference in OSU SIGMA ratings between the ADHD group and the control group of boys. Presence of a developmental disorder or injury could negatively influence OSU SIGMA ranks and exclude a child from participating in the study. Age and sex

represent additional important confounding variables. It is generally accepted, and has been substantiated through logistic regression, that females skip earlier than males and that the ability to skip increases as the child matures (Frego, 1998). Most children have mastery over skipping by age six (Wellman, 1938). To control for both age and sex differences, only boys between the ages of seven and nine will be used in this study. Maturation is another concern addressed by the research design. Only about one month will pass between each OSU SIGMA recording to prevent time for a maturation effect to occur.

Subjects with a background in athletics, gymnastics, or dance may have learned how to skip in their training; this could positively influence OSU SIGMA ratings. This confound was controlled through survey questions given to parents following their consent to participate in the study. Also included in this survey were questions about developmental milestones such as crawling and walking behaviors of the participating children. Because these behaviors could also influence later contralateral abilities, they were built into the study design through the questionnaire.

Basic Assumptions

This investigation grew out of a basic assumption made by educational kinesiologists that contralateral movement ability is associated with focused attentional behavior. This assumption holds despite the current lack of scientific evidence to validate a contralateral deficit within children with learning disorders and attentional

deficits. Therefore, one cannot deduce that improving contralateral ability would impact learning or attention. Much more investigation is needed before that theoretical jump can be substantiated.

This investigation holds the major assumption that contralateral movement is a developmental ability. A child moves from spinal, to homologous, to homolateral, to contralateral maturity in their movement. From a locomotor standpoint this assumes that there is a developmental progression from creeping, to crawling, to walking, to running, to jumping, to galloping, to sliding, to leaping, and finally, to skipping. The early work of Wellman (1938) and Godfrey (1969) corroborates these assumptions.

This study seeks to provide data clarifying the proposition that a contralateral movement deficit exists in children with learning and attentional problems. This study additionally seeks to facilitate future scientific, peer-reviewed research into somatic-based learning methods by focusing on the contralateral movement phenomenon in ADHD as a representative, prevalent, and relevant topic to public health interests.

Definition of Terms

Attention Deficit Hyperactivity Disorder (ADHD)

When problems with attention, hyperactivity, and impulsiveness develop in childhood and persist, in some cases, into adulthood, ADHD may be diagnosed (APA, 2000). Diagnosis using the DSM-IV is based upon 18 criteria divided into 2 domains. One domain is of inattentiveness and one domain is of hyperactivity/impulsivity. In

order to be diagnosed with ADHD combined subtype (ADHD-C), one needs to fulfill six of the nine criteria in both domains. Fulfilling six of nine criteria in either the inattentive or hyperactive/impulsive domains allows for diagnosis of ADHD primarily inattentive subtype (ADHD-PI) or ADHD hyperactive impulsive subtype (ADHD-HI), respectively. Criteria must be met for the six months prior to diagnosis (APA, 2000).

Operationally, ADHD was measured through the diagnostic procedures of child psychologists, psychiatrists, and primary care physicians at local outpatient clinics resulting in a 314.00 to 314.99 diagnostic code. Evaluation of the child, family/parent assessments, school grade reports, teacher surveys, parent surveys, evaluation of social functioning/social skills, and behavioral assessments were used in combinations to diagnose ADHD. Factors such as other psychiatric or learning disorders, psychosocial history, current situational factors, possible current medical problems, history of prenatal or perinatal problems, and the general physiological state of the child are routinely identified or ruled out before a firm diagnosis of ADHD is made (Hales, 2002).

The presence or absence of ADHD is one of the attribute, independent variables for this study. The conventional, physician-directed treatment associated with the diagnosis outcome will serve as the second, also attribute, independent variable.

Basal Ganglia / Basal Nuclei

The basal ganglia, or basal nuclei, are located within the forebrain. They include the striatum, including the caudate nucleus and putamen, the globus pallidus, the subthalamic nucleus, and the substantia nigra. These nuclei are traditionally associated

with motor control and motor learning. They were first known as components of the extrapyramidal motor system. They do not synapse directly in a sensory (afferent) or motor (efferent) relationship with the spinal cord. Instead, the basal ganglia receive their primary input from the cortex and send efferent projections toward the prefrontal, premotor, and motor cortexes via the thalamus. This allows the frontal cortex to negotiate motor functions. More recently, the basal ganglia have been found to have roles in action selection and behavioral switching through their connections with the prefrontal cortex. In this cognitive capacity, they function with the forebrain to drive readily available and established behavioral repertoires while also influencing coordinated movements appropriate to changing behavioral contexts.

Body-Mind Centering (BMC)

Body-Mind Centering was founded by occupational therapist Bonnie Bainbridge Cohen as an experiential process of exploring the ever-changing nature of the human body. Cohen established the school for Body-Mind Centering in 1973. BMC explores patterns that begin in the womb, the neurological system of primitive reflexes, the phylogeny of mimicry, and integrated movement patterns (Brook, 2001). Study of BMC includes experiential and academic learning about the anatomy and physiology of the organ systems, nervous system, developmental movement patterns (both ontogenic and phylogenic), palpation skills, and physical re-patterning in injury recovery processes (Fitt, 1998). BMC practices use reflexes, spirals, and developmental support through movement with the premise that developmental movement can re-pattern and strengthen

neurological firing in the brain, and that developmental movement is key to emergent, consciously integrated movement. This investigation is concerned with the most mature of the developmental movement patterns, contralaterality, which will be operationally defined as ranked OSU SIGMA ratings.

Contralateral Movement

Contralateral movements are the most mature and chronologically the last developmental movement patterns to develop (Cohen, 1989). These patterns are thought to be the biologically universal, not cultural, reflexive recapitulations of ontogeny mimicking phylogeny that have evolved within the human nervous system (Hartley, 1995). During contralateral movements, one moves along one's physical diagonals through space (Bartenieff, 1998). This can be observed during crawling on hands and knees as one does when a baby. The movement involves one moving forward with right upper limb and left lower limb followed by left upper limb and right lower limb. Walking, running, and marching in place with opposite arm swinging to leg are also contralateral examples from an upright positional orientation.

Like contralateral developmental patterns, contralateral locomotor patterns are the most mature and also the last of the locomotor patterns to develop in the child (Frego, 1998). Skipping, which is the most mature of all the locomotor patterns, demonstrates contralaterality (Godfrey, 1969). Contralateral movement is available with the establishment of three-dimensional movement. It differentiates the diagonal quadrants of the body. From a somatic perspective, contralateral movement is thought

to integrate attention, intention, and action (Fitt, 1998). Babies begin contralateral behaviors while supine when reaching for the opposite foot with the opposite hand. This behavior then builds strength, which leads to rolling over. When prone, crawling is the observed contralateral behavior. Then as a child stands, they move through a repetition of homologous (superior-inferior) and homolateral (side-side) movements as they progress through initial walking, to running, to jumping, to galloping, to sliding, to leaping, and finally to skipping.

Operationally, contralateral movement will be defined by rankings of 1 (least mature), 2, 3, or 4 (perfect execution) on the skipping portion of The Ohio State University Scale of Intra Gross Motor Assessment (OSU SIGMA–skipping scale). Contralateral movement is the dependent variable within this study.

Developmental Movement Patterning (DMP)

Developmental Movement Patterning is an area of focus within Body-Mind Centering. The premise holds that development is not linear; it overlaps itself with each stage containing elements of all other stages. The study of DMP includes primitive reflexes, righting reactions, equilibrium responses, and basic developmental, or neurological, patterns. These developmental movement patterns are thought to be the automatic movement responses that underlie all voluntary movement possibilities for an individual.

The somatic perspective asserts that basic developmental patterns establish basic movement patterns and therefore the basic elements of movement, learning, and

communication (Dennison, 1989). The patterns progress from spinal movements (establishing the sagittal plane), to homologous (establishing the transverse plane), to homolateral (establishing the coronal, or frontal, plane), and finally to contralateral movement, which integrates all three planes of movement and allows for diagonal movements through space (Bartenieff, 1998; Fitt, 1998).

Educational Kinesiology

Educational Kinesiology represents a somatic-based system of movement activities intended to facilitate learning potential (Dennison, 1989). The practice uses specific movements to access different parts of the brain in an effort to maximize learning capabilities. Educational kinesiology has been recommended, based upon qualitative evidence, for use with children suffering from learning disabilities despite the fact that few scientific studies validate its effects (Dennison, 1998; Hannaford, 1995).

Homolateral Movement

In homolateral movements one engages either the left half or the right half of the body, such as hopping on one leg or creeping on one's abdomen using the right arm and right leg in a reptilian fashion, then the left arm and left leg to move forward or backwards in space. Through homolateral movement, right and left halves of the body are recognized, thereby establishing movement in the frontal, or coronal, plane. These patterns appear after the establishment of spinal movements, which instill a sense of the sagittal plane, and after homologous movements, which establish kinetic awareness of

the transverse plane, but prior to the establishment of contralateral movement, which encourages full use of the kinesphere and three-dimensional space (Fitt, 1998).

Skipping

Skipping is a combination of two of basic movement patterns, which together form a more advanced pattern. As a highly integrated pattern it is basic to the motor development process (Godfrey, 1969). This developmental skill can also be taught through appropriate sequential activities. Skipping is a contralateral, sequential movement in which each foot performs a walk and a hop before the body's weight is transferred to the other foot. The movement has a triple-beat feeling with the step taking twice as long as the hop. The arms also move reflexively in opposition with the feet. The length of the step, speed, and distance covered will vary within and between children, but the true acquisition of the pattern hinges on the fluidity of contralateral alternation (Godfrey, 1969). Females develop the ability to skip earlier than males (Frego, 1998). Most children have mastered the ability to skip by the approximate age of six years (Wellman, 1938). Skipping will be operationalized through use of The Ohio State University Scale of Intra Gross Motor Assessment's (OSU SIGMA) skipping scale.

Somatics

The founder of somatics, Thomas Hanna, coined the word "Somatics" in 1976 to name the varied approaches to mind-body integration. "Soma" is the Greek word for

body. Hanna redefined the Greek origin of the word as, "the body experienced from within, where mind-body integration takes place." Hanna is quoted to have said that somatics is "… the field of study dealing with somatic phenomena, i.e. the human being as experienced by himself (or herself) from the inside."

Somatics represents an experiential, multidimensional emphasis on body, mind, and environmental factors that provide for bodily-based access to information about the whole system and its interactive patterns. The discipline has developed over the past four decades as the interest in mind-body education has grown. The field encompasses brain function, learning theory, developmental movement education, physical therapy, sports medicine, and holistic health care.

CHAPTER 2: REVIEW OF THE LITERATURE

This literature review focuses on the interdisciplinary aspects of the research questions under investigation. The current understanding of etiological origins to ADHD and frequently comorbid conditions are first discussed followed by conversation of neurotypical attentional processes, theoretical models of what goes wrong in attentional processing, and current knowledge of the neuropathologies associated with attentional dysregulation. Developmental theories of attention, movement, and the interplay between the processes are then explored before concluding the literature review with dialogue surrounding existing assessment and treatment strategies for ADHD.

Etiology of ADHD

Despite a lack of clear neuroanatomical understanding underlying ADHD, the disorder is the most common neurodevelopmental pathology of childhood (Biederman, 1998). Basic information regarding how it varies by race, ethnicity, sex, age, and socio-economic status remains poorly understood. Difficulties diagnosing ADHD have

resulted in problems developing an adequate case definition for epidemiologic studies; difficulties with clinical definitions and epidemiological understanding have hindered diagnosis. Diagnosis of ADHD depends heavily on subjective reports, and there are no laboratory tests to reliably predict ADHD. Prevalence estimates are sensitive to the methods used in their establishment and range from 2%-18% (Rowland, 2002). The DSM-IV states that prevalence is between 3% and 5% in school-aged children (APA, 2000). Diagnosis is also complicated by the frequent occurrence of comorbid conditions, such as learning disabilities, obsessive compulsive disorder, oppositional defiant disorder, Tourette syndrome, depression, bipolar disorder, conduct disorder, and anxiety disorder (Faraone, 2005). Symptoms of these conditions can also mimic or confuse those of ADHD.

History

In 1902, George Still delivered a series of lectures describing lack of moral control among children with no apparent physical impairments (Rowland, 2002). In the years since, many labels and titles for these symptoms have preceded the currently used "attention deficit hyperactivity disorder." Initially, it was called "minimal brain damage," and then the term "minimal brain dysfunction" emerged. "Minimal cerebral dysfunction" evolved next, followed by "minor cerebral dysfunction" and "minimal cerebral insult" (Seitler, 2006).

The 1917-1918 encephalitis epidemic shaped early attempts to link attention deficits and behavioral disturbances to brain dysfunctions. Therefore, the earlier

pathology-oriented nomenclature reflected scientists' perception of the etiology of the disorder (Durston, 2003). Children surviving encephalitis suffered subsequent issues with hyperactivity, learning disabilities, and personality changes (Rowland, 2002). Nomenclature eventually came to reflect the symptom instead of the possible cause, and the term "hyperkinesis" emerged and then evolved into "hyperactivity," which next became "attention deficit disorder" in 1980. Finally, the literature settled on "attention deficit hyperactivity disorder" used today. Over this century-long span of nomenclature tinkering, at least 30 descriptive terms have been used to label the same symptoms (Seitler, 2006).

The assumption held that there must be a neurobiological basis for the disorder, but when the precise nature of this substrate would come into question, the phraseology alone changed, as understanding of the heterogeneous disorder remained elusive. Despite many years of research attempting to find specific etiological correlates of the disorder, a single cause is yet to be identified. At present, ADHD is best understood as a group of behavioral symptoms that reflect excessive impulsivity, hyperactivity, and inattention (Rowland, 2002).

Current Understanding

The fourth edition of the American Psychiatric Association's DSM (APA, 2000) characterizes ADHD as including attentional problems, restlessness and fidgety behavior, increased mood lability, low frustration tolerance, low self-esteem, difficulty sticking with a task, and problems with group situations which are made particularly

difficult when sustained attention is required. Examining the theories and models of ADHD, they appear rather circular; each explains differentially that ADHD must be a neurological entity inferred to exist from its symptoms, which are then inferred to have a neurological origin (Seitler, 2006). While "attention" has been incorporated into the name for nearly three decades, the current diagnostic criteria do not require that attention difficulties or distractibility be central to the disorder. Several researchers have tried to identify core deficits or measurable neuropsychological markers that underlie ADHD with a focus on executive functions (Derks, 2007; Rowland, 2002). Despite such efforts, the heterogeneity is perhaps the best understood aspect of the disorder.

Epidemiological research surrounding ADHD has been hampered by inconsistent diagnosis procedures, numerous name changes, and variations in the definition of the disorder over past decades (Remschmidt, 2004). There are questions about the basis and accuracy of neurobiochemical etiological explanations, weaknesses in the neurobiological hypothesis, problems with definitions, logical fallacies, flawed research methodology, and a problem of serious measurement difficulties surrounding diagnosis. There is no single, reliable, or valid physical, mental, or genetic test available for diagnosing ADHD. A few researchers question whether ADHD even exists (Remschmidt, 2004). Most find this position extreme but agree that, as currently defined, children diagnosed with ADHD comprise a heterogeneous population sharing a cluster of symptoms (Rowland, 2002). Many investigators and clinicians do not regard ADHD as a single diagnostic category, but instead as a syndrome of comoborbid conditions (Seitler, 2006).

Sex Differences

In both clinical and population samples, children diagnosed with ADHD are predominantly male. Boys are identified with ADHD at least four times as often as girls (Rowland, 2002). These differences could be due to a higher liability and therefore referral bias for the disorder within boys, a sex effect in the actual measurement of the phenotype, or to sex differences in the etiology of the disorder. Derks et al. (2007) found that behavior problems relate to the same variables in boys and girls and can therefore be regarded as due to the same latent construct. This supports the contention that reported sex differences are due to a higher liability for the disorder in boys than girls and not due to measurement bias. The group additionally found that half of the variance was attributable to genetic influences, and the remaining variance attributable to unique environmental influences. These magnitudes were the same in boys and girls.

Socioeconomic Variables

Reliable information surrounding socioeconomic variables is not available (Remschmidt, 2005). Rowland (2002) reported higher prevalence rates among urban children compared to rural children although this could be due to differences in access to medical care between the groups. Reliable information surrounding race and ethnicity is also not available and needs further investigation (Faraone, 2005). Preliminary data

suggests little difference between African-American and Caucasian children as far as diagnosis, but shows more substantial racial differences in medication treatment patterns (Remschmidt, 2005; Rowland, 2002). The Rowland study found 8% of white elementary school children being medicated for ADHD, while 5% of African-American children and only 2% of Hispanic children were being pharmacologically managed for their symptoms.

Genetics

Substantial evidence points to a biologically polygenetic basis for ADHD. Most of this evidence is drawn from neuroimaging studies, familial aggregation studies, twin studies, adoption studies, and molecular genetic studies. Collectively, this body of prior research reveals a heritability rate of around .80. Some researchers (Voeller, 1991) interpret this to mean that 80% of ADHD cases are genetic while the rest are environmental. There is no evidence, other than those cases of direct brain damage, to support a purely environmental causality for any cases of ADHD. The greater likelihood is that all ADHD cases are genetic, and the expression of the disease is based upon factors in the environment that exacerbate the underlying vulnerabilities (Wood, 2008).

The 1990s brought a proliferation in the number of neuroimaging studies. Findings converged on dysregulation and dysfunction of striatal, adrenergic-prefrontal circuitry (Castellanos, 2001). Many research groups have implicated abnormal striato-prefrontal anatomy and functions (Durston, 2003). Neuroimaging findings must be

considered preliminary for several reasons: 1) brain regions not implicated in ADHD have not been studied, 2) small sample sizes and inadequate statistical power characterize the majority of neuroimaging studies in ADHD, 3) interpretation of findings is limited by lack of information about normal, age-related developmental changes, and 4) neuroimaging technologies are constantly improving, changing, and in a state of flux (Rowland, 2002).

A primary genetic component to ADHD is strongly suggested because of the clustering of the disorder found within families. Biederman (1998) revealed that, in a clinic-referred sample, 34%-40% of the subjects with ADHD reported a family history of ADHD, whereas only 8% of the control group subjects reported a family history of the disorder. Twin studies suggest a concordance for hyperactivity being greater among monozygotic twins than dizygotic twins (Rowland, 2002). ADHD is likely to involve multiple genes of moderate effect.

A recent review of molecular genetic studies summarized candidate genes under investigation: 1) dopaminergic neurotransmission genes such as DRD4, DRD5, DAT1/SLC6A3, DBH, and DDC; 2) genes associated with the noradrenergic system, or norepinephrine neurotransmission, such as NET1/SLC6A2, ADRA2A, and ADRA2C; 3) genes involved with the serontonergic systems such as 5-HTT/SLC6A4, HTR1B, HTR2A, and TPH2; and finally 4) genes related to neuronal plasticity and neurotransmission, such as SNAP25, CHRNA4, NMDA, BDNF, NGF, NTF3, NTF4/5, and GDNF (Banaschewski, 2010). An earlier review of candidate genes spanning 1991 through 2004 concluded there were significant associations for the dopamine D4 and D5

receptors DRD4 and DRD5, and the serotonin transporters SLC6A genes (Tripp, 2007). Polymorphisms in dopamine transporter and dopamine receptor genes seem to influence the risk of ADHD (Ribases, 2009). Researchers have selected these particular polymorphisms because they are targets of the stimulant medications, which are known to be effective for treating ADHD. The effective pharmacological treatments targeting ADHD symptoms support the involvement of these genes and are discussed in the section in this chapter on pharmacology.

A number of genes involving the catecholaminergic system are being investigated. Candidate gene studies show an inconsistent pattern of replication, and some show non-overlapping chromosomal regions that might contain susceptibility genes. A study by Grady and colleagues (2003) found a significant positive selection for the genetic variation associated with ADHD and novelty-seeking behavior in the human genome. Other studies (Hale, 1998) have also associated the DRD4 gene with thrill-seeking and risk-taking behaviors, while yet other studies do not support these findings (Remschmidt, 2005).

The gene coding for catechol-O-methyltransferase (COMT) has also been studied. It is believed that COMT is important in the metabolism of dopamine in the prefrontal cortex whereas the dopamine transporter is more important in the striatum. While the studies looking at COMT have revealed information of a possible predictive function of dopamine, the studies relating specifically to ADHD have shown limited association between the COMT gene and ADHD presentation (Levy, 2008). This may be a developmental issue related to the late development of the prefrontal cortical

connections in adolescence with a late activation of the COMT gene (Wood, 2008). More research is needed before any conclusions can be drawn.

Environmental Factors

Environmental factors such as low birth weight, perinatal maternal complications, environmental toxicants, and maternal cigarette smoking have all been explored as they relate to ADHD (Seitler, 2006). Studies since the 1950s have implicated low birth weight as having a role in future ADHD diagnosis. Several follow-up and cohort studies over the decades have provided convincing data to this end (Remschmidt, 2005). The general suggestion is that babies born between 1,500 and 2,500 grams, or 3.3 to 5.5 pounds, are at increased risk for developing ADHD (Derks, 2007). More information is needed as to whether the risk varies by gestational age or birth weight alone. Because the brain is still developing at a rapid rate during the third trimester, preterm delivery is a plausibly important risk factor for ADHD (Rowland, 2002). Mothers of ADHD children do report increased rates of pregnancy complications, although this data is difficult to interpret because of the lack of standard epidemiological methods for analyzing and collecting such data (Seitler, 2006). More comprehensive epidemiologic studies examining pregnancy history as a risk factor for ADHD are warranted.

The most suggestive evidence for an environmental toxicant as an etiologic risk factor for ADHD is with lead (Remschmidt, 2005; Seitler, 2006). Studies show that animals dosed with lead are more aggressive and agitated, more distracted by irrelevant

stimuli, and less able to inhibit inappropriate responses. In human subjects, Rowland (2002) reported the dose response relationship between tooth lead levels and teacher ratings of disorganized, hyperactive, and inattentive behaviors among children. The dilemma with pointing too strong a finger at lead is that environmental levels have dropped precipitously since the removal of lead from gasoline, and ADHD levels have not declined in tandem. The literature surrounding lead is nonetheless important because it offers insight as to how environmental toxicants might increase the risk for presentation of the disorder. Other toxicants such as solvents, pesticides, and metals like mercury that can cross the placenta are worthy focuses of investigation but to date have seen few studies devoted to them.

Maternal cigarette smoking has been reported to increase the risk of ADHD as well as other externalizing disorders and psychiatric conditions (Rowland, 2002). However, this evidence could be artifactual. Since nicotine acts as a central nervous system stimulant, it is entirely possible that mothers choosing to smoke throughout their pregnancies are actually self-medicating for their own ADHD symptoms (Wood, 2008). Therefore, maternal smoking status could act as a marker for ADHD status within the family and not as an environmental factor increasing the risk of development for the disorder. Genetic factors are likely responsible for a large portion of the etiologic fraction of ADHD, but environmental, nongenetic factors still warrant further research as they are often preventable and serve as valid targets for intervention. A better understanding of the interplay between genetics and environmental risk factors could prove fruitful for identifying populations where interventions would be of peak value.

Theoretical "Value" of ADHD

With the multitude of studies showing co-segregation within families, overlapping but independent disorders, and underlying heritability factors, there is a clear state of recognition within the literature that ADHD is a genetic disorder that is highly heritable and susceptible to increased expression through exposure to environmental factors. The question then arises: why is ADHD part of the human genome in the first place? This topic is controversial and is strongly opposed by field experts such as Barkley (Barkley, 2000). Within the public and media, this viewpoint of an evolutionary value to ADHD has gained intrigue and relative acceptance. The theory is that genes, such as the DRD4, exist within the human genome due to the adaptive advantage they carried to hunting and gathering societies (Hartman, 2003). The Grady group (2003) found that the 7R allele of the DRD4 gene was created relatively recently in human history. More importantly, multiple studies show the gene with the 7R allele to be present in up to half of all diagnosed ADHD cases (Altink, 2008; Elia, 2010).

While this information is not to imply, as perhaps Barkley interprets, that there is a current evolutionary advantage to ADHD, it is evolutionarily logical that the qualities associated with ADHD did, at some point in time, have value and thus found their way into the human genome. Hartman (2003) refers to the difference between Native American cultures such as the agricultural Pueblo society versus the hunting Navajo society. For the Navajo hunters, it makes adaptable sense to have innate abilities lending toward constant scanning of their environment and more immediate sensitivity

to stimuli. Hunters and gatherers also needed to be great risk takers. Along this line of rationalism, the spread and current dispersion of ADHD's prevalence took place as these hunters and gatherers migrated across the world. This line of thinking has been seriously questioned. When equating etiology across cultures, the rates of ADHD are still higher in the Unites States than Europe (Plizka, 1998), but these cultural differences are most likely attributable to methodological differences and not a true prevalence bias across cultures. The North American rate of 6.2% only slightly exceeds the European rate of 4.6% and the highest rates were found in Africa (8.5%) and South American (11.8%). Finnish and Japanese children had the lowest ADHD prevalence rates whereas Jamaican and Thai children had the highest (Moffitt, 2007).

ADHD as a Lifetime Disorder

This current study, which explores the role of conglomerate contralateral movement and its role in ADHD, is specifically interested in the possibility of ADHD as a developmental disorder with an interest in subsequent developmental interventions. Castellanos (2001) has proposed that ADHD represents a developmental lag. Children with ADHD tend to trail about two years behind their peers in social development. It is known that dopamine metabolite levels in spinal fluid peak around the age of two and then begin to decline. Castellanos hypothesizes that this may be why in neurotypical people hyperactivity decreases after this age. Additionally, the disproportionate male to female ratio in the presentation of the disorder is the same sex ratio seen in many other developmental disorders (Rowland, 2002). This current contralateral movement study is

investigating developmental movement abilities in an attempt to determine if a lag in developmental contralateral motor acquisition is also observable within boys with ADHD.

Longitudinal studies have revealed that ADHD is a lifetime disorder (Biederman, 1998; Durston, 2003; Remschmidt, 2004; Yeh, 2004). Biederman (1998) reported marked increases in behavioral, mood, and anxiety disorders as well as increased impairment in cognitive, family, school, and psychosocial function following a four-year follow up. Many other studies have reported an attenuation of symptoms over longer periods of follow-up but also noted similar rates of increase in other types of impairment as those found by Biederman (Derks, 2007; Remschmidt, 2005). One study of 100 hyperactive 6 to 12 year old boys found 43% met full criteria for ADHD 10 years later, and after 16 years the rate declined to just 8%. When compared to a group of similar controls, this original group had lower educational attainment, lower occupational status, substantially more antisocial personality disorders, and more substance abuse (Rowland, 2002).

Thus, some researchers see the decline in hyperactive behavior and other cardinal symptoms of ADHD coupled with the increase in comorbidity and impairment as the natural progression of ADHD. Viewing the disorder from this perspective, children with ADHD are at a high risk for a large scope of potential problems including learning disabilities, substance abuse, psychopathology, and difficulties in their social relationships, work, and marriages. Knowing that this risk is variable, it is important to continue seeking better epidemiological and etiological understanding, clarifying which

children are at higher risk, and providing routes for untangling the heterogeneity underlying ADHD.

ADHD Comorbidities

Epidemiologic studies have shown that nearly all psychiatric disorders increase the risk of the patient having another disorder. Because comorbidity is the rule rather than the exception, the mere presence of one comorbid condition does not invalidate another condition (Faraone, 2005). Obsessive-compulsive disorder (OCD), Tourette syndrome, depression, disruptive behavior disorders, bipolar disorder, anxiety disorders, and learning disorders are frequently comorbid with an ADHD diagnosis (Pliszka, 1998). Rates of comorbidity are roughly 10% for reading disabilities, 27% for anxiety disorders, and 25%-40% for oppositional defiant disorder/conduct disorder (Rowland, 2002). As with an ADHD diagnosis itself, these comorbidity rates are highly susceptible to methodological choices and definitions. Furthermore, many of these disorders can resemble ADHD, and additional disorders often come to the fore after ADHD has been diagnosed. Distinguishing between ADHD itself and underlying conditions that mimic ADHD is a serious problem for epidemiological studies, diagnosis, and overall treatment (Durston, 2003).

Neuropsychological symptoms and comorbidly occurring disorders have begun and will continue to inform the quest toward understanding completely the neuropathology underlying ADHD. Many factors could explain comorbidity of

disorders: 1) the overlap is not more significant than chance, but the clinicians are seeing the more complicated cases in practice; 2) the comorbid condition is secondary to the ADHD; 3) ADHD has a distinct genetic or environmental etiology, while ADHD with the comorbid condition has a different etiological basis; and 4) ADHD has a unique cause, but environmental influences can cause ADHD children to present differently, inclusive of the potential for comorbid conditions (Angold, 1993).

Usually when disease is effectively treated, the structure(s) underlying the dysfunction is identified and subsequently targeted for enhancement of function. In the case of ADHD, the pharmacology and pathophysiology are constantly informing each other and equally fueling the growing knowledge of this heterogeneous disorder. Information about other disorders known to be pathologically grounded in fronto-striatal networks has proven helpful toward an evolved understanding of ADHD. Bellgrove (2001) studied the bimanual coordination deficit often seen in Huntington's disease and schizophrenia and found a common attentional deficit resulting from disruption to frontal-striatal circuitry. The obsessive-compulsive behavior and Tourette syndrome literature also attribute neurobiology to the basal ganglia and specifically its striato-cortical projections, which are areas known to be important in normal attentional processing (Derks, 2007). Focus is also upon frontocortical executive functions, including measures of sustained attention, impulse control, planning, organization, cognitive flexibility, visuomotor integration processes, various presentations of difficulties at school, and learning disorders (Schultz, 1997).

Analyzing the common clinical features of these concurrent conditions has benefited the search for a neural substrate. Historically, more attention is paid to the motor abnormalities associated with basal ganglia disorders than the mental state and cognitive disturbances. This has been the trend despite the fact that these effects can be as distressing and disabling for patients and caregivers as the motoric disturbances (Ring, 2002). The hyperkinesia of Tourette syndrome and the hyperactivity and clumsiness seen in ADHD both point to motor systems. The premonitory urges of Tourette syndrome, 'chunking' of attention in ADHD (Graybiel, 1997), and compulsive behaviors in obsessive-compulsive disorder all point to substrates interlinking motor capabilities and higher cognitive executive functions. Difficulty inhibiting inappropriate behaviors, seen in many of the comorbid conditions, suggests pathological involvement of inhibitory brain regions (Leckman, 1999).

Motor, association, and inhibitory neural systems therefore are likely to underlie the symptoms of these disorders. The leading neural system subserving these diverse processes is the looping circuitry between the cortical and subcortical brain regions involving the fronto-striatal networks. More specifically, the motor, oculomotor, dorsolateral prefrontal, and limbic loops moving through the basal ganglia process these functions. Schultz (1997) suggests that the visuo-perceptual and visuo-motor integration deficits seen in Tourette syndrome, ADHD, and obsessive-compulsive disorder likely arise from a common abnormality in the circuitry relaying the basal ganglia to visual cortical areas.

Neurotypical Attentional Processing

Before discussing in detail specific circuitries and their potential role in ADHD, a theoretical foundation for normal, healthy attentional processing must be built. From the simplest level of organization in the nervous system to the most complex, information is taken in from the surrounding environment, processed, and appropriate output is provided. This is true from the perspective of a primitive reflex through to the most complicated cognitive processes. The construct of attention could be regarded as the ultimate interaction between such inputs and outputs. Attention, therefore, requires a complex and neuroanatomically distributed system.

In the gestational beginnings of life, the nervous system establishes its functional organization. Throughout life, as one's system adapts to the surrounding world, the nervous system is constantly and relentlessly responding to stimuli in the environment. It is of evolutionary importance that stimuli in the environment are salient and can 'grab' one's attention. This stimuli recognition process is referred to as signal-driven detection, or bottom-up detection. Signal-driven detection forms the basis of attentional capabilities (Sarter, 2005). It is also of crucial importance that the nervous system can process what information is relevant to attend to, make cognitive choices, and devise cognitive strategies so stimuli can become aligned into attentional experiences. This process is referred to as cognitive cholinergic detection, or top-down modulation (Connor, 2004; Sarter, 2005).

Changes in the autonomic state are brought into processing through projections from the brain stem. These projections input, in part, onto the basal ganglia. In the cognitive realm and via nucleus accumbens circuitry with the basal forebrain, the basal ganglia support the abilities of action selection and behavioral switching. Action selection is one's ability to decide which of several behaviors to execute at a given time (Haber, 2000). The ability to select, or inhibit, appropriate cognitive strategies is mediated by circuitry connecting the basal ganglia to many other areas of the forebrain, specifically the prefrontal cortex with its role in executive functioning. The ability to select, or inhibit, appropriate movements and repertoires is mediated by circuitry connecting the basal ganglia to motor and premotor areas within the cortex.

The Basal Ganglia

The basal ganglia are deep-lying structures within the inferior cerebrum. The basal ganglia are formed dorsally by the striatum, globus pallidus, subthalamic nucleus, and substantia nigra and ventrally by a limbic region comprised of the ventral globus pallidus and ventral tegmental area. The dorsal striatum is further divided into the caudate nucleus and the putamen. The ventral striatum contains the nucleus accumbens. The amygdala, found deep within the temporal lobes of the cerebrum, is also often considered a structure of the ventral striatum. Together, these structures form a forebrain system that collects signals from the brain stem and a large part of the cortex, redistributes these inputs with respect to each other, and focuses the integrated inputs to regions of the cortex (Mehler-Wex, 2006).

Basal ganglia nuclei have specific associative structures and operate using multiple neurotransmitter systems. Projections from the substantia nigra carry dopamine to the dorsal striatum, projections from the raphe nuclei of the brain stem carry serotonin to the nucleus accumbens, and projections from the locus coeruleus of the brain stem carry norepinephrine to the nucleus accumbens, amygdala, and basal forebrain (Haber, 2000). These noradrenergic, serotonergic, and dopaminergic projections correspond with autonomic arousal and bias processing toward that of sympathetically activating stimuli.

While such inputs are fundamental to initiate bottom-up processing, they are not capable of sustaining the attentional process once initiated (Sarter, 2005). Nucleus accumbens dopamine is needed to regulate efflux of the basal forebrain cholinergic projections, which mediate acetylcholine-regulated attentional functions (Neigh, 2004). Dopaminergic neurons from the ventral tegmental area have long been identified with the processing of reward value, or saliency, of stimuli. These neurons send dopamine-containing axons to brain structures, such as the striatum and prefrontal cortex, which are involved in motivation and goal-directed behavior (Schultz, 1997). Conditioned stimuli and novel stimuli have been shown to produce increases in striatal dopamine (Tripp, 2007).

Dorsal versus Ventral Strial Functions

The ventral and dorsal striatum have partially dissociable functions. The ventral striatum, containing the amygdala and nucleus accumbens, acts as a "critic," in reward

and motivational aspects, and the dorsal striatum, containing the caudate nucleus and putamen, plays the part of the "actor," coordinating motor and cognitive control (O'Doherty, 2004). The prefrontal cortical/dorsal striatal system is involved in aspects of executive attention to action, whereas the prefrontal cortical/nucleus accumbens system is involved with integration of information about the consequences of action in relation to anticipated reward (Christakou, 2004).

The ventral striatum is a site of convergence for inputs that modulate the recruitment of the basal forebrain corticopetal, cholinergic projections. Neuronal activity in the nucleus accumbens is necessary for attention-related increases in cortical acetylcholine release (Neigh, 2004). The nucleus accumbens receives afferents from the substantia nigra, raphe nuclei, amygdala, locus coeruleus, and prefrontal cortex; it also projects to the basal forebrain and the substantia nigra. These nigral projections bring the information into the motor sphere via the projections from the substantia nigra to the dorsal striatum (Haber, 2000). The nucleus accumbens' projections to the basal forebrain are colored with information regarding the motivational and emotional value of the stimulus via converging inputs from all of these areas. These afferents to the basal forebrain act to stimulate cholinergic, corticopetal outputs. Through this mechanism, the dopaminergic projections to the striatum modulate stimulus-response and stimulus-response-reward associations (O'Doherty, 2004).

Elevated nucleus accumbens dopamine results in enhanced effort to get a reward and sensorimotor response by increasing the incentive value of the stimulus. The increase in dopamine causes one to "want" a stimulus more or find the stimulus to be

more salient. Multiple lines of research have shown the importance of nucleus accumbens dopamine in the process of addictive behaviors due to this incentive "wanting" facilitated by dopamine in the nucleus accumbens (Tripp, 2007; Wyvell, 2000).

The shell and the core of the nucleus accumbens differ histochemically. The core resembles the rest of the striatum and seems important in response selection and organizing the direction of a behavior (Haber, 2000). Ablations of the core have been shown to impact impulse choices in rats (Cardinal, 2001). Christakou and colleagues introduced bilateral lesions to the core and found an effect on attentional accuracy reflecting inhibitory response control. In particular, perseverative or compulsive behaviors and premature or impulsive behaviors were apparent following these core lesions. This suggests that the core is part of circuitry especially implicated in the modulation of attentional performance by affective outcome. The nucleus accumbens, and specifically its core, are involved in behavioral inhibition, including both impulsive and compulsive forms of responding (Christakou, 2004).

The shell of the nucleus accumbens receives limited input from the cortex, midbrain, and thalamus. It acts more to amplify the effects of information and adjust the vigor of response selection. The shell and the core are interconnected, and as such, the shell can exert control over the core as the core functions adjust to the direction and action set for behavior (Haber, 2000). Thus, the frontostriatal projections are organized in a ventromedial, or limbic, to dorsolateral, or associative motor, gradient. Haber and colleagues observed that the dorsal striatum is modulated by the ventral striatum and

proposed this as a mechanism by which limbic circuitry directly affects motor outcome. The shell of the nucleus accumbens receives forebrain input primarily from areas most closely associated with the limbic system. The interface between different striatal regions via dopaminergic projections is organized in an ascending spiral, interconnecting different functional regions of the striatum. So rather than a direct limbic-motor interface, information is mediated through several circuits to reach the motor striatum (Haber, 2000).

Bottom-Up Meets Top-Down Attentional Processing

Complex behavioral situations require one to search for, detect, select, and discriminate amongst abundant, multimodal stimuli within the environment. Such complicated processes have been extensively demonstrated to depend on the activity and integrity of cortical cholinergic inputs (Sarter, 2000). As has been discussed, nucleus accumbens' regulation of cortical acetylcholine efflux mediates the attentional capacities involved in goal-directed behaviors (Neigh, 2004). Once inputs are colored with information about emotional value and importance, the nucleus accumbens projects to the basal forebrain. These basal forebrain projections go to the prefrontal cortex, providing a route for bottom-up mechanisms to take raw input from the environment and bring it to the level of the cortex. This bottom-up mechanism allows for an involuntary means of shifting attention to the salient features of the input (Connor, 2004).

Rarely does signal-driven processing take place in isolation. This would be the case only in situations of a well-practiced task performed in the absence of any novel

distracters. Usually, top-down mechanisms, or cognitive cholinergic modulation of detection, help to implement cognitive strategies and attentional state to the bottom-up process (Connor, 2004; Sarter, 2005). These cognitive detection mechanisms allow for enhancement of processing toward cognitively relevant stimuli based upon cortically derived appreciation for what is being attended to and what is cognitively relevant. It is known that the prefrontal cortex exerts an inhibitory influence over the nucleus accumbens during activation of the amygdala. This modulation may feed back into the prefrontal cortex via the thalamus, providing online integration of affective information into the function of other corticostriatal systems (Christakou, 2004).

Cholinergic inputs to the cortex support executive functioning via adaptive changes in sensory cortical maps allowing for continuously enhanced processing of behaviorally significant stimuli. This enhanced saliency is mediated through the recruitment of the prefrontal cortex via cholinergic projections, dorsolateral prefrontal cortex efferents to the entire striatum, substantia nigra, and ventral tegmental area (Cardinal, 2001). The orbitofrontal cortex afferents from sensory associative cortices, particularly olfactory, gustatory, and visual areas, as well as the hypothalamus and amygdala also facilitate the processing of relevant stimuli (Levy, 2008).

The presence of cortical acetylcholine increases the 'signal-to-noise' ratio of cortical neurons in response to incoming sensory input, activates relevant cortical areas, and supports the neuronal processing of behaviorally salient stimuli (Sarter, 2005). Dopamine can both inhibit background corticostriatal input and facilitate or focus specific corticostriatal synaptic transmissions (Haber, 2000). Although the role of

dopamine and reward is well established, its primary function is to direct attention to important stimuli likely to bring about a goal-directed, desired outcome.

In situations where top-down mechanisms predominate, the major input to the basal forebrain arises from the telencephalon, or cerebrum, and uses glutamate as its neurotransmitter. This provides a steady loop of communications from cortex to basal forebrain and from the basal forebrain back to the cortex. The widespread projections of dopaminergic axons to striatal neurons gives rise to synapses at dendritic spines that are also contacted by excitatory inputs from the cortex. Here, the dopaminergic signaling influences behavioral choices by modulating the level of competition in the striatum. Phasic dopamine signals may lead to an augmentation of excitatory influences in the striatum. There is evidence for striatal plasticity after pulsatile application of dopamine (Schultz, 1997), and this plasticity could mediate the learning of appropriate behaviors (Graybiel, 1997).

At any time, the basal forebrain or any of its afferent projections can modulate the information and thus the processing along this communication pathway, but cholinergic innervation of the prefrontal cortex is necessary to initiate the cognitive mechanisms underlying situations such as the presentation of distracters. This represents the idea that "effort" is ultimately mediated by increases in cortical acetylcholine release. The presence of a distracter alters the uncertainty within the cognitively driven, top-down strategy, and the amount of cholinergic activity reflects the degree of discrepancy between the top-down strategy and the bottom-up inputs (Sarter, 2005).

Due to top-down mechanisms, the nervous system is able to increase the ratio of signal, or what is cognitively intended to be the attentional focus, to noise or the distracters that come at the sensory systems. The prefrontal cortex can input directly to the locus coeruleus, amygdala, basal forebrain, substantia nigra, cerebellum, and the rest of the neocortex (Sarter, 2005). The prefrontal cortex uses representational knowledge and guides overt responses as well as inhibits inappropriate behaviors by gating the processing of irrelevant stimuli (Levy, 2008). Thus, the top-down mechanisms modulate the bottom-up signals when one has a specific knowledge-based or cognitively driven plan for attention. Through this cognitively driven, top-down system, attention is biased toward relevant stimuli that match a given cognitive strategy. Top-down regulation allows sensory inputs to be processed based upon expectations, prior memories, practice, and knowledge associated with the given stimulus (Sarter, 2005). This process consumes cognitive energy and neuronal resources, an idea which will be elaborated upon in the section on models and theories of ADHD.

Summary of Normal Attentional Processes

A model of collaborating signal-driven, bottom-up, and cognitively driven, top-down, mechanisms represents the current understanding of normal attentional processes. At the neural and psychophysical levels, bottom-up attention acts early and top-down attention takes over within the course of 100 milliseconds (Connor, 2004). These parallel time courses help to confirm the functional relationship between the neural events and the behavioral response. The earlier time course for bottom-up attention is

logical, as it allows for salience based upon simple inputs. The subsequent top-down, cognitive control requires operations on the inputs that originate from the cortex.

The cholinergic inputs to the cortex act as the point of integration of these systems. Multisynaptic pathways mediate these cortical cholinergic inputs. This system and its underlying anatomy facilitate the detection, selection, and subsequent processing of stimuli (Neigh, 2004). Repeated administration of psychostimulants causes drug-induced increases in cortical acetylchloline, which corroborates the neuropharmacology used in the treatment of attentional and inhibitory problems. Abnormally reactive cholinergic inputs may limit the shifting of cortical processing, thus biasing toward thalamic, or bottom-up, inputs in the pathology of such disorders. This subjects the system to increased "noise," which would otherwise be filtered by cholinergically mediated inputs (Sarter, 2005). The result would be a movement toward attributing attentional significance to irrelevant or usually unattended-to stimuli.

"Psychology has shown that the complex, dynamic interplay between bottom-up and top-down attention determines what we are aware of from moment to moment" (Connor, 2004). This neurobiological model of neurotypical attentional processing provides a basis for discussing the current models of ADHD, the neurobiology of pathological attentional mechanisms, developmental hypotheses linking cognitive and motoric deficits, and treatments targeting this neuroanatomy.

Theoretical Models of ADHD

At the foundation of deficits seen with ADHD, lies a lack of congruency between cognitive strategy and stimuli recognition. The models that have been devised to theoretically explain ADHD reflect on the neural foci observed in either top-down or bottom-up attentional processes. The primary models considered are the cognitive model of ADHD proposed by Barkley, and the motivational models of ADHD proposed by Sagvolden, Sonuga-Barke, Sergeant, and Nigg.

The most prevalent causal theory of ADHD is the cognitive model, which has been widely discussed by Barkley. In this model, cognitive deficits in executive functioning are the underlying causal mechanism driving ADHD symptoms (Barkley, 2000). The primary roles of executive functioning include the initiating, sustaining, inhibiting, directing, and redirecting of attention. Diminished inhibition of responses to stimuli, or response inhibition, is proposed by Barkley to be the most impacted executive function resulting in the symptoms seen associated with ADHD. A decrease in response inhibition impacts all other executive functions and ultimately results in diminished social intelligence.

The cognitive model refers to additional executive functions that seem impaired in ADHD, such as nonverbal working memory, internalization of speech, self-regulation, and the ability to segment and recombine behaviors into new behavioral sets. Self-regulation and the associated constructs are interlinked phenomena. When on a normal trajectory, self-regulation represents balanced physiological, attentional, emotional, behavioral, and cognitive processes underlying adaptive behaviors with an

interrelated impact on social functioning (Reebye, 2009). When on an abnormal trajectory, self-dysregulation results in the characteristics associated with ADHD.

The anatomy underlying the cognitive model rests in the circuitry connecting the ventral tegmental area and the nearby substantia nigra to the prefrontal cortex. The projections from the substantia nigra travel through the dorsal striatum mediating associative motor functions, and the projections from the ventral tegmental area pass through the ventral striatum, becoming colored with its limbic functions (Schultz, 1997). The main caveat against this model is that research shows not all cases of ADHD involve executive functioning deficits. While the cognitive model may explain theoretical causality of some ADHD cases, there are other cases left unexplained by use of this model alone.

Motivational models are theoretically grounded in poor reward signaling and aversion to delayed reward as the primary mechanism supporting ADHD symptoms. These models suggest an underlying deficit of motivation-based behavior and theorize that ADHD is the outcome of neurobiological impairment in the efficiency of contingencies between immediate action and future rewards. The Dynamic Developmental Hypothesis of ADHD suggests that the dopaminergic system is hypoactive in ADHD and explains the behavioral changes in terms of altered reinforcement and extinction processes (Levy, 2008). Evidence that ADHD children will select immediate smaller rewards even in the presence of greater delayed rewards points to what Sonuga-Barke described in the Motivational Delay Aversion Model of

ADHD (2005). This model suggests that children become averse to delayed rewards and therefore avoid situations wherein delayed reward processes are present.

There is a consistent theme in the literature that children with ADHD have an atypical response to positive reinforcement. There have been a number of studies suggesting that children with ADHD differ from non-ADHD children in their response to reinforcement (Dominey, 1995; Tripp, 2007; Wyvell, 2000). Historically, ADHD children have been described as less able to delay gratification and as failing to respond to discipline. As a group, ADHD children perform more poorly under partial schedules of reinforcement and respond more impulsively to reinforcements (Tripp, 2007).

Sergeant and colleagues (2000) have proposed another branch of the motivational model, the cognitive energy model. In the discussion of normal neurobiological processes, it was proposed that cognitively driven, top-down regulation processes sensory inputs based upon expectations, prior memories, practice, and knowledge associated with the given stimulus. This process consumes cognitive energy and neuronal resources (Sarter, 2005). Overall efficiency of information processing is determined by the interplay of computational mechanisms of attention, state factors, and management of executive functioning. The cognitive energy model encompasses both top-down and bottom-up processes and draws attention to the fact that ADHD causes defects at all three levels. These include motoric mechanisms, such as response output, energetic mechanisms, such as activation and effort, and management of executive functioning deficits. Increasing evidence suggests that inhibition deficits associated

with ADHD may, at least in part, be explained in terms of an energetic dysfunction (Sergeant, 2005).

The cognitive energy model suggests that ongoing information processing is influenced by the availability of cognitive resources. The claim is that dysregulation in available "cognitive energy pools" causes problems in optimization of information processing and attention (Pennington, 2006). This model points to low arousal and diminished activation of dopaminergic pathways, and thereby a behavioral decrease in perceived effort or motivation displayed by ADHD children. The theoretical neurobiology supporting this model stresses the mesolimbic, ventral striatal dopaminergic pathways and their ability to modulate the incoming stimuli and attentional processing with emotional input from the nucleus accumbens and amygdala (Sergeant, 2005).

A fundamental flaw of these relatively simple, singular pathways to ADHD lies in their inability to fully encompass the entire disorder and heterogeneous spectrum of deficits. The expectation of any endophenotype for a disorder is that the genetics will be simpler than the presentation of the disorder observed within the population (Doyle, 2005). While the environmental factors that influence ADHD may change its appearance, none of the current models provide a sufficient, all-encompassing, causal theory for understanding the disorder.

This gap in understanding and clinical presentation led to the proposition by Sonuga-Barke (2005) that both pathways may be incorporated into a dual pathways model of ADHD. This model suggests that the dopaminergic systems of both the

mesocortical circuitry and mesolimbic circuitry are affected and collectively give rise to the deficits observed in ADHD cases. The mesocortical circuits connect the ventral tegmental area and substantia nigra with the prefrontal cortex. The mesolimbic circuits connect the ventral tegmental area to the amygdala and to the prefrontal cortex.

Nigg (2005) elaborates the dual pathways model by suggesting that there are likely multiple pathways that lead to ADHD. In 1995, Morgan and Frith stated that any causal account of a disorder must fully represent the broad nature of the disorder as it is "manifest at the level of the individual child" (Nigg, 2005). Multiple pathways would certainly explain the heterogeneity witnessed in the presentation of the disorder by explaining the disorder with heterogeneous causality.

Both the cognitive model and motivational models share overlapping anatomical correlates as well as support by the genetic, neuropsychological models of neurotypical attention, neuroimaging, and neuropharmaceutical findings. The fundamental challenge in ascribing a causal pathway to ADHD lies in the dilemma of integrating this theoretical and anatomical information subserving cognitive and motivational theories with neurocircuitries supporting pathological attention. The theories pinpoint ends of the spectrum, attributing, on one end the anatomy of the prefrontal cortex and its role in cognitive function to, on the other end loci in the amygdala and ventral striatum dealing with emotional flavoring of stimuli. The dual pathway model allows all models to play a role in the pathologies associated with ADHD and fills in the gray area between the extreme ends of the spectrum. With a clear foundation of the current knowledge surrounding ADHD etiology, normal attentional neurobiology, theoretical models of

ADHD, and anatomical models of ADHD, the neurobiology of pathological attentional processes can be explored.

Pathological Attentional Processing

As discussed in the conversation of normal attentional properties, it is widely suspected that abnormalities in the fronto-striatal networks and in catecholamine dysregulation impact cholinergic, corticopetal inputs underlying the neurobiological substrate of ADHD. This section seeks to combine the psychobiology of normal attention with the theoretical models of ADHD by discussing the known pathophysiologies associated with the disorder. Symptoms of inattention have been linked mainly with the striatum and cingulate gyrus. Symptoms of hyperactivity are predominantly linked with the striatum where motor, cognitive, and motivational systems interact within the caudate and the putamen (Ring, 2002). The impulsivity seen in ADHD has been mostly tied to abnormalities in the nucleus accumbens. Impairments in executive tasks lie mostly within the dorsolateral prefrontal cortex.

The Basal Ganglia

Research into the biological basis of ADHD has focused on the interaction of the brainstem's dopaminergic and noradrenergic reticular activating systems and the prefrontal cortical connections to the basal ganglia. The basal ganglia detect contexts of cortical activity and respond to these contexts by choosing appropriate sets of activity in

the cortex. This plays a vital role for activation and regulation of the motor system and prefrontal functions (Gillies, 2000). The basal ganglia learn to disinhibit only the correct motoric and behavioral responses through motor and behavioral reinforcement learning (Dominey, 1995). The caudate nucleus, a major input nucleus to the basal ganglia, is implicated in cognitive processes such as attention and response selection in specific environmental contexts (Alexander, 1990). Differences in the anatomical dimensions of the caudate nucleus, decreases in blood flow to regions of the striatum, and changes in dopamine transporter binding have all been described in the human striatum of those with ADHD (Tripp, 2007).

Abnormalities in striatal functioning and in the functioning of related association cortical circuits are widely attributed to ADHD pathophysiology. Remembering the functions of the basal ganglia to include adaptive behaviors, movement initiation, motor learning, motor control, sequencing, attentional allocation and filtering, and working memory (Menon, 2001), and given the interconnectivity between the frontal cortex and basal ganglia, it is hypothesized that the basal ganglia are involved in attentional/behavioral switching that lends to executive functioning. By being capable of reordering cortically derived information within the striatum, a more efficient, action-oriented, top-down representation of information may be produced (Graybiel, 1998).

A large number of observations in the human scans, both PET and fMRI, have found the basal ganglia to be active during the learning and execution of sequential behaviors (Biederman, 1998). The caudate nucleus in highly trained monkeys activates

during coding of learned eye and hand-eye movement sequences (Schultz, 1997). Relays between the striatum and thalamus are important for the performance of saccadic eye movements (Alexander, 1990). Hand-eye coordination and ocular tracking are noted deficits within ADHD populations. ADHD children consistently show deficits both in saccade and pursuit of the eyes as well as in the maintenance of steady fixation on a target (Gillies, 2000). During the learning of sensorimotor sequences, it is modeled that the prefrontal cortex architecture is designed so that each point in the sequence holds a unique pattern of activity (Gillies, 2000). Spacial working memory has been associated with the dorsolateral prefrontal cortex while the orbital and ventromedial prefrontal cortex allows recognition and inhibition of emotional responses important for appropriate behavior (Levy, 2008).

Stimulant medications used to treat ADHD increase dopamine within the nigrostriatal circuitry (Leckman, 1999). Primary neurochemical deficits relating to dopaminergic neurons in the nigrostriatal pathways include: executive deficits, difficulties in memory retrieval, visuospatial problems, exacerbation of stereotyped behaviors, disinterest, and environmental dependency. Mood-related symptoms also occur. Loss of dopamine is witnessed through a global reduction in learning rate, which leads to deficits in sequenced learning (Gillies, 2000). This suggests a close relationship between motoric responsivity and executive switching mechanisms.

In 1956, Miller coined the term "chunking" to convey the idea that efficiency in information content can be gained by recoding bits of information to form units of information. These units, once learned, are then treated as learned sequences or entities

of memory. A key neural substrate underlying this process is the basal ganglia (Graybiel, 1998). The slow kinetics of stimulus-response learning offer an advantage for chunking. It is not efficient to collapse all encoded sequences, or temporally ordered acts, into chunks. The behavioral chunk, once consolidated, may be difficult to break apart. Think of trying to stop signing your name halfway through your name or trying to stop reading a word mid-word. Neural coding of sequences appears to occur quickly and to be highly labile. Chunks take on advantage from being manipulatable entities. The intervention of conscious attention, or top-down attentional mechanisms, can alter the implementation of the sequence (Graybiel, 1998).

Disturbed caudate function in ADHD, Parkinson's disease, Huntington's disease, progressive supranuclear palsy, Wilsons's disease, Fahr's disease, and Tourette syndrome result in abnormal activation of the frontal lobes and thalamus via dorsolateral prefrontal and orbitofrontal circuits (Ring, 2001). Single axons from the globus pallidus output from the basal ganglia ipsilaterally, to the same side of the brain, and contralaterally, to the opposite side. The returning, afferent, contralateral projections from the cortex input to the putamen more strongly than those to the caudate. These projections from the cortex to the striatum appear to have important functional consequences. Intracellular recordings demonstrate that stimulation of both the ipsilateral and contralateral frontal cortex can produce monosynaptic, excitatory, postsynaptic potentials in the same cells within the striatum (Leckman, 1999).

Other Cortical Correlates

Global brain volume measured via MRI has shown to be around 5% less in ADHD children (Biederman, 1998). Neuroimaging studies consistently find abnormalities in the basal ganglia and global reductions in grey and white matter, which result in these smaller brain volumes (Rapoport, 2001). The basal ganglia-related imaging literature provides conflicting results. Decreased right caudate volume, reversed caudate asymmetry, decreased volume within the putamen, reversed putamen asymmetry, and decreased globus pallidus volume on one, either, and both sides of the brain have all been reported (Qui, 2009). An in vivo imaging study by Leckman (1999) presents data on the absent normal asymmetry of the caudate nucleus in ADHD patients. In controls, the right sided caudate is usually larger than the left caudate nucleus. In the ADHD subjects, a smaller caudate was observed on the right side. A review of basal ganglia and cortical findings from PET studies indicates decreased activity in the right thalamus, right caudate, right hippocampus, and cingulate gyrus in adult ADHD subjects (Zametkin, 1998).

Looking specifically at the cerebral cortex's morphology in cases of ADHD, Castellanos (2002) found decreased volume across all cerebral cortices. This supports cholinergic models of top-down processing as potentially diminished in ADHD cases. In a quantitative MRI study, Shaywitz et al. (1983) studied children and adolescents with ADHD and found the hyperactive group to exhibit greater sulcal widening throughout the cortex and atrophy within the cerebellum. These children also seem to have impaired metabolism in the frontal cortex and a smaller right prefrontal lobe. The right prefrontal white matter appears specifically diminished in children with ADHD,

which may be attributable to neurodevelopmental delay of the right frontal lobe (Overmeyer, 2001). Cortical volumetric differences have been repeatedly found in the dorsolateral prefrontal cortex and dorsal anterior cingulate cortex (Voeller, 2004).

Interhemispheric coordination appears essential for corticostriatal-thalamic-cortical circuitry. In ADHD, reductions in the size of structures containing interhemispheric axons connecting association, sensorimotor, and parietal cortices further support the symptomatic relevance of these interhemispheric projections. Such reductions have been noted specifically in the corpus callosum, genus and isthmus, and the splenium of ADHD individuals (Faraone, 2005). These cortical areas contain white matter tracts that facilitate regulation of motoric hyperactivity and inattention. Another investigation found hyperkinetic children to have significant gray matter deficits in the right superior frontal gyrus, or Brodmann's area 8/9, right posterior cingulate gyrus, or Brodmann's area 30, and the basal ganglia bilaterally (Overmeyer, 2001). Hypodopaminergic modulation of frontal synapses by the basal ganglia may impair the normal age related development of the gray matter (Rothenberger, 1997).

The role of the frontal cortex, and specifically the anterior cingulated gyrus, in emotional processing has been demonstrated in several imaging studies (Durston, 2003; Faraone, 2004; Rapoport, 2001; Seitler, 2006). These studies indicate an important integrative role for the orbitofrontal cortex and cingulate gyrus in analysis of information that carries emotive, evaluative, and in the long term, survival significance for an individual (Levy, 2008). Experimental evidence supports the idea that the anterior cingulate gyrus, in part, subserves executive functioning, and controls/organizes

visceromotor, endocrine, and skeletomotor behaviors. Lesions confined to the anterior cingulate gyrus can produce indifference, inattention, and disinhibition. The anterior cingulate has been identified in several human imaging studies as important in attentional processes (Leckman, 1999).

Subjects medicated with methylphenidate showed either no change in the basal ganglia or a decrease in activity of the right putamen. Subjects under the effect of dextroamphetamine had increases in right thalamic and right caudate activity or showed no change to the basal ganglia (Zametkin, 1998). Another study also showed decreases in metabolic rates of the striatum (Leckman, 1999). All of these investigations noted cortical and subcortical abnormalities appearing to be part of a fixed, rather than progressive or ongoing, process. Total and regional growth curves for ADHD subjects run parallel to normal brain curves (Rapoport, 2001).

In summary, the body of literature investigating possible neurobiological loci for ADHD is not conclusive, but it draws a picture of the involvement of the same structures known to control normal signal driven, or bottom-up, and cognitively driven, or top-down, mechanisms of a healthy attentional system, namely the fronto-striatal circuitry and the basal ganglia. These areas are associated with attention, motivation, motor sequencing, behavioral repertoire formation, and sequence execution. Abnormalities within these regions have been widely observed in brain regions of those with ADHD when compared to the brains of non-ADHD controls.

Developmental Theories

Many studies have shown smaller total brain volume associated ADHD which is manifest by a global reduction of cortical grey and white matter equally and subtle, inconsistent reductions in the basal ganglia. Longitudinal studies show the anatomical changes to be set and not part of an enduring, progressive pathology. It is also known that the abnormalities are not due to stimulant drug effects (Rapoport, 2001). The total and regional growth curves for ADHD brains parallel those of normal brain curves. Furthermore, normal age-related changes in brain morphology are not seen in children with ADHD (Castellanos, 2002). This knowledge leads to the hypothesis that observed anatomical differences must happen early in neurodevelopment.

The development of individual neurotransmitter systems, from gestation, through infancy and into adulthood, is influential on future behavioral capabilities (Rogeness, 1992). The process of development profoundly molds neuronal maturation. Efficiency within this maturation process may directly affect the future efficiency of the neurotransmitter systems, associated motoric capabilities, and behavioral outcomes. According to Barkley (2000), ADHD should be viewed as a developmental handicap.

The incremental nature of corticostriatal reordering depends on repetition of particular and coherent input patterns. Redundant formation of new associations and striatal inputs leads to an output activation that may underlie the gradual learning mediated by the basal ganglia. Dendrites and their synaptic inputs undergo postnatal maturation, providing the brain a fairly general mechanism for corticostriatal remapping,

development, and learning (Graybiel, 1998). How one matures can impact the mapping of and repertoire formation within these systems.

The basal ganglia and frontal cortex are thought to be involved in the building-up of sequential motor behaviors from movement elements. As a result of repeated pairing between stimulus and response, learning in this manner involves the property of slow acquisition. In humans, acquisition of this sort can occur without conscious awareness (Graybiel, 1998), such as the child naturally learning to reach for things, roll over, sit up, stand, and walk. A growing body of literature concludes that the basal ganglia participate in development and automatic execution of motor acts (Graybiel, 1997) that are far more specific than developmental motor behaviors.

In the macaque monkey, neurons of the caudate and putamen have been shown to fire selectively in relation to particular learned motor sequences. Neurons in the globus pallidus have been shown to behave similarly (Graybiel, 1997). The striatum, supplementary motor area, and related fronto-cortical areas are differentially activated in relation to the acquisition and expression of complex learned movements (Graybiel, 1995). Electrophysiological evidence suggests that as a monkey learns a new sensorimotor behavior through reward conditioning, there are systemic changes in the responsiveness of neurons in the caudate and putamen (Aosaki, 1995). In rodents, the dorsal striatum is essential for the execution of highly patterned grooming responses that are thought to be instinctual. Additionally, these oral stereotypes seen in the rodent can be induced by amphetamine administration (Berridge, 1992).

As discussed above, at the circuit level the basal ganglia are associated with brain structures believed to function in cognitive and motoric processing. Outflow from the basal ganglia is uniquely directed toward the frontal lobes. The basal ganglia receive their primary inputs from the frontal and parietal association areas. These characteristics indicate that the basal ganglia act in conjunction with the cerebral cortex to influence the activity states of such forebrain systems (Graybiel, 1997). The patterns of connectivity suggest that redundant patterns of activity could be established in basal ganglia loop circuits and their affiliated structures. As a consequence, the circuits could function to create and later express behavioral repertoires built up through experience. The patterns generated are organized in relation to future actions. They are not expressed physically in motions of the body, but rather are expressed cognitively. Graybiel's hypothesis states that activity-dependent, repetitive patterns are established equally in the motor system and brain regions subserving cognitive activity. With repetition, dominant modes of activation emerge.

This link between intent and action may also hold a specific developmental function. This set of circuits may prove part of the neural mechanism for building-up cognitive patterns involving recognition of the self. It has been well documented that voluntary motor behaviors develop as feedback. This was termed the "perceptuomotor world of infant development" by Gibson in 1969. The iterative nature of the basal ganglia connections and the apparent involvement of the basal ganglia in forms of sequential learning could provide a mechanism for development of self-awareness (Graybiel, 1997).

It has been argued that the striatum acts to mediate binding in the motor system (Graybiel, 1994). Temporally coordinated firing patterns can be set up in widespread regions of the striatum as a result of sensorimotor learning. This coordinated firing temporally aligns the activity in different motor circuits and thereby produces coordinated motor acts (Graybiel, 1997). Thinking, organizing perceptumotor information, planning, memorizing, and recalling memories depend critically on spatiotemporal coordination and sensorimotor processing capabilities.

The central nervous system has a very limited ability to regenerate but can reorganize the structures and connections it has available. This ability has been witnessed via the functional recovery potential following 6-OHDA lesions in adults and neonates using animal models (Kaneko, 2000). It is possible for the nervous system to compensate for deficits by recruiting novel systems to accommodate necessary functions. This neuronal compensation may utilize enduring and developmentally efficient repertoires or it may necessitate the forging of new behaviorally functional connections. The brains of the lesioned animals have been fundamentally altered in structure and function, forcing the animal to adapt in order to meet the demands of its environment.

Genetic differences or events that result in injury or maldevelopment of the basal ganglia and their connectivity to cortical sites of modulation are likely to exert a lasting impact on individual performance of various neuropsychological measures (Biederman, 1998). Whether an individual with ADHD is genetically predisposed to abnormal circuitry in the fronto-striatal networks, whether this circuitry is impacted by

environmental factors, or whether a combination of genetics and environmental influences play a role in the presentation of ADHD symptoms is still not entirely clear. Regardless of definitive etiology, there is consensus that ADHD can be regarded as a disorder marked by developmental handicap.

Developmental Movement Patterning

As was discussed in the developmental hypotheses section of this chapter, there is evidence from the psychological and child development literature favoring an action plan for movement over a reflexive method of motor development. This premise is mirrored and amplified in the somatics literature. Bartenieff (1997) writes about the simple action of a reach. If two individuals were asked to reach for something, the action itself may look entirely different, but the intention behind the action would be the same. The action can be executed in a potentially infinite number of ways, but the goal driving the action is often rather singular. These converging lines of literature lie at the center of the hypotheses driving this current research study. They imply that the goal is already represented when the action is planned.

Evidence from the neuroscience literature and basic neuroanatomy allows confirmation that areas such at the premotor area, supplementary motor area, and basal ganglia are active milliseconds prior to electromyographic recordings in performing muscle groups (Connor, 2004). Additionally, the supplementary motor area has been shown to be active when a movement is being planning or rehearsed in the mind but not carried out physically (Alexander, 1990; Durston, 2003). In the early writings of

Sherrington (Molnar, 2010), infant motor acquisition was considered reflexive, a simple reaction to a stimulus. Evidence is now showing a much more comprehensive action plan driving early movement behaviors.

Van der Meer (1995) carried out a study wherein infant spontaneous arm movements were measured while the infant was laying supine. In one condition, the room was darkened and a beam of light was cast next to the infant. Interestingly, the infants moved their hand into the light and controlled the position, velocity, and deceleration of their hand to keep it in the light. Furthermore, when the beam was moved the children moved their hands to stay in the light. The function of this early basic skill provides activity-dependent input into specific sensorimotor systems. This implies that the development of action and perception occur in tandem with the development of the nervous system. Each influences the other as the child matures and expands this behavioral repertoire to include sophisticated action patterns that can be used for solving equally sophisticated action problems. Van Hofsten suggests that, as one develops, actions become increasingly future-oriented and integrated. The kind of complex adaptivity needed in daily living engages the coordination of systems supporting cognitive, behavioral, and motoric action planning.

Motivation is a key feature in the idea that actions are purposeful versus reflexive. Motivation marks the developmental movement process from the somatic point of view (Hanna, 1996). For example, before a child has mastered the ability to reach for and grasp for an object, they will try and fail repeatedly. The confirmation of this activity would not take place in the absence of motivation. The child is acting with

a goal to reach for a desired object. It has been shown that once a child can reach and grasp, the child will grab onto a ball differently depending on whether the goal is to throw the ball, or to place it is a hole (Todd, 1977). Again, the goal is the motivator of the action.

Attentional skills and self-regulation emerge around the end of the first year of life and continue to mature toward the preschool and school years (Reebye, 2009). During the course of most neurodevelopmental disorders, it is regularly observed that motor development is also impacted. Fine motor, bimanual, contralateral, and visuomotor skills are not mature and efficient until adolescence. This coincides with the complex cognitive operations of representation, flexibility, working memory, and handling multiple aspects of information that also begin to flourish during the adolescent time (Diamond, 2000). Children who fail to achieve behavioral and attentional self-regulation within these critical periods often find themselves having trouble adapting to academic functioning in school, daycare, and/or at home.

Diamond (2000) reported on the close trajectory of both prefrontal cortex and cerebellar development. She observed that most cognitive tasks require activation of the dorsolateral prefrontal cortex and the cerebellum. As discussed in the sections covering pathological neurobiology of attention, the ADHD imaging literature shows reductions in cerebellar and caudate volume in ADHD children over controls. It has been well established that the cerebellum is responsible for coordination of fine motor, ballistic, and core/vestibular control (Kandell, 1991). The cerebellum is also needed for tasks that require close attention for new learning (Diamond, 2000).

ADHD children have been inconsistently found to have problems associated with key cerebellar functions. They often have trouble with balance, alternating movements, consistency and/or distance of timing movements, handwriting, and representations of fine motor skills (Piek, 2004). Diamond summates her review by stating that the cerebellum plays a role in cognition as well as its known and accepted role in motor function, and that the prefrontal cortex may play a role in motor activities along with its known functions in the cognitive realm. When there are vulnerabilities, either environmental or genetic, that affect the motor system or the cognitive system, it is likely that both systems will ultimately be impacted. Information covered in detail in the pharmacology and pathological attention sections of this chapter suggests a possible etiological role of developmental deficit or delay in the prefrontal cortex level seen in ADHD. This has come to light via the advent of specific noradrenergic therapies, such as guanfacine, which raise the question of the role of working memory in ADHD (Levy, 2008).

Somatic Views on Developmental Movement

There is an abundance of anecdotal evidence, case studies, and phenomenological-based somatic techniques supporting the premise that inefficiency of motor abilities relates to cognitive inefficiencies. Coming from the field of education, educational kinesiology claims to impact the cognitive system through practice and subsequent improvement of the motor system. As discussed in the introductory chapter, an interesting discrepancy between the somatic education community and the

neuropsychology/medical community is their respective approaches and interest in laterality. The somatic community regards laterality as a developmental process that takes place as the child ontogenically moves from homolateral (left sided, right sided) movement to contralateral (coordination of opposite arm to leg and crossing of midline) movements. The latter is considered to be the most mature of all developmental motor and locomotor patterns. Contralateral movement is regarded as important to learning and cognition because it requires the simultaneous recruitment of multiple neurological loci from both hemispheres of the brain to complete (Dennison, 1989).

The somatic theory asserts that the fundamental movement patterns should be acquired in a chronological fashion with the establishment of the prior movement patterns forming a foundation upon which more complex movements can be efficiently built. The patterns begin with navel radiation, which is seen in utero. Next mouthing patterns emerge, which allow for suckling and the establishment of prespinal patterning at the occipito-atlas articulation. Postnatally, and once spinal reaching and pulling capabilities have been established, the infant will discover that they have a top half and a bottom half to their bodies. At this point the child has understood homologous movement. Following homologous movement acquisition, the child will discover homolateral movements. This point marks the beginning of laterality within the child's developing neuromuscular repertoire. Homolateral movement is confined to coordinated movements of just one side of the body at a time. Finally contralateral movements will appear and the child will begin moving opposite leg to arm. This contralaterality should be redundantly integrated into the neural connectivity within the

developing child (Hartley, 1995). From the onset of crawling and creeping during infancy, the child will progress to walking, running, jumping, galloping, sliding, hopping, leaping, and eventually to skipping. Skipping is the most complex of all developmental locomotor patterns. Skipping indicates mastery of contralateral movement integration and acquisition because it is the last of the developmental locomotor patterns to develop (Summers, 1992).

The medical community, on the other hand, regards motor development through a set of developmental milestones that take place as a series of marked events such as rolling over, sitting up, standing, and walking. The Western medical and somatic systems of describing sensorimotor development overlap tremendously and could easily be combined into one cohesive conversation concerning motor development. A primary discrepancy is the difference between "bimanual movement," or "alternating movements" often seen in medical literature, and "contralateral," referred to in the somatic literature. Bimanual and alternating tasks are less developmentally mature than contralateral ones. According to Dennison and educational kinesiologists at large, bimanual movement and alternating movement tasks, as seen in the medical literature in the form of hand crank tests or peg board tests, often represent homologous and homolateral movement patterns, both of which precede and are less mature than contralateral acquisition.

If, in fact, cognition and motor development share common neurological trajectories and substrates, the inability to obtain contralateral fluidity could be associated with diminished cognitive fluidity and/or diminished cognitive abilities could

correlate with diminished contralateral movement deficits. The somatic evidence indicates that improving contralaterality improves a child's performance academically. The first hypothesis of this current study aims to take a first step toward evaluating these claims by specifically investigating if there is a contralateral deficit in children with ADHD. The second hypothesis of this study addresses whether stimulant medications alter the presentation of the child's movement abilities. If contralateral fluidity is developmentally delayed in ADHD children, the addition of medication should not yield a profound improvement in the child's movement performance; if the child never integrated contralaterality into their movement repertoire, medication should not elicit a movement they were not capable of performing prior to medication. If this hypothesis is wrong, children may show improved contralateral movement capabilities when medicated, as the literature shows they do in other types of movement measures, due to an enhanced ability to focus on movement execution and performance.

Contralateral Movement Evaluation in ADHD

Gillberg (2003) conducted a longitudinal study and found children with ADHD-motor dysfunction diagnosis to have a history of prenatal neuropathologic risk factors, such as small and large gestational age and prematurity. Children in this group also had a history of delayed sophisticated motor skill development. Gillberg recommends evaluations for gross motor assessment during psychological diagnosis of children with suspected ADHD. He reasons that observation of gross motor deficits may lead to differing intervention possibilities. Landgren et al. (2000) assert that future studies

should develop and evaluate instruments that assess motor functioning without warranting an expensive and time-consuming formal psychometric approach. Research goals of this current contralateral movement study are to implement The Ohio State University Scale of Intra-Gross Motor Assessment, or OSU SIGMA (Loovis, 1979) and evaluate its efficacy as such a tool, and to introduce the concept of contralaterality to the psychiatric/medical literature in an effort to bridge the academic gaps between somatics, education, and healthcare.

The OSU SIGMA was originally developed to evaluate specific gross motor skills. These skills included walking, climbing stairs, running, throwing, catching, kicking, jumping, hopping, skipping, striking, and ladder climbing (Loovis, 1979). Each motor skill was then assessed along four descriptive levels of skill acquisition. This study evaluated the skill of skipping because skipping requires contralateral coordination and balance and is the last chronological locomotor pattern to develop, making the skipping scale a good indicator of contralateral movement ability. Kindergarten children who have not yet developed the pattern of skipping will tend to gallop, run, hop, or slide. Older children, unable to skip, tend to use only one side of their body, thus changing the movement into a unilateral one (Frego, 1998). Studies setting out to determine the appropriate age level for successful acquisition of motor activities indicate that fundamental locomotor patterns are essential for learning and for development (Summer, 1992).

Roberton (1984) described skipping as a bilateral, sequential movement in which each foot performs a walk and a hop before the body's weight is transferred to the other

foot. The stepping action takes twice as long as the hop, and the arms move reflexively in opposition to the feet (Frego, 1998). Wellman et al. (1938) carried out the first kinesiological research into contralateral acquisition and skipping capability (Wellman, 1937). In that sample of 98 children, most subjects could skip with both feet by the age of five. The motor age determined the age at which half of the same-age children could display acquisition and efficiency of the given locomotor pattern. A review by Frego (1998) adds that females acquire the developmental capability to skip prior to that of matched males.

Ulrich (1985) outlined precisely what is necessary to determine efficiency or fluidity of the skipping pattern: 1) feet must alternate in a rhythmic step-hop combination, 2) the non-weight bearing foot is held near the ground during the hop, and 3) arms must move alternately at the waist level in opposition to the legs. Loovis and Ersing developed the OSU SIGMA to not only evaluate motor tasks but also to describe the movements being used by those who cannot yet skip. The OSU SIGMA appears to be the most comprehensive contralateral movement assessment that the literature has to offer providing accurate and replicable labeling to observable movement behaviors (Frego, 1992).

When viewing movement deficits with such a rating scale the implications for therapeutic interventions look promising. While skipping is a developmental movement pattern, it is also possible to teach skipping through the proper activities. Graybiel (1998) predicts that the striatal projection neurons would only gradually change their firing patterns during active, participatory, sensorimotor behavioral training. Frego

(1992) asserts that with guided practice, the child can learn to use the correct sequence of steps and hops that will eventually lead to skipping. With further repetition, the movement will become fluid and incorporate the contralateral upper limb. It is unknown if such motor learning and motor efficiency can actually improve attentional or cognitive performance. The first hypothesis of this current contralateral movement investigation seeks to begin investigating this unknown by learning if a specific contralateral motor deficit is even observable in children with known attentional issues when compared to neurotypical peers. If a significant contralateral deficit is witnessed, this knowledge will help set a foundation for future studies looking more specifically at improving motor abilities with an intent to improve attentional and cognitive outcomes.

ADHD Assessment

There is no solitary diagnostic instrument that currently meets the needs of all investigators and clinicians working with ADHD. Relatively abbreviated forms are now available that contain the psychometric reliability of the longer and more tedious types of prior testing measures. Commonly utilized instruments, and those used by physicians participating in this study, are the Child Behavior Checklist (CBCL) by Achenback, the Parent and Teacher Rating Scales by Conners, and the Vanderbilt Parent and Teacher Rating Scales. These scales have defined clinically important cut-off points as the

extremes of bell curves, typically 1.4 to 2 standard deviations about the mean or the top 93%-98% of the distribution. Parent scales, teacher scales, and child self-report scales are incorporated together, or in part, to obtain diagnostic information. Additionally, it is important to gather diagnosis information from both home and school settings. There must be impairment present in both settings to confirm an ADHD diagnosis.

The diagnostic process for identifying ADHD individuals is dependent on obtaining a careful history and providing enough time with the child for observation. ADHD assessment can take place over many visits with a clinician or would minimally require one hour for an initial assessment (Greenhill, 1998). Categorical as well as dimensional data must be gathered about each individual. Ideally, the parents and the teachers are available for input and diagnostic benefit. Rating scale information is useful and relatively accurate but not meant to stand alone as the primary tool for diagnosis (Conners, 1998). Children's self-reports of their behavior tend to be less reliable. Symptoms are not always readily present in novel or highly structured situations, making them perhaps difficult to observe during doctor appointments or clinical visits. This makes the use of parent and teacher rating instruments valuable in the diagnostic process (Rowland, 2002).

Continuous performance tests, or computerized measures of attention and impulsivity, are often used but they lack adequate sensitivity and/or specificity (Hales, 2002). Some researchers have tried to quantify ADHD behaviors using systematic observation of "off task" behavior (Greenhill, 1998). Others have tried using mechanical devices to measure leg movements or restlessness (Rowland, 2002).

Measurements of catecholamines or their metabolites in plasma and urine have been implemented with mixed success (Plizka 1998). Despite these efforts to find a solitary, reliable, and valid diagnostic tool, the current consensus, as stated in the DSM-IV, is that no single test available acts as a valid and/or reliable predicator of ADHD. Given the nosology dilemmas surrounding ADHD, there still exists the need for broader and more precise rating instruments. This is especially true in the face of comorbidities that effect both diagnosis and response to treatment (Conners, 1998).

The ADHD sections in the 1987 and 1994 DSM acknowledge the heterogeneity of the disorder by differentiating between presentation with and without the hyperactivity component. The DSM-IV created further separate classifications for children who are primarily inattentive (ADHD-PI), children who are primarily hyperactive and impulsive (ADHD-HI), and a combined category (ADHD-C) for children that are both inattentive and impulsive with hyperactivity (Rowland, 2002). Earlier versions of the DSM allowed ADHD to be present in just one setting, but the current DSM-IV requires that the symptoms and impairment must be present in at least two settings, or "pervasive." Additionally, symptoms must be present for at least six months to be associated with a "clinically significant" impairment (APA, 2000). Diagnosis of ADHD should be made in a developmentally appropriate context with consideration to what would be expected of a child at the same age and cognitive level (Rowland, 2002).

Most medical prescriptions are written by a small number of doctors with 88% being written by primary care physicians and pediatricians (Swanson, 1995). Research

has revealed that many primary care physicians do not use the DSM-IV criteria when evaluating for ADHD. The Pediatric Research in Office Settings (PROS) network reported that the American Psychiatric Association's DSM criteria were only used by 38% of the 3900 clinicians surveyed (Wasserman, 1998). The other 62% were presumed to be diagnosed using "clinical intuition" or some other non-standardized form of assessment. Growing numbers of children being prescribed stimulant medication to treat ADHD elevates concerns as to whether the disorder is being properly diagnosed. Current recommendation guidelines from the American Academy of Pediatrics do suggest the use of DSM-IV-based instruments and criteria when assessing for ADHD (Rowland, 2002). Multiple and various assessment measures along with HMO pressure to keep patient visit time to a minimum cause great inconsistencies in diagnostic assessment protocols and subsequent treatment regimes (Swanson, 1995).

ADHD Treatment

Despite the lack of concise understanding supporting the structural and etiological basis of ADHD, it is one of the most well treated childhood disorders. Nearly three decades of literature and clinical experience support the short-term effectiveness of behavioral and pharmacological interventions to ADHD. Dysfunctions within the catecholaminergic neurotransmitter system involving norepinephrine and dopamine are centrally involved in the pathophysiology of ADHD. Modulation of

dopaminergic and noradrenergic neurotransmission by stimulants and norepinephrine uptake inhibitors appears to be a necessary mechanism for effective treatment of ADHD (Banaschewski, 2010). Considering the complicated and chronic nature of ADHD, the efficiency, availability, and cost of various ADHD treatments are of importance.

Stimulant Medications

Support of stimulant medication for children who have ADHD symptoms with and without other comorbid conditions converges from many studies as a primary treatment strategy (Banaschewski, 2010; MTA, 1999a; MTA, 1999b). Evidence supporting other methods of treatment is weak compared to the robust literature supporting stimulant treatment of symptoms. The Multimodal Treatment Study of ADHD was a multi-site clinical trial designed to determine the relative benefits of three treatment modalities, which were pharmacotherapy, psychosocial treatment, and the combination of the two. Pharmacotherapy alone or pharmacotherapy in combination with behavioral treatments significantly reduced ADHD symptoms and impairment (MTA, 1999a), however children with comborbid internalizing problems like anxiety or externalizing problems like aggression were more likely to benefit from behavioral treatments than children with only ADHD (MTA, 1999b).

Although stimulant medication treatment is generally required for the duration of the disorder, psychosocial treatment may or may not be required for long periods of time. This is because, unlike ADHD, the comorbid conditions that accompany it may respond to short-term interventions or treatments that are delivered on an as-needed

basis. Treating these comborbid conditions may facilitate ADHD medication's ability to exert its full benefit. In the MTA, the group treated with behavior therapy plus medication had the same reduction in ADHD symptoms as the group treated with medication alone. However, this treatment was able to achieve the benefit with doses of medication that were approximately 20% lower.

There are reasons to cautiously interpret the MTA and other studies demonstrating efficacy for medication-only treatment. First, the MTA and other treatment studies usually look at DSM-IV combined subtype ADHD, leaving the efficacy of medication and psychosocial treatment with other subtypes largely unknown. Second, the MTA was a clinical trial where medication was titrated and maintained using data obtained on a regular basis from many predictable and reliable informants with close monitoring and compliance (MTA, 1999a). This is a very different situation from standard prescription and monitoring practices seen in the real world where suboptimal doses are frequent and inconsistent administration of medications is common. In real world situations, additional interventions may be necessary to see the same alleviation of symptoms. Third, stimulant medications are not well tolerated by all children; they can cause sleep disturbances, loss of appetite, and anxiety in some children. For other children, the use of stimulants is unsuitable or ineffective. All of this leads to approximately 20% of children with ADHD who cannot benefit from stimulant medication (Rowland, 2002). Some of these children may benefit from other medications, but psychosocial treatments may also be beneficial and are often overlooked as a second line of defense in the treatment of ADHD.

Estimates indicate that around 2% of all school-aged children in North America receive pharmacological treatments for hyperactivity. More than 150 controlled studies of psychostimulants, observing more than 5000 children, adolescents, and adults have been conducted. The majority of these studies represent a subject population of latency-age, Caucasian boys undergoing short treatment periods (Swanson, 1995). These studies have documented the transient efficacy and safety surrounding psychostimulant intervention, but these same studies have also reported as many as 30% of ADHD children who consistently do not respond to these drugs (Biederman, 1998). Additionally, others are unable to tolerate potential side effects such as decreased appetite, sleep disturbances, mood lability, and exacerbation of comorbid tic disorders (Banaschewski, 2010). For these reasons, other non-stimulant drugs have been investigated and implemented for use in the management of ADHD.

The large number of children being treated with stimulant medication is of concern to the general public and to public health officials. In 2002, over 1.5 million children took methylphenidate, or Ritalin, to treat ADHD. There was a 2.5-fold increase in the number of children treated with stimulants between 1990 and 1995 (Hales, 2002). Two community-based studies indicated that in some parts of the United States, over 15% of all Caucasian boys in upper elementary grade levels were being treated with stimulant medications for their ADHD (Rowland, 2002).

The number of individuals being treated with psychostimulants forces one to question the underlying cause of this rise in children needing medication. Two time-trend and population-based studies from four pharmaceutical databases indicate that a

2.5-fold increase in methylphenidate treatment appeared due to increasing duration of pharmacological treatment within individuals with prior diagnoses. Additionally, the rise was attributed to greater numbers of adolescents, girls, and inattentive children being placed on medication. Parental attitudes toward medication, support at the school level, prescriptions extending to adults, preschoolers, learning-disabled children, and conduct-disordered children with an ADHD diagnosis also contribute to the increase in prescriptive trends (Swanson, 1995).

Zametkin (1987) reviewed the history of pharmacological ADHD interventions and ascertained that no singular neurotransmitter can be responsible for the presentation of symptoms seen with ADHD. Rogeness et al. (1992) reviewing the roles of dopamine, norepinephrine, and serotonin in ADHD concluded that the balance between the norepinephrine and the dopamine systems is critical. This interaction is more important than variation within any singular neurotransmitter system. Effective pharmacological treatments for ADHD share a noradrenergic and dopaminergic mechanism of action (Biederman, 1998). As such, recent directions in the treatment of ADHD have involved both a broadening of pharmacological perspectives to include noradrenergic as well as dopaminergic agents (Levy, 2008).

Stimulants continue to be the first-line drug of choice for uncomplicated variations of ADHD in all ages. Levy (2008) refers to work by Seeman and Madras suggesting that methylphenidate blocks the dopamine transporter, resulting in increased extracellular dopamine, activating autoreceptors, and leading to an attenuation of dopamine release in response to phasic dopamine firing. In this theory, stimulants raise

extracellular levels of dopamine several-fold but reduce the extent to which dopamine is released with nerve impulses compared with the impulse-associated release observed in the absence of the drug. Levy goes on to summarize a second hypothesis proposed by Volkow suggesting that the blocked dopamine transporter overcomes the inhibitory effects caused by activation of the autoreceptors, leading to a net effect of dopamine accumulation in the synapse, which amplifies dopamine signals resulting from tonic as well as phasic dopamine firing (Levy, 2008).

There have been increasing concerns with the older, immediate release preparations of stimulants. Adherence can become an issue due to the need for administration of the medication multiple times during the day. Second doses often need to be given at school, creating potential privacy issues and opposition by children unwilling to take medication at school. Both can lead to missed doses. Concerns also exist regarding the peaks and troughs of stimulant blood levels occurring at the most unstructured times of day when symptom control is most needed. Implications of these variations in blood levels over a long-term perspective as well as the occurrence of the rebound/relapse phenomenon in the evening have spurred interest toward improving immediate release stimulant preparations (Hosenbocus, 2009). Clinicians had to constantly adjust doses to help children function optimally throughout the entire day, including important evening hours for homework, after-school activities, and family time. These problems with immediate release preparations led the pharmaceutical industry to focus on development of more long-acting, once-a-day stimulant

preparations. Commonly prescribed long-acting stimulant medications include Concerta, Adderall-XR, Vyvanse, and Daytrana.

Non-Stimulant Medications

It has been accepted in the past that the beneficial effects of stimulant medications are mediated simply by dopamine transmission, but a more recent suggestion growing from the success of drugs such as atomoxetine, or Straterra, and guanfacine is that the effects are mediated by the alpha-2 adrenoreceptor. Alpha-noradrenergic agents such as atomoxetine, guanfacine, and clonidine represent the more recent pharmacological options for ADHD treatment. Levy (2008) cites Arnsten et al. as having described three different subtypes of alpha-2 adrenoreceptors in humans: the alpha-2A, alpha-2B, and alpha-2C subtypes. The alpha-2A and alpha-2C are the most widely distributed in the human brain, including within the prefrontal cortex. The alpha-2B receptor is most concentrated in the thalamus.

Guanfacine is thought to be the most selective agonist available for the alpha-2A subtype. The sedating effects of clonidine are thought to involve the thalamus, basal forebrain, and other alpha-2B and alpha-2C effects. Atomoxetine, which also has sedating effects in some children, may also have alpha-2B and/or alpha-2C receptor effects (Levy 2008). Atomoxetine likely improves response inhibition via impact to ascending norepinephrine systems. Atomoxetine is a highly selective inhibitor of the presynaptic norepinephrine transporter with a low affinity for serotonin receptors and dopamine transporters (Banaschewski, 2010).

These drugs may act to protect the prefrontal cortex cognitive functions during stress by preventing excessive norepinephrine or dopamine release in the prefrontal cortex (Hosenbocus, 2009). Guanfacine likely acts on the prefrontal cortex, probably postsynaptically, at alpha-2A receptors, to increase cognitive and associated functions. Guanfacine has also been shown to be beneficial in the regulation of locomotor activity via inhibitory control of subcortical brain regions, particularly the caudate, putamen, and nucleus accumbens (Levy, 2008). Atomoxetine and guanfacine may prove to be more protective under conditions of stress wherein high doses of stimulants may impair cognition and induce perseverative or restricted thinking (Levy, 2008). Clonidine has often successfully been used in combination with stimulants for the treatment of sleep disturbances, aggression, and tics associated with ADHD, rather than as monotherapy for ADHD treatment (Banaschewski, 2010).

While some evidence pertaining to atomoxetine and guanfacine is encouraging, there are concerns relating to suicidal thoughts in a small number of patients, raising questions about the actions of atomoxetine, which cause elevated levels of norepinephrine and dopamine in the prefrontal cortex. The selectivity of atomoxetine for norephinephrine was investigated by Bymaster and summated by Levy (2008). These findings showed that atomoxetine increased extracellular levels of norepiniphrine in the prefrontal cortex 3-fold but did not alter serotonin levels. Atomoxetine also increased dopamine concentrations in the prefrontal cortex 3-fold but did not alter dopamine in the nucleus accumbens of the striatum. In contrast, methylphenidate increased dopamine and norepinephrine equally in the prefrontal cortex, but also

increased dopamine in the striatum and nucleus accumbens to the same level. These findings suggest that atomoxetine is less likely than traditional stimulant medications to have motoric or abuse liabilities associated with use of the drug (Hosenbocus, 2009). The relative selectivity of the dopamine increase induced by atomoxetine, in contrast to the more widespread increases in catecholamines caused by stimulants, may explain this lack of abuse potential due to the lack of increased dopamine levels in the reward-mediating nucleus accumbens. The lack of effect on dopamine in striatal motor areas may explain the decreased motor effects, such as unwanted tics, seen with atomoxetine versus those seen with stimulant medications (Banaschewski, 2010).

Tricyclic antidepressants, such as Desipramine, Imipramine, and Nortriptyline, have been demonstrated as efficacious in the treatment of ADHD and are often used as a third line of treatment following stimulants and atomoxetine. The assumption is that their activity in ADHD stems from their actions on catecholaminergic reuptake. Although studies comparing tricyclic antidepressants to stimulants have been generally inconclusive, the conclusion can be drawn that tricyclic antidepressants are superior to placebo but not as effective as stimulants in the treatment of ADHD symptoms (Banaschewski, 2010). Advantages include their relatively long 12-hour half-life, which eliminates the need for dosing after school hours, the absence of abuse potential, and the positive effects on mood, anxiety, sleep, and tics. An additional, unlicensed, medication used in ADHD treatment is Bupropion. While also an antidepressant, it is of the aminoketone class. This drug is not licensed for use in ADHD but is also used as an additional line of treatment. It has noradrenergic, anticholinergic, and indirect

dopaminergic effects. Buprioprion appears to be less effective than stimulants, but studies have shown it do be more effective than placebo and well tolerated. Use of Buproprion may exacerbate tic disorders (Banaschewski, 2010).

Factors in Treatment

Side effects are important considerations in the prescriptive practice of treating ADHD. The short-term side effects of the psychostimulants are well described and appear to be dose related. The most common of the adverse side effects reported with methylphendidate include anorexia, stomachaches, insomnia, headaches, and irritability. Due to the sympathomimetic properties of stimulants, other possible side effects include increased blood pressure, elevated pulse rate, and other cardiac-related problems. Rare cases of leukopenia and psychosis have also been reported. Reebye (2009) reviews concerns of parents related to psychostimulant use in young children and sites the 2004 MTA study, which found changed growth patterns in children receiving psychostimulants. During the first two years of psychostimulant therapy, an approximate height deficit of one centimeter per year was reported. The study also described beneficial growth-related effects to height velocity associated drug holidays, or periods where a child is intentionally not taking ADHD medication. Psychostimulants are, in fact, the most prescribed agents in pediatric psychopharmacology (Findling, 1998). They are generally considered safe and effective for ameliorating the symptoms of ADHD.

Taken together, this information clarifies that there are specificity and tolerability differences amongst the medications used to treat ADHD. This makes the challenge of honing in on the appropriate and most effective treatment for each of the ADHD patients in the heterogenous pool as complex an issue as the etiological and diagnostic ambiguities already discussed in this chapter. Stimulants have a broad effect on vigilance and motor impulsivity. Guanfacine is likely to be the most specific for the treatment of attentional deficits alone. Atomoxetine has effects on attention, social affect, anxiety, and sedation.

The consensus finds that clinicians should continue to follow the rule that medications should not be prescribed in isolation without concurrent psychotherapy and psychosocial interventions, including advocating for adaptations in the child's environment. Tervo (2002) asserts that future studies need to assess the influence of hyperactive, impulsive, and inattentive behaviors on motor functioning along with the degree that fine motor and gross motor skills improve with medication management. This current study hopes to add information specifically about contralateral movement abilities to this conversation. Despite the advances being made to pharmaceutically treat ADHD, many children continue to suffer from the complications of the disorder and long-term follow up of medication regimens is critical. The overreaching clinical goal of treatment is sustained remission in such a way that children can be consistently free of impairing ADHD symptoms, be more available to benefit from other treatment modalities, and enjoy a better quality of life (Hosenbocus, 2009).

CHAPTER 3: PROCEDURES

Research Design

In design, this study is non-experimental, non-randomized, and ex post facto, or causal comparative. Quantitative research of this kind is similar to an experiment, except that the variables are not manipulated because they have already occurred in the natural course of events. In this case, the dependent variable is contralateral movement ability. This developmental movement ability represents an attribute variable. Contralateral movement ability is a variable on which the subjects naturally differed before the study began; maturity of contralateral movement ability is a characteristic that all subjects had before they entered the study. The Ohio State University Scale for Intra-Gross Motor Assessment (OSU SIGMA) operationalizes the concept of contralateral movement. The independent variables are ADHD diagnosis and subsequent diagnosis. These too are attribute variables on which the subjects already differed before the study began. These variables were not manipulated during the course of the study.

The early work of Wellman (1938) established six years of age as the motor age for acquiring the developmental movement of skipping. For this reason, boys aged seven to nine years, upon enrollment to the study, were sought as subjects. By this age,

the variable of interest—contralateral movement observed via skipping—should have already been determined by the natural course of development. This ex post facto research design was implemented to investigate the cause-and-effect relationships between the independent variable of contralateral movement, and the dependent variables of ADHD diagnosis and medication for ADHD. This design had to be used in order to ask the questions underlying this study because randomization and manipulation of the variables under scrutiny are not possible due to their attribute nature.

Ary (2002) states that there are two basic modes of ex post facto research: 1) beginning with subjects who differ on an independent variable and trying to establish the consequences of these differences, and 2) beginning with subjects who differ on a dependent variable and trying to determine the antecedents of this difference. Subjects were recruited into either the experimental group, containing twenty-four boys with ADHD diagnosis, or the control group, containing twenty-five boys confirmed to not have ADHD. Each boy was then recorded skipping on three different occasions. The experimental group of boys skipped twice while on their normal medication and once after a drug washout period to obtain a non-medicated recording. An educational kinesiology expert who was blind to the group assignment of the boys then ranked these recordings.

Non-experimental, ex post facto designs can provide valid and convincing information, but five elements are necessary for such a design to lead to reliable inferences: 1) the treatment must occur naturally, 2) the subjects must provide investigators with valid observations relating to the biological question, 3) the natural

history of the disease, its standard therapy, and its course in the absence of medical intervention must be well-known, 4) the effect of the independent variable must be large enough to overshadow bias and random error, and 5) evidence of efficacy must be consistent with known biological knowledge (Piantadosi, 2005).

The variables and hypotheses in this study meet each of these five criteria: 1) all variables are attribute in nature, and contralateral movement is a developmental phenomenon found to be established by the age of six, 2) the OSU SIGMA has been validated using inter- and intra-rater reliability to assess skipping behaviors in children, 3) the natural history, therapy and course of ADHD are well established, 4) the difference between the experimental and control groups divided by the standard deviation of the control group is large enough to establish significant effect size, and 5) known biological knowledge supports the questions involved in this study as efficacious.

The study consists of three separate phases. Each phase takes the form of a static group comparison between OSU SIGMA ratings for boys diagnosed with ADHD in the experimental group and boys in the control group confirmed to not have ADHD. A static group comparison provides for a design in which a group that has experienced *X*, the experimental group diagnosed with ADHD diagnosis, is compared to a group which has not experienced *X*, the control group confirmed to not have ADHD, for the purpose of establishing the effect of *X*, or ADHD diagnosis. One of the phases allows for a comparison of the experimental group boys off of their medication and the other two phases compare medicated experimental group boys to the control group (see Figure 1).

The order of the phases varied for each boy in the experimental group based upon where they were in their diagnostic process, medication regimen, and convenience to the participating families surrounding the time of the 'drug holiday' wherein the experimental group boys were removed from their ADHD medication in preparation for the unmedicated recordings. Three separate static group comparisons create the basic design of the study.

X: *X* represents the diagnostic assessment(s) carried out to assess for ADHD. In the experimental group, these were physician-initiated assessments that took place prior to the subject's enrollment in the study. In the control group, ADHD was ruled out using the Parent Version of the Children's Interview for Psychiatric Syndromes, or P-ChIPS, (Weller, 1999). All experimental group children were confirmed to have ADHD via physician referral and diagnostic code. All control group children were confirmed not to have ADHD via the P-ChIPS structured interview.

O: This represents the recordings taken of each boy. There were twenty-five boys in the control group and twenty-four in the experimental group. Each boy was recorded three times yielding a total of 147 recordings. The videos were then randomly ordered and given to a collaborating educational kinesiology expert who entered into a data recipient agreement with the Institutional Review Board. This expert was blind to the group allocation of the boys as well as the medication state of the boys. She assigned OSU SIGMA ranks to each of the videos.

O1: *O1* represents the first medicated recording and assigned OSU SIGMA rank for each ADHD child in the experimental group.

O2: *O2* represents the second medicated recording and assigned OSU SIGMA rank for each ADHD child in the experimental group.

OU: *OU* represents the recordings taken of each experimental group boy while not under the influence of their prescribed ADHD medication. Some boys were drug naïve, or had never been treated pharmaceutically for ADHD, during this *OU* recording because it was taken at the end of their diagnostic process and before the onset of medication. Most boys underwent a physician directed drug washout period prior to this recording to clear their system of the medication prior to being recorded for the *OU* assigned OSU SIGMA ranking.

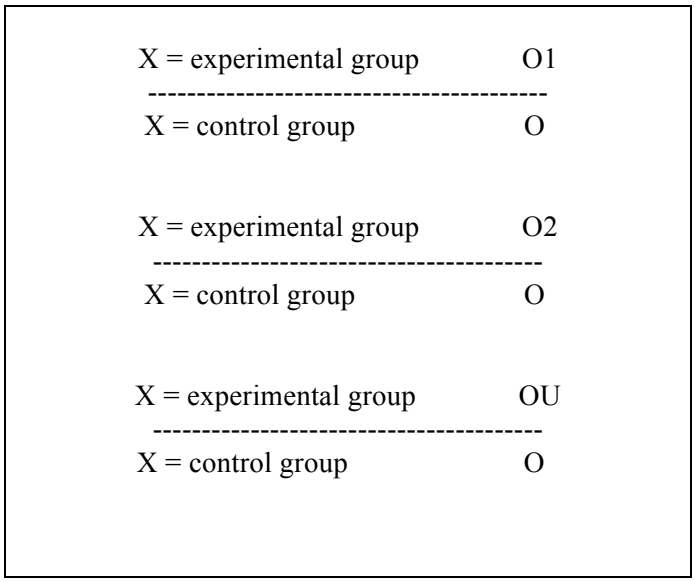

Figure 1. Schematic representation of research design

Internal and External Validity

The ex post facto design is well suited here because the independent variables, ADHD diagnosis, and subsequent medication are attribute. The level of the variables has already been selected naturally in the case of diagnosis and is manipulatable but will not be manipulated in the case of the medication variable. Due to this diminished control of variables and the ordering issue of separate static group comparisons, strong inferences about causal relationships cannot be formed.

Internal Validity

Campbell and Stanley (1963) state that internal validity is the basic requirement to draw correct conclusions from an experimental enquiry. Internal validity of a study

speaks to the extent that the observed differences of the dependent variable, here contralateral movement ability, are the result of the independent variable, here ADHD diagnosis and medication for ADHD, versus being the result of uncontrolled, extraneous variables. At play in this study are many confounding variables impacting internal validity due to the design; included are selection, mortality, maturation, differential selection, common cause, reverse causality, observer bias, and subjective attitudes of the subjects. In an attempt to eliminate alternative explanations from the findings discussed in chapter four, each of the internal validity threats presented by the design of this study were addressed within the design.

Static group comparisons allow for comparison of one group which has experienced X, or ADHD diagnosis, with another group that has not experienced X, or has not been diagnosed with ADHD. The purpose of such a design is to investigate the effect of X, or ADHD diagnosis. A primary concern with this type of design is that one cannot formally certify that the experimental and control groups would have been equivalent had it not been for the diagnostic variable. This leads to the confound of selection. If the experimental and the control groups differ, these differences could very well be through the differential recruitment of the subjects. The groups may have been different anyway regardless of the diagnostic variable.

Ex post facto experimental studies attempt to accomplish a pre-X, or pre-ADHD diagnosis, equation of the experimental and control groups by matching on pre-X attributes. This type of experimental design represents one of the most extended efforts toward quasi-experimental research methodology (Campbell, 1963). In an attempt to

limit pre-X differences, subjects were matched on age and sex. Only boys between the ages of seven and nine were recruited for this study.

Differential dropout rates make mortality another internal validity threat when using static group comparisons. This threat was controlled through measures attempting to make the study easy and convenient for families. Recordings were done at a time and place of the family's convenience. Many families chose to have the recordings done at their home, at the child's school or summer camp, at their church, local park, or coinciding with the boy's sporting event or other after-school activity. The drug washout period for the experimental group was made flexible, so it did not introduce an unnecessary burden to the family. Many of the unmedicated recordings were done on weekends or during school holidays. These attempts to make the study easy for the families were fruitful, resulting in all boys remaining in the study for the full duration of their three recordings. The study had a dropout rate of zero percent. Mortality was well controlled through the incorporation of this flexibility into the study design.

The choice to use males in studies investigating motoric abilities within an ADHD population is justified by Pitcher (2003) on multiple grounds. First, it is estimated that males constitute a greater percentage of children within the ADHD combined hyperactive, impulsive, and inattentive subtype (ADHD-C) and ADHD hyperactive/impulsive (ADHD-HI), subtypes, while females represent a higher proportion in the ADHD primarily inattentive (ADHD-PI) subtype. Second, social environmental and culturally influenced performance expectations have been found to influence motor-skill acquisition and identification of motor problems. Finally, sex

differences can also influence visual-spatial cue processing, vividness of movement imagery, and stimulus configuration. It is fitting with the trends in the literature to control for sex differences by only using male subjects.

Maturation represents another important threat to the internal validity of this study. While skipping is a developmental locomotor pattern that is usually acquired without being taught, it can also be taught and learned by children. Nearly everyone develops the ability to skip at some point throughout life. This maturation threat is controlled in part by the selection of seven to nine year old subjects. These children have matured beyond the established motor age of six years (Frego, 1998; Wellman, 1938) and therefore should already be able to skip upon enrollment into the study. Since the boys enrolled in this study have matured beyond the established motor age, it follows that these subjects are relatively mature as pertains to the contralateral movement, or skipping, variable.

To provide for further control of a possible maturation effect, the expectation placed upon each subject and the timing between the recordings were carefully controlled in the design for the study. The consent and assent forms referred to "looking at developmental movement skills, such as running, walking, skipping, or jumping." Upon each meeting, each boy was told, "today your activity will be skipping." This minimized the expectation that the child would be asked to skip upon each recording and hopefully also minimized any conscious or unconscious effort to practice the skipping skill between recordings. Furthermore, parents were told there was no right or wrong in their child's movement, and it was important for us to see them perform naturally in

whatever form they did the activities. Parents were told that it was important for them not to work with their sons on these named motor skills during their participating in the study. Since it is known that the ability to skip in a developmentally correct manner increases as children age (Frego, 1998), it was important to limit the passage of time between recordings, but at the same time to allow enough time to pass so that the study would not be at the forefront of the child's mind, causing him to practice the developmental skills. A time frame of no less than two weeks and no more than one month was established as a sound interval to pass between each of the recordings. Additionally, the chronology of the recordings was taken into account through the statistical model to reveal any effect of time passage if one was present.

Since the ADHD group was drawn from a clinical population and the non-ADHD group was recruited from the community and local elementary school population, the threat of differential selection also needed to be controlled. It is important to know if the groups are different on extraneous variables that could influence contralateral movement. As has already been addressed, contralateral ability is generally accepted as a developmental skill that matures in all healthy children along a known trajectory. Because of the established attribute, developmental nature of the contralateral movement variable lending the knowledge that most children are able to skip fluidly by the age of six years, and the fact that this internal validity threat has been controlled via design narrowing the age range of subjects, the demographic features become less important to control.

Control instead focused toward confounds that could impede normal motoric development and/or injuries that would prevent normal execution of motoric abilities. Collecting information about confounding variables that could influence contralateral movement abilities can decrease differential selection concerns (Campbell, 1963). Children with a background in athletics or dance may have learned how to skip in their training, which could positively influence OSU SIGMA ratings. This was controlled through a questionnaire that was given to parents following consent to enroll in the study. Also included in this survey were questions about developmental milestones such as crawling and walking behaviors of the participating children. Because these behaviors could also influence later contralateral abilities, they were built into the study through the survey and effect was investigated using analysis of variance. Children with injuries or neurological disorders were excluded from the study.

In the hypothesized presence of contralateral deficits in the ADHD group, it was not asked nor potentially learned through this study whether ADHD could cause the deficit, whether the immature developmental continuum of movements cause ADHD, or whether a completely separate factor influences both ADHD and developmental movement capabilities. The developmental questions driving the investigation make it impossible to establish a method of time order of the variables. Contralateral movement maturity is not recognized until around the first grade. By this time many children have already been diagnosed with ADHD (Biederman, 1998). The pathophysiology supporting delayed contralateral execution may precede ADHD diagnosis or the neurobiology supporting ADHD may precede acquisition and execution of

developmental movement patterns. The psychobiology supporting diagnosis and movement could be one in the same or there could be a separate unknown third variable impacting both diagnosis and movement ability. This study asks only the presence of a relationship, not the direction of that association.

This problem of common cause introduces threats to internal validity. Common cause allows for the possibility that both ADHD and contralateral movement deficits are separate results of a third variable. A common causal pathway linking the heterogeneous aspects of ADHD is a goal for many ADHD investigators, but this study seeks only to clarify if contralateral deficits are predictably associated with ADHD and if there is an effect of medication to contralateral execution operationalized within the skipping skill. The cause underlying these presentations was not under exploration in this study.

Contralateral movement is being used as a benchmark for having progressed through the organic evolution of developmental movement patterns. In the sample of boys, all should be capable of skipping due to their mature ages. Any inability to skip only informs that they are not developmentally on track relative to established motor ages of developmental movement patterning. This may have started in utero, in infancy, or prior to their involvement in this research, but this study will not be able to control the possibility of reverse causality because it will not be possible to know whether the presence of ADHD or the presence of an abnormal developmental movement pattern trajectory came first. This study investigated only the presence of a potential complex functional relationship by asking if a change in one variable is accompanied by a change

in the other. This investigation is not exploring a direct causal relationship between the variables of contralateral ability, ADHD diagnosis, and medication.

Observer bias could be harmful to this study if the assessor assigning OSU SIGMA ratings knew which children had ADHD and/or knew which OSU SIGMA recordings were obtained in a medicated versus an unmedicated state. An educational kinesiology expert and teacher in a local school district entered into a data-recipient agreement with the Institutional Review Board to act as a blind, third-party observer and ranker of the recordings. The expert was blind to diagnostic group allocation and medication state. After all recordings had been obtained, they were organized in a random manner with the subjects indicated only by subject number and handed over to the expert for assignment of OSU SIGMA ranks for each of the 147 recordings.

A final internal validity threat could have been the subjective attitudes of the children while they performed the skipping during each recording. Children were always encouraged to pick the location where they were recorded and reminded they could pick a private setting where nobody would see them. Settings turned out to include hallways in schools, cafeterias, gymnasiums, sidewalks outside schools or homes, living rooms, basements, church gathering rooms, and back yards. Additionally, each boy was asked if he wanted to do a "practice run" of the motor skill before being recorded. Once the camera was recording, each child skipped back and forth between two cones three times to assure any self-conscious feelings of the child would be minimized, and the average of their ability would be seen within the three performances of the skip and not interfere with the expert's ability to provide a sound ranking.

External Validity

Internal validity controls represent a first criterion of a sound research design. External validity controls represent a second important criterion. External validity speaks to the generalizability and representativeness of the findings. To the degree that the observed events in this study can be generalized to unobserved events in populations at large, the study possesses external validity. There are three primary sources of external validity: population external validity, ecological external validity, and external validity of operations (Ary, 2002).

Appropriate sampling procedures, which ensure that the population being studied accurately represents the population to which the findings will be generalized, aids in the establishment of population external validity. The experimentally accessible population is the group of subjects available for enrollment into the study. The experimentally accessible population for this study was seven to nine year old boys within central Ohio communities. The target population is the entirety of subjects to whom the results of the study are intended to apply. For this study, the target population was all seven to nine year old boys with ADHD diagnosis. The major threat to population external validity is the interaction between the characteristics of the subjects and the independent variable of ADHD diagnosis. When the experimentally accessible population is not representative of the target population, it would be hazardous to generalize the findings. The population external validity threat was somewhat controlled by the attribute nature of the variables under investigation. ADHD diagnosis is prevalent throughout the

population of seven to nine year old boys. Furthermore, the partnership with a large metropolitan children's hospital in this study offered a heterogeneous accessible population spanning socio-economic, racial, and demographic strata within the greater central Ohio region. The fact that subjects were volunteers could present an additional threat to population external validity, as volunteers often possess special characteristics not typical of the larger population, but the attribute nature of the variables again provided some safety here. ADHD diagnosis, subsequent medication, and contralateral movement ability have no reason to differ between a volunteer versus a non-volunteer population.

If the experimental environment is very different from the environment to which the results are intended to generalize, the generalizability is diminished by means of ecological external validity. The specific descriptions of experimental settings involved in this study are described below in the Conditions of Testing section. All skipping recordings were made in very real world settings for the children, such as their homes, schools, local parks, churches, and sporting practices. Additionally, the OSU SIGMA requires the child to do the skill three times moving back and forth between a set of cones. This provides the person ranking the recordings with a visual average of their ability. Furthermore the attribute nature of the variables again added protection against a validity threat. Prior OSU SIGMA runs, novelty effects, reactive threats, and experimenter threats are not likely to alter the child's movement ability. Since a hypothetical motor age has been established for skipping, the child should theoretically be able to skip consistently regardless of being recorded for the purposes of research. It

is reasonable to assume that findings learned from recordings made in the situations within this study would generalize to the real world abilities of these boys and of other age-matched boys asked to perform the same skill.

The final external validity threat, external validity of operations, was also controlled through the attribute nature of the variables. The operational definitions provided in chapter one for ADHD and for conglomerate developmental contralateral movement ability are well established. It is likely future investigations working with these variables will be using similar operational definitions of the independent and dependent variables. The definitions are specific, reliable, and have been validated, thus excluding any major threat to the external validity of this design due to external validity of operations.

Subject Selection

It is appropriate that the subjects in this study were children because diagnosis of ADHD usually occurs during childhood, and the maturation effect to skipping is likely to become more of a threat as older and older populations are assessed. It is most relevant to use children for such an inquiry, as the literature focuses almost exclusively on childhood ADHD. Working with children also gives direct access to parents and teachers, thereby providing a better chance of obtaining correct documentation regarding pharmacological regimens, developmental milestone establishment, and academic performance.

ADHD affects predominantly boys in a ratio of 3:1 to 9:1 in clinical samples (Arnold, 1996). Therefore, it would be difficult to recruit enough girls to provide a definitive answer to a possible sex difference. Oversampling of girls, to be balanced and scientific, would have to involve rejecting some available male applicants randomly. Even if it were practical to recruit the same number of girls as boys, making any scientific sense of the comparison would require doubling the sample size. Further, there is no evidence of a sex difference in treatment response (Arnold, 1996) as long as both sexes are diagnosed by the same criteria and within the same subtype. A far larger sample than the one recruited for this study failed to find significant differential treatment effect by sex with four kinds of treatment (MTA Cooperative Group, 1999). Therefore, recruiting extra girls is not likely to yield new information. There is evidence that girls may differ in proportion of different subtypes of ADHD (Arnold, 1996), but if the subtype makes a difference in response, that is the real issue, not sex.

Research into contralateral acquisition and skipping capability has indicated most children could skip leading with both feet by the age of five (Wellman, 1938). This motor age was determined to be that age at which half of the same-age children could display acquisition and efficiency of the given locomotor pattern. A review by Frego (1998) adds that females acquire the developmental capability to skip prior to that of matched males. In light of the available data and literature, the best strategy was to recruit only eligible male children. While the assumption can be made that sex makes no significant difference in ADHD treatment response, the same cannot be said about the relationship of sex and contralateral movement acquisition.

Checking for differences in contralateral abilities and treatment response is a later step in regard to sex and minority representation. For this new uninvestigated focus on contralateral abilities, the first step is to test the hypothesis that there is a significant decrease in contralateral ability within seven to nine year old boys suffering from ADHD. If this study reveals significant contralateral discrepancies between ADHD children and controls, the next step would be to look for differences by sex or race/ethnicity.

It is not possible to calculate a sample size using a power calculation due to the nonparametric nature of the variables involved in these research hypotheses. If it were possible to assume a standard distribution, fourteen subjects would be needed for a 90% chance of rejecting a one-tailed null at the .05 level with an effect size of .8. For this study a sample containing 24 ADHD-diagnosed children formed the experimental group and a sample of 25 boys confirmed to not have ADHD comprised the control group. The total sample size was 49 boys ranging in age from seven to nine years.

The target population was established to be seven to nine year old boys with ADHD. The accessible population for this study was boys within this age range from the area that had been diagnosed with ADHD by physicians at participating outpatient clinics. Subjects were enrolled with informed consent from this accessible population in a nonrandom purposive manner.

The Institutional Review Board approved each clinical facility to release a list of boys diagnosed with ADHD for use in this study. The guardians of the boys were contacted by phone and informed that a study was being conducted investigating ADHD

and movement. The guardians were asked if they had an interest in learning more and participating. Some guardians requested the information flier, the consent forms intended for the guardian, and assent forms intended for the boys to be emailed or sent via standard mail. These families were left to make a secondary contact with research staff on their own if they were interested in participating in the study after reading the materials. Other guardians were comfortable scheduling a meeting to go over these forms in person. Subjects were enrolled into the study upon signing of the consent and assent forms.

A general information flier was created to help recruit subjects. It was dispersed to local physicians, psychologists, researchers, elementary schools, after-school programs, summer camps, community locations, and families participating in the study in an effort to stimulate referrals. Some additional subjects were enrolled after contacting research staff as a result of this flier.

The flier was particularly helpful in the recruitment of non-ADHD subjects for the control group. Initially, the accessible population for the control group was intended to be seven to nine year old boys within a local school district. Seven hundred letters went home with boys in this age range, resulting in just nine of the twenty-four boys needed for the control group. It was at this juncture that the flier was created to help stimulate word-of-mouth referrals into the study. The remainder of the control group was enrolled into the study following signing of assent and consent forms similar to the experimental group but was recruited, in the majority, through word of mouth community referrals.

Regarding the experimental group, the quantity and heterogeneity of children seen through the behavioral health clinics at the participating large metropolitan children's hospital minimizes discrepancies between the experimentally accessible population and the target population within the experimental group. Families choose this hospital because of their expertise, multiple locations through the neighborhood sites, and acceptance of most insurance providers. This ensures that children represented on the hospital's database list obtained for this study come from varied racial, family-educational, socio-economic, and demographic backgrounds.

Regarding the control group, the nonrandom purposive manner of sampling is more problematic. Because so many control group subjects were enrolled via word of mouth, it becomes more difficult to assume that they are typical or representative of the larger population of seven to nine year old boys. For instance, two of the subjects were referred into the study through a common martial arts class. Four others were on the same soccer team. Three boys lived near each other in an upper-class suburban neighborhood. These differences raise concerns about the athletic abilities and the socioeconomic status of the boys in the control group. Theoretically, the attribute nature of all variables under investigation provides some safeguard to the validity of the group. Regardless of sports training or income level, skipping should be solidified in these boys because they have aged beyond the established motor age. Because of the variables under investigation, the convenience, and the low cost associated with purposive sampling, this nonrandom sampling method seemed appropriate to this investigation despite the limitations and caution that should be applied with its use.

Formal stated inclusion criteria for the study were: 1) children seven to nine years of age upon enrollment into the study, 2) boys, and 3) presence of a consistent, reliable, primary guardian. Exclusion criteria included: 1) plans to move which would make them unable to return for follow-up recordings during their study participation, 2) physical disability or injury, and 3) presence of neurological conditions which impact motor skill execution.

Built into the study were convenience measures for the family in the attempt to reduce or prevent subjects from dropping out or not responding to all parts of the study. All forty-nine subjects that enrolled in the study completed all three stages of the study. Guardians, upon consenting into the study, completed a survey describing early movement experiences. One boy declined to participate after reading his assent form and was therefore not enrolled in the study. One boy volunteered for the control group through but was endorsed for seven items in the ADHD section of the PChIPS structured interview, thus meeting the criteria for a potential ADHD diagnosis. His mother was given community referrals to follow up for more comprehensive diagnostic procedures, and he was not enrolled. Another boy was endorsed for three items in the ADHD section of the PChIPS, which concerned his mother and prompted a follow-up with their family physician. He was diagnosed with ADHD and subsequently enrolled into the experimental group. The safeguards built into the research design proved valuable, causing nearly all subjects to enroll once a face-to-face meeting was scheduled and prompting all subjects to participate fully in the tasks associated with study participation.

Outcome Measures

The Ohio State University Scale of Intra Gross Motor Assessment (OSU SIGMA) was used to measure the dependent variable, contralateral ability. This assessment tool is criterion-referenced and permits examination of the qualitative aspects of eleven basic motor skills. A criterion-referenced instrument is necessary for meeting the goals of this study because it allows the description of what abilities a particular individual has mastered without reference to the performance of other children. Only the basic motor skill of skipping was examined, as skipping represents the most mature of all developmental locomotor patterns and is fundamentally grounded in fluid, conglomerate, contralateral abilities.

Each basic motor skill in the OSU SIGMA has four levels of development ranging from the least mature performance, represented as a Level One, to the most mature functional performance, represented as a Level Four. Specific criteria have been established for each level, providing a descriptive assessment of the child's gross motor functioning. Level One children cannot skip but will, run, gallop, hop, or leap. Level Two children present in three ways: 1) they attempt to skip but instead walk or run, 2) skip more often than not on the same leg though not necessarily consecutively, or 3)

hold their arms either down at the sides or slightly bent with hands at approximately waist level. Level 3 will skip in a coordinated manner with the lower limbs but will not use arms in opposition, if at all, and/or will skip slowly in a segmented fashion interjecting a walk or run for brief periods. Finally, Level Four children will alternate feet, use their arms in opposition with the legs and execute skipping with good ease and coordination (Loovis, 1979). The literature on development of motor skills supports the levels of development elaborated for each OSU SIGMA skill.

Two research processes lead to the development of the OSU SIGMA. One evaluated the validity of the OSU SIGMA using a panel of evaluators from the field. A test-retest study was done for reliability and analyzed using Scott's Pi, producing test reliability coefficients for inter- and intra- rater agreement. Median test-retest inter-rater Pi was .5798 and .6697, respectively. Median intra-rater Pi was .8222. Further, the percentage of agreement between judges was computed for inter- and intra-rater relationships. Inter-rater reliability on the test-restest condition was .7500 and .8333, respectively, and intra-rater reliability was initially calculated at .9167 (Loovis, 1979). The OSU SIGMA was subjected to field-testing and has been used in published research since its development in 1979. A pilot study completed initiating this investigation during 2004, found intra-rater reliability, using a kappa coefficient, to be .84 (Frego, unpublished).

Conditions of Testing

Data collection began in September 2009 and concluded in August 2010. Each subject was involved with the study for about three months. For each subject, the study took the form of three phases each marked by a meeting with research staff and a movement recording. For convenience to the families, the study was not time-ordered. This allowed the experimental group to arrange their unmedicated recording at a time that was least burdensome to them and their families.

Before enrolling each child into the study, experimental group boys were confirmed to have ADHD through physician referral and/or diagnostic codes 314.00-314.99 on the NCH database. Control group boys were confirmed to not have ADHD using the PChIPS structured interview. Once enrolled into the study, the guardian completed the questionnaire outlining aspects of the boy's motor history, and the boy was recorded doing his first movement recording. For the experimental group boys, the medication type and dosage was also documented using parent statements and, when possible, the researcher actually seeing the prescription. If the boy was medicated during this first study visit, the recording counted as 01, or first medicated, recording. If the boy was off his medication the day of the first study visit, the recording counted as his 0U, or his one and only unmedicated, recording. For the control group boys, after completing the PChIPS interview to rule out ADHD diagnosis potential, each boy was recorded for his first 0, or control, recording for OSU SIGMA ranking.

On average, three weeks passed between recordings. Some families preferred recordings as close as two weeks apart and some others requested recordings closer to a month apart. Upon the second recording, the boys were again asked to skip. For the

experimental group, parents were asked if the boy had taken his medication that day. If he had, then the recording counted as either his 01 or 02, first or second medicated recording, depending on his medication status during the first recording. If the boy was not medicated and had undergone the directed drug washout period, the recording counted as his 0U, or unmedicated, recording. There were three instances where the boy had not taken his medication upon this second meeting and had also not taken medication for the first recording. Since only one 0U, unmedicated, recording is needed for the study, no recording was made in these circumstances, and an extra study visit was scheduled to obtain the 01, or first unmedicated, recording of these boys. For control group boys, each was recorded in the same manner as the first study visit for their second 0, or control, recording.

Upon the third study visit with each subject a final recording was made. For the experimental group, this recording either counted as the boy's 0U, unmedicated, or 02, second medicated, recording depending upon the medication status of each boy during study visits one and two. Each control group subject was recorded in the same fashion as study visits one and two and the final 0, control, recording was obtained.

During each study visit, each child was recorded skipping for the purpose of assigning an OSU SIGMA rank. Children and their guardians did not know ahead of time that skipping would be their task at all recordings. They were told they would be doing a movement activity "such as running, walking, skipping, or jumping." They were also told repeatedly that there was no "right" or "wrong" that was being investigated as far as the recorded skill. Each boy was going to perform perfectly

regardless of how it looked because the interest was just in seeing how "different kinds of boys move."

Two orange cones were set up approximately twenty feet apart and each boy was recorded skipping between them, back and forth, three times. Each boy was recorded only from his shoulders down to protect his anonymity; children and their guardians were informed of this anonymity safeguard in their consent. Subjects were always asked to decide where to do the recording in an effort to help them feel more confident and less self-conscious. They were always told it was fine to go to a private place where they would not be seen by anyone other than the researcher. As a result, a variety of locations were chosen including busy hallways, private hallways, basements, sidewalks, parking lots, basketball courts, gymnasiums, cafeterias, community meeting rooms, living rooms, hallways in a family home, front or back yards, and local parks.

All recordings were then arranged in a random manner and viewed by an educational kinesiology expert for the purpose of assigning OSU SIGMA ranks. The expert entered into a data-recipient agreement with the Institutional Review Board. She was blind to the conditions and chronology of all three recordings for all children.

Data Analysis

The independent variables of ADHD diagnosis and medication are treated as nominal, or categorical, in this investigation. The dependent variable, the OSU SIGMA ratings, is ordinal, which is also a categorical type of variable but one with a clear

ordering of rank. The order is important, a Level Four child moved more maturely than a Level Three child, but the difference between the values is somewhat arbitrary or qualitative. To analyze this data set an ordinal logistic regression model was applied. The model is:

$$Yijklmnop = \mu + Pi + \alpha k + \beta l + \gamma m + \lambda n + \delta o + \theta p + \Sigma ijklmnop$$

i = subject

j = recording number (first, second, or third)

k = group number (non-ADHD, ADHD with medication, ADHD without medication)

l = question number one from questionnaire

m = question number two from questionnaire

n = question number three from questionnaire

o = medication

p = medication dosage

μ = average effect

Pi = personal, random, effect

αk = effect of group

βl = effect of question number one from questionnaire

γm= effect of question number two from questionnaire

δo = effect of question number three from questionnaire

δo = effect of medication

θp = effect of medication dosage

Dependent Variable = OSU SIGMA ranks (1, 2, 3, or 4)

Independent Variables = ADHD diagnosis and medication (1 = non-ADHD, 2 = ADHD child while medicated, or 3 = ADHD child while not medicated)

The dependent variable of OSU SIGMA rating is the response *Yijklmnop*, or *Yijklmnop* is the value of the rank. If the movement expert ranking the recordings gave the child a ranking 4, then his corresponding *Yijklmnop* would equal 4. The independent variables are ADHD diagnosis and medication. Groups (αk) were defined as follows: group 1 represents the control group of non-ADHD subjects, group 2 represents the experimental group of ADHD subjects when under the effects of their prescribed medication, and group 3 represents the experimental group of ADHD subjects while either drug naïve or following a drug washout period. Group 3 subjects were the same ADHD-diagnosed boys from group 2 but while not under the influence of their prescribed ADHD medication. Additional independent variables include random effect (Pi), the responses from the movement questionnaire (βl, γm, *and* λn), type of medication (δo), and medication dosage (θp). The movement questionnaire contained three questions, which were categorically coded. The first question pertained to if and for how long the child crawled as a baby. Responses were coded as 1: less than or equal to five months, 2: greater than or equal to six months, 3: parent/guardian did not know how long the child crawled, 4: the child did not crawl, and 5: parent/guardian did know if the child crawled. The second question asked for the age that the child started walking. These responses were coded as 1: 8 to 9 months, 2: 10 to 11 months, 3: 12 to 13 months, 4: 14 to 15 months, 5: 16 to 17 months, 6: 18 to 19 months, 7: greater than

20 months, 8: parent/guardian did know when the child started walking. The final questions inquired whether or not the child had ever participated in physical activities such as sports, gymnastics, or dance that may have helped to teach him mature movement skills. These answers were coded as 1: yes, the child did participate in these activities, and 2: no, the child has no history of participating in these activities.

For the remaining independent variable of medication type and dose, each type of medication was coded with a number and, to maintain the categorical nature of the data set, the dosages were divided into a high-dose group and a low-dose group for each medication. Medications included 1: Adderall, 2: Focalin, 3: Vyvanse, 4: guanfacine, 5: Ritalin/methylphenidate, and 6: Concerta. The control group boys and the ADHD boys while unmedicated were coded as a 7. For each medication a high dose was coded as a 2 and a low dose was coded as a 1. These categories were established to divide the dose ranges seen within the group of 24 experimental group subjects within this study and not to depict clinically specified high versus low dose regimens. The low doses were specified as Adderall: less than or equal to 10mg, Focalin: less than or equal to 10mg, Vyvanse: less than or equal to 25mg, guanfacine: less than 2mg, Ritalin/methylphenidate: less than or equal to 5mg, and Concerta: less than or equal to 30mg. The high-dose ranges were Adderall: greater than or equal to 15mg, Focalin: greater than or equal to 20mg, guanfacine: greater than or equal to 2mg, Ritalin/methylphenidate: greater than 5mg, and Concerta: greater than or equal to 35mg. The control group subjects and ADHD subjects while unmedicated were coded as a 3.

This data set was applied to the ordinal logistic regression model created for this study. The model allowed for group comparison with OSU SIGMA ranks and comparisons of medication information and questionnaire responses with OSU SIGMA ranks. Creation of this model provided the best means of applying this data set to the hypotheses under investigation. Analysis of the model and data addressed the primary hypotheses: 1) Contralateral movement ability, as measured using the OSU SIGMA skipping scale, will be less mature in children diagnosed with ADHD compared to children without ADHD, and 2) Children with ADHD will not show improved OSU SIGMA skipping scale scores in the medicated state. The model also provides insight into other interesting independent variables, which may point to future lines of investigation.

CHAPTER 4: RESULTS

Two null hypotheses existed for this study. The primary null hypothesis stated that there would be no significant difference in contralateral locomotor ability between the groups (Ho: $\mu_{2,3} = \mu_1$). Subjects assigned to experimental groups 2 (ADHD diagnosis, taking medication) and 3 (ADHD diagnosis, off medication) would have similar OSU SIGMA ranks to subjects within group 1 (control group, no ADHD diagnosis). The literature strongly suggests that coordination generally improves when the subject or patient is under the effects of pharmacological treatment for ADHD. Conversely, it was a key theory driving this study that such changes should not be observable with developmental movement patterned locomotor skills such as skipping. It is instead conjectured in the secondary research hypothesis that ADHD subjects will display the same movement ability in both medicated and unmedicated scenarios.

The secondary null hypothesis for this study (Ho: $\mu_2 = \mu_3$), being the simpler of the possible outcomes, stated that the ADHD-diagnosed subjects would obtain the same OSU SIGMA ranks, regardless of whether they were in group 2 (ADHD, taking medication) or group 3 (ADHD drug-naïve or following drug washout period). Results indicate that the first null hypothesis is rejected but the second one is not. Tentative conclusions emerge that seven to nine year old boys with ADHD do skip in a less

mature manner than non-ADHD peers and that medication has no impact on the developmental locomotor skill of skipping.

The first hypothesis was addressed by using a Welch Two Sample t-test to learn whether the population means between the control group and experimental group are equal (F = 29.26, p-value = 0.000). The 95% confidence interval for the difference between these two groups (group 2-group 1) was (-1.1545, -0.5366), indicating that the difference is significant. The mean OSU SIGMA score for the ADHD group (group 2) was 2.861, and the mean for the non-ADHD, neurotypical group (group 1) was 3.707 (see Figure 2). There is a statistically significant difference in OSU SIGMA ranks between the ADHD and non-ADHD groups.

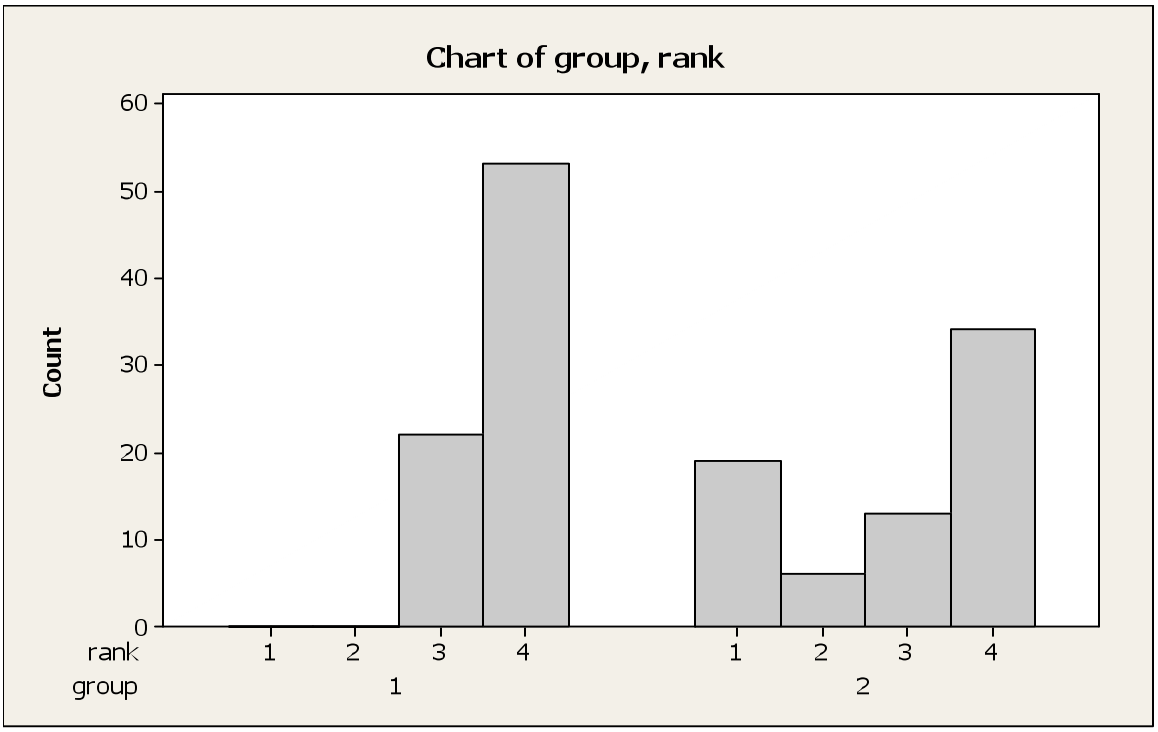

Figure 2. Group one (non-ADHD subjects) and group two (ADHD subjects) compared on OSU SIGMA rank

The second hypothesis was addressed in two parts. For the first part, the Welch's Two Sample t-test was again used. Group 2 subjects from the first analysis were subdivided into group 2 subjects (ADHD diagnosis while on medication) and group 3 subjects (same ADHD-diagnosed subjects but while drug-naïve or following a drug washout period). The groups were again compared with OSU SIGMA ranks. These findings did not reach statistical significance (F = -0.3921, p-value = 0.535) at the 95% confidence interval (-.08801, 0.4634). The mean OSU SIGMA score while medicated was 2.79 and mean rank was 3.0 while not under the effects of medication (see Figure 3). Subjects with ADHD did not rank significantly differently while medicated versus unmedicated.

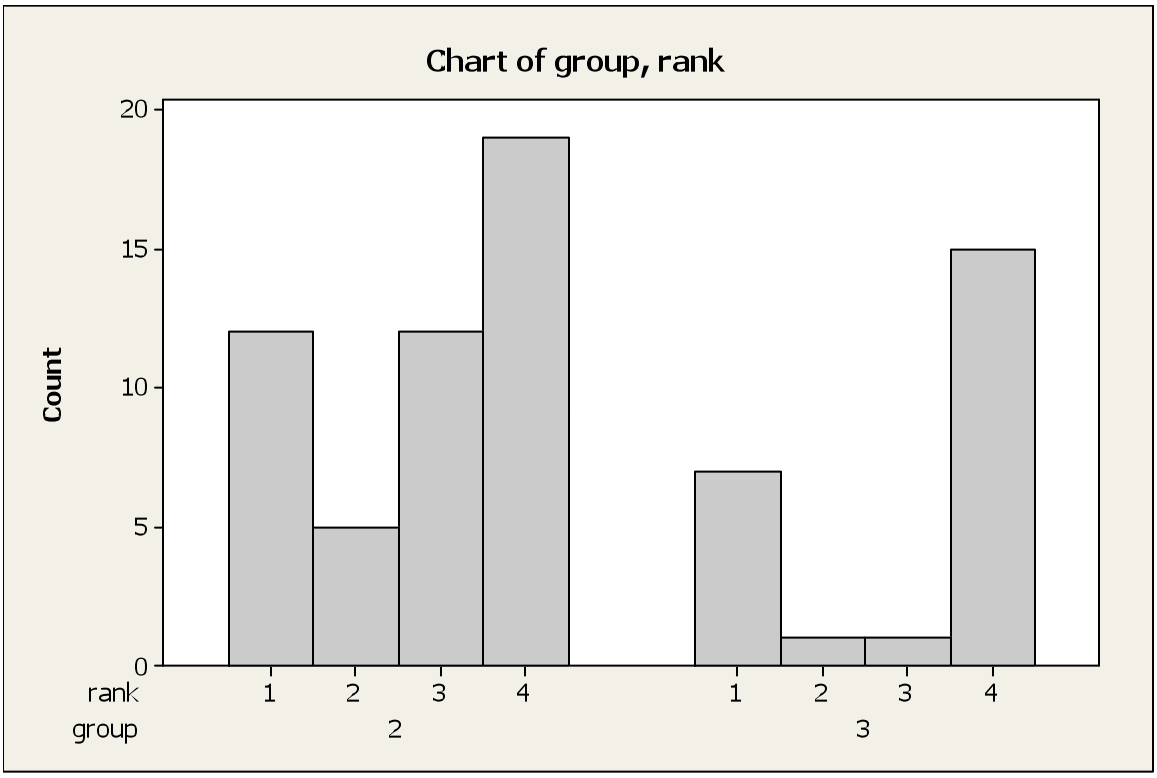

Figure 3. Group two (ADHD/medicated) and group three (ADHD/unmedicated) compared on OSU SIGMA rank

Part of the analysis used likelihood ratio tests of the ordinal regression model to evaluate the effect of medication type and medication dose. Interestingly, there was a medication type/dose effect (p-value = 0.0004). So while medication in general did not elicit a difference in OSU SIGMA ranks, specific types and dosages of medication did have variable effects on movement ranks.

One–way ANOVA were also done to explore OSU SIGMA ranks with answers to survey questions. Pertaining to ranks and survey question number one, eighty-seven subjects had level-one responses (crawled for ≤ five months) with a mean OSU SIGMA rank of 3.5977. Twenty-seven subjects with level-two responses (crawled ≥ six months) had a mean rank of 2.6296. Level three responders (n = 33; crawled, but guardian did

not know how long) had a mean rank of 3.303 (see Figure 4). These results were significant (p-value = 0.000).

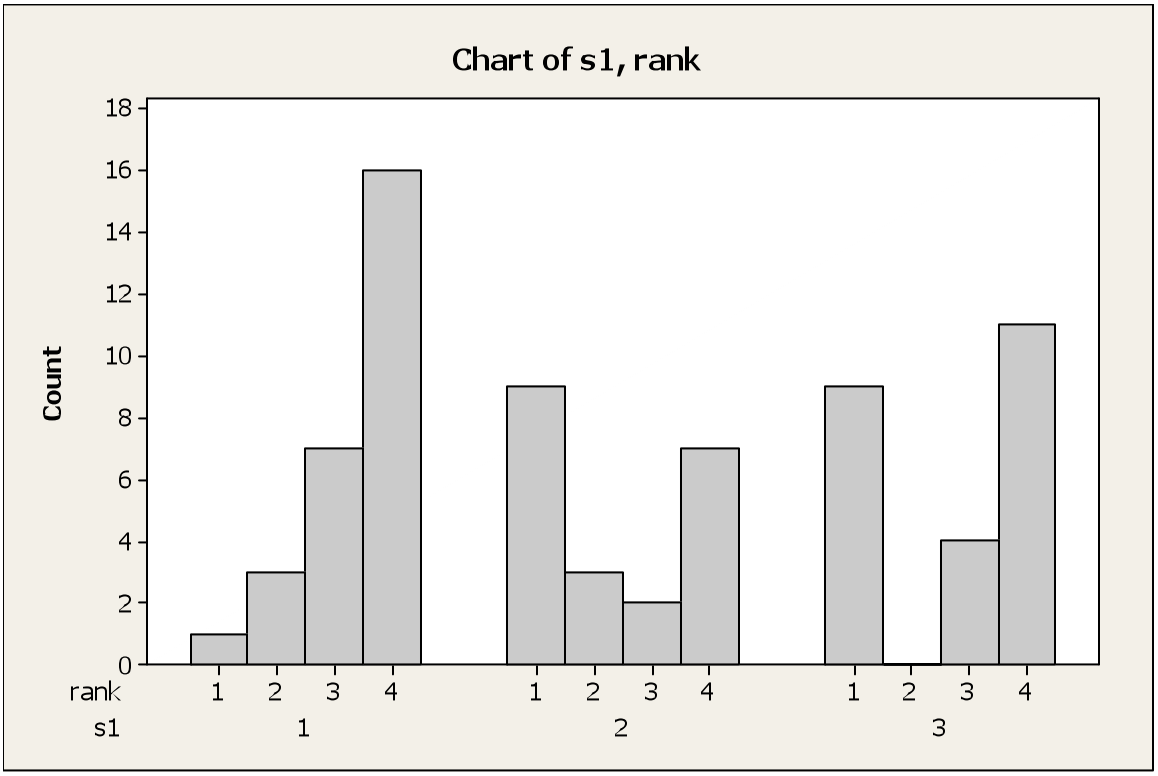

Figure 4. OSU SIGMA rank compared with answers to survey question one. "Did your child crawl and how long?" 1: crawled for 5 months or less, 2: crawled for 6 months or longer, 3: crawled but guardian did not know duration

For survey question number two, results indicate that level two responses (n = 26; mean OSU SIGMA = 3.75; subject walked at 10-11 months of age) were significantly different related to OSU SIGMA ranks from level six (n = 9; mean OSU SIGMA = 2.444; child walked at 18-19 months of age) and level eight (n = 6; mean OSU SIGMA = 2.1667; child walked, guardian did not know at what age). These findings were also significant (p-value = 0.001) (see Figure 5).

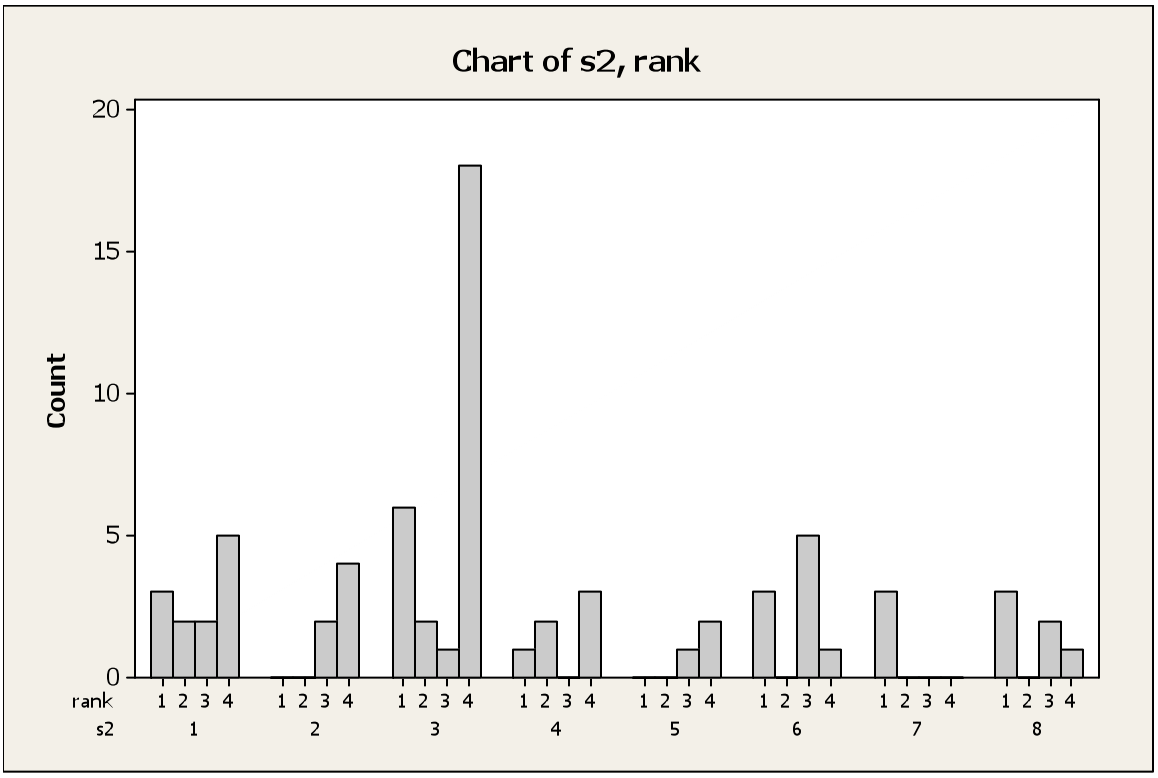

Figure 5. OSU SIGMA ranks compared with answers to survey question two. "At what age did your child start walking?" 1: 8 to 9 months, 2: 10 to 11 months, 3: 12 to 13 months, 4: 14 to 15 months, 5: 16 to 17 months, 6: 18 to 19 months, 7: greater than 20 months, 8: parent/guardian did know when the child started walking

The third survey question asked whether or not the subjects had a history of participation in sports, gymnastics, or dance. Results indicate no significant difference on this variable (p-value = 0.671). Responders that did have a movement history with these activities (n = 123) had a mean OSU SIGMA rank of 3.276 and those without these activities in their movement history (n = 24) displayed a mean rank of 3.375 (see Figure 6).

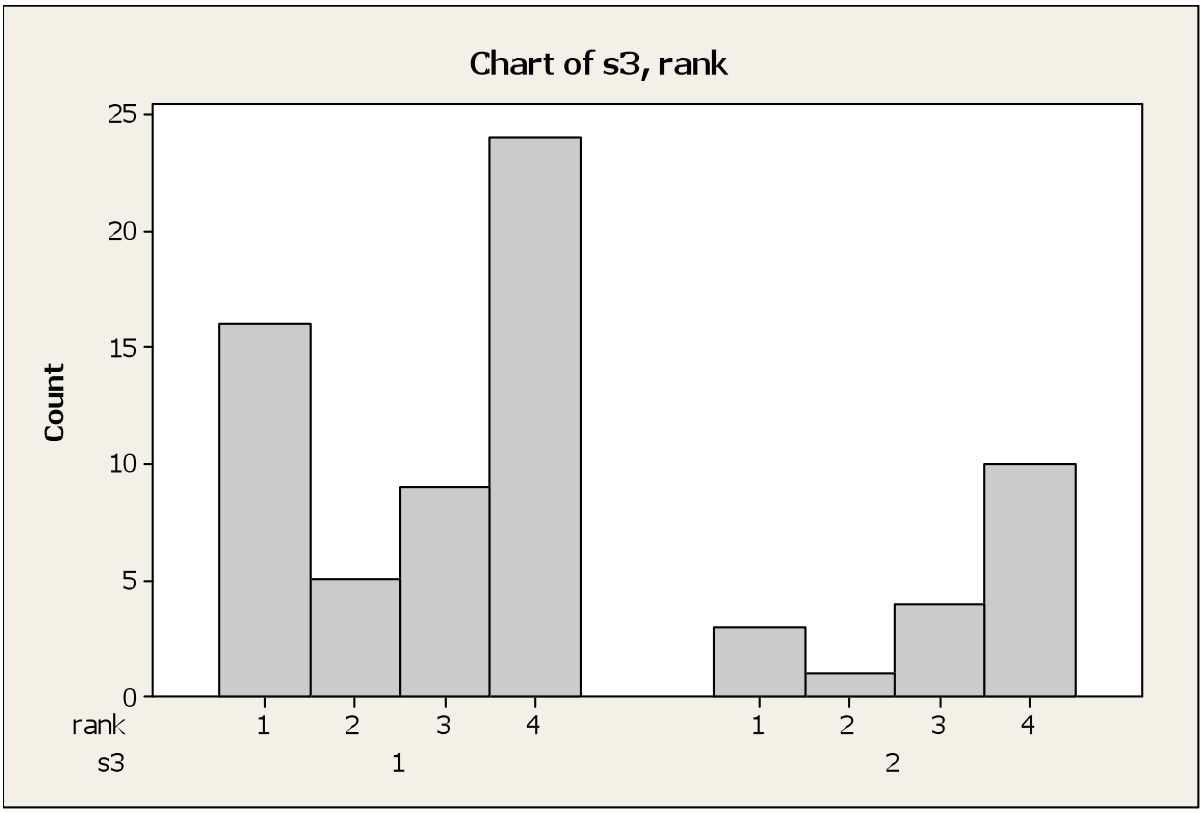

Figure 6. OSU SIGMA ranks compared to answers on survey question three. "Does your child now or has your child ever participated in sports, gymnastics, or dance?" 1: Yes, 2: No

A final series of one-way ANOVA analyses explored the relationship between the same survey question responses and group (coded group 1 = control group, no ADHD diagnosis; group 2 = experimental group, ADHD diagnosis). These results were significant (question 1, p-value = 0.000; question 2, p-value = 0.002; question 3, p-value = 0.005). For the first survey question ("Did your child crawl and for how long?"), control group subjects trended toward the shorter duration of having crawled less than or equal to five months (level 1 mean = 1.32) whereas experimental group subjects (level 2 mean = 1.9583) showed a tendency for the longer duration of crawling for six months or longer (see Figure 7).

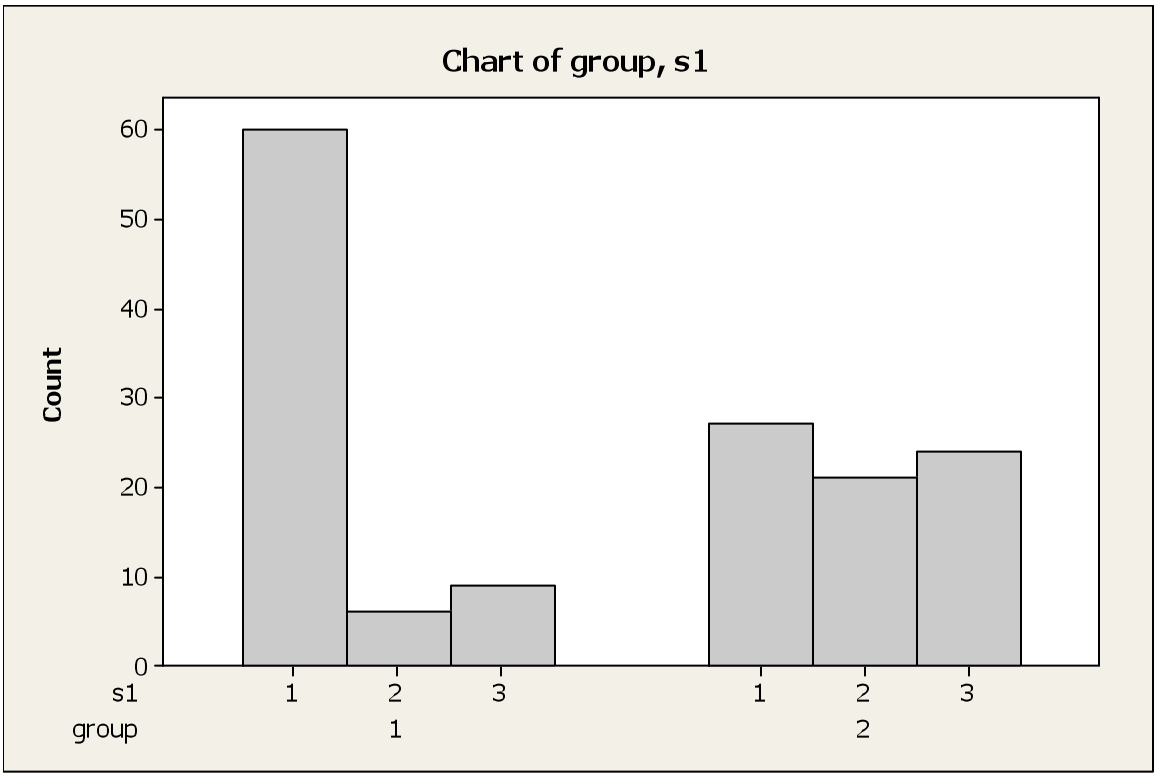

Figure 7. Diagnosis group compared with answers to survey question one. "Did your child crawl and how long?" 1: crawled for 5 months or less, 2: crawled for 6 months or longer, 3: crawled but guardian did not know duration

The second survey question ("At what age did your child walk?") also was significant when compared with group. The control group walked at a mean of level 2.8 (level 2 = walked at 10 to 11 months; level 3 = walked at 12 to 13 months) whereas the experimental group began walking at a mean level of 3.708 (level 3 = 12 to 13 months; level 4 = walked at 14 to 15 months) indicating that subjects with ADHD walked later than subjects without an ADHD diagnosis (see Figure 8).

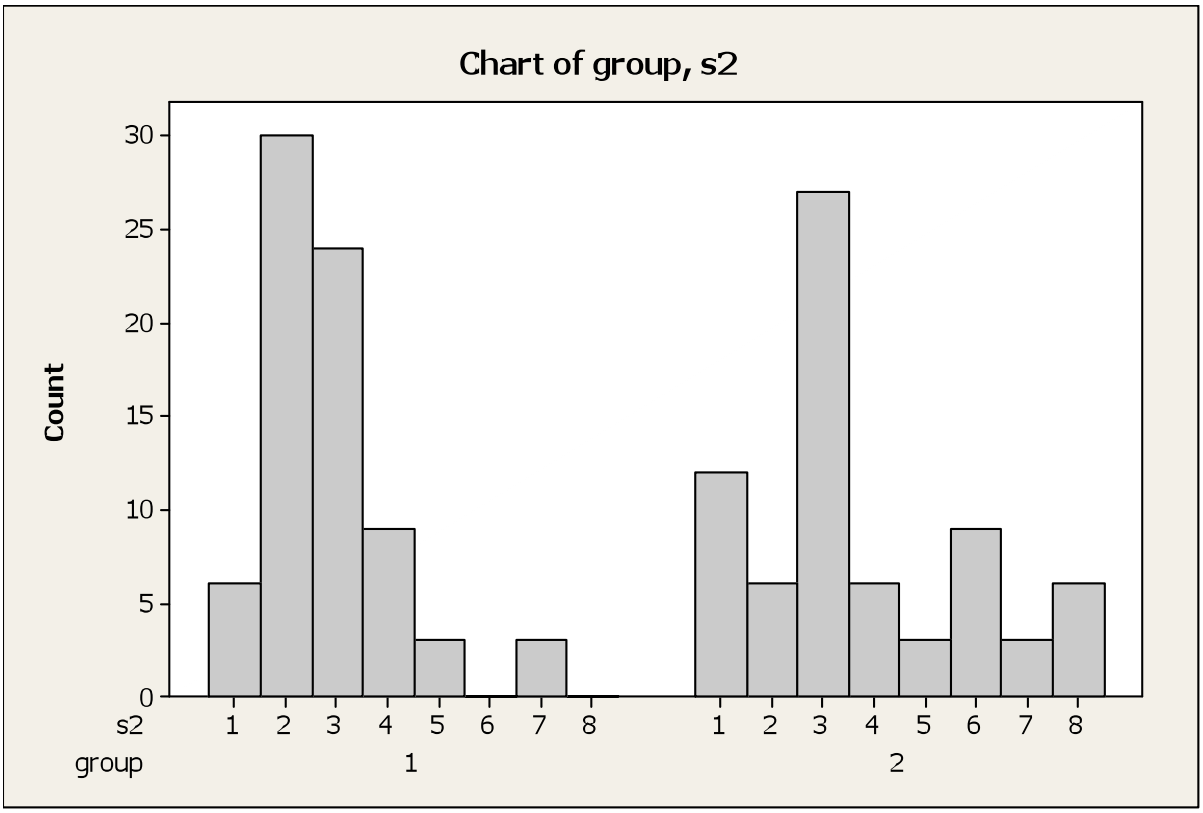

Figure 8. Diagnosis group compared with survey question two. "At what age did your child start walking?" 1: 8 to 9 months, 2: 10 to 11 months, 3: 12 to 13 months, 4: 14 to 15 months, 5: 16 to 17 months, 6: 18 to 19 months, 7: greater than 20 months, 8: parent/guardian did know when the child started walking

The last survey question regarding past or current participation in sports, gymnastics, or dance history was also significant when compared to diagnosis. The control group of non-ADHD subjects contained more boys with a history of movement training than the experimental group of ADHD-diagnosed boys (see Figure 9).

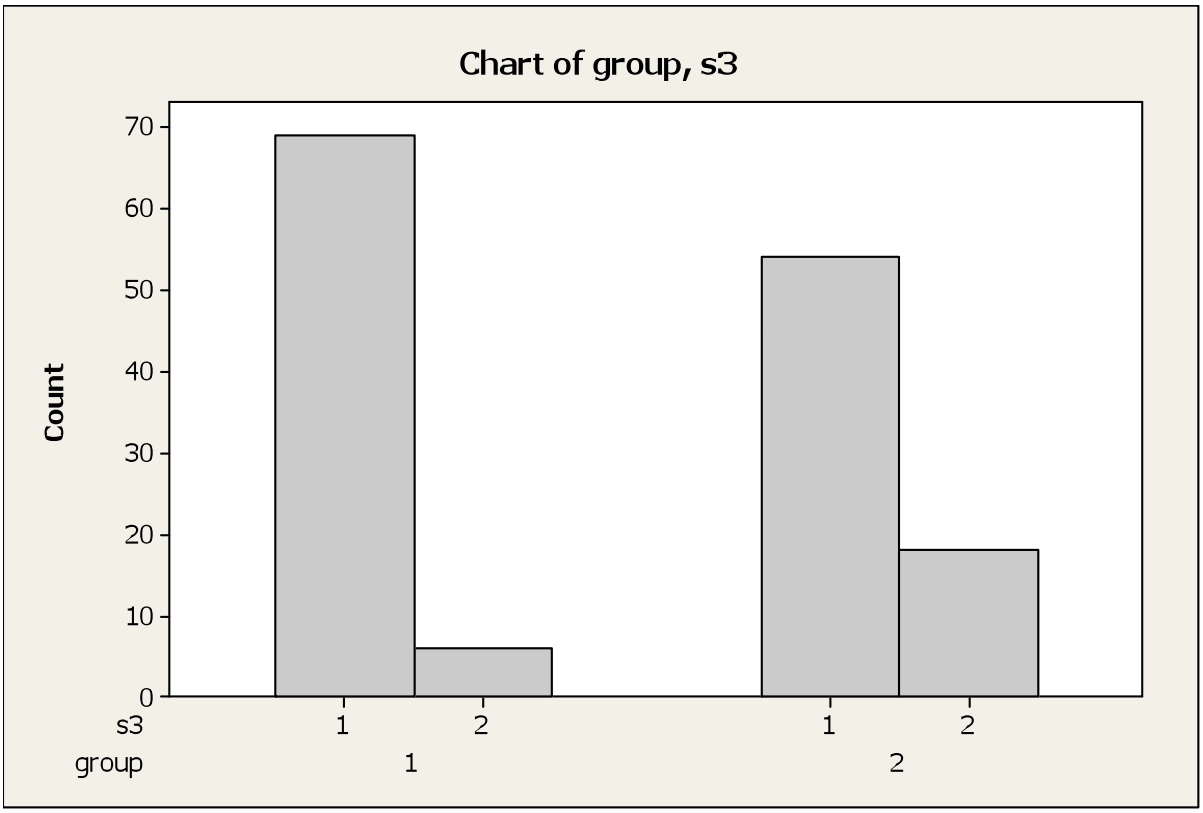

Figure 9. Diagnosis group one (non-ADHD subjects) and group two (ADHD subjects) compared on compared to survey question three. "Does your child now or has your child ever participated in sports, gymnastics, or dance?" 1: Yes, 2: No

CHAPTER 5: DISCUSSION

The ex post facto nature of this study was methodologically limiting. Concerns of differential selection, common cause, reverse causality, and the possibility of alternative explanations diminished control of variables, which makes it unsound to form too strong an inference about causal relationships gleaned from the findings. In the hypothesized and realized presence of contralateral deficits in the ADHD group of children, it still is not known whether ADHD causes the movement deficit, whether the delayed developmental movement continuum causes ADHD, or whether a completely separate factor influences both ADHD and developmental movement capabilities simultaneously or independently.

Progressed beyond the established motor age of six, this sample of seven to nine year old boys all should have been developmentally capable of skipping. The decreased OSU SIGMA ratings found throughout this investigation indicate that many observed children were not developmentally on track relative to this theoretically established motor age. The deficit tendency may have started at any time from conception through enrollment in this study. This study did not attempt to control for reverse causality. It is not possible to know whether the presence of ADHD or the presence of an abnormal developmental movement pattern trajectory came first. This study attempted simply to

investigate the presence of a functional relationship between conglomerate contralateral ability, operationalized by skipping ability, and ADHD diagnosis/treatment. No attempt to explore a direct causal relationship between the variables was made in this study.

Primary Hypotheses: Comparing Group and Rank

At the onset of this project, the expectation was that all children should be able to skip by the age of seven. The primary hypothesis stated that the ADHD boys would yield lower ranks than the non-ADHD boys. This stated difference in OSU SIGMA scores was corroborated in these findings, informing that boys with an ADHD diagnosis perform the skipping skill in a less mature manner than boys without ADHD (see Figure 2). This finding preliminarily suggests that there is a contralateral movement deficit or delay seen in boys with ADHD when compared with neurotypical peers as observed through the skill of skipping.

Medication did not make a significant difference in the OSU SIGMA ranks of the ADHD boys. This contradicts themes in the literature, which assert that ADHD children tend to globally become more coordinated when on appropriate medication regimens for their ADHD symptoms. Within the experimental group, the mean OSU SIGMA score while medicated was 2.79 and unmedicated was 3.00. This difference was not statistically significant indicating tentatively that medication does not improve a boy's ability to skip. This question would be particularly interesting to investigate again with a greater number of subjects because these results inconclusively show OSU SIGMA scores to be slightly lower (2.79) when medicated and slightly higher (3.0)

without medication in the child's system. This is contradictory to the assumption made about medication in the literature but fitting with theories driving this study.

A neurological question driving this study was whether the basal ganglia play a role in the ontogenic recapitulation of phylogenic developmental movement patterns and the storage of those sequences. The basal ganglia are a site where units of behavioral and motoric information are acquired and treated as learned sequences or entities of memory. Temporally ordered acts are collapsed into nearly reflexive, encoded sequences within the basal ganglia. Graybiel reported that intervention of conscious attention, or top-down attentional mechanisms, alters the implementation of behavioral and motor sequences. It would be an interesting next step to explore the question of medication and OSU SIGMA rank in a larger number of subjects to learn if this relationship between medication and decreased OSU SIGMA ranks on developmental skills is significant and predictable. If medication is facilitating top-down attentional mechanism, it is theoretically possible that some ADHD medications may interrupt the execution of developmental sequences such as skipping, leading to poorer ranks while medicated versus unmedicated.

While medication did not make a significant difference to movement rank in either direction, likelihood ratio tests did indicate that medication as a whole had a significant effect on rank. A study design with more subjects and controls for medication type and dose would be required to investigate the nature of this drug effect. Within this study's 24 experimental group subjects, six medications were prescribed: Ritalin (n = 4), Adderall (n = 5), Vyvanse (n = 2), Concerta (n = 6), Focalin (n = 6), and

guanfacine (n = 1). All but guanfacine are either an immediate release or a slow-release form of stimulant medication. The distributions showed more boys skipping at either a level one or level four when unmedicated versus when medicated which displayed a more evenly dispersed spread of ranks (see Figure 3). So while there was no statistically significant difference between the experimental group boys based upon general medication state, future studies with larger subject numbers may help clarify the role that specific medications and dosages have on contralateral movements such as skipping.

Guanfacine is in a class of centrally acting alpha 2A-adrenoreceptor agonists. It is known that drugs have differential influence over various ADHD symptoms. Stimulants have broad effects on vigilance and motor impulsivity whereas guanfacine is likely to be the most specific for the treatment of attentional deficits alone. Preliminary findings from the liklelihood ratio tests of the statistical model used suggest that specific ADHD medications and dosages do have a significant, differential effect on contralateral movement as represented by the skipping skill portion of the OSU SIGMA when looked at as a global medication effect. Other than the knowledge that a drug effect exists, there was not enough data collected on these variables in this study to come to any meaningful understanding of the effect's specificities. A future investigation probing the effect of each medication at various doses to OSU SIGMA rank would be of relevant interest despite the fact that general impact of medication to movement rank was found to be insignificant.

The lack of significant impact the medication state had on OSU SIGMA ranks supports the developmental theories driving this study. Skipping was selected as the movement of interest in this study because it is a developmental locomotor pattern, requires conglomerate contralateral coordination, and has been well documented as a skill that can be taught as well as reflexively acquired. If a child never developed the ability to skip, medicating them did not elicit performance of a skipping pattern. Instead the child to performed a less mature skill such as sliding, galloping, or hopping with potentially differential coordination depending upon medication state. This may be some of what the distributions in Figure 3 are indicating, but future study would be needed to draw any sound assumptions from this preliminary data.

Motor Age

While the primary and secondary hypotheses were substantiated, the results within the control group singularly suggest that the motor age for skipping may be changing, and it is taking longer for children to reach mature integration of their developmental locomotor patterns. It was a surprise to find that even within this group of seven to nine year old boys, not all subjects were capable of level four skipping on the OSU SIGMA (see Figure 2). The early work of Wellman and the later work of Frego established the motor age assumed in the literature today. The survey question relating to boys' involvement in sports, dance, or gymnastics showed no significant correlation with OSU SIGMA ranks, indicating that frequency of these activities or the ability to

teach skipping through such activities in children today versus children of the past does not play an obvious role in the delay.

It would be interesting to also include variables of time in front of televisions, computers, and video games, qualitative aspects of free playtime, musical rhythmicity, cultural and age-related attitudes toward exercise/movement, and diet into future studies relative to contralaterality. A study re-exploring motor age establishment would be helpful toward better understanding this observation. It is appealing to theorize what could underlie this apparent increase in the motor age for skipping. Will additional studies replicate these findings? What has changed that may be impacting boys' ability to skip? Would the same results be found in a similar study investigating contralateral movement in female subjects? While singularly this does not relate to ADHD, it is of key importance to movement educators and does warrant future investigation.

Survey Questions Compared with Rank

The ANOVA information surrounding survey questionnaire responses and rank offers valuable data about how movement history relates to OSU SIGMA rank. In this study's subject population, all control group and all experimental group boys crawled for some duration as babies. It is accepted that around 10% of babies never crawl (Brook, 2001). A study with a larger subject size would hopefully include this representative population of non-crawlers to allow for observation of how/if bypassing this developmental milestone impacts future contralateral skill, acquisition, and performance. Duration of crawling did significantly relate to OSU SIGMA rank.

Subjects that crawled for six months or longer had mean ranks of 2.6296 whereas subjects that crawled for five or fewer months had a mean rank of 3.5977 (see Figure 4). Children who crawled for a shorter period of time became better skippers as older children.

Another significant and interesting relationship was uncovered between ranks and age of walking. Subjects who began walking between 10 and 11 months were significantly different from subjects who walked at 18 to 19 months and subjects whose guardian did not know the age their child walked (see Figure 5). Boys who walked at 10 to 11 months had mean OSU SIGMA ranks of 3.75 whereas subjects that began walking later or walked at an unknown age showed poorer ranks. Boys walking between 18 and 19 months had a mean rank of 2.44 and boys walking at an unknown age had a mean rank of 2.16. It almost reached statistical significance (95% confidence interval -2.5637, 0.0637) that boys who walked at 20 months of age or later had significantly different ranks than boys who walked at 10 to 11 months. These findings imply that boys who begin walking between 10 and 11 months of age show the best skipping skills as 7 to 9 year olds, and that those boys who walk at 18 months or later are poorer skippers than their peers at the ages recruited in this study. It would be of interest to longitudinally investigate these same boys over the next many years to learn if they remain fixed with their contralateral abilities and subsequent OSU SIGMA rank or if they continue to progress and mature, eventually acquiring fully mature developmental locomotor skills as they age. Another way to approach this question would be to create a similar study to this one looking at an older cohort of boys or adults.

The third survey question asked whether or not the subjects had a history of participation in sports, gymnastics, or dance. When comparing answers with OSU SIGMA ranks, results indicate no significant difference on this variable (p-value = 0.671). Responders that did have a history with these activities had a mean OSU SIGMA rank of 3.276 and those without these activities in their movement history displayed a mean rank of 3.375 (see Figure 6). Participation in these activities does not make a child more likely to skip maturely.

Survey Questions Compared with Diagnosis

When the third survey question was compared with diagnosis a significant relationship did emerge (see Figure 9). The control group of non-ADHD subjects (n = 75 recordings, 3 recordings from each of the 25 subjects) contained more boys with a history of movement training than the experimental group (n = 72 recordings, 3 recordings from each of the 24 subjects). The control group and experimental group differ both on OSU SIGMA ranks and their history of participating in physical activities despite the fact that such participation did not have a significant correlation with OSU SIGMA rank. This preferential pattern would be interesting to explore further specific to psychological and psychosocial factors that differentially influence ADHD versus non-ADHD boys.

On the second survey question relating to age of walking the experimental and control groups were also significantly different. Control group subjects walked at an average age of level 2.8 (level 2 = 10 to 11 months, level 3 = 12 to 13 months) whereas

experimental group subjects walked at a later average of level 3.7 (level 3 = 12 to 13 months, level 4 = 14 to 15 months) indicating that control group boys walked a month or two earlier than ADHD group boys on average (see Figure 8). The first survey question again was significantly different relative to diagnosis category. The control group boys crawled for a shorter period of time than the ADHD-diagnosed boys (see Figure 7).

Since somatic theory states that crawling is one of the first iterations of developmental contralateral patterning within the nervous system, future studies examining these crawling durations seem warranted. It is theoretically possible that longer crawling durations could be indicative of difficulties establishing contralateral fluidity. Strong implications cannot be drawn but these preliminary data raise interesting questions for future investigations. These findings suggest that boys who crawl for longer durations and tend to walk later are more likely to also have ADHD diagnosis and not to participate in sports, gymnastics, or dance. This study has shown that boys in the ADHD group have lower movement ranks, which perhaps makes them less interested in sports, gymnastics, or dance. Perhaps the lack of involvement with these activities perpetuates ADHD symptoms, or a third unknown cause may lead to decreased interest and skill in these activities, as well as an ADHD diagnosis. A longitudinal, activity-based research design with conditions of sedentary activities, basic exercise, and more sophisticated developmentally-based exercise would help to clarify the role of specific childhood movement experiences and their relationship to future ADHD diagnosis.

Although it was not of interest at the onset of this study, it became notable that more children in the experimental group were living with guardians instead of biological parents. Within the control group (n = 25) all boys were living with their biological parent(s). Within the experimental group (n = 24), 15 were living with biological parent(s), 9 were living with a grandparent or other relative, and 2 others had a history of being in foster care. Knowing that ADHD is highly heritable, this may point to difficulties that adults with ADHD, and potentially other common comorbidities, have parenting their ADHD children. Guardians who did not know how long their child crawled or walked were more prevalent in the ADHD group over the control group (see Figure 7, see Figure 8). Those who did not know how long their boys crawled had mean OSU SIGMA scores of 3.03 which was significantly different from boys who crawled 5 months or less (mean rank 3.57) and boys who crawled 6 months or longer (mean rank 2.6296). Guardians who did not know the age that their child began walking had boys with a mean OSU SIGMA rank of 2.166.

These ranks cannot be concluded to have any relationship to biological parent versus guardian status for the subjects but the data create a compelling direction for future investigations to explore the role that social factors at home play in developmental movement obtainment and ADHD diagnosis. Is it the heritability factor of ADHD that makes guardianship situations more common for ADHD children? Is the heritability factor of ADHD related to a similar heritability factor of developmental movement patterning progression? Do social factors within the home somehow impact developmental movement acquisition? Would other children living in

foster/guardianship households show different OSU SIGMA ranks from children living with parents? Answers to these and related questions may prove helpful toward enhanced understanding of developmental movement deficits and the heterogeneity and etiological difficulties surrounding ADHD understanding, diagnosis, and treatment.

Summary

This study has presented new knowledge of a definitive type of contralateral deficit in boys with ADHD and provides a foundation for future studies to investigate contralateral movement interventions and their therapeutic efficacy applied to attentional/learning problems and OSU SIGMA ranks. Without prior scientific confirmation that a contralateral movement disparity existed, studies exploring contralateral ability enhancement effects on learning and attention were premature and unfounded. The findings from this study open the arena for more responsible, methodically sound studies researching developmental movement interventions applied toward treating attentional and learning problems.

Beyond the specific hypotheses associated with this study, a foundational goal of this research was to study contralateral movement in an interdisciplinary manner with the intention of bridging conversational gaps between the fields of anatomy, education, kinesiology, neuroscience, psychiatry, and psychology. ADHD was selected as representative of a prevalent and problematic neurodevelopmental disorder within which

to investigate developmental movement. An interdisciplinary and expanded dialogue brings discourse surrounding conglomerate contralateral movement into the scientific arena. This opens doors for more methodologically sound and convincing research within the fields of education and somatics and the introduction of novel, new directions for ADHD research, treatment, and understanding within the medical community.

The Western medical and somatic systems of describing sensorimotor development overlap tremendously and could easily be combined into one cohesive conversation concerning motor development. This study has attempted to introduce disparate disciplines to each other and articulate their differing views of developmental movement and definitions of the word "contralateral." This communication is important primarily toward aiding in options that the public has when considering ADHD treatments and interventions.

Now that this study has reported a conglomerate contralateral deficit in the skill of skipping within boys with ADHD, the question of improving this movement ability and exploring how that impacts measures of ADHD can be launched. Currently, patients have to seek their own paths toward holistic, alternative, somatic-based complements to standard medical care. These findings can begin bringing some of these modalities into the realm of scientific research and clinical care. With the somatic definition of conglomerate contralateral ability and developmental patterning integrated into Western medical education and practice, patients are more likely to receive comprehensive, personalized, and diverse options for living with ADHD.

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APPENDIX A: CONSENT/ASSENT FORMS

CONSENT TO PARTICIPATE IN A CLINICAL RESEARCH STUDY
Control Group

STUDY TITLE: The Role of Contralateral Movement in Boys with Attention Deficit Hyperactivity Disorder (ADHD)

PRINCIPAL INVESTIGATOR: **Dr. Jessica Foster**

CONTACT TELEPHONE NUMBER: **614-563-0997**

SUBJECT'S NAME: _____ DATE OF BIRTH: _____

NOTE: The words "you" and "your" are used in this consent form. These words refer to the study volunteer whether a child or an adult.

1) INTRODUCTION

We invite you to be in this research study because we are looking at how 7 to 9 year old boys with and without ADHD do certain kinds of movements called 'contralateral' movements. Contralateral movement is talked about by physical education and music teachers but is does not come up in psychology and psychiatry. Most tests looking at movement in people with ADHD look at movement issues such as gross motor skills, balance, hand-eye coordination and fine motor skills but contralaterality is a unique kind of movement that is developmental and involves specific coordination of opposite arm to leg. Examples of contralateral movement are crawling as a baby, walking with your arms swinging opposite arm to leg or skipping.

Participation is voluntary. Please learn enough about this research study, its risks and benefits, to decide whether you should agree to participate. We will explain the study to you, and give you a chance to ask questions about anything you do not understand. This process is called "informed consent". It is up to you to choose if you want to be in this study. You may refuse to be in this study or quit this study at any time, and standard medical care will still be available here or at a doctor of your choice without a penalty or loss of benefits to you.

Before agreeing to participate, it is important to read and understand the study information in this consent form**.** By signing the consent form, you agree to be in this study.

This study involves children between 7and 9 years of age. If your child is 9 years of age, he must also agree to be in the study by signing an Assent form.

You will be given a signed and dated copy of the consent and the assent form.

2) WHY ARE WE DOING THIS RESEARCH STUDY?

This study is looking at children's movement abilities, Attention Deficit Hyperactivity Disorder (ADHD) diagnosis and how medicating for treatment of ADHD influences movement. Some teachers use contralateral movement activities in the classroom because they believe that it helps attention, physical control and overall learning. These specific movement activities might be helpful for people with ADHD but we cannot be responsibly research them until we can be sure that there is a contralateral problem seen in the movement of kids with ADHD.

We know from earlier research that most children with ADHD move better when medicated. This study hopes to learn if people with ADHD can also do better at contralateral movements when medicated. We will compare the movement of 7 to 9 year old boys with ADHD to the movement of 7 to 9 year boys without ADHD. If the ADHD boys cannot perform contralateral activities the same way as the non-ADHD boys, this will begin helping us learn if there is a contralateral movement problem in children with ADHD and will open the doors for research into contralateral movement interventions to complement ADHD treatment.

We also know that girls tend to develop contralateral movements earlier than boys. It is generally agreed that most children have mastery over skipping, the most mature of all contralateral movements, by age six. To control for the difference between boys and girls, only boys will be used in this study.

3) WHERE WILL THE STUDY BE DONE AND HOW MANY SUBJECTS WILL TAKE PART?

The study is being done through Nationwide Children's Hospital (NCH) and the Ohio State University (OSU). If you choose for your son to be part of the control group for this study, you will not need to come to either NCH or OSU if that is not convenient. The study coordinator, Melinda Cooksey, can arrange to meet at your child's school (pending school approval), meet you at your home or another location of your convenience.

Forty-eight boys will be participating in this study. Twenty-four will have ADHD and 24 will not have ADHD.

4) WHAT WILL HAPPEN DURING THE STUDY AND HOW LONG WILL IT LAST?

Your child is a potential candidate to participate in the control group of this study. If you choose for your child to participate in this study, you will first participate in an interview that will allow us to rule out the possibility of ADHD or other mental health conditions in your child. This interview will take place between you and the study coordinator, Melinda Cooksey; your child will not need to be present for the interview. The interview will take between 30 and 60 minutes. Once we have confirmed that your child does not have ADHD or other mental health conditions Melinda Cooksey will work with you to schedule a time to digitally record your child doing a contralateral movement. Your child will be recorded only from the shoulders and down to remove his face from the recordings. The study requires 3 such recordings that will take place approximately one month apart.

The location of movement recordings will be specified by you, based upon convenience to your family (NCH, OSU, family home, elementary school). Each recording takes around 60 to 90 seconds. Two cones will be placed on the ground and the child will be asked to do a movement activity traveling between the cones three times (back and forth three times). Children are only recorded from their shoulders down to protect their anonymity. A digital video camera will be used for the recordings. The recordings are then downloaded into a secure, password protected computer and are indicated using alphanumeric codes.

Your participation should not take any longer than 3 months. If you choose to participate, you will meet with Melinda Cooksey once at the beginning for the structured interview and first recording and then 2 more times, approximately one each month, to get all 3 needed recording of your child.

5) WHAT ARE THE RISKS OF BEING IN THIS STUDY?

Some of the questions asked during the interview will be personal regarding you, your child and your family. You may find some of these questions stressful or may feel that some impose on your family's private life. All information will be kept confidential and will never be used for any purposes other than this study.

While not a risk per se, discovery of a previously unknown condition is possible as a result of agreeing to allow your son to participate in this study. It is possible that your child may show symptoms of a mental health condition through our process of ruling out an ADHD diagnosis. If our assessment in any way indicates that your child may have a condition previously unknown to your family we will provide you with proper community referrals. Melinda Cooksey is not a physician but will be the one administering the interview. Since she cannot diagnose your child, the interview only acts as a screen to rule out ADHD. If ADHD cannot be ruled out, or the interview indicates that a diagnosis of any other mental health condition could be possible, we will provide you with community referrals to follow up for more detailed evaluation and potential diagnosis. If your child does show signs of any mental health condition as an outcome of the interview, he will not be able to participate in this study. In this event, the forms completed during the interview will be shredded.

The physical risks are certainly less than your child would face in their daily activities in gym class or during recess play. It is possible that your child could trip or feel silly about how he looked doing the recorded movement, but the recordings will be done in a private area and the recordings will be viewed only at a later time by a member of the research team. Since your child will never be present at the viewing of the recordings and because all recordings will be from the shoulders and below, there is little chance of embarrassment or personal discomfort associated with participation. There is almost no risk of injury or harm coming to your child as a result of his participation.

There may be other risks of being in this research study, which are not known at this time.

6) ARE THERE BENEFITS TO TAKING PART IN THIS STUDY?

There may be no direct benefit to you or your son for participating in this study. After your child is finished participating in the study we will be happy to provide you with a free developmental movement patterning analysis session. Importantly, your participation would help us learn new things about ADHD that could be applied to classroom approaches to dealing with ADHD and clinical practice.

7) WILL THERE BE ANY COSTS TO ME?

It will not cost you anything to be in this study. You will not be paid to be in this study.

8) WHAT HAPPENS IF BEING IN THIS STUDY CAUSES INJURIES?

If your child is hurt by the procedures that are part of the Study, you should seek medical treatment for the injuries and tell the Study Doctor as soon as possible at the number on the first page of this form. If it is an emergency, call 911 or go to the nearest emergency department.

In most cases, this care will be billed to your health insurance company or whoever usually pays for your health care at the usual charges, but some insurance companies will not pay for care related to a study. If the care is provided at Nationwide Children's Hospital, we make no commitment to pay for the medical care provided to you. No funds have been set aside to compensate you in the event of injury. If no one else pays for your care, you may have to pay for the cost of this care. This does not mean that you give up any of your legal rights to seek compensation for your injuries.

9) WHAT HAPPENS IF I DO NOT FINISH THIS STUDY?

It is your choice to be in this study or to stop at any time. If you decide to stop being in this study, it is OK, but you must call the Principal Investigator or the study coordinator. If you stop being in the study, there will not be a penalty or loss of benefits to which you are otherwise entitled.

If at any time the Principal Investigator believes participating in this study is not the best choice of care, the study may be stopped and other care prescribed. If the study instructions are not followed, participation in the study may also be stopped. If unexpected medical problems come up, the Principal Investigator may decide to stop your participation in the study.

10) HOW WILL MY STUDY INFORMATION BE KEPT PRIVATE?

Information collected for this study will be kept confidential to the extent provided by law. Information used and/or disclosed (shared with someone outside of Nationwide Children's Hospital) may include information that can identify you. This is called "protected health information" or PHI. By agreeing to be in this study, you are giving permission or authorizing Dr. Jessica Foster and her study staff to collect, to use, and disclose your PHI for this research study. Information collected is the property of Jessica Foster. In the event of any publication regarding this study, your identity will not be revealed.

If you have a bad outcome or adverse event from being in this study, the Principal Investigator and staff or other health care providers may need to look at your entire medical records.

The PHI collected or created under this research study will be used/disclosed as needed until the end of the study. The records of this study will be kept for an indefinite period of time.

PHI that may be used or disclosed: Address; Telephone/Fax Numbers; Birth Date; E-mail Addresses/URLs

People or Companies authorized to use, disclose, and receive PHI collected or created by this research study:
PI and study staff

- **Dr. Jessica Foster (PI NCH)**
- **Dr. L. Eugene Arnold (PI OSU)**
- **Melinda Cooksey (study coordinator)**

The Nationwide Children's Hospital Institutional Review Board (the committee that reviews all human subject research)

Nationwide Children's Hospital internal auditors

The Office for Human Research Protections (OHRP) (the federal government office that oversees human subject research)

Reason(s) why the use or disclosure is being made: To contact you during the study You may decide not to authorize the use and disclosure of your PHI. However, if it is necessary for this study, you will not be able to be in this study. If you agree to be in this study and later decide to withdraw your participation, you may also withdraw your authorization to use your PHI. This request must be made in writing to the Principal Investigator. If you withdraw your authorization, no new PHI may be collected and the PHI already collected may not be used unless it has already been used or is needed to complete the study analysis and reports.

Dr. Jessica Foster keeps a database of all subjects who participate in a research study. This database may be used to contact people about future studies. Only Jessica Foster and her staff have access to this database. The database will not be disclosed or sold to others outside Nationwide Children's Hospital.

Please initial:
____ I want to be contacted about future research studies.

____ I do not want to be contacted about future research studies.

11) OTHER IMPORTANT INFORMATION

You will be told the results of this study at a later date.

Being in more than one research study at the same time may cause injury. Please tell the Principal Investigator about being in any other research study so a decision can be made about being in more than one study at the same time

12) WHOM SHOULD I CALL IF I HAVE QUESTIONS OR PROBLEMS?

If you have questions about anything while on this study or you have been injured by the research, you may contact the study coordinator, Melinda Cooksey, at 614-563-0997, Monday – Friday, between 8a and 5p.

If you have questions, concerns, or complaints about the research, questions about your rights as a research volunteer, cannot reach the Principal Investigator, or want to call someone else, please call (614) 722-2708, Nationwide Children's Hospital Institutional Review Board, (IRB, the committee that reviews all research in humans at Nationwide Children's Hospital).

Subject's Name _____Date of Birth_____

SUBJECT or SUBJECT'S PARENT OR PERSON AUTHORIZED TO CONSENT ON BEHALF OF THE CHILD (SUBJECT TO THE SUBJECT'S GENERAL MEDICAL CARE)

I have read this consent form and have had a chance to ask questions about this research study. These questions have been answered to my satisfaction. If I have more questions about participation in this study or a research-related injury, I may contact the Principal Investigator. By signing this consent form, I certify that all health information I have given is true and correct to the best of my knowledge.

I have been given a copy of the Nationwide Children's Hospital Notice of Privacy Practices. I understand that my right to my patient information that is created or collected by Nationwide Children's Hospital in the course of this research can be temporarily suspended for as long as the research is in progress. I also understand that my right to access will be reinstated upon completion of this research.

I give permission for my child to participate in this study. I will be given a copy of this consent form with all the signatures for my own records.

CONSENT SIGNATURES

_____

SUBJECT or SUBJECT'S LEGAL REPRESENTATIVE DATE & TIME AM/PM

_____

SUBJECT or SUBJECT'S LEGAL REPRESENTATIVE DATE & TIME AM/PM

Permission of the second parent not obtained because (select all that apply):

_____ Not required by the IRB (risk level 1 or 2).
_____ Other parent is deceased.
_____ Other parent is unknown.
_____ Other parent is not reasonably available.
_____ Only one parent has legal responsibility for the care and custody of subject.

PERSON <u>OBTAINING</u> CONSENT **DATE & TIME AM/PM**

I certify that I have explained the research,it's purposes,and the procedures to the subject or subject's legal representative before requesting their signature.

CONSENT TO PARTICIPATE IN A CLINICAL RESEARCH STUDY
Experimental Group

STUDY TITLE: The Role of Contralateral Movement in Boys with Attention Deficit Hyperactivity Disorder (ADHD)

PRINCIPAL INVESTIGATOR: **Dr. Jessica Foster**

CONTACT TELEPHONE NUMBER: **614-563-0997**

SUBJECT'S NAME: _____DATE OF BIRTH: _____

NOTE: The words "you" and "your" are used in this consent form. These words refer to the study volunteer whether a child or an adult.

1) INTRODUCTION

We invite you to be in this research study because we are looking at how 7 to 9 year old boys with and without ADHD do certain kinds of movements. We are really interested in a kind of movement called 'contralateral' movement. Examples of contralateral movement are crawling as a baby, swinging opposite arm to leg when waking and running, or skipping. Gym and music teachers talk about contralateral movement, but it does not come up in psychology and psychiatry.

Participation is voluntary. Please learn enough about this research study, its risks and benefits, to decide whether you should agree to participate. We will explain the study to you, and give you a chance to ask questions about anything you do not understand. This process is called "informed consent". It is up to you to choose if you want to be in this study. You may refuse to be in this study or quit this study at any time, and standard medical care will still be available here or at a doctor of your choice without a penalty or loss of benefits to you.

Before agreeing to participate, it is important to read and understand the study information in this consent form**.** By signing the consent form, you agree to be in this study.

This study involves children between 7and 9 years of age. If your child is 9 years of age, he must also agree to be in the study by signing an Assent form.

You will be given a signed and dated copy of the consent and the assent form.

2) WHY ARE WE DOING THIS RESEARCH STUDY?

This study is looking at 3 things: 1) how children move, 2) Attention Deficit Hyperactivity Disorder (ADHD) diagnosis and 3) how ADHD medicines might change movement. Some teachers use contralateral movement exercises in the classroom because they think it helps attention, self-control and learning. These exercises might be helpful for people with ADHD but we must first study the contralateral movement in people with ADHD before we can study the specific exercises.

We know from earlier research that most children with ADHD move better when they take their medicine. This study hopes to learn if people with ADHD can also do better at contralateral movements when medicated. We will compare 7 to 9 year old boys with ADHD to 7 to 9 year boys without ADHD.

We also know that girls tend to develop movement abilities earlier than boys. It is generally agreed that most children have mastered all contralateral movements by age six. To control for the difference between boys and girls, we will only look at boys in this study.

3) WHERE WILL THE STUDY BE DONE AND HOW MANY SUBJECTS WILL TAKE PART?

Study visits to record your child doing movement will take place at a site that is convenient for you and your family. Potential sites include NCH, OSU, your home or your child's elementary school (pending approval from their school). Visits to your doctor for diagnosis and medical management of ADHD symptoms will be done at your doctor's office.

Forty-eight boys will be participating in this study. Twenty-four will have ADHD and 24 will not have ADHD.

4) WHAT WILL HAPPEN DURING THE STUDY AND HOW LONG WILL IT LAST?

Your participation should not take any longer than 3 months. If you choose to participate, you will meet with a representative from the study at the beginning to complete a short survey and to address any questions you may have. Your child will meet with a member of the research team just once approximately every month, or every 4 weeks to allow for the recordings of your child. While the care provided to your child by their doctor will certainly involve more time and likely last beyond the duration of this study, contact with your child for the sole purposes of this research will not go beyond this approximated 3 month timeframe. The co-investigator, Melinda Cooksey, will be the one meeting with families to explain the study, obtaining consent and recording the movement activity.

The location of study visits will be specified by you, based upon convenience to your family (NCH, OSU, family home, elementary school, etc.). Each recording takes around 60 to 90

seconds. Two cones will be placed on the ground and the child will be asked to do a movement activity traveling between the cones three times (back and forth three times). Children are only recorded from their shoulders down to protect their anonymity. A digital video camera will be used for the recordings. The recordings are then downloaded into a secure, password protected computer and are indicated using alphanumeric codes.

Study Visit #1:
All study visits will be done at a site convenient to you and your family. Potential sites include NCH, OSU, your home or your child's elementary school (pending approval from their school), or any other site you find most convenient. This first study visit will include this informed consent process, a brief questionnaire and the first recording of your child. This visit may take as long as 30 minutes. If your child has not yet been medicated for ADHD, this will serve as the one non-medicated recording needed for this study. If your child has already begun medication for his ADHD, this will serve as the first of two medicated recordings.

Study Visit #2:
This visit will take place about one month after study visit #1.

- If your child had the first recording taken while medicated, we will ask your child to take a 'drug holiday' or washout period from their prescribed medication prior to this study visit. Washout periods will be between 1 day and 3 weeks depending on the child's medication type and their physician's opinion of a suitable washout period. This will serve as the one non-medicated recording needed for this study.
- If your child had not yet begun his medication for ADHD before the first study visit, he will be able to stay on his medication of this study visit. This will serve as the first of two medicated recordings.

Melinda Cooksey will meet your with your son at a site of your convenience for this study visit #2. She will again record him doing a movement activity. This visit should take no longer than 5 minutes.

Study Visit #3:
About one month after study visit #2, Melinda Cooksey will again meet with your son at a site of your convenience. She will record him doing a movement activity. This study visit should take no longer than 5 minutes. He will stay on his medication for this final recording.

5) WHAT ARE THE RISKS OF BEING IN THIS STUDY?

Washout from ADHD Medications:

In order to take part in this study, it will be necessary to stop some or all medicines being taken for the treatment of ADHD. This is sometimes called a Washout Period, and there is a possibility that the symptoms, hyperactivity and/or inattentiveness, will get worse when these medicines are stopped. We need to record your child doing a movement activity while not under the influence of their prescribed medication. Your child's doctor will help us decide what length of time your child will need to be off his medications for this Washout Period and we will do all we can to make sure that the timing of this drug 'holiday' is as convenient for your family as possible.

Movement Recording:

The physical risks are certainly less than your child would face in their daily activities in gym class or during recess play. It is possible that your child could trip or feel silly about how they looked doing the recorded movement, but the recordings will be done in a private area and the recordings will be viewed only at a later time by a member of the research team. Since your child will never be present at the viewing of the recordings and because all recordings will be from the shoulders and below, there is little chance of embarrassment or personal discomfort associated with participation. There is almost no risk of injury or harm coming to your child as a result of their participation.

6) ARE THERE BENEFITS TO TAKING PART IN THIS STUDY?

There may be no direct benefit to you or your son for participating in this study. After your child is finished participating in the study we will be happy to provide you with a free developmental movement patterning analysis session that may offer you valuable information about your child's movement. Importantly, your participation would help us learn new things about ADHD that could be applied to classroom approaches to dealing with ADHD and clinical practice.

7) WILL THERE BE ANY COSTS TO ME?

It will not cost you anything to be in this study. You will not be paid to be in this study.

8) WHAT HAPPENS IF BEING IN THIS STUDY CAUSES INJURIES?

If your child is hurt by the procedures that are part of the Study, you should seek medical treatment for the injuries and tell the Study Doctor as soon as possible at the number on the first page of this form. If it is an emergency, call 911 or go to the nearest emergency department.

In most cases, this care will be billed to your health insurance company or whoever usually pays for your health care at the usual charges, but some insurance companies will not pay for care related to a study. If the care is provided at Nationwide Children's Hospital, we make no commitment to pay for the medical care provided to you. No funds have been set aside to compensate you in the event of injury. If no one else pays for your care, you may have to pay for the cost of this care. This does not mean that you give up any of your legal rights to seek compensation for your injuries.

9) WHAT HAPPENS IF I DO NOT FINISH THIS STUDY?

It is your choice to be in this study or to stop at any time. If you decide to stop being in this study, it is OK, but you must call the Principal Investigator or the study coordinator. If you stop being in the study, there will not be a penalty or loss of benefits to which you are otherwise entitled.

If at any time the Principal Investigator believes participating in this study is not the best choice of care, the study may be stopped and other care prescribed. If the study instructions are not followed, participation in the study may also be stopped. If unexpected medical problems come up, the Principal Investigator may decide to stop your participation in the study.

10) HOW WILL MY STUDY INFORMATION BE KEPT PRIVATE?

Information collected for this study will be kept confidential to the extent provided by law. Information used and/or disclosed (shared with someone outside of Nationwide Children's Hospital) may include information that can identify you. This is called "protected health information" or PHI. By agreeing to be in this study, you are giving permission or authorizing Dr. Jessica Foster and her study staffs to collect, use, and disclose your PHI for this research study. Information collected is the property of Jessica Foster. In the event of any publication regarding this study, your identity will not be revealed.

If you have a bad outcome or adverse event from being in this study, the Principal Investigator and staff or other health care providers may need to look at your entire medical records.

The PHI collected or created under this research study will be used/disclosed as needed until the end of the study. The records of this study will be kept for an indefinite period of time.

PHI that may be used or disclosed: Address; Telephone/Fax Numbers; Birth Date; E-mail Addresses/URLs

People or Companies authorized to use, disclose, and receive PHI collected or created by this research study:
PI and study staff

- **Dr. Jessica Foster (PI NCH)**
- **Dr. L. Eugene Arnold (PI OSU)**
- **Melinda Cooksey (study coordinator)**

The Nationwide Children's Hospital Institutional Review Board (the committee that reviews all human subject research)

Nationwide Children's Hospital internal auditors

The Office for Human Research Protections (OHRP) (the federal government office that oversees human subject research)

Reason(s) why the use or disclosure is being made: To contact you during the study

You may decide not to authorize the use and disclosure of your PHI. However, if it is necessary for this study, you will not be able to be in this study. If you agree to be in this study and later decide to withdraw your participation, you may also withdraw your authorization to use your PHI. This request must be made in writing to the Principal Investigator. If you withdraw your authorization, no new PHI may be collected and the PHI

already collected may not be used unless it has already been used or is needed to complete the study analysis and reports.

Dr. Jessica Foster keeps a database of all subjects who participate in a research study. This database may be used to contact people about future studies. Only Jessica Foster and her staff have access to this database. The database will not be disclosed or sold to others outside Nationwide Children's Hospital.

Please initial:

____ I want to be contacted about future research studies.

____ I do not want to be contacted about future research studies.

11) OTHER IMPORTANT INFORMATION

Your child's doctor will communicate with your primary care physician regarding diagnosis and treatment. This study is only following your son during his diagnosis and early treatment. We will not interfere with his prescribed medication or treatment plan other than the washout period in phase three.

You will be told the results of this study at a later date.

Being in more than one research study at the same time may cause injury. Please tell the Principal Investigator about being in any other research study so a decision can be made about being in more than one study at the same time

12) WHOM SHOULD I CALL IF I HAVE QUESTIONS OR PROBLEMS?

If you have questions about anything while on this study or you have been injured by the research, you may contact the co-investigator, Melinda Cooksey, at 614-563-0997, Monday – Friday, between 8a and 5p.

If you have questions, concerns, or complaints about the research, questions about your rights as a research volunteer, cannot reach the Principal Investigator, or want to call someone else, please call (614) 722-2708, Nationwide Children's Hospital Institutional Review Board, (IRB, the committee that reviews all research in humans at Nationwide Children's Hospital).

Subject's Name _____ Date of Birth_____

SUBJECT or SUBJECT'S PARENT OR PERSON AUTHORIZED TO CONSENT ON BEHALF OF THE CHILD (SUBJECT TO THE SUBJECT'S GENERAL MEDICAL CARE)

I have read this consent form and have had a chance to ask questions about this research study. These questions have been answered to my satisfaction. If I have more questions about participation in this study or a research-related injury, I may contact the Principal Investigator. By signing this consent form, I certify that all health information I have given is true and correct to the best of my knowledge.

I have been given a copy of the Nationwide Children's Hospital Notice of Privacy Practices. I understand that my right to my patient information that is created or collected by Nationwide Children's Hospital in the course of this research can be temporarily suspended for as long as the research is in progress. I also understand that my right to access will be reinstated upon completion of this research.

I give permission for my child to participate in this study. I will be given a copy of this consent form with all the signatures for my own records.

CONSENT SIGNATURES

_____ _____ _____

SUBJECT or SUBJECT'S LEGAL REPRESENTATIVE DATE & TIME AM/PM

_____ _____ _____

SUBJECT or SUBJECT'S LEGAL REPRESENTATIVE DATE & TIME AM/PM

Permission of the second parent not obtained because (select all that apply):

_____ Not required by the IRB (risk level 1 or 2).
_____ Other parent is deceased.
_____ Other parent is unknown.
_____ Other parent is not reasonably available.
_____ Only one parent has legal responsibility for the care and custody of subject.

_____ _____

PERSON <u>OBTAINING</u> CONSENT DATE & TIME AM/PM

I certify that I have explained the research, it's purposes,and the procedures to the subject or subject's legal representative before requesting their signature.

ASSENT TO PARTICIPATE IN RESEARCH
Control Group
(FOR SUBJECTS 9 YEARS UP TO 18 YEARS OF AGE)

Study Title: The Role of Contralateral Movement in Boys with Attention Deficit Hyperactivity Disorder (ADHD).

Study Doctor: Dr. Jessica Foster

Subject's Name: _____ ***Date of Birth:*** _____

You are being asked to be in a research study. Studies are done to find better ways to treat people or to understand things better.

- **This form will tell you about the study to help you decide whether or not you want to volunteer to participate.**
- **You should ask any questions you have before making up your mind. You can think about it and discuss it with your family or friends before you decide.**
- **It is okay to say "No" if you don't want to be in the study. If you say "Yes" you can change your mind and stop being in the study at any time without getting in trouble.**
- **If you decide you want to be in the study, an adult (usually a parent) will also need to give permission for you to be in the study.**

1. What is this study about? This study is about how different kinds of kids move. We are interested in looking at movement such as walking, running, skipping and jumping in different kinds of children. Some of the boys we will work with will have Attention Deficit Hyperactivity Disorder, or ADHD. The other boys will not have ADHD. We will be recording both groups of boys doing different activities to learn more about the differences in how different kinds of boys can do certain movements.

2. What will I need to do (what will be done to me) if I am in this study? If you choose to be in this study you will be videotaped/recorded doing a movement such as walking, running or skipping. You will be recorded one time each month for three (3) months.

3. How long will I be in the study? You will be in the study for three (3) months, but you will only meet with somebody from the research team one time each month for less than five (5) minutes.

4. Can I stop being in the study? You may stop being in the study at any time.

5. What bad things might happen to me if I am in the study? Only two bad things could happen during this study. It is possible that we could find out that you have symptoms of a disorder when do our assessments. If this were to happen, we would be sure to help your parents find the right doctors to follow up with. If you do show any symptoms, you will not be able to participate in this study. If this happens, any forms with information about you on them will be shredded. It is also possible that you could stumble or trip while doing the movement. We will do everything we can to make sure that you are in a private place when you are recorded so that you would not be embarrassed if you felt silly doing the movement. We will not ask you to do anything that you wouldn't do normally in gym class or doing recess.

6. What good things might happen to me if I am in the study? You will not receive any rewards for being in the study. Your only reward will be knowing that you helped us learn new things. We will provide your parents with information about the study once it is finished.

7. Will I be given anything for being in this study?

You will not be paid for being in this study. When the study is finished you and your parent can come in for a freemovement/exercise session.

8. Who can I talk to about the study?
For questions about the study you may contact ***Melinda Cooksey at 614-563-0997 or Cooksey.6@osu.edu***.

To discuss other study-related questions with someone who is not part of the research team, you may contact the Institutional Review Board Office (the group that reviews all human subject research) at 614-722-2708.

Signing the assent form

I have read (or someone has read to me) this form. I have had a chance to ask questions before making up my mind. I want to be in this research study.

Signature or printed name of subject	**Date and time**	**AM/PM**

Investigator/Research Staff

I have explained the research to the participant before requesting the signature above. There are no blanks in this document. A copy of this form has been given to the participant or his/her representative.

Printed name of person obtaining assent	**Signature of person obtaining assent**
	Date and time **AM/PM**
Printed name of Principal Investigator ***(optional)***	**Signature of Principal Investigator** ***(optional)***
	Date and time **AM/PM**

This form must be accompanied by an IRB approved consent form signed by a parent/guardian.

ASSENT TO PARTICIPATE IN RESEARCH
Experimental Group
(FOR SUBJECTS 9 YEARS UP TO 18 YEARS OF AGE)

Study Title: The Role of Contralateral Movement in Boys with Attention Deficit Hyperactivity Disorder (ADHD).

Study Doctor: Dr. Jessica Foster

Subject's Name: _____ ***Date of Birth:*** _____

You are being asked to be in a research study. Studies are done to find better ways to treat people or to understand things better.

- **This form will tell you about the study to help you decide whether or not you want to volunteer to participate.**
- **You should ask any questions you have before making up your mind. You can think about it and discuss it with your family or friends before you decide.**
- **It is okay to say "No" if you don't want to be in the study. If you say "Yes" you can change your mind and stop being in the study at any time without getting in trouble.**
- **If you decide you want to be in the study, an adult (usually a parent) will also need to give permission for you to be in the study.**

1. What is this study about? This study is about how different kinds of kids move. We are interested in looking at movement such as walking, running, skipping and jumping in different kinds of children. Some of the boys we will work with will have Attention Deficit Hyperactivity Disorder, or ADHD. The other boys will not have ADHD. We will be recording both groups of boys doing different activities to learn more about the differences in how different kinds of boys can do certain movements.

2. What will I need to do (what will be done to me) if I am in this study? If you have ADHD and if you choose to be in this study you will be videotaped/recorded doing a movement such as walking, running or skipping. You will be recorded three (3) times total.

3. How long will I be in the study? You will be in the study for about three (3) months, but you will only meet with a member of the research team, Melinda Cooksey, one time about each month for less than five (5) minutes.

4. Can I stop being in the study? You may stop being in the study at any time.

5. What bad things might happen to me if I am in the study? *Before the third recording, you will need to go off you medicines for ADHD for between 1 day and 3 weeks. Your doctor will decide how long you will need to go off your medicine. During this 'drug holiday' your symptoms of hyperactivity and/or inattentiveness may get worse because you have stopped these medicine. The other thing that could happen to you in this study would be if you were to stumble or trip while doing the movement. We will do everything we can to make sure that you are in a private place when you are recorded so that you would not be embarrassed if you felt silly doing the movement. We will not ask you to do anything that you wouldn't do normally in gym class or during recess.*

6. What good things might happen to me if I am in the study? You will not receive any rewards for being in the study. Your only reward will be the knowledge that you helped us learn new things. We will provide your parents with information about the study once it is finished.

7. Will I be given anything for being in this study? *You will not be paid for being in this study, but if you and your parent want to come in for an exercise/movement session after the study is finished you can come do that for free.*

8. Who can I talk to about the study? **For questions about the study you may contact *Melinda Cooksey at 614-563-0997 or Cooksey.6@osu.edu***.

To discuss other study-related questions with someone who is not part of the research team, you may contact the Institutional Review Board Office (the group that reviews all human subject research) at 614-722-2708.

Signing the assent form

I have read (or someone has read to me) this form. I have had a chance to ask questions before making up my mind. I want to be in this research study.

Signature or printed name of subject	**Date and time**	**AM/PM**

<u>Investigator/Research Staff</u>

I have explained the research to the participant before requesting the signature above. There are no blanks in this document. A copy of this form has been given to the participant or his/her representative.

Printed name of person obtaining	**Signature of person obtaining assent**

assent	
	Date and time **AM/PM**
Printed name of Principal Investigator ***(optional)***	**Signature of Principal Investigator** ***(optional)***
	Date and time **AM/PM**

This form must be accompanied by an IRB approved consent form signed by a parent/guardian.

APPENDIX B: QUESTIONNAIRE

The Role of Contralateral Movement in Boys
With Attention Deficit Hyperactivity Disorder (ADHD)

This brief questionnaire will help us gain important preliminary information about your child's movement history. This information will be stored separately from your consent information to protect the confidentiality of your child.

Thank you for taking a moment to offer us this important information.

1) Did your child crawl as a baby?
a. Yes (if yes, please answer 1b below)
b. No
c. I do not know
d. I prefer not to answer

1b) If your child did crawl, how long did they do so? _____ (in months)
c. I do not know
d. I prefer not to answer

2) At what age did your child start walking? _____ (in months)
c. I do not know
d. I prefer not to answer

3) Does your child now or has your child ever participated in sports, gymnastics or dance?
a. Yes
b. No

APPENDIX C: OSU SIGMA RANKING GUIDELINES

The OSU SIGMA is a criterion referenced assessment tool that permits the examiner to rate qualitative aspects of eleven basic motor skills, one of which is skipping. Each skill has four levels of development that range from Level 1, the least mature performance, to Level 4, the mature functional behavior of that skill. Each skill has specific established criteria that provide the examiner with a descriptive assessment of the child's current gross motor functional capabilities. The scale was developed in 1979 by E. Michael Loovis and Walter F. Ersing to provide a method of descriptive assessment that helps to determine the most logical starting point relative to initiation intervention program planning for working to improve these skills in children.

Level 1:
The child cannot skip but will likely demonstrate any of the following behaviors:

- Running, galloping, hopping, leaping

Level 2:
The child attempts to skip while doing normal walking or running pattern and demonstrates the following behaviors:

- performs skip more often than not on the same leg though not necessarily consecutively
- holds arms either down at sides or slightly bent with hands at approximately waist level

Level 3:
The child skips and demonstrates the following behaviors:

- alternates feet
- does not use arms in opposition, if at all
- does skipping pattern slowly, and it appears segmented (the child may walk or run for brief periods)

Level 4:
The child skips and demonstrates the following behaviors:

- alternates feet
- uses arms in opposition (right arm forward when left leg is forward)
- executes skip with ease and good coordination

APPENDIX D: DATA SET AND RAW DATA

SUBJECT #	RECORDING #	GROUP	SURVEY #1	SURVEY #2	SURVEY #3
959	1	2	2	3	1
94	1	2	2	3	1
513	1	2	2	1	1
503	1	2	3	3	1
628	1	2	2	3	1
990	1	2	1	3	1
122	1	2	1	5	2
829	1	2	1	1	1
551	1	2	1	2	1
456	1	2	2	7	1
676	1	2	1	4	1
747	1	2	1	1	2
900	1	2	3	3	1
523	1	2	2	1	1
157	1	2	3	6	1
288	1	2	3	8	1
180	1	2	1	3	2
481	1	2	3	4	1
867	1	2	1	3	2
3	1	2	1	6	2
639	1	2	3	8	2
757	1	2	3	2	1
97	1	2	3	6	1
289	1	2	2	3	1
968	1	1	1	2	1
227	1	1	1	3	1
977	1	1	1	3	2
923	1	1	1	2	1
350	1	1	1	4	1
919	1	1	1	3	1
113	1	1	3	2	1
368	1	1	1	2	2
697	1	1	1	1	1
550	1	1	3	1	1
391	1	1	1	5	1
93	1	1	1	2	1
821	1	1	2	4	1
654	1	1	1	4	1
423	1	1	1	3	1
119	1	1	1	2	1
142	1	1	1	2	1
671	1	1	2	7	1
317	1	1	1	2	1

574	1	1	1	3	1
813	1	1	3	2	1
499	1	1	1	2	1
344	1	1	1	3	1
791	1	1	1	3	1
988	1	1	1	3	1
959	2	2	2	3	1
94	2	2	2	3	1
513	2	2	2	1	1
503	2	2	3	3	1
628	2	2	2	3	1
990	2	2	1	3	1
122	2	2	1	5	2
829	2	2	1	1	1
551	2	2	1	2	1
456	2	2	2	7	1
676	2	2	1	4	1
747	2	2	1	1	2
900	2	2	3	3	1
523	2	2	2	1	1
157	2	2	3	6	1
288	2	2	3	8	1
180	2	2	1	3	2
481	2	2	3	4	1
867	2	2	1	3	2
3	2	2	1	6	2
639	2	2	3	8	2
757	2	2	3	2	1
97	2	2	3	6	1
289	2	2	2	3	1
968	2	1	1	2	1
227	2	1	1	3	1
977	2	1	1	3	2
923	2	1	1	2	1
350	2	1	1	4	1
919	2	1	1	3	1
113	2	1	3	2	1
368	2	1	1	2	2
697	2	1	1	1	1
550	2	1	3	1	1
391	2	1	1	5	1
93	2	1	1	2	1
821	2	1	2	4	1
654	2	1	1	4	1

423	2	1	1	3	1
119	2	1	1	2	1
142	2	1	1	2	1
671	2	1	2	7	1
317	2	1	1	2	1
574	2	1	1	3	1
813	2	1	3	2	1
499	2	1	1	2	1
344	2	1	1	3	1
791	2	1	1	3	1
988	2	1	1	3	1
959	3	3	2	3	1
94	3	3	2	3	1
513	3	3	2	1	1
503	3	3	3	3	1
628	3	3	2	3	1
990	3	3	1	3	1
122	3	3	1	5	2
829	3	3	1	1	1
551	3	3	1	2	1
456	3	3	2	7	1
676	3	3	1	4	1
747	3	3	1	1	2
900	3	3	3	3	1
523	3	3	2	1	1
157	3	3	3	6	1
288	3	3	3	8	1
180	3	3	1	3	2
481	3	3	3	4	1
867	3	3	1	3	2
3	3	3	1	6	2
639	3	3	3	8	2
757	3	3	3	2	1
97	3	3	3	6	1
289	3	3	2	3	1
968	3	1	1	2	1
227	3	1	1	3	1
977	3	1	1	3	2
923	3	1	1	2	1
350	3	1	1	4	1
919	3	1	1	3	1
113	3	1	3	2	1
368	3	1	1	2	2
697	3	1	1	1	1

550	3	1	3	1	1
391	3	1	1	5	1
93	3	1	1	2	1
821	3	1	2	4	1
654	3	1	1	4	1
423	3	1	1	3	1
119	3	1	1	2	1
142	3	1	1	2	1
671	3	1	2	7	1
317	3	1	1	2	1
574	3	1	1	3	1
813	3	1	3	2	1
499	3	1	1	2	1
344	3	1	1	3	1
791	3	1	1	3	1
988	2	1	1	3	1

SUBJECT #	SIGMA RANK	MED TYPE	MED DOSE
959	3	2	1
94	1	1	1
513	3	1	1
503	4	2	1
628	4	1	2
990	4	6	2
122	4	5	2
829	4	2	2
551	3	5	1
456	1	2	2
676	2	2	1
747	4	6	1
900	1	4	1
523	1	3	2
157	3	6	2
288	3	5	2
180	4	1	2
481	4	1	2
867	4	3	1
3	3	5	2
639	1	6	2
757	4	6	1
97	1	6	2
289	4	2	2
968	3	7	3
227	4	7	3
977	4	7	3

923	4	7	3
350	4	7	3
919	3	7	3
113	3	7	3
368	4	7	3
697	4	7	3
550	4	7	3
391	3	7	3
93	4	7	3
821	4	7	3
654	4	7	3
423	4	7	3
119	4	7	3
142	4	7	3
671	4	7	3
317	4	7	3
574	4	7	3
813	4	7	3
499	4	7	3
344	3	7	3
791	4	7	3
988	4	7	3
959	2	2	1
94	1	1	1
513	2	1	1
503	4	2	1
628	4	1	2
990	4	6	2
122	3	5	2
829	3	2	2
551	3	5	1
456	1	2	2
676	2	2	1
747	4	6	1
900	1	4	1
523	1	3	2
157	3	6	2
288	3	5	2
180	4	1	2
481	4	1	2
867	2	3	1
3	3	5	2
639	1	6	2
757	4	6	1
97	1	6	2
289	4	2	2
968	3	7	3

227	4	7	3
977	4	7	3
923	4	7	3
350	4	7	3
919	3	7	3
113	4	7	3
368	4	7	3
697	4	7	3
550	4	7	3
391	3	7	3
93	4	7	3
821	3	7	3
654	4	7	3
423	3	7	3
119	3	7	3
142	3	7	3
671	4	7	3
317	4	7	3
574	4	7	3
813	4	7	3
499	4	7	3
344	3	7	3
791	3	7	3
988	4	7	3
959	4	2	1
94	1	1	1
513	2	1	1
503	4	2	1
628	4	1	2
990	4	6	2
122	4	5	2
829	4	2	2
551	4	5	1
456	1	2	2
676	1	2	1
747	4	6	1
900	1	4	1
523	1	3	2
157	4	6	2
288	4	5	2
180	4	1	2
481	4	1	2
867	4	3	1
3	3	5	2
639	1	6	2
757	4	6	1
97	1	6	2

289	4	2	2
968	3	7	3
227	3	7	3
977	4	7	3
923	4	7	3
350	4	7	3
919	3	7	3
113	4	7	3
368	4	7	3
697	4	7	3
550	4	7	3
391	3	7	3
93	4	7	3
821	3	7	3
654	4	7	3
423	3	7	3
119	3	7	3
142	4	7	3
671	4	7	3
317	4	7	3
574	4	7	3
813	4	7	3
499	4	7	3
344	3	7	3
791	4	7	3
988	4	7	3

Q1

One-way ANOVA: rank versus group

```
Source  DF      SS     MS     F     P
group    1  26.264 26.264 29.26 0.000
Error  145 130.158  0.898
Total  146 156.422

S = 0.9474   R-Sq = 16.79%   R-Sq(adj) = 16.22%


                    Individual 95% CIs For Mean Based on
                    Pooled StDev
Level   N    Mean   StDev -----+---------+---------+---------+----
1      75  3.7067  0.4584                    (-----*-----)
2      72  2.8611  1.2705  (------*-----)
                    -----+---------+---------+---------+----
                      2.80      3.15      3.50      3.85

Pooled StDev = 0.9474
```

Tukey 95% Simultaneous Confidence Intervals
All Pairwise Comparisons among Levels of group

Individual confidence level = 95.00%

group = 1 subtracted from:

```
group    Lower   Center    Upper  ---------+---------+---------+---------+
2      -1.1545  -0.8456  -0.5366  (-------*-------)
                          ---------+---------+---------+---------+
                              -0.80     -0.40     -0.00      0.40
```

Q2

Part1

Welch Two Sample t-test

data: adhdwm and adhdwom

t = -0.6262, df = 41.306, p-value = 0.5347

alternative hypothesis: true difference in means is not equal to 0

95 percent confidence interval:

-0.8801207 0.4634541

sample estimates:

mean of x mean of y

2.791667 3.000000

Part2:

Likelihood ratio tests of ordinal regression models

Response: as.factor(rank)

Model

1 as.factor(group) + as.factor(s1) + as.factor(s2) + as.factor(s3) + (1 | person)

2 as.factor(group) + as.factor(medtype) + as.factor(meddose) + as.factor(s1) + as.factor(s2) + as.factor(s3) + (1 | person)

	Resid. df	Resid. Dev	Test	Df	LR stat.	Pr(Chi)
1	58	150.1637				
2	52	125.6788	1 vs 2	6	24.48490	0.0004251545

Q3

One-way ANOVA: rank versus s1

```
Source  DF      SS     MS     F     P
s1       2  22.236  11.118  11.93  0.000
Error  144  134.186   0.932
Total  146  156.422

S = 0.9653   R-Sq = 14.22%   R-Sq(adj) = 13.02%


                    Individual 95% CIs For Mean Based on
                    Pooled StDev
Level   N    Mean   StDev  ---+---------+---------+---------+------
1      87  3.5977  0.6187                        (----*----)
2      27  2.6296  1.3344  (--------*--------)
3      33  3.0303  1.3106           (--------*-------)
                   ---+---------+---------+---------+------
                    2.40      2.80      3.20      3.60

Pooled StDev = 0.9653


Tukey 95% Simultaneous Confidence Intervals
All Pairwise Comparisons among Levels of s1

Individual confidence level = 98.08%


s1 = 1 subtracted from:

s1    Lower   Center    Upper   -+---------+---------+---------+--------
2   -1.4718  -0.9681  -0.4643   (------*------)
3   -1.0349  -0.5674  -0.0999         (------*------)
                        -+---------+---------+---------+--------
                      -1.40     -0.70      0.00      0.70


s1 = 2 subtracted from:

s1    Lower  Center   Upper   -+---------+---------+---------+--------
3   -0.1927  0.4007  0.9941                  (--------*-------)
                      -+---------+---------+---------+--------
                    -1.40     -0.70      0.00      0.70
```

One-way ANOVA: rank versus s2

```
Source  DF      SS     MS     F     P
s2       7  26.036  3.719  3.97  0.001
Error  139  130.386  0.938
Total  146  156.422

S = 0.9685   R-Sq = 16.64%   R-Sq(adj) = 12.45%


                    Individual 95% CIs For Mean Based on Pooled StDev
Level   N    Mean   StDev   +---------+---------+---------+---------
1      18  3.1667  1.2005                  (-----*------)
```

```
2      36  3.7500  0.4392                          (----*---)
3      51  3.3529  1.0163                       (---*---)
4      15  3.4000  0.9856                       (------*------)
5       6  3.3333  0.5164                 (-----------*----------)
6       9  2.4444  1.1304          (--------*--------)
7       6  2.5000  1.6432         (----------*----------)
8       6  2.1667  1.3292    (----------*----------)
                      +---------+---------+---------+---------
                     1.40      2.10      2.80      3.50
```

Pooled StDev = 0.9685

Tukey 95% Simultaneous Confidence Intervals
All Pairwise Comparisons among Levels of s2

Individual confidence level = 99.75%

s2 = 1 subtracted from:

```
s2    Lower   Center   Upper  ---------+---------+---------+---------+
2   -0.2767   0.5833  1.4433                (-----*-----)
3   -0.6305   0.1863  1.0030              (----*-----)
4   -0.8082   0.2333  1.2748              (------*-----)
5   -1.2377   0.1667  1.5710           (--------*--------)
6   -1.9384  -0.7222  0.4940       (-------*-------)
7   -2.0710  -0.6667  0.7377      (---------*--------)
8   -2.4044  -1.0000  0.4044     (--------*---------)
                      ---------+---------+---------+---------+
                          -1.5       0.0       1.5       3.0
```

s2 = 2 subtracted from:

```
s2    Lower   Center    Upper  ---------+---------+---------+---------+
3   -1.0456  -0.3971   0.2514            (---*----)
4   -1.2655  -0.3500   0.5655           (-----*-----)
5   -1.7303  -0.4167   0.8970        (--------*--------)
6   -2.4158  -1.3056  -0.1953     (------*-------)
7   -2.5637  -1.2500   0.0637    (--------*-------)
8   -2.8970  -1.5833  -0.2697  (-------*--------)
                      ---------+---------+---------+---------+
                          -1.5       0.0       1.5       3.0
```

s2 = 3 subtracted from:

```
s2    Lower   Center   Upper  ---------+---------+---------+---------+
4   -0.8280   0.0471  0.9221             (-----*-----)
5   -1.3054  -0.0196  1.2661          (--------*-------)
6   -1.9856  -0.9085  0.1686       (------*------)
7   -2.1387  -0.8529  0.4328      (-------*--------)
8   -2.4720  -1.1863  0.0995     (-------*--------)
                      ---------+---------+---------+---------+
                          -1.5       0.0       1.5       3.0
```

s2 = 4 subtracted from:

```
s2   Lower   Center   Upper  ---------+---------+---------+---------+
5   -1.5057  -0.0667  1.3724          (---------*--------)
6   -2.2116  -0.9556  0.3005      (--------*-------)
7   -2.3390  -0.9000  0.5390     (---------*---------)
8   -2.6724  -1.2333  0.2057   (---------*--------)
                      ---------+---------+---------+---------+
                          -1.5       0.0       1.5       3.0
```

s2 = 5 subtracted from:

```
s2   Lower   Center   Upper  ---------+---------+---------+---------+
6   -2.4590  -0.8889  0.6812     (---------*----------)
7   -2.5533  -0.8333  0.8866    (----------*----------)
8   -2.8866  -1.1667  0.5533  (----------*----------)
                      ---------+---------+---------+---------+
                          -1.5       0.0       1.5       3.0
```

s2 = 6 subtracted from:

```
s2   Lower   Center   Upper  ---------+---------+---------+---------+
7   -1.5146   0.0556  1.6257          (---------*----------)
8   -1.8479  -0.2778  1.2923        (---------*----------)
                      ---------+---------+---------+---------+
                          -1.5       0.0       1.5       3.0
```

s2 = 7 subtracted from:

```
s2   Lower   Center   Upper  ---------+---------+---------+---------+
8   -2.0533  -0.3333  1.3866      (----------*----------)
                      ---------+---------+---------+---------+
                          -1.5       0.0       1.5       3.0
```

One-way ANOVA: rank versus s3

```
Source   DF      SS    MS     F      P
s3        1    0.20  0.20  0.18  0.671
Error   145  156.23  1.08
Total   146  156.42
```

S = 1.038   R-Sq = 0.12%   R-Sq(adj) = 0.00%

```
                    Individual 95% CIs For Mean Based on
                    Pooled StDev
Level    N   Mean  StDev  --+---------+---------+---------+-------
1      123  3.276  1.035       (------*------)
2       24  3.375  1.056  (----------------*----------------)
                    --+---------+---------+---------+-------
                    3.00      3.25      3.50      3.75
```

Pooled StDev = 1.038

Tukey 95% Simultaneous Confidence Intervals
All Pairwise Comparisons among Levels of s3

Individual confidence level = 95.00%

s3 = 1 subtracted from:

```
s3   Lower  Center  Upper    +---------+---------+---------+---------
2   -0.359   0.099  0.556           (--------------*--------------)
                          +---------+---------+---------+---------
                        -0.60     -0.30      0.00      0.30
```

Q4

One-way ANOVA: s1 versus group

```
Source   DF      SS     MS     F      P
group     1   14.968  14.968  25.48  0.000
Error   145   85.195   0.588
Total   146  100.163
```

S = 0.7665   R-Sq = 14.94%   R-Sq(adj) = 14.36%

```
                    Individual 95% CIs For Mean Based on
                    Pooled StDev
Level   N    Mean   StDev  ----+---------+---------+---------+-----
1      75  1.3200  0.6810  (------*------)
2      72  1.9583  0.8465                    (------*------)
                    ----+---------+---------+---------+-----
                      1.25      1.50      1.75      2.00
```

Pooled StDev = 0.7665

Tukey 95% Simultaneous Confidence Intervals
All Pairwise Comparisons among Levels of group

Individual confidence level = 95.00%

group = 1 subtracted from:

```
group   Lower  Center   Upper  --------+---------+---------+---------+-
2      0.3884  0.6383  0.8883                   (-------*--------)
                        --------+---------+---------+---------+-
                             0.00      0.30      0.60      0.90
```

One-way ANOVA: s2 versus group

```
Source   DF      SS     MS      F      P
group     1   30.31  30.31  10.11  0.002
Error   145  434.88   3.00
Total   146  465.18

S = 1.732   R-Sq = 6.52%   R-Sq(adj) = 5.87%


                    Individual 95% CIs For Mean Based on
                    Pooled StDev
Level   N   Mean  StDev  --+---------+---------+---------+-------
1      75  2.800  1.273  (-------*-------)
2      72  3.708  2.106                  (-------*-------)
                    --+---------+---------+---------+-------
                    2.50      3.00      3.50      4.00

Pooled StDev = 1.732


Tukey 95% Simultaneous Confidence Intervals
All Pairwise Comparisons among Levels of group

Individual confidence level = 95.00%


group = 1 subtracted from:

group  Lower  Center  Upper    +---------+---------+---------+---------
2      0.344   0.908  1.473                (--------*---------)
                          +---------+---------+---------+---------
                        -0.60      0.00      0.60      1.20
```

One-way ANOVA: s3 versus group

```
Source   DF      SS     MS     F      P
group     1   1.062  1.062  8.09  0.005
Error   145  19.020  0.131
Total   146  20.082

S = 0.3622   R-Sq = 5.29%   R-Sq(adj) = 4.63%


                      Individual 95% CIs For Mean Based on Pooled StDev
Level   N    Mean   StDev   +---------+---------+---------+---------
1      75  1.0800  0.2731   (-------*-------)
2      72  1.2500  0.4361                  (-------*-------)
                     +---------+---------+---------+---------
                    1.00      1.10      1.20      1.30

Pooled StDev = 0.3622


Tukey 95% Simultaneous Confidence Intervals
All Pairwise Comparisons among Levels of group

Individual confidence level = 95.00%
```

group = 1 subtracted from: