EFFECT OF EXTRINSIC MOTIVATION ON ACADEMIC FLUENCY OUTCOMES

IN SURVIVORS OF PEDIATRIC MEDULLOBLASTOMA

APPROVED BY SUPERVISORY COMMITTEE

Peter Stavinoha, Ph.D., ABPP _____

Daniel Bowers, M.D. _____

Lana Harder, Ph.D., ABPP _____

Cheryl Silver, Ph.D. _____

Carroll Hughes, Ph.D., ABPP _____

Dedicated to my parents
with all my love and gratitude.

EFFECT OF EXTRINSIC MOTIVATION ON ACADEMIC FLUENCY OUTCOMES IN SURVIVORS OF PEDIATRIC MEDULLOBLASTOMA

by

ALICE ANN SPURGIN

DISSERTATION

Presented to the Faculty of the Graduate School of Biomedical Sciences

The University of Texas Southwestern Medical Center at Dallas

In Partial Fulfillment of the Requirements

For the Degree of

DOCTOR OF PHILOSOPHY

The University of Texas Southwestern Medical Center at Dallas

Dallas, Texas

August, 2012

ACKNOWLEDGMENTS

After months and months of dissertation planning meetings, more than 300 articles read, and an annotated bibliography that grew to be over 100 pages long single-spaced, I had become increasingly impatient to just pick a topic and get started. Then I read one little sentence buried in one little article that sparked the idea for this dissertation, an idea I might just run with for the rest of my career. So, I'd like to thank Dr. Pete Stavinoha for requiring me to read all those articles and, more to the point, for being the best mentor I ever could have asked for. Your combination of intellect, kindness, and dedication to training make you far superior to *any* hotshot. Thank you for tolerating my endless requests for rec letters and my ridiculous inability to procrastinate—a sometimes painful combination, I know. Thank you for being so consistently supportive, patient, and honest with me. Thank you for always looking for ways to support and enhance my training and my future career. Thank you…thank you…thank you! I will be forever grateful.

I feel incredibly lucky to be able to say that Pete is not the only member of my committee who has been dedicated to giving me as many opportunities as possible. Dr. Lana Harder's offer for me to be involved in demyelinating disorders research went far beyond my initial expectations; it seems as if every other month I get an email about another poster or manuscript submission that will have my name on it! Lana, thank you so much for going above and beyond—you've been a dissertation committee member, clinical mentor, and general research mentor, and I've learned so much from you in all three of those capacities. Having the opportunity to be trained by someone who is so

accomplished and talented in so many ways has been an invaluable experience, and I look forward to continuing to work with you on research projects!

Before asking him to be on this committee, I was told that Dr. Carroll Hughes rarely accepts such requests, so I'm not sure how I was lucky enough to be among those accepted. Dr. Hughes, thank you so much for your expert advice as I was designing the study and your patience in helping me re-learn all the stats I'd forgotten. Your guidance always promoted my independent development as a researcher, and running the analyses for this study made me confident in my ability to be my own statistician in the future.

I suspect (and my apologies if I do so incorrectly) that Dr. Cheryl Silver was my only committee member who read this finished manuscript line by line, cover to cover. Dr. Silver, your keen eye for detail—grammar, spelling, punctuation—was much appreciated! More importantly, your expertise in pediatric neuropsychology was invaluable, as you critically analyzed every detail of the study and provided so many creative and insightful suggestions as to how I might improve it every step of the way. Thank you so much for all the time you dedicated to this student you barely knew—and I'm so glad I've gotten to know you better through this process!

This study would never have gotten off the ground without the support of Dr. Dan Bowers, who has been so patient and kind in putting up with a random grad student hanging around and stealing patients for testing during clinics. Dan, thank you so much for not only supporting my study but also supporting my own training and career. I have so greatly appreciated the time you've taken to teach me about various aspects of neuro-oncology, to offer advice about career decisions, and even to write rec letters. I'm sorry

I'm not going "away" for my fellowship as I know you wanted me to, but I hope you'll forgive me! I'm thrilled to be continuing to collaborate with you in research!

Speaking of stealing patients during clinic, a huge thank-you to the neuro-onc team: Rachel Haarde, Amy Linton, Amy McDonald, Marissa Miller. Thank you for your kindness and patience in allowing me to test patients during clinics and for your support in helping me hunt down treatment variables in patients' charts! I also can't thank Dr. Lynn Gargan enough for digging through databases to identify potential participants and answering questions throughout the process. Dr. Gargan, while you were somewhat behind-the-scenes, everyone should know how essential you were to this study. Your speed in answering emails and compiling patient databases as the target population kept shifting was crucial—and truly impressive. Thank you so much for all of your help!

Other people behind the scenes also deserve many thanks. Dr. Brianne Butcher's enthusiasm in helping me snag participants at clinic was critical in keeping me sane on days I had two or three scheduled. Brianne, thank you also for keeping me "legal" by documenting all of my study visits in the medical records. I'd also like to thank Dr. Paul Glasier and Dr. Alison Wilkinson-Smith for coordinating with me on a couple dual clinical-research cases; I really appreciated your flexibility!

Last but certainly not least, I can't thank my family and friends enough for supporting me throughout this process. To my parents, whose financial support made it possible for me to attend graduate school in the first place, thank you for always being there when I needed encouragement or sympathy, even though you weren't quite sure what exactly I was doing. Many, many thanks to Katie Vick, one of my oldest and dearest friends, for everything from grocery store runs and venting sessions to handmade

cards wishing me luck. It's not every roommate who wouldn't utter a word of complaint when my paperwork took over the coffee table…and the dining table…and the kitchen counter. Your support and enthusiasm for my work means the world to me. Thank you also to Jackie Braud—your friendship would have been reason enough to go to grad school, even if this dissertation process had failed miserably and left me without a degree. Thank you for always being there with an encouraging word or sympathetic ear or baked goods!

In the midst of IRB madness and proposal writing and moonlighting ER shifts, I started dating an amazing guy named Brian Holland, who couldn't have been more patient, supportive, thoughtful, encouraging, and understanding. I am beyond blessed that he stuck with me through this whole process from start to finish and is now my fiancé. Brian, I hope you know that I'm happier to be marrying you than I am to be getting my Ph.D.

To my entire committee, I've valued my research experience here immensely and hope that we can continue to collaborate. I'm thrilled and honored that you invited me to stay, and I know that those of you who wanted me to leave felt that way because you care so much about my training and future career, and that means the world to me. I have never been so well supported and nurtured in my academic career as I was in this dissertation project; it truly never would have happened without each and every one of you. I will always look back on this experience with immense gratitude, and I hope that I'll be able to either return the favor or "pay it forward" to another student some day. Thank you.

EFFECT OF EXTRINSIC MOTIVATION ON ACADEMIC FLUENCY OUTCOMES IN SURVIVORS OF PEDIATRIC MEDULLOBLASTOMA

ALICE ANN SPURGIN, Ph.D.

The University of Texas Southwestern Medical Center at Dallas, 2012

PETER L. STAVINOHA, Ph.D., ABPP

Medulloblastoma is the most commonly diagnosed malignant pediatric brain tumor. While deficits in processing speed, memory, attention, and IQ are well documented in childhood medulloblastoma survivors, impairments in academic functioning have not been adequately studied in this population, despite the fact that most survivors require long-term special education services and are significantly less likely than their healthy peers to finish high school. The present study is the first to identify fluent academic performance as a significant weakness relative to academic skill development in childhood medulloblastoma survivors—thereby isolating fluency as a

major contributing factor to survivors' academic difficulties. The present study is also the first to investigate the effects of enhanced extrinsic motivation on fluent academic performance in pediatric medulloblastoma survivors. As such, this study represents a new direction for research in this population, moving beyond basic documentation of deficits toward intervention-focused research. A previous study indicated that extrinsic motivation enables survivors of childhood medulloblastoma to improve their performance to the normal range on tasks related to processing speed and attention (Riva, Pantaleoni, Milani, & Belani, 1989). However, prior to the present study, there had been no further investigations of this isolated finding. Present results suggest that a performance-based incentive used to enhance extrinsic motivation predicted statistically significant improvement, but not normalization of function, in performance on measures of academic fluency relative to baseline. No demographic, medical, or neuropsychological variables predicted response to incentive. Findings suggest that academic performance of survivors can significantly improve under highly motivating conditions. Recognition of this potential for improvement in light of persisting limitations in fluency, suggesting deficits that cannot be fully overcome, may inform academic supports. Additionally, the findings of this study may provide a rationale for investigations of the effect of varying levels of motivation in other pediatric medical populations and with respect to other areas of neurocognitive functioning. The findings of this study also represent a significant and novel contribution to the debate regarding level of effort and the effect of motivational states on neuropsychological performance.

TABLE OF CONTENTS

PRIOR PUBLICATIONS

Butcher, B., Spurgin, A. A., & Stavinoha, P. L. (2011). Developmental effects on working memory following pediatric stroke. *Journal of the International Neuropsychological Society, 17*(S1), 168.

Glasier, P., Spurgin, A. A., Stavinoha, P. L., Winick, N., & Rollins, N. (2011). Processing speed predicts adaptive living skills in pediatric patients with acute lymphoblastic leukemia treated with chemotherapy only. *The Clinical Neuropsychologist, 25*(4), 585.

Harder, L., Spurgin, A. A., Greenberg, B., & Graves, D. (2011). Cognitive functioning and school performance in pediatric demyelinating diseases: A comparison between multiple sclerosis (MS) and transverse myelitis (TM) groups. *Annals of Neurology, 70*(15), S150-S151.

Spurgin, A. A. (2007). Diffusion tensor imaging in children and adolescents: A technology review. *Stanford Journal of Neuroscience, 1,* 15-17.

Spurgin, A. A. (2007). Stanford's SNAILS: Providing a clearer picture of the brain. *Stanford Scientific, 5(2),* 33-35.

Spurgin, A. A., Benitez, O. J., Hernandez, A. M., van de Bruinhorst, K., Stavinoha, P. L, & Kernie, S. M. (2009). Predicting behavioral versus cognitive executive dysfunction in pediatric TBI. *The Journal of Head Trauma Rehabilitation, 24(5)*, 409.

Spurgin, A. A., Graves, D., Greenberg, B., & Harder, L. (2011). Psychological and clinical predictors of executive dysfunction in pediatric multiple sclerosis. *Archives of Clinical Neuropsychology, 26*(6), 494.

Spurgin, A. A., Krishnan, V., Renthal, W., & McClung, C. (2006). Depression and circadian genes in the mesolimbic dopamine circuit. UT Southwestern Summer Undergraduate Research Fellowship Program Poster Session, Dallas, TX, Aug. 10, 2006.

Spurgin, A. A., Morriss, M., Glasier, P., & Stavinoha, P. L. (2011). Complicated subdural empyema in an adolescent. *Archives of Clinical Neuropsychology, 26*(6), 471-472.

Stavinoha, P. L., Butcher, B., & Spurgin, A. A. (2011.) Pre-morbid functional complications. In J. Apps & K. Walter (Eds.), *Handbook of Pediatric Concussions*. New York: Springer.

Wilkinson, A., Nelson, S., & Spurgin, A. A. (2011). Readability of pediatric neuropsychological reports. *The Clinical Neuropsychologist, 25*(4), 606-607.

LIST OF FIGURES

LIST OF TABLES

LIST OF APPENDICES

CHAPTER ONE
Introduction

Brain tumors are the second most common form of childhood cancer (Gurney, Wall, Jukich, & Davis, 1999; Horner et al., 2009), and medulloblastoma is the most commonly diagnosed form of malignant brain tumor in the pediatric population (Gottardo & Gajjar, 2006). With current five-year survival rates of 70-85%, the majority of children diagnosed with medulloblastoma will become long-term survivors (Palmer, 2008). However, with this improved length of survival, understanding the late effects (i.e., long-term consequences) of medulloblastoma and its treatment has become an important area for research. Survivors of childhood medulloblastoma are at risk of neurocognitive deficits and declines in IQ and academic achievement (Mabbott, Spiegler, Greenberg, Rutka, Hyder, & Bouffet, 2005; Palmer, 2008; Palmer et al., 2001). These detrimental late effects are largely due to the effects of cranial radiation, which is a necessary component of the treatment of this aggressive tumor but is known to be associated with neurocognitive morbidity. In a real-world illustration of this, children who receive cranial radiation are 7 times more likely to require special education compared to survivors of childhood cancers who did not receive such radiation (Mitby et al., 2003; Palmer, 2008).

In particular, impairments in the academic functioning of childhood medulloblastoma survivors deserve more attention, as these impairments represent a real-world consequence of the neuropsychological deficits that have been well-documented in this population. Indeed, the majority of survivors of childhood medulloblastoma require long-term special education services (Hoppe-Hirsch et al., 1995; Maddrey et al., 2005)

and are significantly less likely than their healthy peers to finish high school (Mitby et al., 2003). However, academic performance in pediatric medulloblastoma survivors has not been extensively studied. It is theorized that one of the deficits underlying decreased academic performance in this population is cognitive fluency. While processing speed is a basic cognitive function referring to the speed at which a person can perform automatic, over-learned tasks, cognitive fluency is conceptualized as speed and accuracy of performance on more complex cognitive tasks. Thus, cognitive fluency involves processing speed, as well as other basic functions such as attention.

The only prior study that has addressed the possible effects of motivation in a pediatric brain tumor population noted that posterior fossa tumor patients showed no deficits on computerized reaction time and attention tasks (Riva, Pantaleoni, Melani, & Belani, 1989). However, processing speed and attention are well-documented deficits in this population, particularly in children treated with cranial radiation (Butler & Haser, 2006; Palmer, 2008; Palmer & Leigh, 2009; Steinlin et al., 2003). Thus, the authors of this prior study suggested that the computer, which was particularly fascinating for these children due to its relative novelty at the time, may have enhanced motivation, thus improving performance to the normal range. Although the impact of varying levels of motivation on performance has been shown in a pediatric traumatic brain injury population (McCauley, McDaniel, Pedroza, Chapman, & Levin, 2009), to our knowledge, there have been no prior studies that have explicitly looked at the effects of motivation on cognitive or academic performance in a pediatric brain tumor population. The general similarity between these two patient groups—both having an organic brain

insult—provides sufficient justification for the current investigation on motivation in a medulloblastoma population.

This study was the first to examine the impact of motivation on academic performance among childhood medulloblastoma survivors. The finding of significant effects of motivation could have implications for the design of special education services and other remedial academic programs. Additionally, this study represents a step toward discovering more specific cognitive and intellectual effects of cranial radiation that contribute to poorer academic outcomes in this population. Specifically, this project examined whether simple processing speed or the more complex cognitive fluency is more predictive of fluent academic performance.

CHAPTER TWO
Review of the Literature

POSTERIOR FOSSA TUMORS

Brain tumors are the second most common form of cancer during childhood, accounting for 16.6% of all cancers diagnosed among children younger than 15 years old (Gurney, Wall, Jukich, & Davis, 1999; Horner et al., 2009). In the United States, an estimated 2900 children and adolescents are diagnosed with a brain tumor annually (Horner et al., 2009). The term "posterior fossa" refers to the area where the cerebellum and brain stem are located, near the base of the skull. Tumors in this area are common in a pediatric population—although they comprise less than 1% of all brain tumors in adults (Hubbard, Scheithauer, Kispert, Carpenter, Wick, & Laws, 1989), posterior fossa tumors account for two-thirds of all brain tumors in children (George et al., 2003). Common types of posterior fossa tumors include brainstem glioma, cerebellar astrocytoma, choroids plexus papilloma, ependymoma, hemangioblastoma, primary neuroectodermal tumors, and medulloblastoma. Standard of care treatment for posterior fossa tumors primarily depends on histopathological diagnosis of tumor type. Treatment options generally include surgery, radiation, and chemotherapy. These treatments can be provided independently or in combination.

Late effects of cancer and its treatment include any medical, psychological, or cognitive effects evident months or years following diagnosis. There is a broad, long-term, neurocognitive impact of posterior fossa tumors, with deficits in areas ranging from language to executive functioning (Aarsen et al., 2009; Butler & Haser, 2006; Mabbott et

al., 2008; Palmer, 2008; Palmer & Leigh, 2009; Palmer, Reddick, & Gajjar, 2007; Reeves et al., 2006; Riva & Giorgi, 2000; Steinlin et al., 2003; Vaquero, Gomez, Quintero, Gonzalez-Rosa, & Marques, 2008; Zuzak, Poretti, Drexel, Zehnder, Boltshauser, & Grotzer, 2008). Some studies have noted a temporal progression of these late effects, wherein processing speed is the first noticeably impacted area of functioning, followed by memory and attention deficits, then visuospatial difficulties, and finally deficits in abstract thought and vocabulary (Palmer, 2008; Garcia-Perez et al., 1993). Clearly, such deficits impact the realm of academics, where one study found that 60% of posterior fossa tumor patients were in need of special education programs, yet only 26% were actually enrolled in such programs (Hoppe-Hirsch et al., 1995). Finally, in addition to these neurocognitive and emotional sequelae, Steinlin et al. (2003) noted that 30% of their posterior fossa tumor patient sample had psychopathology (ranging from anorexia to phobias), all but one of whom had a tumor affecting the vermis.

MEDULLOBLASTOMA

Approximately 20% of all pediatric brain tumors are medulloblastomas, making them the most common brain tumor in children (Blaney et al., 2006). Medulloblastomas are typically comprised of poorly differentiated epithelial cells arising from the fourth ventricle (Ris & Abbey, 2010). Standard-risk medulloblastoma describes such a tumor occurring in children older than 3 years, with near-total resection, and no evidence of dissemination (Ris & Abbey, 2010). High-risk medulloblastoma, then, is a tumor

occurring in children younger than 3 years, a tumor with sub-total resection, or a tumor that has metastasized.

Standard of Care

Acute symptoms of medulloblastoma typically include headaches and vomiting due to increased intracranial pressure, as well as gait disturbances due to the cerebellum's role in motor functions. Identification of the presence of a tumor is conducted by CT or MRI scans, while histology enables identification of tumor type. Standard of care is complete surgical resection, generally coupled with adjuvant craniospinal irradiation (CSI) and chemotherapy.

Due to the use of radiation and chemotherapy in the treatment of medulloblastoma, the current five-year survival rate has improved to 70-85% (Palmer, 2008). Increased awareness of the myriad neurocognitive late effects of radiation and chemotherapy has led to efforts to reduce the standard radiation dose in treating medulloblastoma. Thus, until the 1990s, the standard of care for an average-risk patient was 55 Gy delivered to the posterior fossa and 35 Gy delivered to the brain and spinal canal, whereas the current standard of care is 54 to 55 Gy delivered to the posterior fossa and 23.4 Gy (or 35-40 Gy for high-risk patients) delivered to the brain and spinal canal (Mulhern et al., 2001; Oyharcabal-Bourden et al., 2005; Palmer et al., 2003). Some clinicians have even administered a dose as low as 18 Gy to the brain and spinal canal in selected patients (Goldwein et al., 1996), with others relying primarily on chemotherapy and using radiation as a last resort (Ater et al., 1997). Despite these efforts, the majority

of children who have been treated for medulloblastoma still show a wide range of moderate to severe cognitive, academic, and psychosocial deficits.

Risk Factors

Several variables that represent risk factors for worse neurocognitive and psychosocial outcome from pediatric medulloblastoma have been identified. Younger age at diagnosis is the most widely documented risk factor (Dennis, Spiegler, Hetherington, & Greenberg, 1996; George et al., 2003; Hoppe-Hirsch et al., 1990; Kao, Goldwein, Schultz, Radcliffe, Sutton, & Lange, 1994; Kimmings, Kleinlugtebeld, Casey, & Hayward, 1995; Palmer, 2008; Radcliffe, Bunin, Sutton, Goldwein, & Phillips, 1994; Ris & Abbey, 2010; Silverman, Palkes, Talent, Kovnar, Clouse, & Thomas, 1984; Todd & Ruge, 1999). Specifically, diagnosis before age 3 is associated with a significantly lower survival rate as compared to both older children and adults (Kombogiorgas et al., 2007; Curran, Le, Sainani, Propp, & Fisher, 2009a). Higher morbidity rates in children diagnosed prior to age 3 may be due to the fact that radiation interferes with neuronal development, consequently impacting less-developed brains more severely (Monje, Mizumatsu, Fike, & Palmer, 2002). Another potential explanation, the vulnerability theory, states that the effects of the tumor and surgical resection may impact younger children more severely because they have not learned as many basic skills and thus face more of a challenge in developing further cognitive abilities (Mitby et al., 2003). However, Levisohn and colleagues (2000) saw fewer cognitive and affective deficits in younger children among their sample. They offer that this may be due to either neural

plasticity, lack of sensitivity in testing, or the fact that the functional domains in which older children show deficits are not significantly developed until at least age 7.

It is also significant to note that males are diagnosed with medulloblastoma at almost twice the rate as females (Gottardo & Gajjar, 2006; Horner et al., 2009). Females diagnosed with medulloblastoma after age three appear to have higher survival rates (Akyuz et al., 2008; Curran, Sainani, Le, Propp, & Fisher, 2009b). Some studies have suggested that female medulloblastoma survivors are at higher risk for worse neurocognitive outcomes (Butler & Haser, 2006; Nagel et al., 2004; Palmer, Reddick, & Gajjar, 2007; Ris et al., 2001), whereas other studies have failed to replicate this finding (Hardy et al., 2008; Mabbott et al., 2005, 2008; Maddrey et al., 2005).

Low socioeconomic status and high stress levels are two other factors that may increase risk of poor neurocognitive and psychosocial outcome (Palmer, 2008). Time since treatment has a mixed impact on various deficits; for example, IQ, memory, and emotional functioning appear to worsen over time while motor deficits do not show progression (Dennis, Spiegler, Hetherington, & Greenberg, 1996; Hoppe-Hirsch et al., 1990; Packer et al., 1989). Overall, older age at diagnosis and lack of postoperative complications are associated with better neuropsychological functioning, as is shorter time since diagnosis (Maddrey et al., 2005).

Regarding tumor-related variables, brain stem invasion is no longer considered a factor that increases mortality risk, and the prognostic value of residual tumor size has been called into question (Kombogiorgas et al., 2007; Packer et al., 1999). Additionally, medulloblastoma tumor size and local infiltration, described by stages T1 through T4, consistently have been found to have no relation to outcome (Ris & Abbey, 2010).

Nonetheless, children with residual tumors that have metastasized or are more than 1.5 cm^2 in size are considered to be at high risk of mortality (Kombogiorgas et al., 2007). Hydrocephalus has been linked to intellectual declines in medulloblastoma survivors (Hardy, Bonner, Willard, Watral, & Gururangan, 2008; Kao et al., 1994). In a likely related finding, hydrocephalus has been linked to deficits in writing, mathematics, and visuomotor abilities as well (Hardy et al., 2008). Metastasis stages (M0-M4), which measure degree of cerebrospinal fluid dissemination, consistently have been found to be predictive of overall outcome (Ris & Abbey, 2010).

LATE EFFECTS

Mechanisms

White Matter Damage

The primary neuroanatomical consequence of radiation generally thought to be associated with the cognitive deficits seen in posterior fossa patients is damage to white matter (American Cancer Society, 2005; Mabbott, Noseworthy, Bouffet, Rockel, & Laughlin, 2006; Mulhern et al., 1999, 2001, 2004; Palmer et al., 2002; Reddick et al., 2000, 2003). Radiation-induced white matter damage can have widespread effects throughout the brain, especially due to the extensive reciprocal connections between the cerebellum and the frontal lobe (Leiner, Leiner, & Dow, 1986). Imaging studies confirm that cranial radiation reduces the amount of normal-appearing white matter throughout

the brain (Khong et al., 2006; Mabbott et al., 2006; Mulhern et al., 1999; Palmer, 2008; Reddick et al., 2000). The mechanism by which radiation causes late effects in survivors of posterior fossa tumors is not completely understood due to its complicated nature (Wong & Van der Kogel, 2004). Its direct damage to white matter throughout the brain likely occurs by a process of induced apoptosis in which inflammatory and hypoxic-ischemic responses to radiation may mediate cell death (Ris & Abbey, 2010). Both radiation and chemotherapy can destroy oligodendrocytes, astrocytes, microvasculature, and/or mature myelin (Filley & Kleinschmidt-DeMasters, 2001).

Thus, children who receive these treatments display morphologically detected atrophy of white matter and/or fail to develop white matter at an appropriate rate, unlike patients treated with surgery alone (Mulhern et al., 1999; Reddick et al., 2000). The latter effect in particular has been associated with IQ loss—or rather, failure to maintain intellectual trajectories (Palmer et al., 2001). Indeed, children with posterior fossa tumors do not appear to lose previously acquired skills and knowledge; rather, their rate of learning declines. This may in part explain why children diagnosed with medulloblastoma at a younger age generally show greater declines in overall IQ, as they have not had the opportunity to acquire as many skills prior to treatment (Palmer et al., 2001; Radcliffe, Bunin, Sutton, Goldwein, & Phillips, 1994; Ris, Packer, Goldwein, Jones-Wallace, & Boyett, 2001; Silverman, Palkes, Talent, Kovnar, Clouse, & Thomas, 1984). For example, a study by Palmer et al. (2001) found that children treated with radiation for medulloblastoma learn at only 30% of the rate needed to maintain their pre-treatment trajectories.

Relevant to the current study, it is noteworthy that IQ deficits among children treated for leukemia with radiation are largely accounted for by deficits in information processing speed and working memory, unlike children treated without radiation (Schatz, Kramer, Ablin, & Matthay, 2000). Indeed, poor intellectual outcome appears to be strongly related to radiation-induced microscopic damage to normal-appearing white matter, as indicated by a diffusion tensor imaging study conducted by Mabbott and colleagues (2006). Additionally, survivors of pediatric medulloblastoma have significant reductions in cerebral white matter (Khong et al., 2006; Mabbott et al., 2006; Reddick, Mulhern, Elkin, Glass, Merchant, & Langston, 1998; Reddick et al., 2000).

Chemotherapy can also have damaging effects on white matter, as observed by Rutkowski and colleagues (2005) in a population of medulloblastoma patients receiving a chemotherapy-only treatment regimen. In this study, chemotherapy was associated with leukoencephalopathy, or the destruction of the myelin sheaths surrounding nerve fibers, which promote the transmission of nerve signals. However, these effects resolved spontaneously in about 25% of the patients after a year or more. Similar findings of transient leukoencephalopathy have been reported for acute lymphoblastic leukemia patients treated with chemotherapy (Reddick et al., 2005). While changes in cognitive functioning have been observed after chemotherapy, the mechanisms by which chemotherapy can potentially cause late effects are yet unknown (Ahles & Saykin, 2007). Mechanisms that have been proposed include DNA damage by oxidative stress, shortening of telomeres, deregulation of cytokine activity, polymorphisms in proteins and neurotransmitters, reduced levels of estrogen and testosterone, and alterations in the neuroendocrine system (Ahles & Saykin, 2007).

Vascular Damage

In addition to its directly damaging effects on white matter, radiation is hypothesized to damage the cerebral vasculature and disrupt the blood-brain barrier (Ris & Abbey, 2010). This vascular damage may contribute to functional late effects in addition to placing long-term survivors at significantly increased risk for stroke (Bowers et al., 2006). Chemotherapy, as well, is often cardiotoxic and could negatively impact cerebrovascular function (Ahles & Saykin, 2007).

Surgical Lesions

Negative effects of the surgical procedure itself have been minimized in recent years with advances in technology allowing minimal incisions, highly focused resection, and entry routes designed to avoid unnecessary structural damage—such as the infratentorial supracerebellar approach and the cerebellomedullary fissure approach (Hermann, Rittierodt, & Krauss, 2008; Kawashima, Matsushima, Nakahara, Takase, Masuoka, & Ohata, 2009; Kellogg & Piatt, 1997). Risk of poor outcome is increased with multiple surgeries, although this may reflect the more severe nature of a tumor requiring multiple surgeries, such as metastases (Kao et al., 1994). Supporting this theory, multiple surgeries also appear to be associated with increased risk of requiring shunt insertion (Merchant et al., 2004), an indicator of hydrocephalus severity, as discussed in the following section.

Hydrocephalus and Associated Complications

A posterior fossa tumor can often cause hydrocephalus, the abnormal accumulation of cerebrospinal fluid (CSF), typically due to structural impairment of the flow of CSF. Research has shown that hydrocephalus, independent of any associated medical condition or treatment, can cause long-term cognitive deficits, especially if untreated (Hardy et al., 2008; Mirazayan, Luetjens, Borremans, Regel, & Krauss, 2010). With regard to structural effects, hydrocephalus often causes ventricular enlargement and increased intracranial pressure (ICP), which may be contributing mechanisms to neurocognitive deficits. Increased ICP can also be caused by mass effect from a tumor compressing and deforming the adjacent areas of the brain, which is another potential mechanism of cognitive impairment in children with posterior fossa tumors.

Severity of hydrocephalus may be more relevant to the neurocognitive deficits of posterior fossa tumor patients than duration (Aarsen et al., 2004). Shunt insertion to alleviate hydrocephalus can worsen cognitive outcomes in pediatric medulloblastoma survivors, possibly by contributing to structural neural damage or due to infections (Hardy et al., 2008; Merchant et al., 2004). However, insertion of a shunt, which is not required in all cases, may simply be an indicator of more severe hydrocephalus (Merchant et al., 2004). Additionally, posterior fossa tumor patients with permanent shunts may be more susceptible to the neurotoxic effects of radiation (Merchant et al., 2004). Overall, the example of hydrocephalus and its associated complications illustrates the difficulty in attempting to isolate the various mechanisms underlying the late effects of posterior fossa tumors and their treatments.

Endocrinopathies

Between 50% and 80% of children who receive craniospinal irradiation for brain tumors exhibit primary endocrine deficiencies, most commonly growth hormone deficiencies and primary hypothyroidism (Bahl et al., 2009, Duffner, 2004; Muirhead, Hsu, Grimard, & Keene, 2002). The role of craniospinal radiation in this process was highlighted by a recent study that found 71% of posterior fossa tumor survivors treated with craniospinal radiation developed endocrinopathies within 5 years of treatment, compared to 18% of those treated only with focal radiation to the tumor bed (Bahl et al., 2009). More than 80% of a sample of posterior fossa tumor patients receiving craniospinal irradiation developed permanent growth hormone deficiencies within one year of treatment (Duffner et al., 1985). Growth hormone deficiencies have been hypothesized to be a consequence of radiation-induced white matter damage to the hypothalamus, although chemotherapy has also been shown to play a role in such endocrinopathies (Duffner, 2004). However, a limitation of the Duffner et al. (1985) study is that radiation doses administered to their sample were significantly greater than the current standard of care dosage. A more recent study found that only about 17% of a sample of medulloblastoma patients developed growth hormone deficiencies (Dolezal-Oltarzewska, Wojcik, Korab-Chrzanowska, Kwiatkowski, & Starzyk, 2009). Finally, Merchant and colleagues (2002b) have suggested that the incidence of radiation-induced endocrinopathy may be overestimated, citing findings that pediatric brain tumor patients commonly display endocrinopathy prior to receiving radiation. They note that pre-

radiation endocrinopathy "may be an early indicator of central nervous system damage that will influence the functional outcome related to radiotherapy."

Surgery

Surgical resection of a posterior fossa tumor occurs in the majority of cases. Although this is typically a necessary component of treatment in order to reduce the risk of mortality, surgical resection is associated with a wide variety of cognitive deficits in children. Language deficits—ranging from word-finding difficulties to "cocktail party speech"—are often observed (Levisohn, Cronin-Golomb, & Schmahmann, 2000; Aarsen et al., 2004; Riva & Giorgi, 2000). An extreme example of this is the phenomenon of cerebellar mutism, speech loss that can last up to several months, associated with 10% of midline cerebellar tumor resections and often accompanied by a spectrum of neurobehavioral deficits such as emotional lability (Pollack, 2001). Memory deficits are also a common side effect of surgical resection of posterior fossa tumors in a pediatric population (Aarsen et al., 2004; Ater et al., 1996; Levisohn, Cronin-Golomb, & Schmahmann, 2000; Riva & Giorgi, 2000; Steinlin et al., 2003). Surgical resection is often associated with visuospatial and visuomotor deficits as well (Aarsen et al., 2004; Levisohn, Cronin-Golomb, & Schmahmann, 2000; Steinlin et al., 2003). In regard to intellectual abilities, some studies have found deficits in IQ and academic achievement associated with surgical resection (Beebe et al., 2005; Steinlin et al., 2003). However, a study by Karatekin, Lazareff, & Asarnow (2000) did not report evidence of intellectual deficits associated with surgery in a general posterior fossa population. Attentional

deficits are commonly associated with surgical resection of posterior fossa tumors in children (Aarsen et al., 2004; Riva & Giorgi, 2000; Steinlin et al., 2003). It is uncertain whether tumor location plays a role in the cognitive deficits of posterior fossa patients treated with surgery only, as some studies support this hypothesis (Levisohn et al., 2000; Riva & Giorgi, 2000; Vaquero et al., 2008), while others have found no location-specific findings (Beebe et al., 2005; Lazaroff & Castro-Sierra, 1996).

Surgical resection of posterior fossa tumors has also been shown to have psychological, behavioral, and emotional consequences in children. A large body of research indicates that behavioral changes are merely transient in a posterior fossa tumor population (Pollack, Polinko, Albright, Towbin, & Fitz, 1995; Siffert et al., 2000; Riva, 2000a; Riva, 2000b; Van Dongen, Catsman-Berrevoets, & Van Mourik, 1994). However, Meyer & Kieran (2002) found that 56% of children treated with surgical resection of brain tumors experienced both short-term and long-term disturbances in mood, behavior, and academic adjustment. Notably, not all tumors in this study involved the cerebellum, and no distinctions were made between cerebellar and non-cerebellar tumor groups. Overall, surgical resection of posterior fossa tumors in a pediatric population is associated with suboptimal psychological well-being and reduced quality-of-life (Pompili et al., 2002). Areas that seem to be particularly affected are socializing behaviors and school experience. However, a significant limitation of this study is that it investigated subjects whose tumors were resected from 1970 to 1985, when surgical treatments were more invasive.

Risk factors for poor outcome related to surgery include subtotal resection and damage to cerebellar structures. Larger residual tumor size appears to be correlated with

worse cognitive outcomes in medulloblastoma survivors (Dennis et al., 1996; Gottardo & Gajjar, 2006). Damage to cerebellar structures such as the vermis also may predict poorer outcomes, particularly a transient loss of speech termed "cerebellar mutism," although surgical techniques have been developed to avoid this in most cases (Hermann, Rittierodt, & Krauss, 2008; Kawashima, Matsushima, Nakahara, Takase, Masuoka, & Ohata, 2009; Kellogg & Piatt, 1997; Steinlin et al., 2003; Van Dongen, Catsman-Berrevoets, & Van Mourik, 1994).

Radiation and Chemotherapy

Radiation is associated with increased survival rates in children with posterior fossa tumors (Curran, Le, Sainani, Propp, & Fisher, 2009a). However, children with posterior fossa tumors treated not only with surgical resection but also with radiation and/or chemotherapy often show more severe cognitive deficits than do those treated solely with surgical resection. Thus, it is believed that radiation and/or chemotherapy often produce neurocognitive deficits above and beyond the effects of the tumor and surgical resection (Briere et al., 2008; Garcia-Perez, Sierrasesumaga, Narbona-Garcia, Calvo-Manuel, & Aguirre-Ventallo, 1994; Lafay-Cousin, Bouffet, Hawkins, Amid, Huang, & Mabbott, 2009; Mabbott et al., 2008; Maddrey et al., 2005; Palmer, 2008). One study has suggested that the distinguishing feature between the two groups is that the group treated additionally with radiation and/or chemotherapy displays more severe deficits in processing speed (Mabbott, Penkman, Witol, Strother, & Bouffet, 2008). Since processing speed is a foundation for many other cognitive functions and academic

performance, this may explain the overall severity of broad deficits in childhood medulloblastoma survivors.

Progression of deficits with increased time since cranial radiation has been documented in areas such as intellectual ability, academics, attention, and processing speed (Mabbott et al., 2005; Merchant et al., 2002; Palmer et al., 2001, 2003; Spiegler et al., 2004; Reeves et al., 2006). Regarding academics, survivors of posterior fossa tumors treated with cranial radiation have been noted to demonstrate performance declines in areas of spelling, mathematics, and reading; this finding is supported by declining parent and teacher ratings of academic ability (Mabbott et al., 2005; Mulhern et al., 2005). At least in part contrary to a study that reported significant declines in attention with increased time since radiation in posterior fossa tumor survivors (Mabott et al., 2005), other research has shown that increased time since radiation is not associated with worse performance on sustained attention in medulloblastoma survivors (Mulhern et al., 2001). However, this same study indicated that measures of intellect and verbal memory did show a time-related decline. The latter finding is supported by a number of other studies, which generally report a non-linear decline in IQ associated with cranial radiation (Fuss, Poljanc, & Hug, 2000; Hoppe-Hirsch et al., 1995; Kieffer-Renaux et al., 2005; Lafay-Cousin et al., 2009; Palmer et al., 2003; Spiegler et al., 2004). With regard to the non-linear pattern of decline, Spiegler et al. (2004) explain that losses of 2 to 4 points in IQ scores appear to occur annually in children with radiated posterior fossa, although the losses are generally steeper in the first few years after radiation.

Many studies have found that childhood medulloblastoma survivors have poor intellectual outcomes, generally with performance IQ being more impaired than verbal IQ

(Reimers et al., 2003). However, any apparent trends in the intellectual functioning of medulloblastoma survivors must be qualified by the fact that the progression of intellectual deficits appears to be affected by variables such as age at radiation and dose of radiation, the latter of which varies between patients and across time, as radiation and chemotherapy standard of care has changed greatly with advances in technology.

In terms of the dose-effect relationship, Kieffer-Renaux et al. (2005) found that patients treated with focal posterior fossa (PF) radiation alone did not demonstrate declines in IQ, whereas patients receiving full craniospinal radiation of varying dosages all displayed progressive intellectual deficits over a four-year period. On a positive note, the degree of decline lessened with each year post-radiation. Contrary to the Kieffer-Renaux study, Grill et al. (1999) indicated that at one year post-radiation, most pediatric posterior fossa patients displayed long-term cognitive impairment, even after PF radiation alone, with a significant correlation between the full-scale IQ score (FSIQ) and the dose of cranio-spinal irradiation. In another illustration of the differences between PF-only radiation and full cranial radiation, at 10-year follow-up only 10% of full-cranial radiated medulloblastoma patients had an FSIQ above 90, whereas 60% of the PF-only radiated ependymoma patients met this criterion (Hoppe-Hirsch et al., 1995). It is generally accepted that, at equal dose levels, partial brain radiation (such as PF-only) is less damaging than whole brain radiation. However, Mabbott et al. (2005) found that academic and behavioral outcomes were not related to radiation dose, extent of surgery, or treatment with chemotherapy.

Age at cranial radiation has a well-established role in determining cognitive outcomes—overall, the younger the child, the more pronounced the decline in intellectual

ability as a result of radiation. There appears to be a steeper intellectual decline both for patients with higher baseline IQ and for younger patients (George et al., 2003; Hoppe-Hirsch et al., 1990; Palmer et al., 2003; Radcliffe et al., 1994; Reimers et al., 2003; Ris et al., 2001; Roncadin, Dennis, Greenberg, & Spiegler, 2008). For children younger than 8 years of age at diagnosis, intellectual deficits may be immediate and plateau at 6 years post-diagnosis, whereas older children's deficits appear only after a delay (Palmer et al., 2003). One study even indicated that whole brain radiation doses of 18 and 24 Gy appear to have no major impact on the intellectual outcome of children as long as they are older than age 6, whereas doses greater than 24 Gy pose a substantial risk for FSIQ decline, even in older children (Fuss, Poljanc, & Hug, 2000).

Thus, in recent years, there has been a movement toward balancing the efficacy of the dose of radiation with fewer negative side effects, with current standard of care moving from a craniospinal dose of 54 Gy toward a reduced dose of approximately 23.4 Gy (Mulhern et al., 2001; Oyharcabal-Bourden et al., 2005; Ris & Abbey, 2010). Reddick and colleagues (2000) found that child medulloblastoma patients who received this reduced-dose craniospinal radiation (23.4 Gy) lost normal-appearing white matter at a 23% slower rate when compared to those who received a conventional dose of radiation (36 Gy). The functional consequences of this reduction in dosage were illustrated by a study wherein Mulhern and colleagues (1998) randomized the administration of these two radiation doses to a group of pediatric medulloblastoma patients and found that cognitive and academic functioning was less impaired in the reduced-dose group. For example, the reduced-dose group had IQ scores averaging 10 to 15 points higher, although both groups still displayed declining IQ. However, the same group later found

that younger age was more predictive of significant IQ deficits than radiation dose (Mulhern et al., 2005).

It should be noted that chemotherapy alone has been found to be much less neurotoxic than radiation, and thus it is not often isolated in discussions of late effects (Ris & Abbey, 2010). A longitudinal study of medulloblastoma patients showed that chemotherapy did not predict late effects on IQ (Palmer et al., 2003). However, this is not to say that chemotherapy does not contribute to the late effects observed in children who receive this adjuvant treatment. As previously noted, chemotherapy often has specific effects such as leukoencephalopathy, which can potentially contribute to poorer functional outcomes (Reddick et al., 2005; Rutkowski et al., 2005). Indeed, medulloblastoma survivors treated with a combination of chemotherapy and radiation report greater need for educational support than those treated with radiation alone (Bull, Spoudeas, Yadegarfar, & Kennedy, 2007). A review by Anderson & Kunin-Batson (2009) concluded that children treated with chemotherapy alone display poor outcomes related to attention, executive functioning, visual processing, and visuomotor functioning. These effects are more pronounced in younger children and females.

Academic, Psychosocial, and Neurocognitive Outcomes

Academic Outcomes

Although survivors of childhood medulloblastoma generally display limited awareness of their cognitive deficits, self-reports regarding their academic performance

have been shown to be quite accurate (Maddrey et al., 2005, O'Donnell, De Soto, & De Soto, 1993). This may be due to the receipt of poor grades, feedback from teachers and parents, or objective awareness of being enrolled in special education programs. Likely related to their declines in IQ and other cognitive functions, survivors of childhood medulloblastoma exhibit significant declines in academic performance (Dennis, Spiegler, Hetherington, & Greenberg, 1996; Palmer et al., 2001; Palmer, Reddick, & Gajjar, 2007; Ris, Packer, Goldwein, Jones-Wallace, & Boyett, 2001). Children who receive cranial radiation, such as medulloblastoma patients, are 7 times more likely to require special education services compared to survivors of childhood cancers who do not receive cranial radiation (Mitby et al., 2003; Palmer, 2008). At ten-year follow-up, 80% of medulloblastoma survivors may require special education services (Hoppe-Hirsch et al., 1995). Furthermore, survivors of childhood medulloblastoma are significantly less likely than their healthy peers to finish high school (Mitby et al., 2003). Despite these statistics, impairments in the academic functioning of this population have not been adequately studied.

Limited research in the realm of academic achievement testing with pediatric medulloblastoma survivors shows that these children display deficits in reading, spelling, and mathematics (Mabbott et al., 2005; Muhern et al., 2005; Reeves et al., 2006). Furthermore, even when accounting for declines in IQ, survivors of childhood medulloblastoma exhibit significant continuing declines in spelling and arithmetic at a mean of 5 years post-diagnosis, with the rate of decline from year to year significantly increasing with time (Mabbott et al., 2005). Declining parent and teacher ratings of academic ability corroborate the real-world applicability of these findings (Mabbott et al.,

2005). By controlling for IQ, the researchers were able to assert that academic declines are at least in part due to additional factors, potentially related to the general neurocognitive late effects of treatment. Although there does not appear to be a significant difference between the academic performance of average-risk and high-risk survivors of childhood medulloblastoma (corresponding to differential doses of radiation), patients younger than 7 years at diagnosis experience significantly worse declines in reading (Mabbott et al., 2005; Mulhern et al., 2005). This is hypothesized to be a result of impairments in more fundamental language skills such as orthographic and phonologic analysis. Overall, however, research thus far suggests that children diagnosed with medulloblastoma at any age typically exhibit deficits in most if not all areas of academic achievement testing.

Psychosocial Outcomes

In addition to the cognitive, intellectual, and academic difficulties faced by survivors of childhood medulloblastoma, there are myriad negative impacts on psychosocial functioning. Due to impairments in motor functioning, these survivors often are unable to drive or participate in many recreational and vocational activities (Maddrey et al., 2005). One study showed that 36% of childhood medulloblastoma survivors were unemployed 10 years post-surgery, and none of that sample had "normal employment" (Hoppe-Hirsch, Renier, Lellouch-Tubiana, Sainte-Rose, Pierre-Kahn, & Hirsch, 1990). More broad cognitive impairments may be responsible for their difficulties with independent living, dating, and emotional functioning (Maddrey et al.,

2005). Five years post-surgery, 47% of childhood medulloblastoma survivors display some sort of emotional or behavioral disorder, and this percentage increases with time—10 years post-surgery, 78% of the population had some sort of psychopathology (Hoppe-Hirsch et al., 1990). Other studies have replicated the finding that behavioral and social problems worsen with time in this population (Aarsen et al., 2006; Boman, Hoven, Anclair, Lannering, & Gustafsson, 2009). Research suggests that radiation may be responsible for declines in social skills (Mabbott et al., 2005), although the full etiology of these various psychosocial difficulties is still uncertain.

Inability to live independently has been noted in a population of childhood medulloblastoma survivors, although this finding was not statistically significant when compared to the general population (Maddrey et al., 2005). However, neither participants nor caregivers reported decreased quality of life at ten years post-diagnosis (Maddrey et al., 2005). This may reflect habituation to the limitations faced by children with chronic medical disorders (Leonard, Bower, Petterson, & Leonard, 1999), or it may be associated with the finding that, over time, survivors' satisfaction with life increases as they accept those limitations (Nordeson, Engstrom, & Norberg, 1998). Of course, lack of awareness of deficits, as has been noted in this population (Maddrey et al., 2005), may be responsible for the high quality of life reported.

Executive Functions

Executive functioning refers to a broad integration of cognitive abilities such as initiation, inhibition, self-monitoring, planning, organization, problem-solving, and

cognitive shifting/flexibility. The exact role of the cerebellum in executive functioning is unclear, as some studies have found executive function deficits in children and adults with cerebellar pathology (Exner, Weniger, & Irle, 2004; Gottwald, Wilde, Mihajlovic, & Mehdorn, 2004; Karatekin, Lazareff, & Asarnow, 2000; Malm, Kristensen, Karlsson, Carlberg, Fagerlund, & Olsson, 1998), while others have found that executive function is intact in such patients (Bracke-Tolkmitt et al., 1989; Daum, Ackermann, Schugens, Reimold, Dichgans, & Birbaumer, 1993; Fiez, Peterson, Cheney, & Raichle, 1992). However, a growing body of evidence indicates that survivors of pediatric medulloblastoma appear to have difficulties with planning, organization, problem-solving, and cognitive flexibility, making these children vulnerable to difficulties in effectively completing complex tasks or to inappropriate social behavior (Aarsen, Van Dongen, Paquier, Van Mourik, & Catsman-Berrevoets, 2004; Levisohn, Cronin-Golomb, & Schmahmann, 2000; Maddrey et al., 2005; Riva & Giorgi, 2000; Spiegler et al., 2004; Vaquero, Gomez, Quintero, Gonzalez-Rosa, & Marquez, 2008). One study has shown that childhood medulloblastoma survivors have significantly more executive dysfunction if the medulloblastoma affected the vermis (Vaquero et al., 2008). Furthermore, Schmahmann and Sherman (1998) have described a "cerebellar cognitive affective syndrome" sometimes seen in patients with lesions to the cerebellum. This syndrome includes impairments in executive functions such as planning, cognitive shifting/flexibility, abstract reasoning, and working memory.

Language

Deficits in language production, particularly with regard to prosody, grammar, and word-finding abilities (mild anomia), have been documented in the medulloblastoma survivor population (Dennis et al., 1996; Levisohn, Cronin-Golomb, & Schmahmann, 2000; Maddrey et al., 2005; Riva & Giorgi, 2000). This is not one of the more common findings among survivors of childhood medulloblastoma; in fact, language appears to be the least impaired neurocognitive domain (Maddrey et al., 2005). However, it is estimated that up to 58% of pediatric medulloblastoma survivors display some sort of language deficit (Maddrey et al., 2005).

Relative strengths in language skills among childhood medulloblastoma survivors are reflected in IQ scores, with verbal IQ scores typically higher than performance IQ (Dennis et al., 1996; Hardy, Bonner, Willard, Watral, & Gururangan, 2008; Kieffer-Renaux, Bulteau, Grill, Kalifa, Viguier, & Jambaque, 2000; Maddrey et al., 2005; Mulhern et al., 1998; Riva, Milani, Pantaleoni, Ballerini, & Giorgi, 1991; Silverman et al., 1984). This relative strength in language skills can cause others to overestimate these individuals' general abilities, which is particularly problematic in educational and occupational settings (McCabe, Getson, Brasseux, & Johnson, 1995).

Visuospatial Skills

Visuospatial skills can be divided into two categories: visual perception, referring to accurate visual intake of the world, and visual construction, which involves the

reproduction of visual elements. Difficulties in either of these areas can cause real-world problems, such as becoming lost easily.

Brain tumor patients who receive radiation, including medulloblastoma patients, display worse visuospatial skills than surgery-only brain tumor populations (Copeland et al., 1999; Spiegler et al., 2004). Indeed, impairments in visuospatial skills have been widely noted in survivors of childhood medulloblastoma (Levisohn, Cronin-Golomb, & Schmahmann, 2000; Spiegler et al., 2004; Steinlin et al., 2003; Maddrey et al., 2005). One study has suggested that these visuospatial difficulties may be a consequence of executive function deficits, particularly related to planning and organization (Levisohn, Cronin-Golomb, & Schmahmann, 2000). White matter damage is a likely explanation for visuospatial deficits in medulloblastoma patients, whether by directly affecting visual processes or by contributing to related executive dysfunction. Potentially reflecting the more extensive white matter damage in patients with hydrocephalus, medulloblastoma patients with a history of receiving a shunt display even more severe visuospatial deficits than those who do not require a shunt (Hardy, Bonner, Willard, Watral, & Gururangan, 2008). Notably, visuospatial impairments in medulloblastoma survivors have been observed independent of motor skills, as subjects with varying motor skills did not display significant differences in visuospatial performance (Maddrey et al., 2005).

Motor Skills

Survivors of childhood medulloblastoma display motor skills deficits as well (Grill et al., 2004; Jain, Krull, Brouwers, Chintagumpala, & Woo, 2008; Maddrey et al.,

2005). As measured by a finger-tapping task, up to 77% display motor impairments (Maddrey et al., 2005). Motor deficits can cause difficulties in performing a number of recreational and vocational tasks, from driving a car to typing. However, processing speed deficits can cause impairments in motor-based reaction time and thereby can confound measurements of motor functioning. Since processing speed deficits have been extensively documented in childhood medulloblastoma survivors, it is important to consider the potential impact of these deficits on motor performance in this population (Dennis, Hetherington, & Spiegler, 1998; Kieffer-Renaux et al., 2000; Mabbott et al., 2008; Mulhern et al., 2004; Nagel et al., 2006; Riva & Giorgi, 2000; Reeves et al., 2006; Schatz, Kramer, Ablin, & Matthay, 2000; Spiegler et al., 2004; Vaquero, Gomez, Quintero, Gonzalez-Rosa, & Marquez, 2008).

More broadly, in a general population of posterior fossa patients, it has been found that surgical resection of the tumor may be the primary cause of observed motor deficits (Aarsen et al., 2004; Levisohn, Cronin-Golomb, & Schmahmann, 2000; Steinlin et al., 2003). This conclusion is supported by the finding that motor deficits in this population remain constant over time, neither worsening nor improving (Dennis, Spiegler, Hetherington, & Greenberg, 1996; Hoppe-Hirsch et al., 1990; Packer et al., 1989). Peripheral neuropathy, often linked to chemotherapy agents such as vincristine and cisplatin, is another potential cause of late motor deficits in pediatric medulloblastoma survivors, although this is rare (Hockenberry, Krull, Moore, Pasvogel, Gregurich, & Kaemingk, 2006; Packer et al., 1994; Quastoff & Hartung, 2002). The use of amifostine in chemotherapy regimens has been shown to reduce the incidence of peripheral neuropathy in childhood medulloblastoma survivors (Fisher et al., 2004).

Memory

Pediatric medulloblastoma survivors display a wide variety of memory deficits in areas such as verbal, visual, short-term, long-term, and working memory (George et al., 2003; Hardy, Bonner, Willard, Watral, & Gururangan, 2008; Maddrey et al., 2005; Nagel, Delis, Palmer, Reeves, Gajjar, & Mulhern, 2006; Palmer, 2008; Palmer, Reddick, & Gajjar, 2007; Roman & Sperduto, 1995; Steinlin et al., 2003; Timmann & Daum, 2007). A wide variety of verbal memory deficits in survivors of childhood medulloblastoma have been documented, with several studies suggesting that verbal memory impairment may be due to poor encoding of information rather than difficulties in storage or retrieval (Levisohn, Cronin-Golomb, & Schmahmann, 2000; Maddrey et al., 2005). However, Nagel and colleagues (2006) report both retrieval and recognition deficits in this population, while other studies indicate no evidence of verbal memory decline at all—merely visual memory deficits (Johnson et al., 1994; Spiegler et al., 2004). Hardy and colleagues (2008), on the other hand, found both verbal and visual memory to be impaired in childhood medulloblastoma survivors. Within the realm of short-term memory, research has suggested that verbal short-term memory is more frequently impaired in this population than is visual short-term memory (Riva & Giorgi, 2000; Steinlin et al., 2003).

Overall, the findings that suggest verbal memory impairments in this population are more clear and consistent than findings regarding visual memory impairments, with most studies indicating some form of verbal memory deficit (George et al., 2003; Hardy et al., 2008; Levisohn, Cronin-Golomb, & Schmahmann, 2000; Maddrey et al., 2005;

Nagel et al., 2006; Palmer, 2008; Palmer, Reddick, & Gajjar, 2007; Riva & Giorgi, 2000; Steinlin et al., 2003; Timmann & Daum, 2007). The inconsistency of findings on visual memory in survivors of pediatric medulloblastoma may be due to the methodology of visual memory testing. For example, Steinlin and colleagues (2003) note that the Rey-Osterrieth Complex Figure, which is often used to test visual-spatial memory, can be impacted by difficulties with planning and organization, which their study found to be the actual explanation for apparent visual-spatial memory deficits in childhood medulloblastoma survivors.

Structural neural damage due to both surgical resection and radiation likely impacts these varied functional deficits in memory among survivors of childhood medulloblastoma and to a certain extent may explain some of the commonalities in memory outcome research in this population. For example, the past two decades of research have particularly implicated the cerebellum as having a role in verbal working memory, a function that is often impaired among childhood medulloblastoma survivors (Timmann & Daum, 2007). Imaging research also has shown that survivors of childhood medulloblastoma, especially females, have hippocampal atrophy (Nagel et al., 2004). In particular, volumes of both the left and right hippocampus—particularly in the posterior regions—appear to steadily decrease until about two or three years post-diagnosis, at which point normal growth patterns seem to resume (Nagel et al., 2004). This period of decreasing hippocampal volume in childhood medulloblastoma survivors may be due to the long-lasting toxic effects of chemotherapy and radiation (Palmer, Reddick, & Gajjar, 2007). Indeed, cranial radiation dose has been found to be negatively correlated with both verbal and nonverbal memory functions (Roman & Sperduto, 1995). However, the

impact of radiation on memory as a broad domain remains unclear, as there have been inconsistent findings regarding the impact of radiation on various types of memory, such as visual or verbal memory (Konczak et al., 2005; Spiegler, Bouffet, Greenberg, Rutka, & Mabbott, 2004, Timmann & Daum, 2007). Some researchers have suggested that radiation-induced deficits in processing speed, which impact learning and retrieval, may be at the core of memory impairments in childhood medulloblastoma survivors (Mabbott et al., 2008; Palmer, 2008).

Attention

Deficits in attention are common among childhood medulloblastoma survivors, with research indicating that up to 92% of these patients have difficulties with basic attention (Maddrey et al., 2005). Teachers report increasing attentional difficulties in this population over time (Mabbott et al., 2005). Supporting these teacher reports, increased time since radiation is generally associated with worsening attentional problems in medulloblastoma survivors (Briere, Scott, McNall-Knapp, & Adams, 2008; Palmer et al., 2001, 2003; Reeves et al., 2006; Spiegler et al., 2004), although this finding has not consistently been replicated (Mulhern et al., 2001). With regard to specific types of attentional deficits, medulloblastoma survivors often exhibit deficits in both selective and sustained attention (Dennis, Hetherington, & Spiegler, 1998; Mulhern et al., 2004; Reddick et al., 2003; Roman & Sperduto, 1995; Steinlin et al., 2003). Naturally, broad attentional deficits present challenges for everyday life skills such as driving. However, in this population, impaired attention is particularly problematic due to its negative

impact on the development of cognitive abilities, which consequently affects academic achievement (Briere, Scott, McNall-Knapp, & Adams, 2008; Dennis et al., 1998; Maddrey et al., 2005; Palmer, 2008; Palmer, Reddick, & Gajjar, 2007; Roman & Sperduto, 1995).

Dennis and colleagues (1998) note that "the main finding is that it is not the presence of a tumor as such that appears to place the child at risk for poor attention but, rather, the combination of a tumor and adjuvant radiation treatment" (p. 31). This is a possible explanation for the attentional difficulties faced by childhood medulloblastoma survivors. This finding of radiation causing attentional difficulties has been replicated in other studies (Butler, Kerr, & Marchand, 1999; Garcia-Perez, Sierrasesumaga, Narbona-Garcia, Calvo-Manuel, & Aguirre-Ventallo, 1994; Merchant, Kiehna, Miles, Zhu, Xiong, & Mulhern, 2002a; Reeves et al., 2006). Attentional deficits are particularly pronounced in patients less than 8 years of age at diagnosis and in patients who receive higher doses of craniospinal irradiation (Mulhern, Kepner, Thomas, Armstrong, Friedman, & Kun, 1998; Schmidt, Copeland, & Fletcher, 1989). Those children who have had fewer years of schooling may be the least able to benefit from further schooling due to worse attentional difficulties. The findings by Mulhern and colleagues (1998) and Schmidt et al. (1989) also suggest that radiation plays a large role in exacerbating neurocognitive deficits. Indeed, according to a diffusion tensor imaging study, the primary consequence of radiation-reduced healthy ("normal-appearing") white matter appears to be decreased attentional abilities (Mulhern, Merchant, Gajjar, Reddick, & Kun, 2004). Just as in the domain of memory, researchers have suggested that attention impairments in childhood medulloblastoma survivors may be due to processing speed deficits thought to be caused

by radiation-induced white matter damage (Mabbott et al., 2008; Palmer, 2008). Indeed, a large number of studies have found that childhood medulloblastoma survivors have more significant attention deficits than survivors of non-irradiated tumors, implying that the use of radiation in the treatment of medulloblastoma is largely responsible for such deficits (Butler, Kerr, & Marchand, 1999; Mabbott et al., 2008; Merchant, Kiehna, Miles, Zhu, Xiong, & Mulhern, 2002a; Reeves et al., 2006; Ronning, Sundet, Due-Tonnessen, Lundar, & Helseth, 2005).

Processing Speed

Several studies have noted deficits in processing speed in survivors of pediatric medulloblastoma (Dennis, Hetherington, & Spiegler, 1998; Kieffer-Renaux et al., 2000; Mabbott et al., 2008; Mulhern et al., 2004; Nagel et al., 2006; Riva & Giorgi, 2000; Reeves et al., 2006; Schatz, Kramer, Ablin, & Matthay, 2000; Spiegler et al., 2004; Vaquero, Gomez, Quintero, Gonzalez-Rosa, & Marquez, 2008). A review article by Palmer (2008) has suggested that in this population, processing speed deficits are the first to emerge post-treatment, whereas difficulties in attention emerge later. In a general posterior fossa population, processing speed was distinguished as the only significantly worse deficit in a radiated group as compared to a non-radiated group (Mabbott et al., 2008). Furthermore, medulloblastoma patients younger than 12 years of age show decreased reaction times after radiation therapy (Merchant et al., 2002a). These findings suggest that radiation may play a large role in the processing speed deficits seen in childhood medulloblastoma survivors.

The presence of processing speed deficits in childhood medulloblastoma survivors may be due to the fact that the tumor and/or the process of surgical resection causes lesions in close proximity to the ascending reticular activating system of the brainstem (Riva, Pantaleoni, Milani, & Belani, 1989). However, another hypothesis proposed by Riva and Giorgi (2000) is that slow processing speed is reflective of a disruption of the intrinsic structure of the cerebellum, "which is constituted in a crystalline manner by a micromodular structure that works in parallel" (p. 1057). Since each module is essentially a microprocessor working within a large array, the cerebellum is thus an extremely powerful network that processes information at high speeds. By recruiting modules in rapid succession, the cerebellar network can reach "exceptional speeds of execution" (p. 1057). Thus, Riva and Giorgi have posited, any lesion that affects a part of this cerebellar network reduces the speed at which modules may be recruited, in turn reducing the speed at which the cerebellum can participate in the execution of cognitive processes (Ito, 1984; Kandel, Schwartz, & Jessell, 2000). One of the most prominent deficits associated with any sort of damage to the cerebellum is decreased processing speed (Aarsen et al., 2009; Butler & Haser, 2006; Mabbott et al., 2008; Palmer, 2008; Palmer & Leigh, 2009; Palmer, Reddick, & Gajjar, 2007; Reeves et al., 2006; Riva & Giorgi, 2000; Steinlin et al., 2003; Vaquero, Gomez, Quintero, Gonzalez-Rosa, & Marques, 2008; Zuzak, Poretti, Drexel, Zehnder, Boltshauser, & Grotzer, 2008). The larger the lesion, the more processes may be slowed. Indeed, as first noted by Vernon (1983), processing speed deficits can have widespread negative effects on other cognitive functions and intellectual functioning. Riva and Giorgi (2000) clarify that motor-based deficits such as dysmetria and tremor were not responsible for the slow

processing speed of the children with medulloblastoma observed in their study, as patients with such deficits were excluded.

Processing speed quite likely is impaired by the reduced functionality of cerebral white matter pathways, as damage to these neural tracts is another known effect of the cranial radiation received by most medulloblastoma patients (Khong et al., 2006; Mabbott et al., 2006; Mulhern et al., 1999; Palmer, 2008; Reddick et al., 2000). Briere and colleagues (2008) note that the functional impact of radiation on the prefrontal cortex may not be immediately apparent, as this area of the brain is still developing into early adulthood. However, they hypothesize that radiation-induced cerebral white matter damage impairs processing speed by disrupting the brain's overall development of its structural network, which is designed to enhance speed of processing. White matter damage specific to the cerebellum, caused by radiation focused on the posterior fossa, also can have widespread effects throughout the brain, due to the extensive reciprocal connections between the cerebellum and the frontal lobe (Leiner, Leiner, & Dow, 1986). Thus, it may be assumed that the damaging effects of radiation on normal-appearing white matter in both the cerebrum and the cerebellum contribute to childhood medulloblastoma survivors' significant deficits in processing speed. Indeed, deficits in processing speed appear to be positively correlated with radiation dose (Kieffer-Renaux et al., 2000), while treatment with surgical resection alone does not appear to significantly impact processing speed (Mabbott et al., 2008). Thus, it is likely that survivors of childhood medulloblastoma typically have deficits in cognitive fluency, although there are no prior studies explicitly addressing this construct in this population.

COGNITIVE FLUENCY

Definition and Neural Basis

It has been proposed that the ability to perform complex cognitive operations quickly and accurately—that is, cognitive fluency—enables minimization of resource allocation and maximization of performance (Rypma et al., 2006). From a developmental perspective, cognitive fluency appears to govern improvements in task performance (Kail, 1986, 1988, 1991; Keating & Bobbitt, 1978). In terms of the Cattell-Horn-Carroll theory, cognitive fluency is a combination of *Gs*, or processing speed, and *Glr*, or long-term retrieval (Mather & Woodcock, 2001; McGrew & Woodcock, 2001). Thus, cognitive fluency is distinguished from cognitive efficiency, which is a combination of *Gs* and *Gsm*, or working memory. In layman's terms, cognitive fluency is a measure of ease, speed, and accuracy in performing complex cognitive processes that rely more on long-term knowledge acquisition than short-term memory. Processing speed is a necessary but not sufficient component of cognitive fluency. In addition to short-term memory, other cognitive abilities such as visuospatial skills or attention may also be needed to complete a given task quickly and accurately. As reported previously, many such components of cognitive fluency are often significantly impaired subsequent to treatment for childhood medulloblastoma.

Studies of both development and aging suggest that one of the determinants of cognitive fluency is axonal maturation of the prefrontal cortex (Gomez-Perez, Ostrosky-Solis, & Prospero-Garcia, 2003; Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli,

2002; Fisk, Fisher, & Rogers, 1992; Medina et al., 2006; Rypma, Prabhakaran, Desmond, & Gabrieli, 2001; Rypma, Berger, Genova, Rebbechi, & D'Esposito, 2005; Rypma & D'Esposito, 1999; Salthouse, 1992; Sawamoto, Honda, Hanakawa, Fukuyama, & Shibasaki, 2002; Small, Kemper, & Lyons, 2000; West, 1996). However, the cerebellum also has been shown to play a large role in cognitive fluency, particularly in the sense that it may improve the efficiency of communication between various cortical regions (Ackerman, Mathiak, & Ivry, 2004; Desmond, 2001; Ivry & Baldo, 1992; Leiner, Leiner, & Dow, 1986, 1989; Schmahmann & Sherman 1998; Weaver, 2005). Leiner and colleagues (1989) mention that "increased speed and skill in the control of mental manipulation" (p. 1005) is the primary impact of the cerebellum on the association areas of the prefrontal cortex, thereby enhancing the efficiency of a wide range of cognitive functions. These authors provide an extremely detailed summary of the reciprocal connections between the cerebellum and the frontal lobe to support this assertion. In particular, they highlight the role of the dentate nucleus and ventrolateral thalamus in transmitting efferent signals from the cerebellum to the prefrontal cortex and the role of the pontine nuclei and the red nucleus in transmitting signals the opposite direction.

Comparison to Processing Speed

Cognitive fluency is conceptualized as the simultaneous maximization of speed and accuracy of performance on complex cognitive tasks, typically involving previously acquired knowledge. Thus, it is hypothesized that cognitive fluency builds on basic functions such as processing speed and attention. Processing speed refers to the speed at

which a person can perform automatic, over-learned tasks. Tests that measure processing speed often involve simple skills such as visual scanning, whereas cognitive fluency tests typically consist of more involved tasks such as math calculations. Furthermore, cognitive fluency measures typically have less dependence on motor functioning and more emphasis on cognitive demands than do measures of processing speed. Both constructs are measured by timed performance, reflecting a scenario commonly seen in real-world academic testing. However, cognitive fluency, involving speed and accuracy on complex cognitive tasks, may be more representative of the neurocognitive skill set necessary for real-world academic success than is processing speed.

In Medulloblastoma

While processing speed refers to the speed at which a person can perform automatic, simple tasks, cognitive fluency is conceptualized as speed of performance on more complex cognitive tasks. Thus, deficits in these two areas most likely contribute to the declines in IQ and academic performance observed in childhood medulloblastoma survivors. While deficits in processing speed have been clearly documented in survivors of childhood medulloblastoma, specific studies of cognitive fluency in this population have not been conducted previously.

In a general posterior fossa population, processing speed—but not working memory or sustained attention—was distinguished as the only significantly worse deficit in a radiated group as compared to a non-radiated group (Mabbott et al., 2008). Furthermore, medulloblastoma patients younger than 12 years of age show slower

reaction times after radiation therapy (Merchant et al., 2002a). These findings suggest that radiation may play a large role in the cognitive fluency deficits seen in childhood medulloblastoma survivors. This is most likely due to the negative effects of radiation on normal-appearing white matter in the cerebrum and cerebellum (American Cancer Society, 2005; Mabbott et al., 2006; Mulhern et al., 1999, 2001, 2004; Palmer et al., 2002; Reddick et al., 2000, 2003). White matter is particularly crucial to cognitive fluency due to its function in facilitating the rate of signal transmission along axons (Schmithorst, Wilke, Dardzinski, & Holland, 2002). It is theorized that impaired processing speed, a consequence of radiation damage to white matter in the brain, is responsible for poor acquisition of skills and knowledge in childhood medulloblastoma survivors (Mabbott et al., 2008). This theory may be extended to include the impact of impaired cognitive fluency, a more complex application of processing speed that involves accuracy and parsimony, vital to real-world learning and academic performance.

MOTIVATION

Definition

Motivation is an elusive construct, defined and operationalized in a wide variety of manners, with no one theory uniting its study. Many definitions of motivation have been offered over the decades, and attempts to define motivation remain wide-ranging. Marin and Wilkosz (2005) describe motivation as "the psychological domain concerned with goal-directed behavior." Evolutionary psychologists discuss motivation in terms of

"purposeful behavior that is ultimately directed toward the fundamental goal of inclusive fitness" (Bernard, Mills, Swenson, & Walsh, 2005). Many simply define it as "what makes one work to obtain a reward or to avoid punishment" (Pessoa, 2008). This final definition simplifies and integrates the others, addressing the issue of goal-directed behavior while at the same time stating in real-world terms how this might appear.

Locke & Braver (2008) note three sources of variation in motivation: an individual's motivational state, individual differences in motivation-related traits, and the efficacy of the pathway that translates increased motivation into optimized cognitive processing. The former cannot always be assumed, even in the presence of an experimental manipulation of motivational state, such as an incentive or reward. This is where individual differences in motivation-related traits become relevant. However, even increased motivation may not imply successful attainment of a cognitive performance goal, which is where the final source of variation—the efficacy of that pathway—is of the utmost importance. In operationalizing and studying motivation, it is prudent to keep these three aspects in mind. The latter, the efficacy of neural pathways connecting motivation and cognition, deserves further discussion, as there exists strong empirical support for the role of various neural structures in motivation.

Neural Structures of Reward

Reward, the notion itself ranging from financial gain to a sense of personal satisfaction, has long been known to have an impact on motivation. Consequently, external incentives are frequently used to manipulate levels of motivation in experimental

designs, enabling fairly straightforward investigation into at least one aspect of motivation. The brain's reward system "is responsible for defining goals, encoding incentive value, and motivating goal directed behavior" (Engelmann & Pessoa, 2007, p. 668; Robbins & Everitt, 1996; Schultz, 2000). A model outlined by Marin and Wilkosz (2005) lists the core neural structures of the reward system as the anterior cingulate cortex, nucleus accumbens, ventral palladium, and the medial dorsal nucleus of the thalamus (with supporting evidence from Kalivas & Barnes, 1993 and Marin, 1996). They theorize that the prefrontal cortex interacts with this core reward system to modify an individual's current motivational state.

Evidence for this theory is abundant. The possibility of monetary gain has been shown to activate regions of the ventral striatum—particularly the nucleus accumbens—and ventral tegmental area to a degree proportional to the amount of potential gain (Carter, MacInnes, Huettel, & Adcock, 2009; Bjork et al., 2004; Knutson et al., 2001, 2003; O'Doherty, Critchley, Deichmann, & Dolan, 2003). The ventral striatum also appears to respond differentially based on a reward's perceived predictability (Berns, McClure, Pagnoni, & Montague, 2001; McClure, Berns, & Montague, 2003; O'Doherty et al., 2003). Striatal fibers, in conjunction with the orbitofrontal and prefrontal cortices, also appear to form circuits associated with motivation for rewards (Bjork et al., 2004; Cohen, Schoene-Bake, Elger, & Weber, 2009; Knutson et al., 2001, 2003; O'Doherty et al., 2001; Pochon et al., 2002; Schultz, 2000; Taylor, Welsh, Wager, Phan, Fitzgerald, & Gehring, 2004). As a result of the neural convergence of both cognitive and affective/motivational neural pathways in the lateral prefrontal cortex, this region is thought to play a large role in making motivation-based decisions regarding behavior.

Several primate studies support the role of the lateral prefrontal cortex as the neural region that integrates cognition and motivation (Kobayashi, Lauwereyns, Koizumi, Sakagami, & Hikosaka, 2002; Roesch & Olson, 2005; Tsujimoto & Sawaguchi, 2005; Watanabe, 1990, 1996, 2002, 2007). Meanwhile, the amygdala appears to code reward intensity and respond to learned predictors of reward (O'Doherty, 2004), while the anterior cingulate cortex plays a role in reward anticipation, decision-making, and negative emotional responses to monetary losses (Bush, 2000; Marin, 1996; Small, Gitelman, Simmons, Bloise, Parrish, & Mesulam, 2005).

Although the ventral striatum and prefrontal cortex appear to be the primary structures involved in reward-based motivation, rewards also seem to preferentially activate a number of regions throughout the brain. Locke and Braver (2008) note that "results so far show that brain systems associated with reward, with cognitive control structures, and with the individual tasks themselves all show increased activity under reward conditions, sometimes in the same way as for increased task demands" (p. 99). This statement is based on findings from a number of studies in addition to their own (e.g., Pochon et al., 2002; Taylor et al., 2004). In their study, Locke & Braver (2008) found that a monetary incentive produced enhanced reaction times and increased activity in the right lateral prefrontal cortex, the right parietal cortex, the dorsal medial frontal cortex, and the left cerebellum. In the caudate nucleus and putamen, reward-processing regions that comprise the dorsal striatum, motivation-related increases proportional to the amount of a monetary incentive have also been noted (Delgado, Stenger, & Fiez, 2004; Mizuno et al., 2008; Taylor et al., 2004). In general, various structures related to the mesocortico-limbic dopamine system, including the orbitofrontal and prefrontal cortices,

anterior cingulate, amygdala, insula, striatum, thalamus, mesencephalon, and cerebellum have been implicated in secondary (e.g., monetary) reward processing (e.g., Thut et al., 1997; O'Doherty, Deichmann, Critchley, & Dolan, 2002; Kirsch et al., 2003; McClure et al., 2003, 2004).

In commenting on their finding that monetary incentives cause increased neural activity in the cerebellum, Locke & Braver (2008) mention that the nature of the task, a continuous performance task (CPT), may be one explanation. Stronger cerebellar responses in this context could be related to this structure's hypothesized roles in verbal working memory, error-detecting in verbal rehearsal, or precise interval timing. However, a study in rats found that removal of the cerebellum reduced motivation-related behaviors independent of the modulatory influence of resultant motor deficits (D'Agata, Drago, Serapide, & Cicirata, 1993). And in healthy adults, cerebellar activity has been shown to decline as a function of cognitive workload, interpreted as related to decreased motivation (Weich et al., 2005). Schutter and van Honk (2005) theorize that the cerebellum acts as a convergence zone for multiple streams of information, noting that "the highly topographically organized information flow from and to the cerebellum […] suggests that the cerebellum plays an important role in the regulation of adaptive emotional and motivational behavior" (p. 292).

Additionally, it is important to note that "reward is suggested to have specific influences on cognitive function—as opposed to general effects, such as arousal" (Pessoa, 2009). That is, although motivational processes impact a wide range of neural structures, this is theorized to occur with relative precision, with the goal of maximizing reward by specifically recruiting the necessary structures for the task at hand. Engelmann

and Pessoa (2007) observed that attention, as reflected by performance on a response-time task, improved linearly as a function of enhanced motivation, as quantified by absolute monetary incentive value. They hypothesize that increased motivation impacts the allocation of resources available for various executive functions in a manner intended to maximize potential reward. Overall, these findings from imaging research on motivation may be interpreted as illustrative of the interwoven nature of cognition and emotion/motivation.

Extrinsic Motivation

After defining motivation as a whole, the construct then commonly is split into two distinct types—intrinsic and extrinsic. Intrinsic (or internal) motivation can be explained as "the doing of an activity for its inherent satisfactions rather than for some separable consquence" (Ryan & Deci, 2000, p. 56) or, in layman's terms, "the motivation to engage in work primarily for its own sake, because the work itself is interesting, engaging, or in some way satisfying" (Amabile, Hill, Hennessey, & Tighe, 1994, p. 950). In contrast, extrinsic (or external) motivation may be defined as engaging in an activity "in order to attain some separable outcome" (Ryan & Deci, 2000, p. 60) or more specifically, "the motivation to work primarily in response to something apart from the work itself, such as reward or recognition or the dictates of other people" (Amabile, Hill, Hennessey, & Tighe, 1994, p. 950). Much debate has focused on the relationship between the two, ever since deCharms (1968) proposed that they are indeed related and two seminal studies shortly thereafter suggested that increasing extrinsic motivation

decreases intrinsic motivation (Deci, 1971; Lepper, Green, & Nisbett, 1973). The debate regarding the relationship between intrinsic and extrinsic motivation will be examined in a following section.

The formal study of extrinsic motivation began with B. F. Skinner's study of operant conditioning, using primary rewards such as food as a reward to train animals to perform certain behaviors. Although easily and primarily studied in humans by means of monetary incentives for performance, extrinsic motivation can be based on a variety of motivational factors, even as simple as a smile. Indeed, the facial expressions of caregivers in particular appear to serve a "communicatory function" (Blair, 2003), giving an infant a sense of social reward or punishment, and thus being capable of altering future behavior (Chakrabarti, Bullmore, & Baron-Cohen, 2006; O'Doherty, Winston, Critchley, Perrett, Burt, & Dolan, 2003; Schultz, 2004; Sorce, Emde, Campos, & Klinnert, 1985). However, as early as age 5, children begin to develop greater interest in and understanding of the concept of money; by age 8, these are fully established (Berti & Bombi, 1981; Grunberg & Anthony, 1980). This corresponds with the much-replicated finding of a decline in intrinsic motivation and enhanced salience of extrinsic motivation as children mature (Harter, 1981; Harter & Jackson, 1992; Lepper, Corpus, & Iyengar, 2005; Newman, 1990; Ryan & Deci, 2000; Tzuriel, 1989). Just like intrinsic motivation, various forms of extrinsic motivation can have performance-enhancing consequences for children, adolescents, and adults (Casey, Tottenham, & Fossella, 2002; Schultz, 2004; Maxwell, Shackman, & Davidson, 2005; Miller & Cohen, 2001; Hare & Casey, 2005; Hare, Tottenham, Davidson, Glover, & Casey, 2005; Latham & Kinne, 1974; Latham & Yukl, 1975; Lea & Webley, 2006; Lee, Locke, & Phan, 1997; Vohs, Mead, & Goode,

2006; Watanabe, 2007). Thus, the majority of research on motivation has looked at extrinsic (rather than intrinsic) motivation, as it is easily operationalized in varying degrees with money, food, or social rewards.

The nature-versus-nurture (trait-versus-context) debate often arises in discussions of extrinsic motivation. One fMRI study showed that subjects who self-reported a high level of extrinsic motivation had enhanced activity in the prefrontal regions, parietal and temporal cortex, amygdala, and striatum during a gambling task with monetary win/loss potential, whereas subjects with low extrinsic motivation had diminished activity in these areas during the same task (Linke et al., 2010). Similarly, a study of children and adolescents by Kohls and colleagues (2009) found that personality traits related to reward seeking were associated with enhanced responsiveness to monetary rewards. While these and other similar findings (e.g., Pailing & Segalowitz, 2004) may suggest biologically based neural differences, it is just as likely that these differences are a function of environmental conditions or learning.

Monetary Incentives

Money is a "secondary" external incentive in that it increases extrinsic motivation for behaviors after its value has already been learned via its utility in obtaining "primary" reinforcers such as food. However, that is not to say that money is not a strongly motivating incentive—in fact, many researchers assert that money may be the most influential reward possible for humans (Lea & Webley, 2006; Vohs, Mead, & Goode, 2006). Neuroimaging studies of adults comparing various types of external

incentives have confirmed this assertion, while also corroborating the common finding of a positive correlation between activity in reward areas of the brain and the amount of monetary incentive (Delgado, Stenger, & Fiez; 2004; Kirsch et al., 2003; Thut et al., 1997). In children, at least one study has shown that monetary rewards enhance performance more powerfully than social rewards (e.g., smiles) in males ages 8 through 12 (Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009). One explanation for the strength of money as an extrinsic motivator may be that money has a constant utility, regardless of fluctuations in "satiation" of desire for it, whereas reinforcers such as food or even social rewards (e.g., smiles) may not have such a constant utility (Estle, Green, Myerson, & Holt, 2007).

Locke and Latham (2002) note that goal-directed performance is enhanced when people are more strongly committed to those goals, and that monetary incentives are a practical means of enhancing such goal commitment for a task. However, they caution that the use of monetary incentives for this purpose must take into account the fact that if a goal cannot be reached due to extreme difficulty, monetary rewards can actually hurt performance due to a sense of decreased self-efficacy. However, even with a very difficult task, if rewards are provided on a sliding scale based on performance rather than a single reward for reaching the goal, performance will remain highly motivated (Latham & Kinne, 1974; Latham & Yukl, 1975; Lee, Locke, & Phan, 1997). Thus, the present study has been designed in this manner.

Effects of Extrinsic on Intrinsic

It is frequently implied that within motivational orientation (regardless of whether it is viewed as a trait or a state), intrinsic and extrinsic motivation are oppositional forces. This view originated from studies by Deci (1971, 1972a, 1972b) showing that tangible rewards such as money could reduce intrinsic motivation in college students, a finding that has been often replicated (e.g., Kruglanski, Friedman, & Zeevi, 1971; Lepper, Greene, & Nisbett, 1973). A landmark meta-analysis by Deci, Koestner, and Ryan (1999) indicated an overall negative effect of external rewards on intrinsic motivation, with a few situation-specific exceptions, some of which will be discussed shortly.

Relevant to the present study, Deci, Koestner, and Ryan (1999) found that expected, performance-contingent, external rewards can negatively impact the "free choice" operationalization of intrinsic motivation (that is, a person's free choice to engage in a task) on inherently interesting tasks. However, such rewards did not significantly impact self-reported interest or enjoyment, the other manner in which intrinsic motivation is typically operationalized (p. 644, 653). Additionally, such rewards did not have a negative impact on intrinsic motivation when given for dull, boring, uninteresting tasks (p. 651). Clearly, this has implications for academic tasks, as children and adolescents from kindergarten through college generally find schoolwork dull and uninteresting (Anderman & Maehr, 1994; Eccles & Midgley, 1990; Epstein & McPartland, 1976; Haladyna & Thomas, 1979; Harter, 1981; Lepper, Sethi, Dialdin, & Drake, 1997; Newman, 1990; Sansone & Morgan, 1992; Tzuriel, 1989). And even the

finding regarding "free choice" has had its dissenters, the most prominent being several meta-analyses indicating that external rewards generally do *not* have negative effects on intrinsic motivation (Cameron, 2001; Cameron & Pierce, 1994; Eisenberger & Cameron, 1996). Cameron (2001) asserts that external rewards actually enhance intrinsic motivation (as measured by either interest/enjoyment *or* "free choice") for boring tasks and even for interesting tasks, as long as the rewards are explicitly tied to performance standards. Granted, the statistical methodology of Cameron's group has been resoundingly criticized (Deci, Ryan, & Koestner, 2001; Kohn, 1996; Lepper, Hinderlong, & Gingras, 1999; Lepper, Keavney, & Drake, 1996; Ryan & Deci, 1996). However, the point remains that even methodologically strong meta-analyses have not found a negative effect of expected, performance-contingent, external rewards on self-reported interest for interesting tasks—nor on either form of operationalization of intrinsic motivation (interest/enjoyment or "free choice") with regard to dull, uninteresting tasks (Deci, Koestner, & Ryan, 1999; Deci, Koestner, & Ryan, 2001; Tang & Hall, 1995). Furthermore, one of these methodologically strong meta-analyses did in fact confirm Cameron's findings that external, expected, performance-contingent rewards may enhance interest for boring tasks (Tang & Hall, 1995).

There has not been much research on the real-world implications of external rewards on intrinsic motivation, despite the fact that a survey of 136 college students found that 72-74% of the subjects had received some sort of external rewards for academic achievement in elementary school, middle school, and high school (Davis, Winsler, & Middleton, 2006). Slightly more than half of the subjects (51%) had received money for good grades in high school, while 90% of the subjects reported receiving

external rewards for achievement from teachers in elementary school (81% of those in middle school and 75% in high school)—such as stickers, prizes, extra recess, and even money. One study has suggested that such rewards are associated with poorer academic performance (Ginsberg & Bronstein, 1993). However, it should be noted that the researchers recruited participants for this self-report study by paying them $100, thus potentially skewing the subject pool toward more extrinsically motivated subjects in the first place. Davis and colleagues (2006), retrospectively investigating the impact of external rewards on long-term intrinsic motivation in the realm of academics, found that college-age males who had received more performance-based external rewards for academic performance in lower grades displayed greater extrinsic *and* intrinsic motivation than those who had not. Furthermore, males who had received external rewards from teachers in the earlier years of their schooling were setting higher academic goals for themselves in college. For female college students, a history of receiving external rewards for achievement was *negatively* associated with current levels of extrinsic motivation, and there was no association with intrinsic motivation.

Thus, some feel that the debate remains open regarding the overall effect of extrinsic rewards on intrinsic motivation, as there appear to be a number of situation-specific caveats to this finding. Trait-based aspects of intrinsic motivation are likely an important factor as well, as findings from Amabile and colleagues (1994) suggest that "highly autonomous individuals, while retaining high levels of intrinsic motivation toward their work, might also be highly motivated to achieve compensation for that work" (pp. 964-965). On a similar note, many researchers (e.g., Deci & Ryan, 1985, 1991; Harter, 1978; Maslow, 1954; Rigby, Deci, Patrick, & Ryan, 1992) have theorized

that various forms of intrinsic motivation "may be produced or augmented by a process of progressive 'internalization' of initially external incentives and constraints" (Lepper, Sethi, Dialdin, & Drake, 1997, p. 39). Lepper and colleagues suggest that "perhaps too exclusive a preoccupation with either intrinsic or extrinsic motivation may have deleterious effects—though presumably of predictably different sorts" (p. 44).

Why Study Motivation?

Locke and Braver (2008) said it best: "It is a truism of cognitive research that participants perform experimental tasks with varying levels of motivation. Some participants appear to show little interest and exert minimal effort in their task performance, whereas others seem to approach the task as a critical test, exhibiting a burning desire to perform to their utmost ability. Yet, even though this variation in motivation is a well-known phenomenon, it seems to be underappreciated and underexplored" (p. 99).

Indeed, both internal and external motivation can fluctuate on both an individual and an environmental basis, thus impacting performance not only in research tasks but also in real-life situations such as school. An individual's level of motivation at any given time may result in performance that either reflects his or her true ability or falls short of the individual's true capabilities. Thus, when conducting neuropsychological tests, is it enough to record an individual's scores without taking both his or her trait- and context-based motivation into account? Can neuropsychological functioning be significantly altered—either negatively or positively—by motivation to the point that

diagnoses or recommendations might be affected? Are certain populations capable of significantly improving their performance on neuropsychological tests based on alterations in motivation? These are questions that need to be asked, explored, and answered—but have not yet been given proper consideration in neuropsychological research.

Motivation and Performance in Children

Experimentally increased motivation (usually tapping the extrinsic side) has been shown to significantly improve performance on a variety of cognitive tasks from childhood through adulthood, in both healthy and disordered populations (Casey, Tottenham, & Fossella, 2002; Engelmann & Pessoa, 2007; Engelmann, Damaraju, Padmala, & Pessoa, 2009; Hare & Casey, 2005; Hare, Tottenham, Davidson, Glover, & Casey, 2005; Locke & Braver, 2008; Maxwell, Shackman, & Davidson, 2005; McCauley, McDaniel, Pedroza, Chapman, & Levin, 2009; Miller & Cohen, 2001; Pailing & Segalowitz, 2004; Riva, Pantaleoni, Milani, & Belani, 1989; Schultz, 2004; Taylor, Welsh, Wager, Phan, Fitzgerald, & Gehring, 2004; Thornton, Boudreau, Griffiths, Woodward, Fawkes-Kirby, & Honer, 2007; Watanabe, 2007). Particularly relevant to populations with psychopathology, it has been found that "emotional distress does not have a significant effect on neuropsychological performance provided adequate motivation is present" (Meyers & Diep, 2000, p. 137; Reitan & Wolfson, 1997).

With regard to the present study, the impact of motivation is particularly relevant to children's academic performance. Schoolwork is a child's primary occupation, and

success or failure in school can have lifelong consequences. However, children and adolescents generally consider most academic tasks inherently dull and consequently report low intrinsic motivation for such tasks (Anderman & Maehr, 1994; Eccles & Midgley, 1990; Epstein & McPartland, 1976; Haladyna & Thomas, 1979; Harter, 1981; Lepper, Sethi, Dialdin, & Drake, 1997; Newman, 1990; Sansone & Morgan, 1992; Tzuriel, 1989). In fact, these studies all show that intrinsic motivation toward academics linearly decreases with years of schooling. Notably, intrinsic motivation toward other activities, such as sports and social relationships, does not show this decline (Harter, 1981; Sansonse & Morgan, 1992). The concepts of cognitive evaluation theory and overjustification theory have been highlighted as potential theoretical explanations for this phenomenon, but no clear conclusion has been reached. In particular, cognitive evaluation theory's proposal that the increasing control of the school setting (such as through tests and grades) is responsible for this decline in intrinsic motivation is questionable, as sports are increasingly controlled as well, with structured practices, coaching, and "grading" in the forms of wins and losses.

Children and adolescents' low intrinsic motivation for schoolwork is more often than not supplemented extrinsically by their parents or teachers (Davis, Winsler, & Middleton, 2006). How, then, do external rewards for academics impact children and adolescents' performance? Recall that there is conflicting evidence from two real-world studies. One has suggested a negative impact of external rewards on academic performance (Ginsberg & Bronstein, 1993). The other found that receiving external rewards at some point in early schooling has gender-dependent neutral to positive effects on college-age intrinsic motivation and academic goal-setting—and most importantly,

current academic performance was positively associated with both current educational goals and intrinsic motivation (Davis, Winsler, & Middleton, 2006). Another study has shown that overall motivation, both intrinsic and extrinsic, has been shown to influence classroom achievement, without any additional effects attributable to increased effort (Brookhart, Walsh, & Zientarski, 2006). Males in particular may perceive extrinsic rewards for academic performance as informational, internalizing the rewards as an indication of their abilities, enhancing confidence, and creating healthier motivational patterns (Davis, Winsler, & Middleton, 2006). Finally, specific studies have shown that external incentives can improve reading fluency and math performance (Codding, Baglici, Gottesman, Johnson, Kert, & Lebeouf, 2009; Eckert et al., 2002; Noell et al., 2001) and that externally motivated elementary and junior high students perform just as well in mathematics and reading as internally motivated students (Schultz & Switzky, 1993). Jones and colleagues (2009) note that "although lack of motivation and insufficient practice are not considered core deficits in reading disorders, they are often essential components of *any* educational intervention" (p. 53) and indicate that external incentives can provide such motivation. Thus, there are several compelling arguments for a further study of motivation in relation to academic performance.

It is particularly important to study motivation in children because it appears to have a powerful impact on their behavior and performance. Children and adolescents appear more responsive to monetary rewards than to intangible rewards (Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009). In fact, both young children and adolescents appear to respond more strongly to monetary incentives than do adults (Hardin et al., 2007; Jazbec et al., 2005; Kohls et al., 2009)—that is, their performance improves more.

This seems contradictory to knowledge that one of the most notable and long-lasting developmental differences between the growing and the mature brain is in the frontal lobe, which continues developing through the third decade of life (Sowell, Thompson, Holmes, Jernigan, & Toga, 1999). Thus, the amygdala and other "emotional" areas of the developing brain respond more strongly to the affective valence of rewards, as they are less restrained by inputs from the more "rational" frontal regions. Yet despite the underdevelopment of the frontal lobe in children and adolescents, individuals in this age range show a surprising capacity for behavioral control if the motivation is there. For example, while adolescents showed lower performance accuracy than did adults in a particular antisaccade control task, monetary incentives brought that performance up to the level of adults (Jazbec et al., 2005). This may be due the "emotional" brain regions of children and adolescents responding so strongly to external incentives that they are able to enhance the neural activation of high-order control structures in order to overcome developmental neural deficits, improve performance, and obtain the reward. Granted, there may be some neural deficits that cannot be overcome—particularly depending on the type of task and which brain regions are required to perform it. The tumor- and treatment-related brain damage of childhood medulloblastoma survivors may fall into this category, although this has yet to be determined and will be investigated by the present study.

In Medulloblastoma Survivors

The study of motivation in the context of academic performance may be particularly relevant for survivors of pediatric medulloblastoma for a number of reasons. First, survivors of childhood medulloblastoma exhibit significant declines in academic performance (Dennis, Spiegler, Hetherington, & Greenberg, 1996; Palmer et al., 2001; Palmer, Reddick, & Gajjar, 2007; Ris, Packer, Goldwein, Jones-Wallace, & Boyett, 2001). As previously mentioned, children who receive cranial radiation, such as medulloblastoma patients, are 7 times more likely to require special education services compared to survivors of childhood cancers who do not receive cranial radiation (Mitby et al., 2003; Palmer, 2008). The declines in IQ and academic performance of childhood medulloblastoma survivors are likely related to underlying neurocognitive deficits in areas ranging from memory and attention to processing speed (Palmer, 2008). Thus, it may be valuable to further investigate the aforementioned general impact of motivation on academic performance in this specific population.

Since the well-documented declines in IQ among childhood medulloblastoma survivors appear to be due to a failure to continue learning information at their pre-treatment rate (Palmer et al., 2001), these survivors may be considered as similar to learning disabled children in this respect. It has been shown that learning disabled and other "academically problematic" children have more extrinisic motivation than their healthy peers (Lepper, Sethi, Dialdin, & Drake, 1997; Lincoln & Chazan, 1979). Furthermore, it has been theorized that motor deficits are responsible for a generalized disruption of motivation in a population of cerebellar patients (Bracke-Tolkmitt et al.,

1989). These points highlight additional reasons to investigate the response to external incentives among childhood medulloblastoma survivors. However, the two most compelling arguments for such a study are the prior investigations by Riva and colleagues (1989) and McCauley and colleagues (2009).

Riva et al. (1989) did not intend to investigate the effects of extrinsic motivation on the neuropsychological performance of pediatric medulloblastoma survivors. But when these children failed to show deficits on a task involving processing speed and attention, despite the fact that a large body of research has indicated they should, the authors came to an intriguing conclusion. Due to the relative novelty of computers at that time, and noting that such technology was "known to be a source of irresistible attraction for children" (p. 110), Riva and colleagues hypothesized that these children did not display deficits due to heightened motivation. They did not delve into the distinctions of extrinsic versus intrinsic motivation. This study remains the only known documentation of the potential impact of motivation on childhood medulloblastoma survivors at this time. However, in a study of children with traumatic brain injury (TBI), McCauley and colleagues (2009) showed that monetary incentives significantly improved performance on a vigilance/memory task. Mild TBI patients were even able to perform at the same level as an orthopedic control group, although this was not the case for children with severe TBIs. This suggests that childhood medulloblastoma survivors may be similarly responsive to monetary incentives, as the neurocognitive effects of medulloblastoma and its treatment are quite similar to those of traumatic brain injury in children. This study also provides a rationale for using monetary incentives in a medulloblastoma population.

A final piece of evidence supporting the present investigation is the fact that in healthy populations, brain structures associated with academically relevant cognitive tasks—including tasks involving reaction time, a measure of processing speed—show increased activation during motivational conditions (Locke & Braver, 2008; Pochon et al., 2002; Taylor et al., 2004). The brains of medulloblastoma survivors may function similarly, allowing these children to overcome deficits seen under low-motivation conditions, or the damage caused by the tumor and its treatment may be too significant for them to overcome.

CONCLUSION

Brain tumors are the second most common form of childhood cancer (Gurney, Wall, Jukich, & Davis, 1999; Horner et al., 2009), and medulloblastoma is the most commonly diagnosed form of malignant brain tumor (Gottardo & Gajjar, 2006). With current five-year survival rates of 70-85%, the majority of these children will become long-term survivors (Palmer, 2008). An abundance of research suggests that radiation plays a large role in the various intellectual, academic, psychosocial, and neurocognitive deficits seen in childhood medulloblastoma survivors. These children generally display declines in IQ and academic achievement, likely related to neurocognitive deficits in areas ranging from attention to processing speed (Mabbott et al., 2005; Palmer, 2008; Palmer et al., 2001). Processing speed is theorized to be a component of cognitive fluency, which refers to speed of performance on more complex tasks. This study

represents the first comprehensive evaluation of cognitive fluency and the impact of motivation on academic performance among childhood medulloblastoma survivors.

Medulloblastoma survivors exhibit significant declines in academic performance, and the potential lifelong implications of such difficulties make academics an important area for the type of research involved in the present study, potentially bearing implications for future interventions (Dennis, Spiegler, Hetherington, & Greenberg, 1996; Hoppe-Hirsch et al., 1995; Palmer, 2008; Palmer et al., 2001; Palmer, Reddick, & Gajjar, 2007). Relevant to the present study, it has been shown that monetary incentives can be used to enhance goal commitment/motivation (Locke & Latham, 2002). Furthermore, children and adolescents appear more responsive to monetary rewards than to intangible rewards (Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009). Since "academically problematic" children display more extrinsic motivational orientation, monetary incentives are a natural starting point for a study of the impact of motivation on the academic performance of childhood medulloblastoma survivors (Lepper, Sethi, Dialdin, & Drake, 1997). Monetary incentives are commonly used to enhance academic performance, with one study indicating that 90% of children receive monetary or other extrinsic incentives for academic performance in elementary school, while 51% receive monetary rewards through high school (Davis, Winsler, & Middleton, 2006).

Even though extrinsic motivation has been shown to decrease intrinsic motivation in certain situations, no research has argued against the use of extrinsic motivation in scientific studies of motivation. In fact, many have used external incentives in studies to improve academic performance (Codding, Baglici, Gottesman, Johnson, Kert, & Lebeouf, 2009; Eckert et al., 2002; Noell et al., 2001; Jones et al.,

2009). It appears likely that expected, performance-contingent, external rewards would not decrease intrinsic motivation toward inherently boring tasks (Cameron, 2001; Cameron & Pierce, 1994; Deci, Koestner, & Ryan, 1999, 2001; Eisenberger & Cameron, 1996; Tang & Hall, 1995). The academic tasks used in the present study are likely seen as inherently uninteresting to most children. Furthermore, it is necessary to use extrinsic motivation in the present study because it is uncertain whether childhood medulloblastoma survivors will be able to increase their performance to normal levels as a result of enhanced motivation. Since monetary rewards produce the strongest changes in performance (Estle, Green, Myerson, & Holt, 2007; Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009; Latham & Kinne, 1974; Latham & Yukl, 1975; Lea & Webley, 2006; Lee, Locke, & Phan, 1997; Locke & Latham, 2002; Vohs, Mead, & Goode, 2006), using a monetary incentive is the most efficient and reasonable starting point.

Other arguments for the use of a monetary incentive include the fact that in general, intrinsic motivation decreases as children grow older, while extrinsic motivation appears to change very little (Lepper, Corpus, & Iyengar, 2005), making an extrinsic manipulation less susceptible to individual differences. Additionally, childhood medulloblastoma survivors may have similar learning profiles to learning disabled children, who have been shown to be more extrinsically motivated (Lepper, Sethi, Dialdin, & Drake, 1997; Lincoln & Chazan, 1979). If childhood medulloblastoma survivors exhibit significant improvement in performance in response to monetary incentive, this finding would perhaps provide a rationale for looking at other, more intrinsic types of motivation manipulations. If childhood medulloblastoma survivors do

not exhibit performance improvements, this would suggest that their tumor- and treatment-related deficits in cognitive efficiency may be too severe for them to overcome.

Relevant to the population of interest, a previous study has suggested that enhanced motivation enables survivors of childhood medulloblastoma to improve their performance to the normal range on tasks related to processing speed and attention, although no studies since have investigated this further (Riva, Pantaleoni, Milani, & Belani, 1989). Additionally, this study did not intentionally manipulate motivation, so the interpretation that enhanced motivation was responsible for performance improvements deserves further investigation. Furthermore, the study included only eight medulloblastoma subjects and seven posterior fossa astrocytoma subjects of unknown severity. Thus, a study with a larger and more explicitly uniform sample size would be advantageous for drawing more certain conclusions.

Recently, a study of children with traumatic brain injury (TBI) showed that monetary incentives significantly improved performance on a vigilance/memory task, addressing neurocognitive functions known to be at risk following brain injury (McCauley et al., 2009). This study is remarkable in that it demonstrated that children with TBIs are capable of overcoming their brain-based deficits. Since the neurocognitive effects of medulloblastoma and its treatment are quite similar to those of traumatic brain injury in children, this finding suggests that the academic performance of childhood medulloblastoma survivors may be enhanced by monetary incentives. However, the extent to which monetary incentives can improve functioning within the boundaries of neurocognitive limitations is unknown.

The majority of the current literature on medulloblastoma survivors is confounded by sampling mixed tumor types and making broad generalizations about "posterior fossa tumors" that cannot definitively be attributed to any specific tumor type. However, the most significant criticism of the current literature is simply that there is a lack of research on the topics of cognitive fluency, academic performance, and the effects of motivation in survivors of childhood medulloblastoma. While many studies have found deficits in processing speed in this population (Mabbott et al., 2008; Merchant et al., 2002a; Mulhern et al., 2004; Palmer, 2008; Riva & Giorgi, 2000; Schatz, Kramer, Ablin, & Matthay, 2000; Spiegler et al., 2004; Vaquero, Gomez, Quintero, Gonzalez-Rosa, & Marquez, 2008), none have extended this to a more comprehensive evaluation of cognitive fluency, which is arguably more meaningful in terms of cognitive and academic performance. Likewise, while many studies have noted academic difficulties in childhood medulloblastoma survivors, this is generally a passing observation, peripheral to the primary aims of the studies. None have examined the nature of these difficulties in much detail, particularly in terms of academic fluency versus basic academic skills. Finally, despite the intriguing indication that enhanced motivation may allow these children to overcome the neurocognitive deficits caused by surgery, chemotherapy, and radiation (Riva, Pantaleoni, Milani, & Belani, 1989), there has been no further research on the possibility. This is not because researchers have dismissed their claims—in fact, Dennis et al. (1998), citing that very study, have called for further investigation.

This study is the first to investigate the effects of monetary incentives on academic fluency tasks involving speed and cognitive efficiency in survivors of childhood medulloblastoma. As such, this study represents a new direction for research

in this population, moving beyond basic documentation of deficits toward interventional research. Specifically, this study investigates enhancement of motivation as a possible intervention to address the declining academic performance of childhood medulloblastoma survivors. Other studies have indicated that extrinsic motivation can enhance reading fluency and math performance in a healthy population (Codding, Baglici, Gottesman, Johnson, Kert, & Lebeouf, 2009; Eckert et al., 2002; Noell et al., 2001; Jones et al., 2009). It seems only logical, then, to investigate the effect of enhanced motivation on the academic performance of childhood medulloblastoma survivors, a population clearly in need of more effective interventions. This study represents the first step toward creating a better future for these children, as high academic failure rates in this population can negatively impact long-term quality of life (Mitby et al., 2003).

PURPOSE OF STUDY AND HYPOTHESES

Purpose of Study

The purpose of the present study will be to examine the effect of an external incentive on the academic fluency of childhood medulloblastoma survivors, which has never been studied previously. An additional goal will be to explore the nature of academic impairments in this population as relates to processing speed and cognitive fluency, the latter of which has never been studied in this population. These investigations will include an examination of potential covariates such as IQ and

radiation dose. Overall, it is the researchers' hope that the present study will provide new levels of insight onto the long-term cognitive deficits experienced by survivors of childhood medulloblastoma. Most importantly, the proposed study will set a new direction for research in this population, investigating motivation as a potential mechanism for the cognitive and academic rehabilitation of childhood medulloblastoma survivors. In a broad sense, this study also may have implications regarding the veracity of neuropsychological test results when conducted in the typical manner—that is, in a low motivation state without incentive.

Aims and Hypotheses

Specific Aim 1

The first aim of the present study will be to determine the effect of external incentives (representing a source of extrinsic motivation) on the academic performance of childhood medulloblastoma survivors.

Hypothesis 1A: External incentives will significantly improve the performance of childhood medulloblastoma survivors on academic fluency tasks.

Hypothesis 1B: Performance on measures of cognitive fluency and processing speed will influence the impact of external incentives on academic fluency performance in childhood medulloblastoma survivors.

Rationale 1: The effect of external incentives has never been studied in survivors of childhood medulloblastoma. Thus, these hypotheses are tentative, being primarily

based on two studies that did not directly investigate this primary aim of the present study. The first of those two studies indicated that a computer, a particular "source of irresistible attraction for children" (p. 110) due to its relative novelty at the time, may have externally enhanced the motivation of childhood medulloblastoma survivors to the extent that they did not exhibit deficits as expected on a task related to processing speed and attention, instead performing in the normal range (Riva, Pantaleoni, Milani, & Belani, 1989). No studies since have investigated this finding further. The second study, conducted by McCauley and colleagues (2009), indicated that a monetary incentive enabled children aged 6 to 19 who had suffered a traumatic brain injury to improve their performance on a vigilance/memory task. As the neurocognitive effects of medulloblastoma are quite similar to those of traumatic brain injury in children, this finding suggests that the cognitive functioning of childhood medulloblastoma survivors may similarly benefit from monetary incentives. Pediatric medulloblastoma survivors are theorized to have a deficit in cognitive fluency, based on their deficit in processing speed (Dennis, Hetherington, & Spiegler, 1998; Kieffer-Renaux et al., 2000; Mabbott et al., 2008; Mulhern et al., 2004; Nagel et al., 2006; Riva & Giorgi, 2000; Reeves et al., 2006; Schatz, Kramer, Ablin, & Matthay, 2000; Spiegler et al., 2004; Vaquero, Gomez, Quintero, Gonzalez-Rosa, & Marquez, 2008). This deficit is likely at the root of their declines in IQ and academic performance (Hoppe-Hirsch et al., 1995; Palmer et al., 2001, 2007; Ris et al., 2001). Thus, the severity of brain-based deficits in cognitive fluency and processing speed may limit the extent to which external incentives can enhance performance in childhood medulloblastoma survivors.

Specific Aim 2

The second aim of the present study will be to investigate how the fluent academic performance of childhood medulloblastoma survivors compares to their academic skill development, cognitive fluency, and processing speed.

Hypothesis 2A: Survivors of childhood medulloblastoma will display a relative weakness in fluent academic performance as compared to academic skill development.

Hypothesis 2B: Performance on measures of cognitive fluency will better predict fluent academic performance in childhood medulloblastoma survivors than performance on measures of processing speed.

Rationale 2: The first hypothesis is based on a pilot study that has indicated that survivors of childhood medulloblastoma have a relative weakness in fluent academic performance as compared to academic skill development (Stavinoha & Burrows, 2004). The second hypothesis is based on the conceptualization of cognitive fluency as an ability that promotes maximization of performance (Kail, 1986, 1988, 1991; Keating & Bobbitt, 1978; Rypma et al., 2006). Whereas processing speed is a relatively simple construct, cognitive fluency is conceptualized as a combination of speed and accuracy on complex cognitive tasks. Thus, it is hypothesized that cognitive fluency would be more relevant than would be pure processing speed to fluent academic performance.

CHAPTER THREE
Methodology

DESIGN

The study has a two-group, repeated measure, within-subjects design. The repeated measure involves administration of a pre- and post-test of fluent academic achievement to each participant. The administration of this repeated measure is central to the investigation of Specific Aim 1, involving the study of the incentive and no-incentive conditions. The subjects were randomized to condition, with half receiving a monetary incentive for performance on the post-test (incentive condition), and half completing the post-test without any monetary incentive (no-incentive condition).

SUBJECTS

The study consisted of 30 children between the ages of 7 years, 0 months and 18 years, 11 months who have been treated for medulloblastoma. Participants were recruited from the departments of Neurosurgery and Neuro-Oncology at Children's Medical Center in Dallas. Participants were recruited by phone prior to coming in for a routine follow-up visit at the Neuro-Oncology Clinic at Children's Center for Cancer and Blood Disorders. Twenty-one participants were enrolled in the study upon arrival for a routine follow-up visit and participated in the study at that time. Nine participants requested to participate in the study outside of their routine follow-up visits; those participants were enrolled and participated in the study either at the department of

Neuropsychology at Children's Medical Center in Dallas or at the department of Psychology at Children's Medical Center at Legacy.

Inclusion Criteria

1. Radiographically diagnosed cerebellar region brain tumor
2. Histographically diagnosed medulloblastoma
3. Current age 7.0 to 18.11 years
4. Proficiency in English (tests are not validated on non-English-speaking populations)
5. Prior treatment with cranial radiation
6. Completion of the signed informed consent by a parent or legal guardian
7. Patient's assent to participate in the protocol
8. No history of traumatic brain injury, stroke, recurrent disease, neurological disorder unrelated to medulloblastoma, or major medical difficulties unrelated to medulloblastoma

MATERIALS

Medical Record Review

Medical information was obtained by retrospective chart review. Data collected included medical information required for standard of care treatment for pediatric brain tumor patients, including name, gender, date of birth, date of diagnosis, preoperative conditions, diagnostic and imaging results, date of surgery, surgical procedure, other treatment modalities, duration of treatment, dosage of treatment received, medical

complications (e.g., hydrocephalus, shunt insertion), and current tumor status. Telephone numbers also were collected in order to conduct telephone screening of potential participants. The medical chart review form can be found in Appendix A.

Measures Unique to the Study

Parents or legal guardians completed a three-page questionnaire detailing the patient's developmental, educational, and medical history, as well as family educational history and current parental employment information. This data was collected in order to describe the sample's psychological, educational, and demographic characteristics. The patient history questionnaire can be found in Appendix B.

At various time points in the study protocol, a Likert scale of cognitive fatigue was administered to the participant. This scale was developed for the present study and consists of visual and written descriptions of cognitive fatigue. Other parent- and self-report measures that were administered are listed below, followed by the measures that comprised the core battery for the study.

Child Behavior Checklist

The Child Behavior Checklist (CBCL; Achenbach, 1991) is a questionnaire that rates a child's problem behaviors and competencies. It was used because it includes a calculation of a child's cognitive tempo. The CBCL was standardized on parents of children ages 6 to 18 and has established validity and reliability. Test-retest reliability

coefficient ranges from 0.95 to 1.00, inter-rater reliability ranges from 0.93 to 0.96, and the internal consistency coefficient ranges from 0.73 to 0.97. The CBCL takes about 15 minutes to complete.

Behavior Rating Inventory of Executive Function

The Behavior Rating Inventory of Executive Function (BRIEF; Gioia, Isquith, Guy, & Kenworthy, 2000) is a questionnaire designed to assess executive functioning in the home and school environments. It was used because it includes scales of inhibition, initiation, and planning/organization, which can provide an estimate of a child's general motivational tendencies in terms of goal-setting theory (Locke & Latham, 1990). The BRIEF was standardized on parents of children ages 5 to 18 and has established validity and reliability. Internal consistency reliability coefficients are high, ranging from 0.80 to 0.98. The BRIEF takes approximately 10 to 15 minutes to complete.

Behavior Assessment System for Children 2

The Behavior Assessment System for Children 2 (BASC-2; Reynolds & Kamphaus, 2007) includes a Self-Report of Personality (SRP). The SRP provides insight into a child's thoughts or feelings. Some of its resulting score clusters relate to self-reliance, locus of control, and attitudes toward school, which provided insight into each child's inherent motivation to perform well on academic tasks. The SRP was standardized on a population ranging from 6 years to college age. It has established

validity and reliability, with internal consistency reliability ranging from .71 to .94 for each scale, and with a median test-retest reliability of .70. The SRP takes approximately 30 minutes to complete and was completed during any down time a patient had during his or her visit to the Center for Cancer and Blood Disorders.

Woodcock-Johnson III Tests of Achievement

The Woodcock-Johnson III Tests of Achievement Standard Battery (WJ-ACH; Riverside Publishing, 2001) is comprised of 12 subtests designed to assess academic progress and assist in the diagnosis of learning disabilities and determination of individual learning variations. Two of these subtests—Calculation and Letter-Word Identification—were administered in the current study to evaluate each participant's level of academic skill development. Two other subtests—Math Fluency and Reading Fluency—were administered to evaluate each participant's academic fluency in that particular modality.

The WJ-ACH is a very efficient measure, with each aforementioned subtest's administration time ranging from 3 to 10 minutes. Math Fluency and Reading Fluency both have a 3-minute time limit. The WJ-ACH was standardized on a stratified random sample of persons aged 24 months to 90+ years, with 4,784 of the 8,818 normative sample subjects in kindergarten to twelfth grade. The WJ-ACH provides age-based norms by month from ages 24 months to 19 years, by year from ages 2 to 90+ years, and by grade from kindergarten through graduate school. One-day test-retest reliability coefficients for Math Fluency and Reading Fluency range according to age from .89 to

.95 and from .80 to .94, respectively. The Calculation and Letter-Word Identification subtests have a median internal consistency reliability of .86 and .94, respectively.

Woodcock-Johnson III Tests of Cognitive Abilities

The Woodcock-Johnson III Tests of Cognitive Abilities (WJ-COG; Riverside Publishing, 2001) is comprised of 20 subtests designed to assess both general intellectual ability and specific cognitive abilities. Three of these subtests—Retrieval Fluency, Decision Speed, and Rapid Picture Naming—were used in the current study. These subtests were selected due to the fact that they comprise the Cognitive Fluency cluster and enable an assessment of this ability, which is essentially a more complex processing speed measure. The WJ-COG was standardized on a stratified random sample of persons aged 24 months to 90+ years, with 4,784 of the 8,818 normative sample subjects in kindergarten to twelfth grade. The WJ-COG provides age-based norms by month from ages 24 months to 19 years, by year from ages 2 to 90+ years, and by grade from kindergarten through graduate school. It has established validity and reliability, with median internal consistency reliability of .85 for Retrieval Fluency, .87 for Decision Speed, and .97 for Rapid Picture Naming.

Wechsler Abbreviated Scale of Intelligence

The Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) is a brief measure of intelligence that can be administered in a four-form or a two-form

version. Both versions provide a Full-Scale IQ score. The two-form version, consisting of Vocabulary and Matrix Reasoning, was administered in this study to provide an estimate of general intellectual ability. The WASI was standardized on a stratified random sample of persons aged 6 to 89 years. It has established validity and reliability, with internal consistency reliability ranging from .92 to .98 and test-retest reliability ranging from .85 to .88 on a mean interval of 31 days. Administration took approximately 15 minutes.

Weschler Intelligence Scale for Children IV

The Weschler Intelligence Scale for Children IV (WISC-IV; Pearson, 2003) is a measure of intellectual ability for children aged 6:0 to 16:11. The current study included the administration of the Processing Speed cluster subtests—Coding and Symbol Search—to all participants younger than 16 in order to determine each participant's basic processing speed abilities. Both of these subtests are timed and took approximately 4 minutes total to complete. The WISC-IV was standardized on 2200 children ages 6:0 to 16:11. The Coding and Symbol Search subtests have a median internal consistency reliability of .85 and .79, respectively.

Weschler Adult Intelligence Scale IV

The Weschler Adult Intelligence Scale IV (WAIS-IV; Pearson, 2008) is a measure of intellectual ability for persons aged 16:0 to 90:11. The current study included

the administration of the Processing Speed cluster subtests, Coding and Symbol Search, to all participants ages 16:0 to 18:11 in order to determine each participant's basic processing speed abilities. Both of these subtests are timed and took approximately 4 minutes total to complete. The WAIS-IV was standardized on a sample of 2200 persons ages 16:0 to 90:11. The Coding and Symbol Search subtests have a median internal consistency reliability of .86 and .81, respectively.

PROCEDURES

Participant Recruitment

Approval to conduct the present study was obtained from the Institutional Review Boards (IRB) of the University of Texas Southwestern Medical Center at Dallas (UTSW) and Children's Medical Center of Dallas (CMCD). Scientific merit approval was obtained from the UTSW Simmons Cancer Center's Protocol Review and Monitoring Committee (PRMC). The names of the children who met eligibility criteria were obtained from the Neurosurgery/Neuro-Oncology patient database at the CMCD Center for Cancer and Blood Disorders (CCBD). Parents or legal guardians of the identified children were contacted by phone at least one week prior to their scheduled follow-up visit to CCBD. A description of the study was provided, and the parent or legal guardian was asked if the child is fluent in English and capable of reading at a first-grade level. If the parent or legal guardian of an eligible child expressed interest in having his or her child participate in the study, the child typically was enrolled upon

arrival for his or her follow-up visit to CCBD. Nine children participated in the study outside of their follow-up visits to CCBD due to scheduling conflicts; eight of these children were enrolled and participated in the study at the Neuropsychology Department at CMCD, and one child was enrolled and participated at the Psychology Department at Children's Medical Center at Legacy. Written, informed consent from the parent and assent of the minor child were obtained at the time of enrollment and participation.

In order to obtain consent and assent, the examiner provided the parent(s) or guardian(s) and the child with a full description of the study via a detailed discussion of the IRB-approved informed consent document. The study's purpose, benefit, and potential risks were described. The consent form is included in Appendix C. Parents and children were encouraged to ask questions regarding their participation in the study. They were informed that their participation was voluntary and that they might withdraw from the study at any time. After answering any and all questions, the examiner obtained informed, written consent from the parent(s). The examiner then verified that the child understood his or her role in the study. Informed, written assent from the child stating an understanding of his or her voluntary participation was obtained by the examiner. The parent(s) then completed the Health Insurance Portability and Accountability Act (HIPAA) Release document, which authorizes the use of the child's private health information for the study. Signed copies of the consent form and the HIPAA Release form were provided to the parent(s), CMCD Medical Records, and the departments of Neurosurgery/Neuro-Oncology. Children were enrolled in the study upon completion of the informed consent process.

Randomization

Patients that met inclusion criteria were randomized to an incentive condition or a no-incentive condition. Approximately half received a monetary incentive for performance on the post-test (incentive condition). The other half completed the post-test without any monetary incentive (no-incentive condition). A stratified randomization procedure was used. Separate randomization lists were constructed for males and females. Each randomization list used random permuted blocks. Block sizes varied and were determined by the statistician.

Data Collection

Each subject completed the study in a single session lasting approximately 90 minutes, conducted by an examiner trained in the administration of the test battery. During the session, a parent or legal guardian completed the patient history form, CBCL, and BRIEF. Each session proceeded as follows: (1) a self-report of situational cognitive fatigue on a visual analog scale, repeated four times throughout the session; (2) administration of Form A of the WJ-ACH Basic Math Calculation (paper-and-pencil math test) and Letter-Word Identification (oral reading of individual words) subtests; (3) WJ-ACH Form A Reading Fluency (rapid comprehension of simple sentences) and Math Fluency (rapid calculation of single-digit addition, subtraction, and multiplication) subtests; (4) WJ-COG cognitive fluency subtests (Retrieval Fluency, Decision Speed, and Rapid Picture Naming); (5) WASI two-subtest form, unless IQ testing had been

conducted within 6 months prior, in which case the General Ability Index (GAI) derived from the previously obtained IQ score was used; (6) WISC-IV or WAIS-IV processing speed subtests (Coding and Symbol Search); (7) a five-minute break with the opportunity to use the restroom if desired; (8) Form B WJ-ACH Reading Fluency and Math Fluency subtests. The time elapsed from step 1 through step 8 was recorded. Prior to the administration of Form B of the WJ-ACH Reading Fluency and Math Fluency (step 8), each subject in the incentive condition was informed that he or she would receive $1 per every 10 items correct, up to $10 per test, on the next two tests. This information was presented with a standardized script and included a fidelity check to ensure the subject understood the incentive. Subjects in the no-incentive condition did not receive this information. All subjects ultimately were compensated equally. At the conclusion of the session, each subject completed a BASC-2 self-report form.

Statistical Analyses

Data were double-entered into a Predictive Analytics Software (PASW) 18.0 database and were checked for potential outliers, normality of distribution, linearity, homoscedasticity, and homogeneity. Linearity among dependent variables and between covariates and dependent variables was evaluated via visual examination of scatterplots. The Shapiro-Wilk test was conducted to assess normality of distribution. The assumptions of homoscedasticity and homogeneity of variance were evaluated with the Box's M test and Levene's test, respectively. For data that did not meet the assumption of homogeneity of variance, appropriate data transformations (e.g., logarithmic and

square root) were applied depending on the characteristics of the non-normal distribution (Tabachnick & Fidell, 2007). When statistical assumptions were not met, even after appropriate data transformations were attempted, and when violations of such assumptions were identified in the literature as creating an invalid statistical test, equivalent non-parametric tests were applied (e.g., Mann-Whitney U).

Prior to covariate selection, candidate variables were identified based on their perceived potential as predictors of performance, their relevance to the stated hypotheses, and their ecological validity (i.e., common availability and usefulness) in research and clinical settings. In order to narrow the field to the most promising predictors while minimizing problems with multicollinearity, a full correlation matrix was developed employing these covariates. As both continuous and dichotomous variables were involved, correlational procedures included Pearson product-moment, phi, biserial, and point-biserial correlations. Variables identified as possible covariates were then analyzed with stepwise multiple regression to determine their inclusion as covariates in the analysis of hypothesis 1A.

Hypotheses 1A and 1B were explored with two-group analysis of variance (ANOVA) and analysis of covariance (ANCOVA), respectively, run with Bonferroni corrections to control for Type I errors. Repeated-measures ANOVAs were conducted to confirm findings of two-group ANOVAs or ANCOVAs. As previously mentioned, equivalent nonparametric tests were applied when statistical assumptions were not met. Analysis of hypothesis 2A was conducted with pairwise t-tests. Exploratory analyses and analysis of hypothesis 2B were conducted with linear regression.

Calculation of Reliable Change Scores

ANOVAs and ANCOVAs for Hypotheses 1A and 1B were conducted two ways—using the basic change score (post-test minus pre-test) for Reading Fluency or Math Fluency as the dependent variable and, alternately, using the reliable change (RC) score, a z-score. First developed by Jacobson and Truax (1991), RC scores are algebraically manipulated calculations of the difference between a pre-test and a post-test score, accounting for the standard deviation of the normal population and the test-retest reliability of the measure. Since the development of Jacobson and Truax's basic algebraic equation for calculating RC scores, a plethora of variations on that equation has been developed, primarily sparked by the fact that the original equation did not account for practice effects (Hinton-Bayre, 2010). Fundamentally, all RC equations attempt to determine the statistical significance of a difference in test scores while controlling for practice effects or poor reliability of the measure.

A recent comparison of all existing models for calculating RC scores determined that "it is still unclear whether a particular model is to be preferred" (Hinton-Bayre, 2010). However, some guidelines have been provided for selection of a model. The most commonly used models in neuropsychological research are those developed by Chelune, Naugle, Luders, Sedlak, and Awad (1993) and by McSweeny, Naugle, Chelune, and Luders (1993). Those two models have been shown to perform similarly (Heaton, Temkin, & Dikmen, 2001). Thus, given the lack of consensus on a preferred model, the Chelune et al. (1993) model (Figure 1) was selected for use in the present study because it appears to be a more conservative model (Hinton-Bayre, 2010), making it more

appropriate for a study with a small sample size. The extensive debate regarding the various RC models is the reason the analyses for the present study were calculated twice, once with RC scores and once with basic (non-adjusted) change scores.

Figure 1
Equation for Calculation of RC Scores

$$RC = \frac{Y - Y'}{SE}$$

Y = individual's actual post-test score
Y' = individual's predicted score (see below)
SE = standard error expression (see below)

Predicted score (Y')	Components
$X + (M_Y - M_X)$	X = individual's initial test score, M_X = control group initial test mean, M_Y = control group retest mean

Standard error (SE) expression	Components
$SE_{diff} = \sqrt{2S_X^2(1 - r_{XY})}$	S_X = control group initial test standard deviation, S_Y = control group retest standard deviation, r_{XY} = Pearson's correlation coefficient for initial and retest score

Regardless of the equation used to calculate it, an RC score is typically considered to represent meaningful change if it exceeds ± 1.645, corresponding to a 90% level of confidence (Hinton-Bayre, 2010). When calculated using the Chelune et al. (1993) model, an RC score exceeding ± 1.645 represents change beyond that which might be expected due to practice effects or random variance.

For the present study, each participant's scores on Forms A and B of the Math and Reading Fluency subtests of the WJ-ACH were used to calculate an RC score according to the Chelune et al. (1993) model. Those scores were then used in the

analysis of hypothesis 1A with ANOVAs and the analysis of hypothesis 1B with ANCOVAs.

CHAPTER FOUR
Results

DEMOGRAPHIC CHARACTERISTICS

Thirty participants recruited from the departments of Neurosurgery and Neuro-Oncology at Children's Medical Center in Dallas met inclusion criteria for and were enrolled in this study. Medical and demographic information is provided below in Tables 1, 2, and 3. Per the inclusion criteria, all subjects were treated with surgery, radiation, and chemotherapy. Mean age at evaluation for the overall sample was 13.99 years (SD = 3.46), with ages ranging from 7.75 to 18.75 years. Mean age at evaluation for the no-incentive group was 12.89 years (SD = 3.24), with ages ranging from 7.75 to 18.5 years. Mean age at evaluation for the incentive group was was 15.08 years (SD = 3.43), with ages ranging from 9.33 to 18.75 years. Independent t-tests and chi square tests indicated that the two groups did not significantly differ on any medical or demographic variables except for age at diagnosis and age at surgery (p=.03). However, stepwise linear regression indicated that neither variable met criteria for inclusion as a covariate.

Table 1
Medical Characteristics of Sample

	No Incentive (n=15)		Incentive (n=15)		Overall Sample (n=30)	
	n	**%**	**n**	**%**	**n**	**%**
Type of resection						
Gross total	8	53.3	12	80.0	20	66.7
Subtotal	7	46.7	3	20.0	10	33.3
Complications						
Hydrocephalus	14	93.3	11	73.3	25	83.3
Ventriculostomy	13	86.7	9	60.0	22	73.3
Shunt	2	13.3	0	0.0	2	6.7

Table 1 (Cont.)
Medical Characteristics of Sample (Cont.)

	No Incentive (n=15)		Incentive (n=15)		Overall Sample (n=30)	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Age at diagnosis	7.00	3.54	10.57	4.78	8.79	4.51
Age at surgery	7.01	3.54	10.58	4.78	8.79	4.51
Months post surgery	70.52	53.78	54.21	34.27	62.36	45.08
Craniospinal radiation	2912.86	690.27	2672.00	645.42	2788.28	666.71
PF radiation boost[a]	2545.71	646.53	2762.67	621.24	2657.93	631.83
Total radiation dose	5430.67	184.84	5434.67	82.28	5432.67	140.59
Months post radiation	67.53	53.16	51.80	33.87	59.67	44.52
Months post chemo	57.27	52.60	40.90	34.16	49.08	44.37

Note: Ages are given in years; radiation doses are given in centigray (cGy).
[a]PF = posterior fossa.

Table 2
Demographic Characteristics of Sample

	No Incentive (n=15)		Incentive (n=15)		Overall Sample (n=30)	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Age at evaluation	12.89	3.24	15.08	3.43	13.99	3.46
	n	**%**	**n**	**%**	**n**	**%**
Gender						
Male	11	73.3	11	73.3	22	73.3
Female	4	26.7	4	26.7	8	26.7
SES						
Low	1	6.7	3	20.0	4	13.3
Lower Middle	0	0.0	2	13.3	2	6.7
Middle	2	13.3	2	13.3	4	13.3
Upper Middle	7	46.7	5	33.3	12	40.0
High	5	33.3	3	20.0	8	26.7
Ethnicity						
Caucasian	8	53.3	6	40.0	14	46.7
African-American	4	26.7	4	26.7	8	26.7
Hispanic	1	6.7	4	26.7	5	16.7
Asian-American	2	13.3	0	0.0	2	6.7
Other	0	0.0	1	6.7	1	3.3

NOTE: Age at evaluation is given in years.

Table 3
Academic Characteristics of Sample

	No Incentive (n=15)		Incentive (n=15)		Overall Sample (n=30)	
	n	**%**	**n**	**%**	**n**	**%**
History of ECI[a]	3	20.0	1	6.7	4	13.3
History of PPCD[b]	0	0	0	0	0	0
Grade retention	4	26.7	5	33.3	9	30.0
Special education[c]	11	73.3	9	60	20	66.7
Section 504 plan[c]	4	26.7	4	26.7	8	26.7
English performance[d]						
Failing	0	0.0	0	0.0	0	0.0
Below Average	4	26.7	3	20.0	7	23.3
Average	7	46.7	8	53.3	15	50.0
Above Average	4	26.7	4	26.7	8	26.7
Math performance[d]						
Failing	0	0.0	0	0.0	0	0.0
Below Average	5	33.3	4	26.7	9	30.0
Average	9	60.0	5	33.3	14	46.7
Above Average	1	6.7	6	40.0	7	23.3
Science performance[d]						
Failing	0	0.0	0	0.0	0	0.0
Below Average	4	26.7	4	26.7	8	26.7
Average	8	53.3	6	40.0	14	46.7
Above Average	1	6.7	5	33.3	7	23.3
History performance[d]						
Failing	0	0.0	0	0.0	0	0.0
Below Average	4	26.7	3	20.0	7	23.3
Average	6	40.0	7	46.7	13	43.3
Above Average	5	33.3	5	33.3	10	33.3

Note: All participants with a history of grade retention were retained one year, with the exception of one participant in the no-incentive group, who was retained two years.
[a]ECI = Early Childhood Intervention.
[b]PPCD = Preschool Programs for Children with Disabilities.
[c]The special education and Section 504 variables indicated whether or not a child was receiving special education services or Section 504 accomodations, respectively, at the time of the evaluation.
[d]Parent-reported academic performance. For science performance, overall n=29, as one child was not enrolled in science classes.

Descriptive analyses of neuropsychological characteristics of the sample are detailed in Table 4 below. It should be noted that FSIQ-2 was calculated for 23 (77%) of

the participants, whereas 7 (23%) participants had been administered a WISC-IV or WAIS-IV within the past six months. Per study protocol, the latter participants were not administered the WASI to obtain the FSIQ-2 score. Instead, the General Ability Index (GAI) was calculated based on their index scores from the previous WISC-IV or WAIS-IV administration. The GAI was calculated because it excludes processing speed and working memory subtests, which are not captured in the FSIQ-2, thus making it a more direct comparison to the FSIQ-2. There is no established method for deriving comparisons between the FSIQ-2 and the FSIQ from the WISC-IV or WAIS-IV.

Table 4
Neuropsychological Characteristics of Sample

	No Incentive (n = 15)		Incentive (n = 15)		Overall Sample (n = 30)	
Variable	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
FSIQ-2 / GAI	88.93	13.76	90.60	13.63	89.77	13.48
Processing Speed Index	78.73	12.61	76.13	15.19	77.43	13.78
Cognitive Fluency	69.80	16.63	75.07	15.06	72.43	15.82
WJ-III Achievement						
Letter-Word	91.53	18.31	89.00	15.94	90.27	16.92
Calculation	86.73	24.75	86.73	18.22	86.73	21.35
Reading Fluency A	82.33	13.79	77.33	12.74	79.83	13.29
Reading Fluency B	82.07	14.40	81.07	14.68	81.57	14.29
Math Fluency A	77.00	17.37	74.80	14.57	75.90	15.79
Math Fluency B	77.53	18.55	79.20	14.89	78.37	16.55
BASC-2						
Attitude to School	48.53	10.04	55.07	13.98	51.80	12.41
Attitude to Teachers	47.27	14.03	50.00	11.04	48.63	12.48
School Problems	45.57	10.15	52.80	12.07	49.31	11.58
Locus of Control	49.79	7.42	49.27	8.10	49.52	7.65
Self Esteem	51.14	9.11	52.87	9.76	52.03	9.32
Self Reliance	45.29	6.97	47.13	12.24	46.24	9.92
BRIEF						
Initiate	50.07	10.57	51.80	11.49	50.93	10.89
Inhibit	46.33	9.12	48.40	5.96	47.37	7.65
Plan/Organize	48.67	10.87	55.27	13.55	51.97	12.52
CBCL						
Cognitive Tempo	52.53	4.61	59.73	8.93	56.13	7.89

Mean FSIQ-2/GAI for the overall sample was 89.77 (SD = 13.48), with scores ranging from 58 to 113. Mean FSIQ-2/GAI for the no-incentive group was was 88.93 (SD = 13.76), with scores ranging from 58 to 109. Mean FSIQ-2/GAI for the incentive group was 90.60 (SD = 13.63), with scores ranging from 66 to 113. Independent t-tests and chi square tests indicated that incentive and no-incentive groups did not significantly differ on any neuropsychological characteristics.

For the overall sample, mean performance on a measure of basic reading skills was average, while mean performance on a measure of basic mathematics skills was low average. Mean performance on measures of reading and math fluency were below average for the overall sample. For the incentive group, mean reading fluency performance improved from below average to low average, with scores ranging from 56 to 96 on Form A and ranging from 58 to 109 on Form B. Mean math fluency performance for the incentive group was in the below average range at both timepoints, with scores ranging from 55 to 104 on Form A and ranging from 60 to 109 on Form B.

HYPOTHESES

Specific Aim 1

Hypothesis 1A

This hypothesis stated that external incentives would significantly improve the performance of childhood medulloblastoma survivors on academic fluency tasks.

After performing initial data checks and prior to conducting statistical analyses of this hypothesis, a correlation matrix was developed as described previously, using both basic change scores and RC scores for both math and reading fluency change. To calculate the RC scores, Pearson's correlation coefficients were calculated for initial and retest scores. The Pearson's correlation coefficient for reading fluency was 0.952, while the Pearson's correlation coefficient for math fluency was 0.964. Using standard deviations for mean fluency scores of the control (no incentive) group, the standard error (SE) expression was calculated. The SE expression for reading fluency was 4.27, while the SE expression for math fluency was 4.66. The equation represented previously in Figure 1 was then used to calculate each individual participant's math fluency and reading fluency RC scores.

Relevant findings of the correlational procedures are illustrated in Table 5. Pearson's correlation coefficient (r) is presented for parametric variables, while Spearman's correlation coefficient (rho) is presented for non-paramatric variables. Statistics for both basic change and RC change scores were identical; thus, only statistics for RC scores are presented here.

Table 5
Correlation Matrix for Academic Fluency

	Reading Fluency RC		Math Fluency RC	
Variable	**r/rho**	**p**	**r/rho**	**p**
Testing variables				
Location Tested	.068	.72	.013	.95
Age Tested	.269	.15	.112	.56
Grade Tested	.306	.10	.136	.47
Demographics				
Gender	-.035	.85	.206	.28
Ethnicity	.049	.80	.050	.79
FSIQ	.382	.04*	.329	.08
SES	.054	.78	-.119	.53

Table 5 (Cont.)
Correlation Matrix for Academic Fluency (Cont.)

	Reading Fluency RC		Math Fluency RC	
Variable	**r/rho**	**p**	**r/rho**	**p**
Demographics				
Years ECI	-.318	.09	-.110	.56
Years Retained	-.178	.35	-.138	.47
Medical				
Age at Diagnosis	.442	.01*	.229	.22
Age at Surgery	.444	.01*	.229	.22
Type of Resection	-.279	.14	-.201	.29
Months Post Surgery	-.282	.13	-.169	.37
Months Post Radiation	-.277	.14	-.166	.38
Total Radiation	.102	.60	-.290	.12
Months Post Chemo	-.287	.12	-.177	.35
Hydrocephalus	-.088	.64	-.005	.99
Ventriculostomy	.053	.78	-.131	.49
Shunt	-.202	.29	-.287	.12
Processing Speed	.097	.61	.211	.26
Cognitive Fluency	.341	.07	.285	.13
WJ-III Achievement				
Letter-Word	.267	.15	.084	.66
Calculation	.324	.08	.178	.35
BASC-2				
Attitude to School	.199	.29	.058	.76
Attitude to Teachers	-.015	.94	-.029	.88
School Problems	.187	.33	.173	.37
Locus of Control	-.127	.51	.093	.63
Self Esteem	.091	.64	-.106	.58
Self Reliance	.204	.29	.136	.48
BRIEF				
Initiate	-.031	.87	-.008	.97
Inhibit	-.094	.62	-.126	.51
Plan/Organize	-.152	.42	-.081	.67
CBCL				
Cognitive Tempo	.341	.07	.285	.13

Significant correlations indicated a few potential covariates, which were then entered as predictors into stepwise multiple regression models. Results of those stepwise regression analyses indicated that none of the variables identified by the correlation matrix met criteria to be included as covariates in the analyses of hypothesis 1A.

Thus, analysis of hypothesis 1A was conducted with two-group ANOVAs. Those ANOVAs were run in three different ways. First, an ANOVA was run using the RC change scores for both math and reading fluency. There was a significant effect for incentive with regard to reading fluency change ($F = 7.74$, $p = .01$). The effect size was large (partial $\eta^2 = .217$), with an observed power of .766. With regard to math fluency change, there was a significant effect for incentive ($F = 6.895$, $p = .01$). The effect size was large (partial $\eta^2 = .198$), with an observed power of .717. However, Levene's test indicated that the variances of the Math Fluency basic and RC change scores were not homogeneous, thus violating one of the assumptions for analysis of variance (ANOVA). Various appropriate data transformations (logarithmic, square root, and z-score) were applied but did not correct this issue. Therefore, an equivalent non-parametric test, the Mann-Whitney U Test, was used to test for significant differences in mean Math Fluency change between the incentive and no-incentive groups. The Mann-Whitney U analysis supported the results of the ANOVA for math fluency change, indicating a significant effect for incentive ($p = .03$). ANOVAs using basic change scores for both math and reading fluency also confirmed the findings of the ANOVAs using the RC change scores, with identical statistical outcomes. In the process of running the ANOVAs, mean change scores for the two groups on both reading and math fluency were calculated; see Table 6.

Table 6
Mean Change Scores

	Reading Fluency		Math Fluency	
Variable	**Mean**	**SD**	**Mean**	**SD**
Basic change scores				
No incentive	-0.27	2.71	0.53	5.139
Incentive	3.73	4.86	4.40	2.473
RC scores				
No incentive	-0.01	0.64	0.01	1.10
Incentive	0.94	1.14	0.83	0.53

As reported in Table 4, the mean scores for math and reading fluency at baseline differed between the incentive and no-incentive groups. To investigate any possible impact of the differences between the two groups on baseline fluency means, the same ANOVAs were repeated with baseline scores entered as a covariate. For reading fluency change, there remained a significant effect for incentive ($F = 8.531$, $p < .01$) with a large effect size (partial $\eta^2 = .240$) and observed power of .804. Likewise, for math fluency change, there remained a significant effect for incentive ($F = 6.798$, $p = .02$) with a large effect size (partial $\eta^2 = .201$) and observed power of .710.

Finally, repeated-measures ANOVAs were performed using the baseline and post-test fluency scores. Findings were identical to the outcomes of the two-group ANOVAs. That is, there was a significant effect for incentive with regard to reading fluency change ($F = 7.74$, $p = .01$), associated with a large effect size (partial $\eta^2 = .217$) and an observed power of .766. With regard to math fluency change, there was a significant effect for incentive ($F = 6.895$, $p = .01$), associated with a large effect size (partial $\eta^2 = .198$) and an observed power of .717. Plots were created to demonstrate the changes in fluency scores for each group (see Figures 2 and 3).

Figure 2
Change in Math Fluency Scores

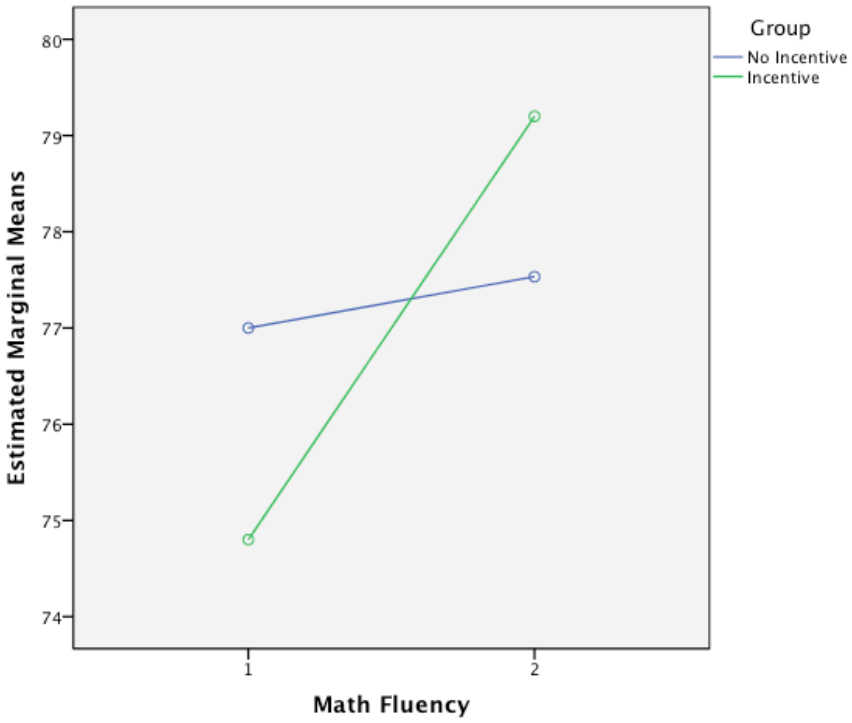
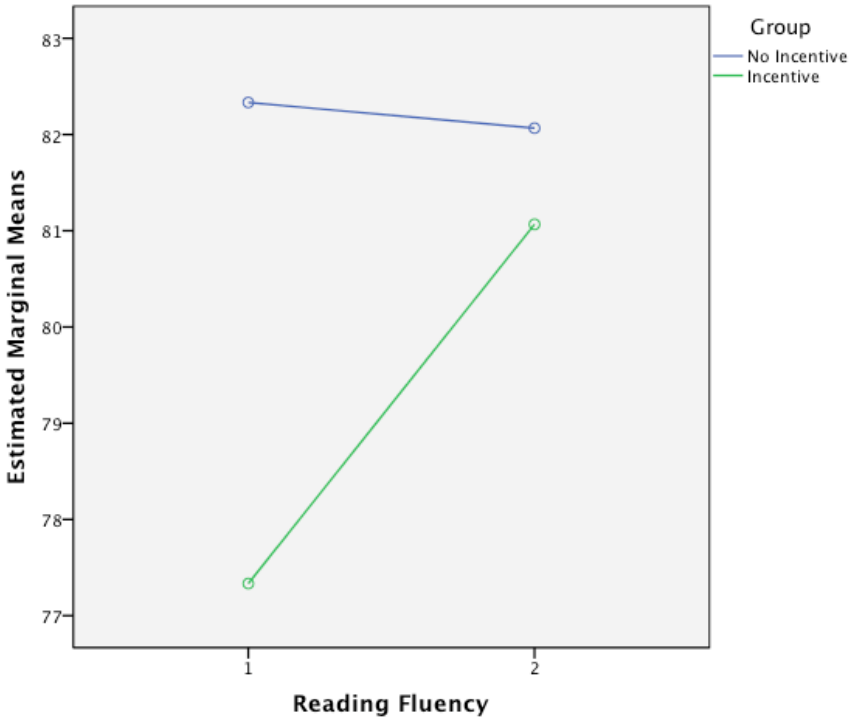


Figure 3
Change in Reading Fluency Scores



In summary, hypothesis 1A was supported via multiple methods of statistical analysis. Findings indicated that external incentives significantly improved the performance of childhood medulloblastoma survivors on academic fluency tasks. This was true for both math fluency and reading fluency.

Hypothesis 1B

It was predicted that performance on measures of cognitive fluency and processing speed would influence the impact of external incentives on academic fluency performance in childhood medulloblastoma survivors. As an initial step in this investigation, ANOVAs were run to determine whether the incentive and no-incentive groups differed with respect to performance on processing speed and cognitive fluency measures. The groups did not differ with respect to processing speed ($F = .260$, $p = .61$) or cognitive fluency ($F = .827$, $p = .37$).

Subsequently, the analyses described for the investigation of hypothesis 1A were repeated with processing speed and cognitive fluency as covariates, alternately. An analysis of covariance (ANCOVA) using the RC change scores for both math and reading indicated that neither processing speed nor cognitive fluency significantly contributed to the model. That is, neither processing speed nor cognitive fluency significantly influenced the effect of external incentives on academic fluency performance. This finding was confirmed with repeated-measures ANOVA. See Tables 7 and 8 for a description of the findings.

Table 7
Analyses of Processing Speed as Covariate

	Reading Fluency		Math Fluency	
Measure	**F**	**p**	**F**	**p**
ANCOVA				
with RC Score				
covariate	0.717	.41	2.377	.14
group	8.050	< .01*	7.982	< .01*
with Basic Change Score				
covariate	0.717	.41	2.377	.14
group	8.050	< .01*	7.982	< .01*
Repeated Measures ANOVA				
covariate	0.717	.41	2.377	.14
group	8.050	< .01*	7.982	< .01*

Table 8
Analyses of Cognitive Fluency as Covariate

	Reading Fluency		Math Fluency	
Measure	**F**	**p**	**F**	**p**
ANCOVA				
with RC Score				
covariate	2.691	.11	1.620	.21
group	6.485	.02*	5.765	.02*
with Basic Change Score				
covariate	2.691	.11	1.620	.21
group	6.485	.02*	5.765	.02*
with Rank Transformed Scores				
covariate	N/A	N/A	3.001	.10
group	N/A	N/A	4.680	.04*
Repeated Measures ANOVA				
covariate	2.691	.11	1.620	.21
group	6.485	.02*	5.765	.02*

It should be noted that although Levene's Test was not significant (p = .08) for the ANCOVA of math fluency with processing speed as a covariate, Levene's Test was significant (p = .03) for the ANCOVA of math fluency with cognitive fluency as a covariate. This indicated a violation of the assumption of homogeneity of variances for that analysis. Since the Mann-Whitney U Test cannot be run with covariates, the data for

math fluency change was rank transformed and subsequently analyzed with an ANCOVA. This is the recommended method for running an analysis of covariance (ANCOVA) on data that violates the assumption of homogeneity of variances (Conover & Iman, 1982). Levene's Test was not significant ($p = .13$) for the ANCOVA of the rank transformed math fluency change data with cognitive fluency as covariate.

In summary, hypothesis 1B was not supported via multiple methods of statistical analysis. Findings indicated that neither processing speed nor cognitive fluency significantly influenced the effect of external incentives on academic fluency performance. This was true for both math fluency and reading fluency.

Specific Aim 2

Hypothesis 2A

This hypothesis stated that survivors of childhood medulloblastoma would display a relative weakness in fluent academic performance as compared to academic skill development. Paired sample t-tests identified a significant difference between reading fluency and basic reading skills ($p < .01$). Consistent with the hypothesis, analysis of means to determine directionality indicated a relative weakness in reading fluency as compared to basic reading skills (see Table 9). Calculation of Cohen's d indicated a medium to large effect size of .69 for the difference between reading fluency and skills. Paired sample t-tests also identified a significant difference between mathematics fluency and basic mathematics skills ($p < .01$). Analysis of means to

determine directionality indicated a relative weakness in mathematics fluency as compared to basic mathematics skills (see Table 9). Calculation of Cohen's d indicated a medium effect size of .58 for the difference between math fluency and skills.

Table 9
Means of Academic Fluency and Skills Measures

	Overall Sample	
Measure	**Mean**	**SD**
Letter-Word Identification	90.27	16.92
Reading Fluency	79.83	13.29
Calculation	86.73	21.35
Math Fluency	75.90	15.79

In summary, statistical analysis with paired sample t-tests supported hypothesis 2A. Survivors of childhood medulloblastoma displayed a relative weakness in fluent academic performance as compared to basic academic skill development. This was true for both reading and mathematics.

Hypothesis 2B

It was predicted that performance on measures of cognitive fluency would better predict fluent academic performance in childhood medulloblastoma survivors than performance on measures of processing speed. As in the investigation of hypothesis 2A, statistical analyses of hypothesis 2B were run on Form A of the Reading Fluency and Math Fluency subtests, rather than Form B, in order to eliminate any possible influence of practice effects and/or incentive.

Multiple linear regression analyses conducted with simultaneous entry indicated that, contrary to the stated hypothesis, performance on measures of processing speed accounted for more variance in the prediction of fluent academic performance in childhood medulloblastoma survivors. This finding was consistent both for math and reading fluency. Processing speed significantly accounted for 45% of the variance in fluent math performance [$F(2,27) = 11.203$, $p = .01$] and 55% of the variance in fluent reading performance [$F(2,27) = 16.752$, $p < .01$]. In contrast, cognitive fluency did not significantly contribute to the predictive value of the regression models for fluent math performance ($p = .32$) or fluent reading performance ($p = .22$).

In summary, hypothesis 2B was not supported by linear regression analyses. Findings indicated that performance on measures of processing speed better predicted fluent academic performance in childhood medulloblastoma survivors than performance on measures of cognitive fluency. This was true for both reading and math fluency.

EXPLORATORY ANALYSES

Academic Fluency and Skills

Single linear regression analyses were performed to test whether individual risk factors predicted performance on measures of academic fluency and skills among childhood medulloblastoma survivors. Findings are presented in Tables 10 through 21. For this portion of the exploratory analyses, the entire sample was analyzed as a whole, without division into groups. Since analysis of predictors for fluent academic

performance was performed using Form A of the fluency subtests of the WJ-ACH, there was no rationale for dividing the sample into groups for analysis. Likewise, analysis of risk factors predicting academic skill development was performed using the Calculation and Letter-Word Identification subtests, which were administered prior to any incentive.

Table 10
Medical Factors Predicting Academic Fluency Performance

	Math Fluency			Reading Fluency		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
Age at diagnosis	0.436	.51	.124	0.005	.94	.014
Age at surgery	0.426	.52	.122	0.006	.94	.014
Months post surgery	3.708	.06	.342	1.598	.22	.232
Type of resection	0.961	.34	.182	0.690	.41	.155
Complications						
Hydrocephalus	0.523	.48	.135	0.218	.64	.088
Ventriculostomy	1.814	.19	.247	0.081	.78	.054
Shunt	0.528	.47	.136	0.222	.64	.089
Craniospinal radiation	0.060	.81	.047	0.490	.49	.134
PF radiation boost	0.050	.82	.043	0.275	.60	.100
Total radiation dose	2.031	.17	.260	4.761	.04*	.381
Months post radiation	3.841	.06	.347	1.674	.21	.238
Months post chemo	3.935	.06	.351	1.850	.19	.249

In a single linear regression, total radiation dose significantly predicted variance in reading fluency performance ($p = .04$). Because total radiation dose was significantly correlated with craniospinal radiation ($p = .05$), those two variables were then entered into a stepwise multiple regression. Both variables were excluded from the analysis, indicating that total radiation dose did not significantly predict variance in reading fluency performance when considered together with craniospinal radiation.

Table 11
Medical Factors Predicting Academic Skill Development

	Calculation			Letter-Word Identification		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
Age at diagnosis	1.883	.18	.251	3.259	.08	.323
Age at surgery	1.894	.18	.252	3.267	.08	.323
Months post surgery	5.279	.03*	.398	6.820	.01*	.443
Type of resection	0.010	.92	.019	1.130	.30	.197
Complications						
Hydrocephalus	0.727	.40	.159	1.070	.31	.192
Ventriculostomy	0.594	.45	.144	0.006	.94	.014
Shunt	0.696	.41	.156	1.132	.30	.197
Craniospinal radiation	2.252	.15	.277	1.566	.22	.234
PF radiation boost	2.203	.15	.275	1.714	.20	.244
Total radiation dose	2.000	.17	.258	0.426	.52	.122
Months post radiation	5.317	.03*	.399	6.777	.02*	.441
Months post chemo	5.799	.02*	.414	7.411	.01*	.457

In a single linear regression, months post chemotherapy significantly predicted variance in performance on measures of math ($p = .02$) and reading ($p = .01$) skill development. Because months post chemotherapy was significantly correlated with months post surgery ($p < .01$) and months post radiation ($p < .01$), all three variables were then entered into a stepwise multiple regression for both math and reading skill performance, respectively. Months post surgery and months post radiation were excluded from the analysis, indicating that months post chemotherapy was responsible for the majority of the variance in both math skill development [$F(1,28) = 5.799$, $p = .02$, $R = 0.414$] and reading skill development [$F(1,28) = 7.411$, $p = .01$, $R = .457$]. The relationship was such that as months post chemotherapy increased, performance on measures of academic skill development decreased—a clear demonstration of late effects.

Table 12
Demographic Factors Predicting Academic Fluency Performance

	Math Fluency			Reading Fluency		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
Testing location	0.054	.82	.044	0.149	.70	.073
Age at evaluation	11.167	< .01*	.534	1.673	.21	.237
Grade at evaluation	7.186	.01*	.452	1.083	.31	.193
SES	0.105	.75	.061	0.998	.33	.186
Gender	0.775	.39	.164	1.688	.20	.238
Ethnicity	0.293	.59	.102	1.600	.22	.233
Years of ECI	0.014	.91	.023	0.242	.63	.093
Years retained	0.591	.45	.144	1.624	.21	.234
Special education	2.179	.15	.269	2.906	.10	.307
Section 504 accomodations	0.923	.35	.179	1.143	.29	.198

In single linear regressions, variance in math fluency was significantly predicted by both age at evaluation ($p < .01$) and grade at evaluation ($p = .01$) such that greater age or grade predicted worse math fluency. Because age at evaluation was significantly correlated with grade at evaluation ($p < .01$), both variables were then entered into a stepwise multiple regression for math fluency (see Table 13).

Table 13
Stepwise Regression for Math Fluency Performance

	Math Fluency		
Variable	**F**	**p**	**R**
Model 1			
Age at evaluation	11.167	.002	.534
Model 2			
Age at evaluation	11.426	.001	.677
Grade at evaluation	11.426	.007	.677

The results of the stepwise multiple regression shown in Table 13 indicate that both age at evaluation and grade at evaluation significantly predicted math fluency, although age at evaluation appears to be the better predictor. As in the single linear regression, greater age predicted worse performance on the measure of math fluency.

Table 14
Demographic Factors Predicting Academic Skill Development

	Calculation			Letter-Word Identification		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
Testing location	0.001	.97	.007	0.005	.94	.014
Age at evaluation	0.327	.57	.107	0.105	.75	.061
Grade at evaluation	0.012	.91	.021	0.049	.83	.042
SES	0.144	.71	.071	1.127	.30	.197
Gender	0.010	.92	.018	0.897	.35	.176
Ethnicity	0.914	.35	.178	0.944	.34	.181
Years of ECI	0.001	.98	.005	0.317	.58	.106
Years retained	1.715	.20	.240	6.676	.02*	.439
Special education	5.993	.02*	.420	5.786	.02*	.414
Section 504 accommodations	5.078	.03*	.392	5.719	.02*	.412

In single linear regressions, variance in math and reading skill development was significantly predicted by both special education status ($p = .02$ for both) and Section 504 status ($p = .03$ for Calculation and $p = .02$ for Letter-Word Identification). Enrollment in special education predicted worse academic skill performance, whereas receipt of Section 504 accommodations predicted better academic skill performance relative to that of participants not receiving Section 504 accommodations (i.e,. either enrolled in special education or not receiving any services). Since special education and Section 504 statuses significantly correlated with each other ($p < .01$), both variables were then entered into a stepwise linear regression for both math and reading skill development, respectively. Section 504 status was excluded from the equation, indicating that special education status better predicted variance in academic skill development, $F(1,28) = 5.786$, $p = .02$, $R = 414$.

A single linear regression also indicated that years retained significantly predicted performance on a measure of reading skill development. Since years retained was significantly correlated with FSIQ-2/GAI ($p = .02$), both variables were entered into

a stepwise multiple regression for reading skill development. Years retained was excluded from the resulting equation, indicating that FSIQ-2/GAI better predicted variance in reading skill development, $F(1,28) = 32.543$, $p < .01$, $R = .733$.

Table 15
Neuropsychological Factors Predicting Academic Fluency Performance

	Math Fluency			Reading Fluency		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
FSIQ-2 / GAI	8.956	< .01*	.492	24.764	< .01*	.685
Processing Speed Index	21.362	< .01*	.658	31.281	< .01*	.726
Cognitive Fluency	12.450	< .01*	.555	16.974	< .01*	.614
WJ-III Achievement						
Calculation	34.823	< .01*	.745	41.759	< .01*	.774
Letter-Word	13.783	< .01*	.574	26.280	< .01*	.696
BASC-2						
Attitude to School	0.278	.60	.099	0.003	.96	.010
Attitude to Teachers	0.022	.88	.028	2.305	.14	.276
School Problems	0.527	.47	.138	0.163	.69	.077
Locus of Control	1.453	.24	.226	0.011	.92	.020
Self Esteem	4.295	.05*	.370	4.350	.05*	.373
Self Reliance	0.045	.83	.041	0.532	.47	.139
BRIEF						
Initiate	0.218	.64	.088	1.839	.19	.248
Inhibit	0.523	.48	.135	3.696	.07	.341
Plan/Organize	1.269	.27	.208	3.257	.08	.323
CBCL						
Cognitive Tempo	1.571	.22	.231	1.177	.29	.201

Self-esteem as self-reported on the BASC-2 significantly predicted variance in performance on the measures of math and reading fluency. Although self-esteem was significantly correlated with attitude to school ($p = .04$), stepwise multiple regressions with both variables excluded attitude to school from the equation—unsurprisingly, as attitude to school was not a significant predictor for math fluency according to a single linear regression.

Because of significant correlations between FSIQ-2/GAI, Processing Speed Index, Cognitive Fluency, Calculation, and Letter-Word Identification (see Table 16), these variables were entered into a stepwise multiple regression. Although each variable independently predicted both math and reading fluency in single linear regressions, the results of the stepwise multiple regression (see Table 17) excluded all variables except Calculation and Processing Speed Index.

Table 16
Correlation Matrix for Academic Fluency Stepwise Regression

	PSI[a]		Cognitive Fluency		FSIQ-2/ GAI		Calculation		Letter-Word ID[b]	
	r	**p**	**r**	**p**	**r**	**p**	**r**	**p**	**r**	**p**
PSI[a]	--	--	.683	<.01	.581	<.01	.566	<.01	.424	.02
Cognitive Fluency	.683	<.01	--	--	.600	<.01	.585	<.01	.468	<.01
FSIQ-2/GAI	.581	<.01	.600	<.01	--	--	.762	<.01	.733	<.01
Calculation	.566	<.01	.585	<.01	.762	<.01	--	--	.779	<.01
Letter-Word ID[b]	.424	.02	.468	<.01	.733	<.01	.779	<.01	--	--

[a]PSI = Processing Speed Index
[b]Letter-Word ID = Letter-Word Identification

Table 17
Stepwise Regression for Academic Fluency Performance

	Math Fluency			Reading Fluency		
Variable	**F**	**p**	**R**	**F**	**p**	**R**
Model 1						
Calculation	34.823	< .01	.745	41.759	< .01	.774
Model 2						
Calculation	23.626	< .01	.798	34.861	< .01	.849
Processing Speed Index	23.626	.02	.798	34.861	< .01	.849

The results of the stepwise multiple regression shown in Table 17 indicate that both Calculation and Processing Speed Index significantly predicted math fluency, although Calculation appears to be the better predictor. The relationship was such that

better performance on the measure of Calculation and a greater Processing Speed Index predicted better performance on the academic fluency measures.

Table 18
Neuropsychological Factors Predicting Academic Skill Development

	Calculation			Letter-Word Identification		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
FSIQ-2 / GAI	38.859	< .01*	.762	32.543	< .01*	.733
Processing Speed Index	13.232	< .01*	.566	6.149	.02*	.424
Cognitive Fluency	14.539	< .01*	.585	7.837	< .01*	.468
WJ-III Achievement						
Math Fluency	34.823	< .01*	.745	13.783	< .01*	.574
Reading Fluency	41.759	< .01*	.774	26.280	< .01*	.696
BASC-2						
Attitude to School	0.628	.44	.148	0.115	.74	.064
Attitude to Teachers	1.284	.27	.209	6.009	.02*	.420
School Problems	0.127	.73	.068	0.959	.34	.185
Locus of Control	0.012	.92	.021	0.210	.65	.088
Self Esteem	2.934	.10	.313	0.629	.43	.151
Self Reliance	0.000	.99	.002	0.026	.87	.031
BRIEF						
Initiate	0.066	.80	.049	0.078	.78	.053
Inhibit	0.211	.65	.086	0.151	.70	.073
Plan/Organize	1.600	.22	.233	1.145	.29	.198
CBCL						
Cognitive Tempo	0.197	.66	.035	0.026	.87	.057

Because of significant correlations between FSIQ-2/GAI, Processing Speed Index, Cognitive Fluency, Math Fluency, and Reading Fluency (see Table 19), these variables were entered into a stepwise multiple regression. Although each variable independently predicted performance on measures of both math and reading skill development in single linear regressions, the stepwise multiple regression excluded some of these variables (see Tables 20 and 21).

Table 19
Correlation Matrix for Academic Skills Stepwise Regression

	PSI[a]		Cognitive Fluency		FSIQ-2/ GAI		Math Fluency		Reading Fluency	
	r	**p**	**r**	**p**	**r**	**p**	**r**	**p**	**r**	**p**
PSI[a]	--	--	.683	<.01	.581	<.01	.658	<.01	.726	<.01
Cognitive Fluency	.683	<.01	--	--	.600	<.01	.555	<.01	.614	<.01
FSIQ-2/GAI	.581	<.01	.600	<.01	--	--	.492	<.01	.685	<.01
Math Fluency	.658	<.01	.555	<.01	.492	<.01	--	--	.752	<.01
Reading Fluency	.726	<.01	.614	<.01	.685	<.01	.752	<.01	--	--

[a]PSI = Processing Speed Index

Table 20
Stepwise Regression for Calculation

	F	**p**	**R**
Model 1			
Reading Fluency	41.759	< .01	.774
Model 2			
Reading Fluency	31.549	< .01	.837
FSIQ-2/GAI	31.549	< .01	.837
Model 3			
Reading Fluency	28.873	.35	.877
FSIQ-2/GAI	28.873	< .01	.877
Math Fluency	28.873	.01	.877
Model 4			
FSIQ-2/GAI	43.015	< .01	.872
Math Fluency	43.015	< .01	.872

The results of the stepwise multiple regression shown in Table 20 indicate that Reading Fluency, FSIQ-2/GAI, and Math Fluency all significantly predicted Calculation performance, although Reading Fluency appears to be the single best predictor among those. Better performance on all three variables predicted better performance on the measure of math skill development.

Table 21
Stepwise Regression for Letter-Word Identification

	F	p	R
Model 1			
FSIQ-2/GAI	32.543	< .01	.733
Model 2			
FSIQ-2/GAI	20.948	.01	.780
Reading Fluency	20.948	.04	.780

The results of the stepwise multiple regression shown in Table 21 indicate that FSIQ-2/GAI and Reading Fluency both significantly predicted performance on Letter-Word Identification, although FSIQ-2/GAI appears to be the better predictor. Higher FSIQ-2/GAI scores and better reading fluency performance predicted better performance on the measure of reading skill development.

Overall, the exploratory analyses regarding academic fluency and skills indicate that survivors of pediatric medulloblastoma may demonstrate declining performance on measures of basic academic skills and fluency as they grow older. Those enrolled in special education may demonstrate worse academic skill performance. Greater processing speed predicted better academic fluency performance, while greater overall cognitive ability and academic fluency performance predicted better performance on measures of basic academic skill development.

Response to Incentive

In addition to the single and multiple regression analyses described above, single linear regression analyses were also performed to test whether individual risk factors predicted change in academic fluency performance associated with incentive. Findings

were identical for RC and basic change scores; thus, only the findings for RC score analyses are presented here (see Tables 22 through 27). For this portion of the exploratory analyses, simple linear regression analyses were conducted for the incentive and no-incentive groups independently. No analyses were conducted for the overall group given that the difference in experimental conditions meant that such analyses would have been uninformative and potentially misleading.

Table 22
Medical Predictors of Reliable Change in Math Fluency Performance

	No Incentive (n=15)			Incentive (n=15)		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
Age at diagnosis	0.286	.60	.147	2.471	.14	.400
Age at surgery	0.289	.60	.147	2.492	.14	.401
Months post surgery	0.000	.98	.006	2.547	.13	.405
Type of resection	0.005	.94	.020	0.208	.66	.126
Complications						
Hydrocephalus	0.469	.51	.187	1.855	.20	.353
Ventriculostomy	1.054	.32	.274	1.359	.27	.308
Shunt	0.194	.67	.121	N/A	N/A	N/A
Craniospinal radiation	6.918	.02*	.605	0.527	.48	.197
PF radiation boost	4.999	.05*	.542	0.438	.52	.181
Total radiation dose	1.971	.18	.363	0.463	.51	.185
Months post radiation	0.000	.99	.003	2.576	.13	.407
Months post chemo	0.001	.97	.009	2.808	.12	.421

[a]PF = Posterior fossa

In single linear regressions, change in math fluency performance for the no-incentive group was significantly predicted by both craniospinal radiation ($p = .02$) and posterior fossa radiation boost ($p = .05$) such that greater radiation predicted less change in math fluency performance. Since craniospinal radiation and posterior fossa boost were significantly correlated with each other ($p < .01$), both variables were then entered into a stepwise multiple regression for the no-incentive group. Posterior fossa radiation boost

was excluded from the regression equation, indicating that craniospinal radiation was the better predictor of change in math fluency performance for the no-incentive group, $F(1,12) = 6.918$, $p = .02$, $R = .605$. As in the single linear regression, greater craniospinal radiation predicted less change in math fluency performance for the no-incentive group.

Table 23
Medical Predictors of Reliable Change in Reading Fluency Performance

	No Incentive (n=15)			Incentive (n=15)		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
Age at diagnosis	1.157	.30	.286	1.590	.23	.330
Age at surgery	1.131	.31	.283	1.637	.22	.334
Months post surgery	0.080	.78	.078	2.676	.13	.413
Type of resection	0.605	.45	.211	0.295	.60	.149
Complications						
Hydrocephalus	1.096	.31	.279	0.000	.99	.002
Ventriculostomy	1.637	.22	.334	0.626	.44	.214
Shunt	0.159	.70	.110	N/A	N/A	N/A
Craniospinal radiation	0.119	.74	.099	1.208	.29	.292
PF radiation boost	0.068	.80	.075	1.373	.26	.309
Total radiation dose	0.574	.46	.206	0.028	.87	.046
Months post radiation	0.065	.80	.070	2.645	.13	.411
Months post chemo	0.055	.82	.065	2.913	.11	.428

Table 24
Demographic Predictors of Reliable Change in Math Fluency Performance

	No Incentive (n=15)			Incentive (n=15)		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
Testing location	0.009	.93	.027	0.248	.63	.137
Age at evaluation	0.384	.55	.169	0.626	.44	.214
Grade at eval	0.352	.56	.162	0.634	.44	.216
SES	1.677	.22	.338	0.735	.41	.231
Gender	0.787	.39	.239	2.534	.14	.404
Ethnicity	0.000	.99	.005	0.093	.77	.084
Years of ECI	0.038	.85	.054	0.059	.81	.067
Years retained	0.647	.44	.218	0.000	.99	.000
Special education	0.119	.74	.095	0.015	.90	.034
Section 504 plan	0.119	.74	.095	1.086	.32	.278

As reported in Table 25 below, in single linear regressions, change in reading fluency performance for the no-incentive group was significantly predicted by both special education status ($p < .01$) and receipt of Section 504 accommodations ($p < .01$). Specifically, for the no-incentive group, enrollment in special education predicted less change in reading fluency performance, whereas receipt of Section 504 accommodations predicted more change in reading fluency.

Table 25
Demographic Predictors of Reliable Change in Reading Fluency Performance

	No Incentive (n=15)			Incentive (n=15)		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
Testing location	0.000	.99	.003	0.127	.73	.099
Age at evaluation	0.556	.47	.203	0.178	.68	.116
Grade at eval	1.084	.32	.277	0.334	.57	.158
SES	0.291	.60	.148	1.557	.23	.327
Gender	0.038	.85	.054	0.012	.92	.030
Ethnicity	1.044	.33	.273	1.060	.32	.275
Years of ECI	3.452	.09	.458	0.322	.58	.156
Years retained	3.399	.09	.455	0.033	.86	.050
Special education	12.108	< .01*	.694	0.127	.73	.099
Section 504 plan	12.108	< .01*	.694	0.934	.35	.259

Since Section 504 and special education statuses correlated significantly with each other ($p < .01$), both variables were then entered into a stepwise multiple regression for the no-incentive group. Special education status, which independently predicted less change in reading fluency, was excluded from the equation, indicating that Section 504 status was the better predictor for the no-incentive group, $F(1,13) = 12.108$, $p < .01$, $R = .694$. Section 504 accommodations predicted more change in reading fluency.

Table 26
Neuropsychological Predictors of Reliable Change in Math Fluency Performance

	No Incentive (n=15)			Incentive (n=15)		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
FSIQ-2 / GAI	1.962	.19	.362	1.748	.21	.344
Processing Speed Index	2.417	.14	.396	0.385	.55	.170
Cognitive Fluency	0.475	.50	.188	2.286	.15	.387
WJ-III Achievement						
Calculation	0.420	.53	.177	1.066	.32	.275
Letter-Word	0.099	.76	.087	0.895	.36	.254
BASC-2						
Attitude to School	0.622	.44	.214	0.180	.68	.117
Attitude to Teachers	0.067	.80	.071	0.255	.62	.139
School Problems	0.030	.87	.050	0.000	.99	.005
Locus of Control	0.779	.40	.247	0.064	.81	.070
Self Esteem	0.211	.65	.132	1.096	.31	.279
Self Reliance	2.953	.11	.444	0.716	.41	.228
BRIEF						
Initiate	0.286	.60	.147	0.210	.65	.126
Inhibit	0.840	.38	.246	0.141	.71	.104
Plan/Organize	0.913	.36	.256	0.921	.36	.257
CBCL						
Cognitive Tempo	3.136	.10	.419	0.002	.97	.032

As reported in Table 27 below, in single linear regressions, change in reading fluency performance for the no-incentive group was significantly predicted by self-reported attitude toward school ($p = .05$), self-reported school problems ($p = .02$), and parent-reported planning/organizational difficulties ($p = .03$). Worse attitude toward school, school problems, or planning/organizational skills predicted less change in reading fluency performance.

Since attitude toward school, school problems, and parent-reported planning/organizational skills were significantly correlated with each other ($p < .01$ for all correlations), all three variables were entered into a stepwise multiple regression for

the no-incentive group. Attitude toward school and school problems were excluded from the regression equation, indicating that the scale representing parent-reported planning and organizational skills from the BRIEF was the better predictor of change in reading fluency performance for the no-incentive group, $F(1,12) = 8.540$, $p = .01$, $R = .645$. As in the single linear regression, more parent-reported difficulty with planning and organization predicted less change in reading fluency performance for the no-incentive group.

Table 27
Neuropsychological Predictors of Reliable Change in Reading Fluency Performance

	No Incentive (n=15)			Incentive (n=15)		
Variable	**F**	**p**	**r**	**F**	**p**	**r**
FSIQ-2 / GAI	2.953	.11	.430	2.596	.13	.408
Processing Speed Index	1.979	.18	.363	0.066	.80	.071
Cognitive Fluency	2.095	.17	.373	1.142	.31	.284
WJ-III Achievement						
Calculation	1.410	.26	.313	3.689	.08	.470
Letter-Word	3.103	.10	.439	1.444	.25	.316
BASC-2						
Attitude to School	4.870	.05*	.522	1.614	.23	.332
Attitude to Teachers	1.512	.24	.323	0.101	.76	.088
School Problems	7.968	.02*	.632	1.914	.19	.358
Locus of Control	2.066	.18	.383	0.000	.99	.004
Self Esteem	3.648	.08	.483	0.317	.58	.154
Self Reliance	1.064	.32	.285	0.302	.59	.151
BRIEF						
Initiate	3.040	.11	.435	0.138	.72	.103
Inhibit	0.147	.71	.106	1.166	.30	.287
Plan/Organize	5.742	.03*	.553	0.718	.41	.229
CBCL						
Cognitive Tempo	2.069	.17	.394	2.214	.16	.782

Overall, for the no-incentive group, craniospinal radiation, receipt of Section 504 accommodations, and parent-reported difficulty with planning and organization were the

strongest significant predictors of variance in change in academic fluency performance. It is worth noting that in the incentive group, no significant neuropsychological, medical, or demographic predictors were identified for either reading fluency change or math fluency change. However, inclusion in the incentive group significantly predicted improvement in both math fluency [$F(1,28) = 6.895$, $p = .01$, $r = .445$] and reading fluency [$F(1,28) = 7.744$, $p = .01$, $r = .465$].

CHAPTER FIVE
Discussion

This is the first study to intentionally examine the effects of external incentive on any neuropsychological outcome measure among pediatric medulloblastoma survivors. Based on the suggestion of Riva et al. (1989) that enhanced motivation may allow these children to overcome the neurocognitive deficits caused by medulloblastoma and its treatment, the present study was the first to heed the urging of Dennis et al. (1998) for further investigation of motivation and its effects in this population. As such, this study represents a new direction for research in this population, moving beyond basic documentation of deficits toward interventional research. Specifically, this study investigated enhancement of motivation as a possible intervention to address the declining academic performance of childhood medulloblastoma survivors. Thus, this study represents the first step toward creating a better future for these children, as high academic failure rates in this population can negatively impact long-term quality of life (Mitby et al., 2003).

This is also the first study of cognitive fluency in a population of pediatric medulloblastoma survivors, despite well-documented deficits in processing speed (Dennis et al., 1998; Kieffer-Renaux et al., 2000; Mabbott et al., 2008; Mulhern et al., 2004; Nagel et al., 2006; Riva & Giorgi, 2000; Reeves et al., 2006; Schatz et al., 2000; Spiegler et al., 2004; Vaquero et al., 2008). Additionally, this is one of few neuropsychological studies of pediatric medulloblastoma survivors to be conducted with a homogeneous sample, excluding all other types of brain tumors.

The primary objective of the present study was to determine the effect of external incentives on the fluent academic performance of childhood medulloblastoma survivors. A secondary objective of the present study was to investigate relationships between academic fluency, basic academic skill development, cognitive fluency, and processing speed in this population. To accomplish these objectives, 30 survivors of pediatric medulloblastoma between the ages of 7:0 and 18:11 years of age were administered a brief neuropsychological battery. Half of the sample was randomized to receive an incentive prior to completing repeated measures of academic fluency. Reliable change in performance across the repeated measures of academic fluency was calculated and compared between experimental groups. Parents of the participants also completed questionnaires that measured various constructs theorized to estimate aspects of intrinsic motivation and cognitive fluency.

Findings indicated that a performance-based incentive resulted in improvement in performance on measures of academic fluency relative to baseline, with response to incentive observed regardless of the severity of deficits in processing speed or cognitive fluency. Additionally, as hypothesized, a relative weakness in fluent academic performance as compared to academic skill development was identified. However, contradicting a secondary hypothesis, processing speed was determined to be better than cognitive fluency in predicting academic fluency. The interpretation and relevance of these findings are considered below.

EFFECT OF INCENTIVE

The primary goal of the present study was to determine whether the performance of childhood medulloblastoma survivors on academic fluency tasks would significantly improve as a result of external incentive. This was the first study to attempt such an investigation in this population specific to academic fluency. Additionally, this was the first study to intentionally examine the effects of external incentive on any neuropsychological outcome measure among pediatric medulloblastoma survivors. Findings supported the hypothesis that external incentive, theorized to create enhanced extrinsic motivation, resulted in significant improvements in the performance of childhood medulloblastoma survivors on academic fluency tasks. Moreover, no variables other than inclusion in the incentive group predicted change in academic fluency associated with incentive, suggesting that the effect of the external incentive washed out all other factors that may have otherwise influenced change in performance. This finding also suggests that enhanced extrinsic motivation as a result of the incentive may be solely responsible for the observed improvements in performance—improvements that were observed regardless of the degree of cognitive impairment. The potential implications of this finding are substantial, being relevant not only to pediatric medulloblastoma survivors but also to the issue of neuropsychological testing validity, and thus are described in subsequent sections.

It is worth noting that the study that served as a model for the present study (Riva et al., 1989) found that pediatric medulloblastoma survivors demonstrated normal performance on a computer-based measure of reaction time and a computerized

continuous performance test (CPT), despite demonstrating deficits in these areas in other studies and in that same study on pencil-and-paper measures. This caused some speculation among the research team on the present study that incentive might cause deficits in fluent academic performance to normalize. However, despite significant improvements in fluent academic performance, mean scores did not improve to the average range for the incentive group in the present study (see Table 4). That said, in several instances, external incentive improved academic fluency performance from one descriptive range (e.g., below average) to another (e.g., average).

Such observations raise the question of what may have caused some participants to respond more to incentive than others. As a corollary to the primary goal of this study, it was hypothesized that performance on measures of cognitive fluency and processing speed would influence the impact of external incentives on academic fluency performance in childhood medulloblastoma survivors. However, neither processing speed nor cognitive fluency was found to have a significant influence on the effect of external incentives for either math fluency or reading fluency. This suggests that response to incentive was not affected by any existing brain-based deficits in processing speed or cognitive fluency. That is, pediatric medulloblastoma survivors' ability to improve their performance on measures of academic fluency does not appear to be influenced by their performance on measures of processing speed or cognitive fluency. Rather, the degree of improvement in performance may have been determined by the degree to which the monetary incentive motivated a participant, which likely depends on differences in within-child factors determining motivation.

Thus, exploratory analyses were conducted to determine whether any constructs theorized to estimate aspects of intrinsic motivation (e.g., locus of control, initiation) or relevant variables related to personality (e.g., attitude to school, self-esteem, etc.) predicted response to incentive. In summary, no neuropsychological, demographic, or medical variables significantly predicted change in performance on academic fluency measures for the incentive group. The motivating effects of the external incentive may have washed out any potential influence of other factors. This finding is particularly significant with regard to processing speed or cognitive fluency deficits, indicating that a positive response to incentive on a task requiring those cognitive functions was observed regardless of the severity of deficits.

ACADEMIC FLUENCY VERSUS SKILLS

The hypothesis that survivors of childhood medulloblastoma would display a relative weakness in fluent academic performance as compared to basic academic skill development was supported. The observed deficits in fluent academic performance are likely due to radiation-induced white matter damage, which is believed to impair processing speed by disrupting the brain's overall structural network (Kandel et al., 2000; Leiner et al., 1986; Riva & Giorgi, 2000). Additionally, pediatric medulloblastoma patients receive their largest total dose of radiation at the posterior fossa, which likely causes extensive white matter damage to the cerebellum. This is another likely contribution to fluency deficits in this population, as one of the most prominent deficits associated with any sort of damage to the cerebellum is decreased processing speed

(Aarsen et al., 2009; Butler & Haser, 2006; Mabbott et al., 2008; Palmer, 2008; Palmer & Leigh, 2009; Palmer, Reddick, & Gajjar, 2007; Reeves et al., 2006; Riva & Giorgi, 2000; Steinlin et al., 2003; Vaquero, Gomez, Quintero, Gonzalez-Rosa, & Marques, 2008; Zuzak, Poretti, Drexel, Zehnder, Boltshauser, & Grotzer, 2008). Given that processing speed is a component of cognitive fluency and that the study sample demonstrated impairments on measures of both constructs, the finding that this population also demonstrated impairments in fluent academic performance seems quite logical. Finally, the finding that fluent academic performance was significantly weaker than basic academic skill development supports the conclusions of a pilot study (Stavinoha & Burrows, 2004).

Interestingly, in both the present study and the pilot study, basic academic skill development was low average to average. Contrary to this finding, previous research generally has indicated deficits in basic academic skill development. This could be due to the fact that previous studies sampled mixed tumor types (e.g., Mabbott et al., 2005) or overstated findings (e.g., Reeves et al., 2006). Specifically, the Reeves et al. study (2006) emphasized that for their sample of 38 pediatric medulloblastoma survivors, mean performances on the Wechsler Individual Achievement Test were "significantly below the standardization sample" despite reporting mean scores of 92.50 for basic reading, 89.18 for mathematical reasoning, and 91.24 for spelling—all of which fall well within one standard deviation of the normative mean and which would be considered "average" performances relative to the general population. One possible explanation for the findings of relatively average academic skill development in the present study, the pilot study, and the Reeves et al. study is that school difficulties experienced by survivors of

childhood medulloblastoma are primarily associated with deficits in academic fluency rather than poor academic skill development. It may also be possible that the continuous refinement of medical treatment protocols and increasing emphasis on academic interventions are contributing to improvements in academic skill development in pediatric medulloblastoma survivors.

It is worth noting that previous studies have found little to no predictive value for clinical factors such as radiation dose, extent of surgery, or treatment with chemotherapy in determining academic outcomes (Dennis et al., 1996; Mabbott et al., 2005; Palmer et al., 2007; Reeves et al., 2006). The only significant findings have been that time since radiation predicted reading skill development (Reeves et al., 2006) and younger age at diagnosis/treatment predicted worse academic outcomes (Dennis et al., 1996; Palmer et al., 2007). The present study found that time since surgery, radiation, and chemotherapy—essentially, time elapsed since treatment—significantly predicted performance on measures of both math and reading skill development. Specifically, as time since treatment (surgery, radiation, or chemotherapy) increased, performance on basic academic skill measures decreased, with time post chemotherapy responsible for the majority of the variance. Based on findings from previous research regarding IQ changes in pediatric medulloblastoma survivors, this is likely not a true decrease but instead a failure to make expected gains (Palmer et al., 2001).

Exploratory analyses found that many of the neuropsychological predictors of academic skill development—specifically, overall cognitive ability, processing speed, and cognitive fluency—also predicted academic fluency. Performance on measures of academic skills also predicted performance on measures of both math and reading

fluency—a reasonable finding given that academic fluency by definition requires some degree of academic skill development and given the moderate relationship among performances on these measures in the general population (McGrew & Woodcock, 2001). Among all these neuropsychological variables, math skill development and processing speed best predicted academic fluency. It is particularly logical that processing speed was one of the best predictors given that processing speed is a significant component of academic fluency by definition. Possible construct validity issues related to the cognitive fluency measure, to be discussed in the following section, may explain why cognitive fluency was not identified as one of the best predictors of academic fluency.

Regarding medical variables, the only treatment-related predictor of any academic fluency performance was the amount of total craniospinal radiation, which significantly predicted reading fluency such that with greater total radiation, reading fluency scores decreased. This finding is consistent with existing literature, which highlights radiation as the single most significant predictor of many outcome measures (e.g., Mulhern et al., 1999; Palmer et al., 2001; Reddick et al., 2000), and is logical given the known adverse effects of craniospinal radiation on white matter, which can in turn negatively impact processing speed and cognitive fluency. Finally, regarding demographic predictors, older age and higher grade at evaluation significantly predicted worse performance on the measure of math fluency, with age at evaluation emerging as the better predictor. This is likely a reflection of the well-documented finding that pediatric medulloblastoma survivors fail to make expected gains in cognitive and academic areas as they mature (Palmer et al., 2001).

COGNITIVE FLUENCY

This study was the first to conduct a direct investigation of cognitive fluency in childhood medulloblastoma survivors, particularly doing so in relation to their academic performance. The hypothesis that performance on measures of cognitive fluency would better predict fluent academic performance of childhood medulloblastoma survivors than performance on measures of simple processing speed was not supported. In fact, while processing speed was a significant predictor for academic fluency outcomes, cognitive fluency did not significantly contribute to the prediction of variability in fluent academic performance for either math or reading. However, this finding does not necessarily stipulate a complete rejection of the stated hypothesis. Instead, it is possible that this finding is simply a reflection of poor construct validity of the Cognitive Fluency cluster of the WJ-COG. While it is widely acknowledged that this cluster of subtests measures speed and accuracy in performing more complex cognitive processes than are involved in measures of processing speed (Floyd, Shaver, & McGrew, 2003), it also has been suggested that the Cognitive Fluency cluster of the WJ-COG measures more verbal cognitive fluency than nonverbal (Gregg, Coleman, & Knight, 2003; Gregg et al., 2005), given the subtests that comprise the Cognitive Fluency cluster involve word recall, rapid picture naming, and semantic organization. Thus, it is possible that the Cognitive Fluency cluster of the WJ-COG does not accurately or effectively measure the construct of cognitive fluency. Indeed, the Cognitive Fluency cluster was not predictive of fluent academic performance in the present study. Additionally, in a sample of 100 normally achieving college students serving as a control group for a study of students with learning

disabilities, the Cognitive Fluency cluster did not significantly predict performance on the WJ-ACH Reading Fluency subtest (Gregg et al., 2005). Given the lack of other measures proven to be valid and/or reliable in measuring cognitive fluency, at this time there is no ideal solution to the possibility that the WJ-COG Cognitive Fluency cluster does not effectively measure its intended construct.

Additionally, given that this is merely a hypothesized explanation for the failure of the Cognitive Fluency cluster to predict academic fluency performance in this population of childhood medulloblastoma survivors, further studies are needed prior to declaring this cluster to have poor construct validity. An alternate possibility is that processing speed truly is a better predictor of academic fluency performance than cognitive fluency, at least when measured by the tests used in the present study. It is possible that the Reading Fluency and Math Fluency subtests of the WJ-ACH measure academic knowledge that is fairly over-learned and simple, thus drawing more on processing speed (the speed at which a person can perform automatic, over-learned tasks) than cognitive fluency. The findings of the present study suggest a possible lack of functional clarity in conceptualizations of cognitive fluency and processing speed, rendering it virtually impossible to explain those findings without further investigation.

The WJ-COG Cognitive Fluency cluster remains the only measure of cognitive fluency that is widely used in neuropsychological research, hence the decision to use it as a measure of that construct in the present study. Discussions of other findings of this study therefore include considerations of performance on the Cognitive Fluency cluster based on the indication of satisfactory validity based on the Cattell-Horn-Carroll theory,

according to studies conducted by the test developers (Mather & Woodcock, 2001; McGrew & Woodcock, 2001).

CLINICAL AND THEORETICAL IMPLICATIONS

The primary aim of the present study was to determine whether pediatric medulloblastoma survivors might respond similarly to healthy populations under motivational conditions, showing increased processing speed and enhanced academic performance (Codding et al., 2009; Eckert et al., 2002; Jones et al., 2009; Locke & Braver, 2008; Noell et al., 2001; Pochon et al., 2002; Taylor et al., 2004), or if the damage caused by the tumor and its treatment would be too significant to overcome. The finding of a significantly greater improvement in fluent academic performance for the incentive group without regard to severity of initial deficit suggests that there are conditions under which survivors of medulloblastoma can improve their performance on neurocognitive dimensions known to be at great risk following treatment. Given that significant effects of motivation were found, this could have implications for the design of special education services and other remedial academic programs for pediatric medulloblastoma survivors. Alternatively, it must be noted that despite the finding that incentive significantly predicted improvement in performance on a measure of academic fluency, the vast majority of participants still demonstrated deficits in this domain. Such deficits being observed even under strongly motivating conditions reinforces that this is an area of true neurocognitive impairment in pediatric medulloblastoma survivors. This observation also suggests that provision of relevant academic accommodations, such as

extended time and abbreviated assignments, is likely of primary importance for this population, particularly as it may be argued that fluency cannot be "taught." However, secondary to accommodations, the findings of this study support the use of extrinsic motivation to improve academic fluency performance in pediatric medulloblastoma survivors, at the very least on a one-time basis. Guidelines regarding the practical implementation of external incentives for this purpose will require further investigation, though some initial suggestions are provided here.

Academic remediation is greatly needed in this population, given that at ten-year follow-up, 80% of medulloblastoma survivors may require special education services (Hoppe-Hirsch et al., 1995). Indeed, close to 70% of the present sample was receiving such services, and yet the participants were on average just four years post treatment. Furthermore, even when accounting for declines in IQ, survivors of childhood medulloblastoma have been shown to exhibit significant continuing declines in spelling and arithmetic (Mabbott et al., 2005), possibly due to a failure to continue learning information at their pre-treatment rate (Palmer et al., 2001). In this respect, pediatric medulloblastoma survivors may be considered similar to children with learning disabilities. Notably, learning disabled and other "academically problematic" children tend to be more extrinisically motivated than their healthy peers (Lepper et al., 1997; Lincoln & Chazan, 1979). This lends support to the argument that remedial academic programs and services, based on the findings of the present study, should incorporate attempts to enhance pediatric medulloblastoma survivors' motivation for academic performance.

The use of extrinsic motivation to improve academic performance is a polarizing subject, particularly as it has been shown to decrease intrinsic motivation in certain situations (Deci, 1971; Lepper et al., 1973). However, it appears likely that expected, performance-contingent, external rewards would not decrease intrinsic motivation toward inherently boring tasks (Cameron, 2001; Cameron & Pierce, 1994; Deci et al., 1999, 2001; Eisenberger & Cameron, 1996; Tang & Hall, 1995) such as schoolwork. To prevent cognitive exhaustion and minimize the risk of incentives losing value, such incentives may be most judiciously used for situations such as high-stakes testing rather than being used on a constant basis. While recommendations regarding the specific nature of the external incentives used in remedial academic programs and services are beyond the scope of this study, it should be noted that these incentives do not necessarily have to be monetary in nature. That said, many researchers assert that money may be the most influential reward possible for humans (Lea & Webley, 2006; Vohs et al., 2006)—a finding that applies to children and adolescents as well (Kohls et al., 2009), particularly those who are "academically problematic" (Lepper et al., 1997). Realistically, most remedial academic programs and services for pediatric medulloblastoma survivors likely would not have the funding necessary to provide monetary incentives for academic performance. However, parents of these children may be able to create such incentives on an individual basis.

One caveat should be noted in designing programmatic interventions—research suggests that if a goal cannot be reached due to extreme difficulty, monetary rewards can actually hurt performance due to a sense of decreased self-efficacy (Locke & Latham, 2002). However, even with a very difficult task, if rewards are provided on a sliding

scale based on performance rather than a single reward for reaching the goal, performance will remain highly motivated (Latham & Kinne, 1974; Latham & Yukl, 1975; Lee, Locke, & Phan, 1997). Any remedial academic programs or services for pediatric medulloblastoma survivors should be designed with these recommendations in mind.

Additionally, this study's finding that pediatric medulloblastoma survivors demonstrated a relative weakness in academic fluency with respect to basic academic skill development suggests that in addition to the provision of relevant academic accommodations, there may be value in implementing programs or services targeted toward fluent academic performance. That is, the emphasis of academic remedial programs should be on helping these children learn strategies to help them process information and perform academic tasks more quickly, not just simply teaching them the material. Exploratory analyses suggest that a focus on teaching basic academic skills may become more relevant with increasing time since treatment, yet such teaching still should not be to the exclusion of remediation targeted toward improving academic fluency. Time since treatment was not a significant predictor of academic fluency outcomes, suggesting that deficits in fluent academic performance are observed both immediately after treatment and long-term. This meshes with the established finding that processing speed is the first deficit to emerge after treatment for pediatric medulloblastoma (Palmer, 2008).

In addition to the clinical implications of the present study regarding the design of academic interventions for pediatric medulloblastoma survivors, the findings of the present study also have broad theoretical implications for neuropsychological testing in

general. Recall the admonition of Locke and Braver (2008): "It is a truism of cognitive research that participants perform experimental tasks with varying levels of motivation. Some participants appear to show little interest and exert minimal effort in their task performance, whereas others seem to approach the task as a critical test, exhibiting a burning desire to perform to their utmost ability. Yet, even though this variation in motivation is a well-known phenomenon, it seems to be underappreciated and underexplored" (p. 99). Indeed, few would argue that fluctuations in both intrinsic and extrinsic motivation impact performance in real-life situations such as school, yet the majority of neuropsychological research and clinical evaluations are conducted without overt or objective acknowledgment of this ever-present factor. Given that the typical neuropsychological battery is likely to be viewed as tedious and boring by most children, adolescents, and even adults (e.g., Lepper et al., 1997), there is a strong possibility that even neuropsychological testing conducted under optimal conditions (referring both to the physical setting and to the patient's inner state with regard to hunger, mood, restedness, etc.) may not reflect optimal performance or consistently motivated performance.

Some may argue that neuropsychologists take this into account when interpreting performance and presenting results. But that caveat may not be heard by a distressed parent, and it may not be noticed by a school administrator or medical professional flipping through a report. And even when heard or noticed, that caveat may not be understood or appreciated, as it has no real definition or boundaries, in contrast to the apparent simplicity and clarity of a score value or range. Additionally, though with experience neuropsychologists may become more able to detect poor motivation, there

are always cases where the patient's level of motivation is uncertain. After all, can another person's precise levels of intrinsic and extrinsic motivation ever be known with any degree of certainty? The closest the field has come presently is through symptom validity tests (SVTs), which have been reported to detect suboptimal effort in a pediatric population when clinical judgment alone would have failed to do so (Kirkwood, Kirk, Blaha, & Wilson, 2010). However, even a failure on an SVT can be due to any number of reasons other than motivational factors, including the underlying medical condition or verbal fluency deficits (Donders, 2005; Kirkwood & Kirk, 2010; Rienstra, Spaan, & Schmand, 2010). Additionally, even if an SVT was a perfect test for motivation, it would assess motivation only at the time of that particular test, leaving the validity of the results on the rest of the neuropsychological battery open to question. Regardless, the crux of the matter is that, more often than not, neuropsychological testing—particularly when conducted for research purposes—is not conducted with more than a fleeting consideration of the potential effects of motivation.

Others may argue that the issue of motivation is irrelevant because neuropsychological testing should reflect typical rather than optimal performance. However, consider that the definition of "typical" performance is quite unclear given that the average neuropsychological evaluation lasts several hours—plenty of time for fluctuations in motivation and resultant effort—and certainly is not "typical" with regard to a classroom setting or even a child's daily/weekly schedule. Ideally, neuropsychological assessment should provide information regarding a child's full capabilities. So when administering neuropsychological measures, is it sufficient to record an individual's scores without taking both his or her intrinsic and extrinsic

motivation into account? Can motivation alter neuropsychological functioning to the point that diagnoses or recommendations might be affected? Are certain populations capable of significantly improving their performance on neuropsychological tests based on alterations in motivation? Given the potential implications of these questions, it is surprising that they have not yet been sufficiently explored in research or considered on a clinical level.

Granted, a growing amount of research has been devoted to investigating the effects of extrinsic incentives on a variety of cognitive tasks, finding that performance can significantly improve as a result of external motivation from childhood through adulthood, in both healthy and disordered populations (Casey et al., 2002; Engelmann & Pessoa, 2007; Engelmann et al., 2009; Hare & Casey, 2005; Hare et al., 2005; Locke & Braver, 2008; Maxwell et al., 2005; McCauley et al., 2009; Miller & Cohen, 2001; Pailing & Segalowitz, 2004; Schultz, 2004; Taylor et al., 2004; Thornton et al., 2007; Watanabe, 2007). However, the majority of these studies have been focused on identifying biological correlates of motivation through neuroimaging (e.g., Locke & Braver, 2008; Taylor et al., 2004) or have been devoted to esoteric topics such as "exogenous spatial attention" (Engelmann & Pessoa, 2007) without highlighting issues relevant to neuropsychological testing or real-world functioning. For the most part, the field of neuropsychology has yet to examine these findings in the context of neuropsychological testing. One of the first studies to address such issues was published recently (Duckworth, Quinn, Lynam, Loeber, & Stouthamer-Loeber, 2011), reporting a finding that intelligence tests measure both intelligence and motivation and thus can be confounded by poor motivation. That study represents significant progress toward an

understanding of the impact of motivational states on the validity of neuropsychological testing and highlights the need for further research. Given the findings of the present study and Duckworth et al. (2011), the assumption heretofore of the validity of neuropsychological assessment clearly comes into question.

The findings of the present study suggest that motivation can, in fact, influence neuropsychological performance—and in a population with significant brain-based deficits, at that. How much more, then, might fluctuations in motivation affect the neuropsychological performance of a child who has not undergone neurosurgery, chemotherapy, and full craniospinal radiation? And to what extent might similar populations—from survivors of various pediatric brain tumors to children who have experienced a traumatic brain injury—display a similar performance-based response to incentive? The present study represents a significant contribution to the small but developing body of research on the potential variance in neuropsychological performance due to fluctuations in motivation—an issue relevant to the evaluation of all individuals, not just that of pediatric medulloblastoma survivors.

LIMITATIONS

Studies examining childhood medulloblastoma survivors are often limited by a small sample size consisting primarily of males, due to the fact that medulloblastoma is a relatively low-incidence condition and is more common in males (Gottardo & Gajjar, 2006; Horner et al., 2009). Those limitations also apply to the present study, although a sample size of 30 could arguably be classified as medium to large in the context of

studies of childhood medulloblastoma survivors, especially among those studies that do not include heterogenous tumor types. Previous studies have attempted to increase sample size at the cost of clarity of findings by including survivors of tumors similar to medulloblastoma. However, the present study maintained strict inclusion guidelines in order to avoid the limitations inherent in studies of heterogeneous tumor types. Although a sample of 30 participants is particularly strong for a study of this population involving a homogeneous sample, randomizing the participants into two groups for the purpose of the primary aim of the study resulted in two groups of just 15 participants. To enhance statistical power, recruitment for the present study is being continued at a second site, with the goal of obtaining a sample of approximately 50 participants for analysis. Regarding gender distribution, the sample for this study was 73% male. In anticipation of this, stratified randomization procedures were used to ensure equal numbers of males and females in each group. However, due to the relative preponderance of males in the sample, the findings of the present study are still less easily generalized to female survivors of pediatric medulloblastoma.

Due to the design of the study involving the reading of a standardized script to participants in the incentive group to inform them of the incentive, it was not feasible to keep the trained examiner administering the test battery blinded to condition. Thus, the potential exists that this could have inadvertently contributed to examiner bias. In an attempt to reduce such influence, randomization of subjects was conducted during the five-minute break in the study protocol, rather than prior to the administration of the majority of the research battery. The fact that the Math Fluency and Reading Fluency subtests of the WJ-ACH do not require subjective scoring also may have helped reduce

the potential impact of knowledge of condition. However, under ideal circumstances, the study would have had sufficient personnel to enable presentation of the standardized script by a non-examiner researcher, without the trained examiner knowing whether the script had been presented prior to the administration of the WJ-ACH Form B subtests, and scoring of the protocols would have been performed by a third researcher, similarly blinded to condition. However, the consistency in administration afforded by the utilization of a single examiner may offset some of the weakness of this approach, particularly for a study in which participant motivation is such a significant piece of the design.

Limitations of Measures

A primary limitation of this study is the lack of preexisting data regarding any practice effects between the two forms of the WJ-ACH within an approximate 60-mintute interval. Attempts to address this limitation in the present study included the presence of a control group and the calculation of reliable change scores using a model designed to adjust for practice effects (Chelune et al., 1993). Additionally, to strengthen the analysis of practice effects in the control group for use in the analysis of incentive effects in the experimental group, WJ-ACH Forms A and B were not counter-balanced. Counter-balancing was not necessary, as each participant served as his or her own control.

The lack of an appropriately normed, reliable, valid measure of intrinsic trait motivation for the age range of the present sample represents another limitation of this study. Such measures have been developed only for college-age populations and/or are

specific to academic motivation. Thus, approximations of intrinsic motivation such as initiation and self-reliance had to be obtained from outcome factors of the BRIEF and BASC-2, as described previously. Those two measures captured both parent- and self-report and are therefore potentially influenced by any biases of the responder, although it is difficult to imagine a measure of intrinsic motivation that would not require parent- or self-report.

Possible difficulties with construct validity of the WJ-COG Cognitive Fluency cluster, as previously discussed, also represents a limitation for the present study. This cluster appears to be biased toward verbal fluency (Gregg, Coleman, & Knight, 2003; Gregg et al., 2005) and therefore may not accurately or effectively measure its intended construct. It is also possible that conceptualizations of cognitive fluency and processing speed need to be further clarified and potentially more clearly differentiated, which is beyond the scope of the present study. As the WJ-COG Cognitive Fluency cluster is the only measure of cognitive fluency that is widely used in neuropsychological research, it appears that there is no satisfactory alternative at this time. Additionally, the WJ-COG test developers have reported statistical analyses indicating that the Cognitive Fluency cluster accurately measures the construct of cognitive fluency as described by the Cattell-Horn-Carroll theory (Mather & Woodcock, 2001; McGrew & Woodcock, 2001). Further research is needed to clarify this.

Finally, limitations regarding the ecological validity of the measures utilized in this study must be acknowledged. Although widely used in clinical evaluations and neuropsychological research, the WJ-ACH is merely an approximation of "true" academic performance as would be observed on an everyday basis in a classroom

environment. Clearly, academic successes and failures are determined by multiple factors, yet this study focused on a brief examination of basic academic skill development and academic fluency.

DIRECTIONS FOR FUTURE RESEARCH

The findings of the present investigation highlight several areas in need of additional research, both specific to the population of pediatric medulloblastoma survivors and applicable to the field of neuropsychology in general. Specific to childhood medulloblastoma survivors, further research is needed to determine the extent of basic academic skills deficits versus fluency deficits in pediatric medulloblastoma survivors, particularly given the large standard deviations in performance reported in this and previous studies. Findings of the present study suggest that academic fluency deficits may play a larger role in school difficulties than do basic academic skills deficits. However, the majority of research on academic performance in pediatric medulloblastoma survivors is focused on real-world outcomes such as graduation rate and special education utilization rather than skills and fluency. More investigations specific to those individual components of academic performance are greatly needed. Additionally, given the finding of the present study that performance on measures of basic academic skills decreased as time since treatment increased, studies of basic academic skills and fluency that are longitudinal in nature would also represent a significant contribution to the literature. Finally, studies with greater ecological validity—for instance, those integrated into a classroom setting or involving analysis of

actual grades and/or standardized test performance—also would enhance the understanding of the nature of academic deficits in childhood medulloblastoma survivors.

The potential lifelong implications of these academic difficulties underscore the importance of the type of research involved in the present study, potentially bearing implications for future interventions. Much more neuropsychological research with an interventional focus is needed for this population, even going beyond the more theoretical nature of the present study to directly investigate outcomes of neurocognitive and academic interventions. Furthermore, extrapolating on the findings of the current study, it is possible that external incentives may enhance performance not only for pediatric medulloblastoma survivors but also for children with other medical conditions or learning disabilities. However, a disproportionately small amount of research in the field of neuropsychology is conducted with the explicit focus of improving neuropsychological interventions. More neuropsychological research with interventional applications is needed in a variety of medical and psychiatric pediatric populations. Extrinsic motivation may play a large role in such intervention-focused research, regardless of the population or the targeted cognitive functions. For instance, Jones and colleagues (2009) have noted that "although lack of motivation and insufficient practice are not considered core deficits in reading disorders, they are often essential components of *any* educational intervention" (p. 53) and indicate that external incentives can provide such motivation. There is a clear need for research investigating the manipulation of extrinsic motivation in academic and neuropsychological interventions for any number of populations.

In considering the findings of the present study, a pressing question clearly is the issue of what caused some participants to respond more strongly to incentive than others.

Absent the identification of any significant neuropsychological, medical, or demographic predictors of response to incentive in the present study, there is room for speculation that within-child characteristics contributing to intrinsic motivation may be the distinguishing factors. It is possible that the variables selected in the present study to represent those factors, though selected based on theory (e.g., Pailing & Segalowitz, 2004), may not have adequately represented the intended constructs. As the existing research on intrinsic motivation is quite divergent, with no consensus as to which of the many models is preferable, theoretical research is needed to clarify this construct in order to then shape the development of valid measures specific to intrinsic motivation. Such investigation of the various components of intrinsic motivation could lead to increased awareness and measurement of motivation as a factor in neuropsychological performance, which could in turn lead to the development of clinically relevant measures of motivation, enabling more accurate interpretation of test performance.

Measurement issues aside, there remains a great need for further investigation into intrinsic factors that may cause certain individuals to be more strongly motivated by external incentive than others—a need echoed by Duckworth and colleagues (2011). This is the case not only for the population of pediatric medulloblastoma survivors but also for other pediatric medical populations. Such knowledge could inform the design of remedial academic programs or services for these populations. In combination with the aforementioned guidelines derived from this study's findings, regarding the design of such programs or services for pediatric medulloblastoma survivors, the ability to identify which children might be better candidates for motivation-focused interventions would

further enhance efforts to ensure that academic difficulties do not lead to lifelong issues for these children.

CONCLUSION

The present study was the first to intentionally examine the effects of external incentive on any neuropsychological outcome measure among pediatric medulloblastoma survivors. As such, this study represents a new direction for research on pediatric medulloblastoma survivors, moving beyond basic documentation of deficits toward intervention-focused research. Despite indications that survivors of childhood medulloblastoma may not be able to overcome deficits in academic fluency even under highly motivating conditions, the findings of this study suggest that there is a potential role for external incentives in the design and implementation of academic interventions for these individuals. Thus, this study represents a first step toward creating a better future for pediatric medulloblastoma survivors, as high academic failure rates in this population can cause numerous lifelong difficulties. Additionally, the findings of this study may provide a rationale for investigations of motivation in other pediatric medical populations and with respect to other areas of neurocognitive functioning.

The study of motivation has traditionally remained within the realm of personality psychology, where it has been the elusive subject of numerous and diverse theories on which no consensus has been reached. The present study demonstrates that no branch of psychology stands alone. In order to fully understand neuropsychological performance, consideration must be given to motivational factors. However, as such

factors are far from being fully understood, significant progress must made in terms of conceptualizing motivation as a functional construct to advance knowledge within the realms of both personality psychology and neuropsychology. The field of neuropsychology, long interdisciplinary in nature with regard to medical fields, is now making slow but steady progress toward adopting a more interdisciplinary approach with regard to other branches of psychology itself. This study is the first of just a few (e.g., Duckworth et al., 2011) to investigate the effects of motivation on neurocognitive performance, and it is hoped that many more will follow. This new direction for research will not only contribute to the advancement of neurocognitive interventions but also will enhance the validity and utility of neuropsychological assessments, which is vital to both the reputation and the continued relevance of the field of neuropsychology.

APPENDIX A
Patient Medical Chart Review Form

For Office Use Only Patient ID: _____ Date of Evaluation: _____

MEDICAL CHART REVIEW FORM

Patient's Name: _____ MRN: _____

Date of Birth: _____ Gender: Male _____ Female ____

Date of Evaluation: _____ Age at Evaluation: _____

INCLUSION CRITERIA MET

_____ Histopathologically diagnosed medulloblastoma
_____ Prior treatment with cranial radiation
_____ Current age: 7 to 18 years
_____ Proficiency in English
_____ No history of tumor recurrence
_____ No history of traumatic brain injury
_____ No history of stroke
_____ No history of neurological disorder unrelated to medulloblastoma
_____ No history of major medical difficulties unrelated to medulloblastoma
_____ Completion of signed informed consent by parent/guardian or 18-yo subject
_____ Subject's assent to participate in the protocol if minor

Date of Diagnosis: _____ Age at Diagnosis: _____

Date of Surgery: _____ Age at Surgery: _____

Resection:
_____ Gross total resection
_____ Subtotal resection
_____ Not Available

Dates of Radiation: _____ Age at Radiation: _____

Type of Radiation:
_____ Whole brain Amount: _____
_____ Craniospinal Amount: _____
_____ Posterior Fossa Amount: _____

Treated with chemotherapy: _____ No _____ Yes, dates: _____

If yes, type of chemotherapy: _____ Vincristine
_____ Cisplatin
_____ Cyclophosphamide
_____ Lomustine
_____ Other: _____

Perioperative/Postoperative Procedure and Complications:

_____ Shunt Insertion, # of Revisions: _____
_____ Ventriculostomy
_____ Infection
_____ Brain swelling

Date of last neuropsychological testing: _____

APPENDIX B
Patient History Form

For Office Use Only Patient ID: _____ Date of Evaluation: _____

HISTORY FORM

Please complete the following information as completely as you can. This information will be helpful in gaining a better understanding of your child.

Child's Name: _____

Age: _____ Date of Birth: _____ Race/Ethnicity: _____

Sex: _____ Male _____ Female Handedness: _____ Right _____ Left

Languages spoken: _____ Preferred language: _____

Address: _____

Home Phone: _____ Hours since child last ate: _____

Quality of child's sleep last night: ____ Good ____ Poor Explain: _____

PARENT HISTORY

Mother's Name: _____ Age: _____

Occupation (please provide specific job title): _____

Highest grade completed: _____ Degree obtained: _____

Father's Name: _____ Age: _____

Occupation (please provide specific job title): _____

Highest grade completed: _____ Degree obtained: _____

SCHOOL HISTORY

Current grade: _____ Name of School: _____

Did your child attend…
Pre-School? No _____ Yes _____, ages: _____
Early Childhood Intervention Program (ECI)? No _____ Yes _____, ages: _____
Preschool Program for Children with Disabilities (PPCD)? No _____ Yes _____, ages: _____

Has your child ever been retained? No _____ Yes _____ If yes, which grade? _____

Has your child ever received special education services, i.e., ever had an ARD meeting?
Have an Individualized Education Program (IEP)? No _____ Yes _____

If yes, classification?
____ Speech/Language Impairment
____ Vision Impairment
____ Auditory Impairment
____ Orthopedic Impairment
____ Other Health Impairment
____ Learning Disabled
____ Mental Retardation
____ Emotional Disturbance
____ Traumatic Brain Injury
____ Autism

Does your child receive any of the following services?

____ Special Education	____ Content Mastery	____ Speech/Language Therapy
____ Resource Room	____ Tutoring	____ Visual Impairment Services
____ 504 Accommodations	____ Physical Therapy (PT)	____ Occupational Therapy (OT)
____ Inclusion Classroom	____ Individual Aide	____ Tutoring (in or out of school)
____ Special Testing	____ Summer School	____ Special School for LD

If yes, how long has your child received these services, and how often does he/she currently?

Service: _____ received _____ days/week, _____ hours/day since _____
Service: _____ received _____ days/week, _____ hours/day since _____
Service: _____ received _____ days/week, _____ hours/day since _____
Service: _____ received _____ days/week, _____ hours/day since _____

Please indicate with an "X" your child's performance in each of the following subjects:

	Failing	Below Average	Average	Above Average
Reading/English/Language Arts				
History/Social Studies				
Arithmetic/Math				
Science				

MEDICAL HISTORY

Diagnosis/Disease (Present and Past)	Date	Treatment
_____	_____	_____
_____	_____	_____
_____	_____	_____

Medications (past and present): ____ Check if none

Name	Reason	Date
_____	_____	_____
_____	_____	_____
_____	_____	_____

Does your child have vision problems? No _____ Yes _____
If yes, does your child wear corrective lenses? No _____ Yes _____

Does your child have hearing problems? No _____ Yes _____
If yes, does your child use hearing aids? No _____ Yes _____

If there is anything else that you feel we need to know about your child, please feel free to explain here:

_____

_____

_____

Form completed by: _____

Relationship to child: _____

Date:_____

Thank you for your participation in this research study!

APPENDIX C
Consent Form

The University of Texas Southwestern Medical Center at Dallas
Parkland Health & Hospital System
Children's Medical Center
Texas Scottish Rite Hospital for Children
Presbyterian Hospital of Dallas

CONSENT TO PARTICIPATE IN RESEARCH

Title of Research:	Academic Performance in Survivors of Pediatric Medulloblastoma
Funding Agency/Sponsor:	UT Southwestern Medical Center
Study Investigators:	Pete Stavinoha, Ph.D. Lana Harder, Ph.D. Daniel Bowers, M. D. Alice Ann Spurgin, B.A. Lynn Gargan, Ph.D. Brianne Butcher, Ph.D.

You may call these study doctors or research personnel during regular office hours at 214-456-8985. At other times, you may call them at 214-456-7000.

Note: If you are a parent or guardian of a minor and have been asked to read and sign this form, the "you" in this document refers to the minor.

Instructions:
Please read this consent form carefully and take your time making a decision about whether to participate. As the researchers discuss this consent form with you, please ask him/her to explain any words or information that you do not clearly understand. The purpose of the study, risks, inconveniences, discomforts, and other important information about the study are listed below. If you decide to participate, you will be given a copy of this form to keep.

Why is this study being done?
This study is being done to learn more about the cognitive abilities and academic performance of survivors of childhood medulloblastoma.

Why is this considered research?
This is a research study because the researchers are investigating the effects of medulloblastoma on cognitive abilities and academic performance in child and adolescent survivors.

The following definitions may help you understand this study:

- Standard medical care means the regular care you would receive from your personal doctor if you choose not to participate in this research.
- Researchers means the study doctor and research personnel at the University of Texas Southwestern Medical Center at Dallas and its affiliated hospitals.
- Neuropsychological means related to cognitive abilities such as memory, attention, and processing speed.

Why am I being asked to take part in this research study?
You are being asked to take part in this study because you have undergone treatment for a childhood medulloblastoma.

Do I have to take part in this research study?
No. You have the right to choose whether you want to take part in this research study. If you decide to participate and later change your mind, you are free to stop participation at any time.

If you decide not to take part in this research study it will not change your legal rights or the quality of health care that you receive at this center.

How many people will take part in this study?
About 60 people will take part in this study at Children's Medical Center.

What is involved in the study?
If you volunteer to take part in this research study, you will be asked to sign this consent form and will have the following tests and procedures. Some of the procedures may be part of your standard medical care, but others are being done solely for the purpose of this study.

Screening Procedures
To help decide if you qualify to be in this study, the researchers may ask you questions about your background history, including presence of any neurological or developmental disorders. The researchers may also ask you questions about your health, including medications you take and any surgical procedures you have had.

Procedures and Evaluations during the Research
You will be administered a series of academic and neuropsychological tests. These pen-and-paper tests will take approximately 1 hour to complete. The tests will evaluate academic abilities and functioning, processing speed, and cognitive fluency.

Parents will complete four questionnaires. Three questionnaires will be used to collect information regarding the child's typical behavior. The final questionnaire will be used to collect information regarding the child's developmental, educational, and medical history, family medical and educational history, and relevant demographic information such as parent education and employment.

Parents will be asked to provide consent for the researchers to obtain records from the child's school(s) regarding the child's grades, standardized test scores, and any special education plans or curriculums.

The participant's medical charts will be reviewed to obtain data concerning medical information required for standard of care treatment for pediatric brain tumor patients. Information will include preoperative conditions, tumor type, brain imaging, surgical procedures, treatment type, and medical complications (i.e., shunt insertions, infections).

The academic and neuropsychological tests in this study are designed for research, not for medical purposes. They are not useful for finding problems or diseases. Even though the researchers are not looking at your academic and neuropsychological tests to find or treat a medical problem, they will tell you if they notice something unusual. You and your regular doctor can decide together whether to follow up with more tests or treatment. Because the academic and neuropsychological tests done in this study are not for medical purposes, the research results will not be sent to you or to your regular doctor. However, parents will be provided with a written summary of their child's performance.

How long can I expect to be in this study?
The study will be completed in 1 visit lasting approximately 1 hour. You can choose to stop participating for any reason at any time. If you decide to stop participating in the study, you may be asked if you are willing to complete some study termination questionnaires.

What are the risks of the study?
Neuropsychological and academic tests do not involve any medical procedures and have no inherent risks. However, you may experience mild frustration or nervousness due to cognitively challenging tasks.

Loss of Confidentiality
Any time information is collected, there is a potential risk for loss of confidentiality. Every effort will be made to keep your information confidential; however, this cannot be guaranteed.

You should be aware that the researchers are not prevented from reporting information to authorities in order to prevent serious harm to you or others. If the researchers suspect abuse of a child, elder, or disabled person, they will report such concerns to proper authorities as required by law.

How will risks be minimized or prevented?
If at any point during the evaluation you experience discomfort as a result of the testing, you may refuse to answer any of the questions, take a break, or stop your participation in this study at any time.

What will my responsibilities be during the study?
While you are part of this study, the researchers will follow you closely to determine whether there are problems that need medical care. It is your responsibility to do the following:

- Ask questions about anything you do not understand.
- Keep your appointments.
- Follow the researchers' instructions.
- Let the researchers know if your telephone number or address changes.

If I agree to take part in this research study, will I be told of any new risks that may be found during the course of the study?
Yes. You will be told if any new information becomes available during the study that could cause you to change your mind about continuing to participate or that is important to your health or safety.

What should I do if I think I am having problems?
If you have unusual symptoms, pain, or any other problems while you are in the study, you should report them to the researchers right away. Telephone numbers where they can be reached are listed on the first page of this consent form.

This study is not expected to cause serious problems. However, if you have a sudden, serious problem, like difficulty breathing or severe pain, go to the nearest hospital emergency room, or call 911 (or the correct emergency telephone number in your area). Tell emergency personnel about any medications you are taking.

What are the possible benefits of this study?
If you agree to take part in this study, there may not be direct benefits to you. The researchers cannot guarantee that you will benefit from participation in this research. However, you will receive a Target gift card for your work. You will also receive a free screening of IQ and academic skills, which may be useful in academic planning. You will be provided with a written summary of your performance. This can help you learn more about your specific strengths and weaknesses in certain areas of academics and cognitive abilities.

We hope the information learned from this study will benefit others with childhood medulloblastoma in the future. Information gained from this research could lead to better understanding of the consequences of childhood medulloblastoma and better academic programming for survivors. Information gained from this research could also contribute to the improved identification of childhood medulloblastoma survivors in need of academic or cognitive interventions.

What options are available if I decide not to take part in this research study?
This is not a treatment study. You do not have to be part of it to get treatment for your condition.

Will I be paid if I take part in this research study?
Yes. If you take part in this research, you will be given the following:

- A Target gift card with a value of up to $20, to be received after completing the study procedures

There are no funds available to pay for transportation to and from the research center, lost time away from work and other activities, or lost wages.

Will my insurance provider or I be charged for the costs of any part of this research study?
No. Neither you, nor your insurance provider, will be charged for anything done only for this research study (i.e., the Screening Procedures, Experimental Procedures, or Monitoring/Follow-up Procedures described above).

However, the standard medical care for your condition (care you would have received whether or not you were in this study) is your responsibility (or the responsibility of your insurance provider or governmental program). You will be charged, in the standard manner, for any procedures performed for your standard medical care.

What will happen if I am harmed as a result of taking part in this study?
It is important that you report any illness or injury to the research team listed at the top of this form immediately.

Compensation for an injury resulting from your participation in this research is not available from the University of Texas Southwestern Medical Center at Dallas or Children's Medical Center.

You retain your legal rights during your participation in this research.

Can I stop taking part in this research study?
Yes. If you decide to participate and later change your mind, you are free to stop taking part in the research study at any time.

If you decide to stop taking part in this research study, it will not affect your relationship with the UT Southwestern staff or doctors. Whether you participate or not will have no effect on your legal rights or the quality of your health care.

If you are a medical student, fellow, faculty, or staff at the Medical Center, your status will not be affected in any way.

Your doctor is a research investigator in this study. He is interested in both your medical care and the conduct of this research study. At any time, you may discuss your care with another doctor who is not part of this research study. You do not have to take part in any research study offered by your doctor.

If I agree to take part in this research study, can I be removed from the study without my consent?

Yes. The researchers may decide to take you off this study if:

- Your medical problem remains unchanged or becomes worse.
- The researchers believe that participation in the research is no longer safe for you.
- The researchers believe that other treatment may be more helpful.
- The sponsor or the FDA stops the research for the safety of the participants.
- The sponsor cancels the research.
- You are unable to keep appointments or to follow the researcher's instructions.

Will my information be kept confidential?

Information about you that is collected for this research study will remain confidential unless you give your permission to share it with others, or if we are required by law to release it. You should know that certain organizations that may look at and/or copy your medical records for research, quality assurance, and data analysis include:

- Children's Medical Center;
- Representatives of government agencies, like the U.S. Food and Drug Administration (FDA), involved in keeping research safe for people; and
- The UT Southwestern Institutional Review Board.

In addition to this consent form, you will be asked to sign an "Authorization for Use and Disclosure of Protected Health Information." This authorization will give more details about how your information will be used for this research study, and who may see and/or get copies of your information.

Whom do I call if I have questions or problems?

For questions about the study, contact Dr. Pete Stavinoha at 214-456-8197 during regular business hours and at 214-418-6498 after hours and on weekends and holidays.

For questions about your rights as a research participant, contact the UT Southwestern Institutional Review Board (IRB) Office at 214-648-3060.

SIGNATURES:

YOU WILL BE GIVEN A COPY OF THIS CONSENT FORM TO KEEP.

Your signature below certifies the following:

- You have read (or been read) the information provided above.
- You have received answers to all of your questions and have been told whom to call if you have any more questions.
- You have freely decided to participate in this research.
- You understand that you are not giving up any of your legal rights.

_____

Participant's Name (printed)

_____ _____

Participant's Signature Date

_____

Legally Authorized Representative's Name (printed)

_____ _____

Legally Authorized Representative's Signature Date

_____

Name of person obtaining consent (printed)

_____ _____

Signature of person obtaining consent Date

ASSENT OF A MINOR:

I have discussed this research study with my parent or legal guardian and the researchers, and I agree to participate.

_____ _____

Signature of participant (age 10 through 17) Date

INTERPRETER STATEMENT:

I have interpreted this consent form into a language understandable to the participant and the participant has agreed to participate as indicated by his or her signature on the associated short form.

_____

Name of Interpreter (printed)

_____

Date

_____

Signature of Interpreter

BIBLIOGRAPHY

Aarsen, F. K., Paquier, P. F., Arts, W., Van Veelen, M., Michiels, E., Lequin, M., & Catsman-Berrevoets, C. E. (2009). Cognitive deficits and predictors 3 years after diagnosis of a pilocytic astrocytoma in childhood. *Journal of Clinical Oncology*, 27(21), 3526-3532.

Aarsen, F. K., Paquier, P. F., Reddingius, R. E., Streng, I. C., Arts, W. M., Evera Preesman, M., & Catsman-Berrevoets, C. E. (2006). Functional outcome after low-grade astrocytoma treatment in childhood. *Cancer*, 106, 396-402.

Aarsen, F. K., Van Dongen, H. R., Paquier, P. F., Van Mourik, M., Catsman Berrevoets, C. E. (2004). Long-term sequelae in children after cerebellar astrocytoma surgery. *Neurology*, 62, 1311-1316.

Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for the ASEBA school-age forms & profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families.

Ahles, T. A., & Saykin, A. J. (2007). Candidate mechanisms for chemotherapy-induced cognitive changes. *Nature Reviews Cancer*, 7, 192-201.

Akyuz, C., Varan, A., Kupeli, S., Akalan, N., Soylemezoglu, F., Zorlu, F., Kutluk, T., & Buyukpamukcu, M. (2008). Medulloblastoma in children: A 32-year experience from a single institution. *Journal of Neuro-Oncology*, 90, 99-103.

Amabile, T. M., Hill, K. G., Hennessey, B. A., & Tighe, E. M. (1994). The Work Preference Inventory: Assessing intrinsic and extrinsic motivational orientations. *Journal of Personality and Social Psychology*, 66(5), 950-967.

Anderman, E. M., & Maehr, M. L. (1994). Motivation and schooling in the middle grades. *Review of Educational Research*, 64, 287-309.

Anderson, F., & Kunin-Batson, A. (2009). Neurocognitive late effects of chemotherapy in children: The past 10 years of research on brain structure and function. *Pediatric Blood and Cancer*, 52, 159-164.

Ater, J. L., Moore, B. D., Francis, D. J., Castillo, R., Slopis, J., & Copeland, D. R. (1996). Correlation of medical and neurosurgical events with neuropsychological status in children at diagnosis of astrocytoma: Utilization of neurological severity score. *Journal of Clinical Neurology*, 11(6), 462-469.

Ater, J. L., van Eys, J., Woo, S. Y., Moore, B., Copeland, D. R., & Bruner, J. (1997). MOPP chemotherapy without irradiation as primary postsurgical therapy for brain tumors in infants and young children. *Journal of Neurooncology*, 32, 243-252.

Bahl, G., Urbach, S., Bartels, U., Hodgson, D. C., Millar, B., Parent, A., Le, L., Awrey, S., & Laperriere, N. (2009). Endocrine complications in children treated for medulloblastoma or ependymoma using radiation therapy: Outcomes in the CT-planning era. *Journal of Clinical Oncology*, 27(15s), 10064.

Beebe, D. W., Ris, D. M., Armstrong, F. D., Fontanesi, J., Mulhern, R., Holmes, E., & Wisoff, J. H. (2005). Cognitive and adaptive outcome in low-grade pediatric cerebellar astrocytomas: Evidence of diminished cognitive and adaptive functioning in national collaborative research studies (CCG 9891/POG 9130). *Journal of Clinical Oncology*, 23(22), 5198-5204.

Bernard, L. C., Mills, M., Swenson, L., & Walsh, R. P. (2005). An evolutionary theory of human motivation. *Genetic, Social, and General Psychology Monographs*, 131(2), 129-184.

Berns, G. S., McClure, S. M., Pagnoni, G., & Montague, P. R. (2001). Predictability modulates human brain response to reward. *Journal of Neuroscience*, 21, 2793-2798.

Berti, A. E., & Bombi, A. S. (1981). The development of the concept of money and its value: A longitudinal study. *Child Development*, 52(4), 1179-1182.

Bjork, J. M., Knutson, B., Fong, G. W., Caggiano, D. M., Bennett, S. M., & Hommer, D. W. (2004). Incentive-elicited brain activation in adolescents: Similarities and differences from young adults. *Journal of Neuroscience*, 24(8), 1793-1802.

Blair, R. J. (2003). Facial expressions, their communicatory functions and neuro cognitive substrates. *Philosophical Transactions of the Royal Society of London*, 358(1431), 561-572.

Blaney, S. M., Kun, L. E., Hunter, J., Rorke-Adams, L. B., Lau, C., Strother, D., & Pollack, I. F. (2006). Tumors of the central nervous system. In P. A. Pizzo & D. G. Poplack (Eds.), *Principles and practices of pediatric oncology* (5th ed., pp. 786-864). Philadelphia: Lippincott Williams & Wilkins.

Boman, K. K., Hoven, E., Anclair, M., Lannering, B., & Gustafsson, G. (2009). Health and persistent functional late effects in adult survivors of childhood CNS tumours: A population-based cohort study. *European Journal of Cancer*, 45(14), 2552-2561.

Bowers, D. C., Liu, Y., Leisenring, W., McNeil, E., Stovall, M., Gurney, J. G., et al. (2006). Late-occurring stroke among long-term survivors of childhood leukemia and brain tumors: A report from the Childhood Cancer Survivor Study. *Journal of Clinical Oncology*, 24, 5277-5282.

Bracke-Tolkmitt, R., Linden, A., Canavan, A. G. M., Rockstroh, B., Scholz, E., Wessel, K., & Diener, H. C. (1989). The cerebellum contributes to mental skills. *Behavioral Neuroscience*, 103(2), 442-446.

Briere, M.-E., Scott, J. G., McNall-Knapp, R., & Adams, R. L. (2008). Cognitive outcome in pediatric brain tumor survivors: Delayed attention deficit at long-term follow-up. *Pediatric Blood and Cancer*, 50, 337-340.

Brookhart, S. M., Walsh, J. M., & Zientarski, W. A. (2006). The dynamics of motivation and effort for classroom assessments in middle school science and social studies. *Applied Measurement in Education*, 19(2), 151-184.

Bull, K. S., Spoudeas, H. A., Yadegarfar, G., & Kennedy, C. R. (2007). Reduction of health status 7 years after addition of chemotherapy to craniospinal irradiation for medulloblastoma: A follow-up study in PNET 3 trial survivors on behalf of the CCLG (formerly UKCCSG). *Journal of Clinical Oncology*, 25(27), 4239-4245.

Bunge, S. A., Dudukovic, N. M., Thomason, M. E., Vaidya, C. J., & Gabrieli, J. D. E. (2002). Immature frontal lobe contributions to cognitive control in children: Evidence from fMRI. *Neuron*, 33, 301-311.

Bush, G. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Science*, 4, 215-222.

Butler, R. W. & Haser, J. K. (2006). Neurocognitive effects of treatment for childhood cancer. *Mental Retardation and Developmental Disabilities Research Reviews*, 12, 184-191.

Butler, R., Kerr, M., & Marchand, A. (1999). Attention and executive functions following cranial irradiation in children. *Journal of the International Neuropsychological Society*, 5, 108.

Cameron, J. (2001). Negative effects of reward on intrinsic motivation: A limited phenomenon: Comment on Deci, Koestner, and Ryan (2001). Review of Educational Research, 71(1), 29-42.

Cameron, J., & Pierce, W. D. (1994). Reinforcement, reward, and intrinsic motivation: A meta-analysis. *Review of Educational Research*, 64, 363-423.

Casey, B. J., Tottenham, N., & Fossella, J. (2002). Clinical, imaging, lesion, and genetic approaches toward a model of cognitive control. *Developmental Psychobiology*, 40(3), 237-254.

Chakrabarti, B., Bullmore, E., & Baron-Cohen, S. (2006). Empathizing with basic emotions: Common and discrete neural substrates. *Social Neuroscience*, 1(3), 364-384.

Chelune, G. J., Naugle, R. I., Luders, H., Sedlak, J., & Awad, I. A. (1993). Individual change after epilepsy surgery: Practice effects and base-rate information. *Neuropsychology*, 7(1), 41-52.

Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry*, 50, 975-990.

Codding, R. S., Baglici, S., Gottesman, D., Johnson, M., Kert, A. S., Lebeouf, P. (2009). Selecting intervention strategies: Using brief experimental analysis for mathematics problems. *Journal of Applied School Psychology*, 25(2), 146-168.

Cohen, M. E., Schoene-Bake, J. C., Elger, C. E., & Weber, B. (2009). Connectivity based segregation of the human striatum predicts personality characteristics. *Nature Neuroscience*, 12, 32-34.

Conover, W. J., & Iman, R. L. (1982). Analysis of covariance using the rank transformation. *Biometrics, 38*, 715-724.

Copeland, D. R., deMoor, C., Moore, B. D. III, & Ater, J. L. (1999). Neurocognitive development of children after a cerebellar tumor in infancy: A longitudinal study. *Journal of Clinical Oncology*, 17, 3476-3486.

Curran, E. K., Le, G. M., Sainani, K. L., Propp, J. M., & Fisher, P. G. (2009a). Do children and adults differ in survival from medulloblastoma? A study from the SEER registry. *Journal of Neuro-Oncology*, 95, 81-85.

Curran, E. K., Sainani, K. L., Le, G. M., Propp, J. M., & Fisher, P. G. (2009b). Gender affects survival for medulloblastoma only in older children and adults: A study from the surveillance epidemiology and end results registry. *Pediatric Blood and Cancer*, 52, 60-64.

D'Agata, V., Drago, F., Serapide, F., & Cicirata, F. (1993). Effects of cerebellectomy on motivation-related behavior: A time-course study. *Physiology and Behavior*, 53, 173-176.

Daum, I., Ackermann, H., Schugens, M. M., Reimold, C., Dichgans, J., & Birbaumer, N. (1993). The cerebellum and cognitive function in humans. *Behavioral Neuroscience*, 107, 411-419.

Davis, K. D., Winsler, A., & Middleton, M. (2006). Students' perceptions of rewards for academic performance by parents and teachers: Relations with achievement and motivation in college. *The Journal of Genetic Psychology*, 167(2), 211-220.

deCharms, R. (1968). *Personal causation: The internal affective determinants of behavior*. New York: Academic Press.

Deci, E. L. (1971). Effects of externally mediated rewards on intrinsic motivation. *Journal of Personality and Social Psychology*, 18, 105-115.

Deci, E. L. (1972a). Effects of contingent and non-contingent rewards and controls on intrinsic motivation. *Organizational Behavior and Human Performance*, 8, 217-229.

Deci, E. L. (1972b). Intrinsic motivation, extrinsic reinforcement, and inequity. *Journal of Personality and Social Psychology*, 22, 113-120.

Deci, E. L., Koestner, R., & Ryan, R. M. (1999). A meta-analytic review of experiments examining the effects of extrinsic rewards on intrinsic motivation. *Psychological Bulletin*, 125, 627-668 discussion 692-700.

Deci, E. L., Koestner, R., & Ryan, R. M. (2001). Extrinsic rewards and intrinsic motivation in education: Reconsidered once again. *Review of Educational Research*, 71, 1-27.

Deci, E. L., & Ryan, R. M. (1985). *Intrinsic motivation and self-determination in human behavior*. New York: Plenum.

Deci, E. L. & Ryan, R. M. (1991). A motivational approach to the self: integration in personality. In R.A. Dienstbier (Ed.). *Perspectives on motivation*. Nebraska Symposium on Motivation, 1990. Vol. 38. Lincoln: University of Nebraska Press. 237-88.

Delgado, M. R., Stenger, V. A., & Fiez, J. A. (2004). Motivation-dependent responses in the human caudate nucleus. *Cerebral Cortex*, 14(9), 1022-1030.

Dennis, M., Hetherington, C. R., & Spiegler, B. J. (1998). Memory and attention after childhood brain tumors. *Medical and Pediatric Oncology Supplement* (1), 25-33.

Dennis, M., Spiegler, B. J., Hetherington, C. R., & Greenberg, M. L. (1996). Neuropsychological sequelae of the treatment of children with medulloblastoma. *Journal of Neurooncology*, 29(1), 91-101.

Desmond, J. E. (2001). Cerebellar involvement in cognitive function: Evidence from neuroimaging. *International Review of Psychiatry*, 13, 283-294.

Dolezal-Oltarzewska, K., Wojcik, M., Korab-Chrzanowska, E., Kwiatkowski, S., & Starzyk, J. (2009). Endocrine sequelae after treatment for childhood medulloblastoma. *Endocrinology, Obesity, and Metabolic Disorders*, 5(1), 1-6.

Donders, J. (2005). *Performance on the Test of Memory Malingering in a mixed pediatric sample*. Child Neuropsychology, 11, 221-227.

Duckworth, A. L., Quinn, P. D., Lynam, D. R., Loeber, R., & Stouthamer-Loeber, M. (2011). Role of test motivation in intelligence testing. *Proceedings of the National Academy of Sciences, 108*(19), 7716-7720.

Duffner, P. K. (2004). Long-term effects of radiation therapy on cognitive and endocrine function in children with leukemia and brain tumors. *The Neurologist*, 10, 293-310.

Duffner, P. K., Cohen, M. E., Voorhess, M. L., MacGillivray, M. H., Brecher, M. L., Panahon, A., & Gilani, B. B. (1985). Long-term effects of treatment on endocrine function in children with brain tumors: A prospective study. *Cancer*, 56, 2189-2193.

Eccles, J. S., & Midgley, C. (1990). Changes in academic motivation and self-perceptions during early adolescence. In R. Montemayer, G. R. Adams, & T. P. Gullotta (Eds.), *Early adolescence as a time of transition* (pp. 1-29). Beverly Hills, CA: Sage Publishing Co.

Eckert, T. L., Ardoin, S. P., Daly, E. J., III, & Martens, B. K. (2002). Improving oral reading fluency: A brief experimental analysis of combining an antecedent intervention with consequences. *Journal of Applied Behavior Analysis*, 35, 271-281.

Eisenberger, R., & Cameron, J. (1996). Detrimental effects of reward: Reality or myth? *American Psychologist*, 51, 1153-1166.

Engelmann, J. B., Damaraju, E., Padmala, S., & Pessoa, L. (2009). Combined effects of attention and motivation on visual task performance: Transient and sustained motivational effects. *Frontiers in Human Neuroscience*, 3, 1-17.

Engelmann, J. B., & Pessoa, L. (2007). Motivation sharpens exogenous spatial attention. *Emotion*, 7(3), 668-674.

Epstein, J. L., & McPartland, J. M. (1976). The concept and measurement of quality of school life. *American Educational Research Journal*, 13, 15-30.

Estle, S. J., Green, L., Myerson, J., & Holt, D. D. (2007). Discounting of monetary and directly consumable rewards. *Psychological Sciences*, 18(1), 58-63.

Exner, C., Weniger, G., & Irle, E. (2004). Cerebellar lesions in the PICA but not SCA territory impair cognition. *Neurology*, 63, 2132-2135.

Fiez, J. A., Petersen, S. E., Cheney, M. K., & Raichle, M. (1992). Impaired non-motor learning and error detection associated with cerebellar damage. *Brain*, 115, 155-178.

Filley, C. M., & Kleinschmidt-DeMasters, B. K. (2001). Toxic leukoencephalopathy. *New England Journal of Medicine*, 345, 425-432.

Fisher, M. J., Lange, B. J., Needle, M. N., Janss, A. J., Shu, H. G., Adamson, P. C., & Phillips, P. C. (2004). Amifostine for children with medulloblastoma treated with cisplatin-based chemotherapy. *Pediatric Blood and Cancer*, 43, 780-784.

Fisk, A. D., Fisher, D. L., & Rogers, W. A. (1992). General slowing alone cannot explain age-related search effects: Reply to Cerella (1991). *Journal of Experimental Psychology: General*, 120, 215-223.

Floyd, R. G., Shaver, R. B., & McGrew, K. S. (2003). Interpretation of the Woodcock-Johnson III Tests of Cognitive Abilities: Acting on evidence. In F. A. Schrank & D. P. Flanagan (Eds.), *WJ III clinical use and interpretation: Scientist-practitioner perspectives* (pp. 3-37). San Diego: Academic Press.

Fuss, M., Poljanc, K., & Hug, E. B. (2000). Full Scale IQ (FSIQ) changes in children treated with whole brain and partial brain irradiation: A review and analysis. *Strahlenther und Onkologie*, 176(12), 573-581.

Garcia-Perez, A., Narbona-Garcia, J., Sierrasesumaga, L., Aguirre-Ventallo, M., & Calvo-Manuel, F. (1993). Neuropsychological outcome of children after radiotherapy for intracranial tumours. *Developmental Medicine and Child Neurology*, 35, 139-148.

Garcia-Perez, A., Sierrasesumaga, L., Narbona-Garcia, J., Calvo-Manuel, F., & Aguirre-Ventallo, M. (1994). Neuropsychological evaluation of children with intracranial tumors: Impact of treatment modalities. *Medical and Pediatric Oncology*, 23, 116-123.

George, A. P., Kuehn, S. M., Vassilyadi, M., Richards, P. M. P., Parlow, S. E., Keene, D. L., & Ventureya, E. C. G. (2003). Cognitive sequelae in children with posterior fossa tumors. *Pediatric Neurology*, 28(1), 42-47.

Ginsberg, G. S. & Bronstein, P. (1993). Family factors related to children's intrinsic/extrinsic motivational orientation and academic performance. *Child Development*, 64, 1461-1474.

Gioia, G.A., Isquith, P.K., Guy, S. C., & Kenworthy, L. (2000). *Behavior Rating Inventory of Executive Function: Professional manual*. Lutz, FL: Psychological Assessment.

Goldwein, J. W., Radcliffe, J., Johnson, J., Moshang, T., Packer, R. J., Sutton, L. N., Rorke, L. B., & D'Angio, G. J. (1996). Updated results of a pilot study of low dose craniospinal irradiation plus chemotherapy for children under five with cerebellar primitive neuroectodermal tumors (medulloblastoma). *International Journal of Radiation Oncology, Biology, Physics*, 34, 899-904.

Gomez-Perez, E., Ostrosky-Solis, F., & Prospero-Garcia, O. (2003). Desarrollo de la atencion, la memoria, y los procesos inhibitorios: Relacion temporal con maduracion de la estructura y function cerebral. *Revista de Neurologia*, 37, 561-567.

Gottardo, N. G., & Gajjar, A. (2006). Current therapy for medulloblastoma. *Current Treatment Opinions in Neurology*, 8(4), 319-334.

Gottwald, B., Wilde, B., Mihajilovic, Z., & Medhorn, H. M. (2004). Evidence for distinct cognitive deficits after focal cerebellar lesions. *Journal of Neurology, Neurosurgery, and Psychiatry*, 75, 1524-1531.

Gray, J. A. (1987). *The psychology fear and stress* (2nd ed.). Cambridge, UK: Cambridge University Press.

Gregg, N., Coleman, C., & Knight, D. (2003). Use of the Woodcock-Johnson III in the diagnosis of learning disabilities. In F. A. Schrank & D. P. Flanagan (Eds.), *WJ III clinical use and interpretation: Scientist-practitioner perspectives* (pp. 126-167). San Diego: Academic Press.

Gregg, N., Hoy, C., Flaherty, D. A., Norris, P., Coleman, C., Davis, M., & Jordan, M. (2005). Decoding and spelling accommodations for postsecondary students with dyslexia: It's more than processing speed. *Learning Disabilities: A Contemporary Journal*, 3(2), 1-17.

Grill, J., Renaux, V. K., Bulteau, C., Viguier, D., Levy-Piebois, C., Sainte-Rose, C., Dellatolas, G., Raquin, M. A., Jambaque, I., & Kalifa, C. (1999). Long-term intellectual outcome in children with posterior fossa tumors according to radiation doses and volumes. *International Journal of Radiation Oncology, Biology, Physics*, 45(1), 137-145.

Grill, J., Viguier, D., Kieffer, V., Bulteau, C., Sainte-Rose, C., Hartmann, O., Kalifa, C., & Dellatolas, G. (2004). Critical risk factors for intellectual impairment in children with posterior fossa tumors: the role of cerebellar damage. *Journal of Neurosurgery*, 101(2 Supplement), 152-158.

Grunberg, N. E., & Anthony, B. J. (1980). Monetary awareness in children. *Basic and Applied Social Psychology*, 1(4), 343-350.

Gurney, J.G., Wall, D.A., Jukich, P.J., & Davis, F.G. (1999). The contribution of nonmalignant tumors to CNS tumor incidence rates among children in the United States. *Cancer Causes and Control*, 10, 101-105.

Haladyna, T., & Thomas, G. (1979). The attitudes of elementary school children toward school and subject matters. *Journal of Experimental Education*, 48, 18-23.

Hardin, M. G., Schroth, E., Pine, D. S., & Ernst, M. (2007). Incentive-related modulation of cognitive control in health, anxious, and depressed adolescents: Development and psychopathology related differences. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 48(5), 446-454.

Hardy, K. K., Bonner, M. J., Willard, V. W., Watral, M. A., & Gururangan, S. (2008). Hydrocephalus as a possible additional contributor to cognitive outcome in survivors of pediatric medulloblastoma. *Psychooncology*, 17(11), 1157-1161.

Hare, T. A., & Casey, B. J. (2005). The neurobiology and development of cognitive and affective control. *Cognition, Brain, Behavior*, 9(3), 273-286.

Hare, T. A., Tottenham, N., Davidson, M. C., Glover, G. H., & Casey, B. J. (2005). Contributions of amygdala and striatal activity in emotion regulation. *Biological Psychiatry*, 57(6), 624-632.

Harter, S. (1978). Effectance motivation reconsidered: Toward a developmental model. *Human Development*, 1, 34-64.

Harter, S. (1981). A new self-report scale of intrinsic versus extrinsic orientation in the classroom: Motivational and informational components. *Developmental Psychology*, 17, 300-312.

Harter, S., & Jackson, B. K. (1992). Trait vs. nontrait conceptualizations of intrinsic/extrinsic motivational orientation. *Motivation and Emotion*, 16, 209-230.

Heaton, R. K., Temkin, N. R., & Dikmen, S. S. (2001). Detecting change: A comparison of three neuropsychological methods using normal and clinical samples. *Archives of Clinical Neuropsychology*, 16, 75-91.

Hermann, E. J., Rittierodt, M., & Krauss, J. K. (2008). Combined transventricular and supracerebellar infratentorial approach preserving the vermis in giant pediatric posterior fossa midline tumors. *Neurosurgery*, 63(1 Suppl. 1), ONS30-35.

Hinton-Bayre, A. D. (2010). Deriving reliable change statistics from test-retest normative data: Comparison of models and mathematical expressions. *Archives of Clinical Neuropsychology*, 25, 244-256.

Hockenberry, M. J., Krull, K., Moore, I. M., Pasvogel, A., Gregurich, M., & Kaemingk, K. (2006). Longitudinal evaluation of fine motor deficits in children with leukemia. *Journal of Clinical Oncology*, ASCO Annual Meeting Proceedings Part I, 24(18S), 8559.

Hoppe-Hirsch, E., Brunet, L., Laroussinie, F., Cinalli, G., Pierre-Kahn, A., Renier, D., Sainte-Rose, C., & Hirsch, J. F. (1995). Intellectual outcome in children with malignant tumors of the posterior fossa: Influence of the field of irradiation and quality of surgery. *Child's Nervous System*, 11(6), 340-345.

Hoppe-Hirsch, E., Renier, D., Lellouch-Tubiana, A., Sainte-Rose, C., Pierre-Kahn, A., & Hirsch, J. F. (1990). Medulloblastoma in childhood: Progressive intellectual deterioration. *Child's Nervous System*, 6(2), 60-65.

Horner, M. J., Ries, L. A. G., Krapcho, M., Neyman, N., Aminou, R., Howlader, N., Altekruse, S.F., Feuer, E.J., Huang, L., Mariotto, A., Miller, B. A., Lewis, D. R., Eisner, M. P., Stinchcomb, D. G., Edwards, B. K. (eds). (2009). SEER Cancer Statistics Review, 1975-2006, National Cancer Institute. Bethesda, MD, http://seer.cancer.gov/statfacts/html/brain.html, based on November 2008 SEER data submission, posted to the SEER web site.

Hubbard, J. L., Scheithauer, B. W., Kispert, D. B., Carpenter, S. M., Wick, M. R., & Laws, E. R. (1989). Adult cerebellar medulloblastomas: The pathological, radiographic, and clinical disease spectrum. *Journal of Neurosurgery*, 70, 536-544.

Ito, M. (1984). *The cerebellum and neural control*. New York: Raven Press.

Ivry, R. B., & Baldo, J. V. (1992). Is the cerebellum involved in learning and cognition? *Current Opinion in Neurobiology*, 2, 212-216.

Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology*, 59, 12-19.

Jain, N., Krull, K. R., Brouwers, P., Chintagumpala, M. M., & Woo, S. Y. (2008). Neuropsychological outcome following intensity-modulated radiation therapy for pediatric medulloblastoma. *Pediatric Blood and Cancer*, 51(2), 275-279.

Jazbec, S., McClure, E., Hardin, M., Pine, D. S., & Ernst, M. (2005). Cognitive control under contingencies in anxious and depressed adolescents: An antisaccade task. *Biological Psychiatry*, 58(8), 632-639.

Johnson, D. L., McCabe, M. A., Nicholson, H. S., Joseph, A. L., Getson, P. R., Byrne, J., Brasseux, C., Packer, R. J., & Reaman, G. (1994). Quality of long-term survival in young children with medulloblastoma. *Journal of Neurosurgery*, 80, 1004-1010.

Jones, K. M., Wickstrom, K. F., Noltemeyer, A. L., Brown, S. M., Schuka, J. R., & Therrien, W. J. (2009). An experimental analysis of reading fluency. *Journal of Behavioral Education*, 18, 35-55.

Kail, R. (1986). Sources of age differences in speed of processing. *Child Development*, 57, 969-987.

Kail, R. (1988). Developmental functions for speeds of cognitive processes. *Journal of Experimental and Child Psychology*, 45, 339-364.

Kail, R. (1991). Developmental change in speed of processing during childhood and adolescence. *Psychological Bulletin*, 109, 490-501.

Kalivas, P. W., & Barnes, C. D. (1993). *Limbic motor circuits and neuropsychiatry*. Boca Raton, FL: CRC Press.

Kandel, E., Schwartz, J., & Jessell, T. (2000). *Principles of Neural Science*. New York: McGraw-Hill.

Kao, G. D., Goldwein, J. W., Schultz, D. J., Radcliffe, J., Sutton, L., & Lange, B. (1994). The impact of perioperative factors on subsequent intelligence quotient deficits in children treated for medulloblastoma/posterior fossa primitive neuroectodermal tumors. *Cancer*, 74(3), 965-971.

Karatekin, C., Lazareff, J. A., & Asarnow, R. F. (2000). Relevance of the cerebellar hemispheres for executive function. *Pediatric Neurology*, 22(2), 106-112.

Kawashima, M., Matsushima, T., Nakahara, Y., Takase, Y., Masuoka, J., & Ohata, K. (2009). Trans-cerebellomedullary fissure approach with special reference to lateral route. *Neurosurgical Review*, 32(4), 457-464.

Keating, D. P., & Bobbitt, B. L. (1978). Individual and developmental differences in cognitive processing components of mental ability. *Child Development*, 49, 155-169.

Kellogg, J. X., & Piatt, J. H. (1997). Resection of fourth ventricle tumors without splitting the vermis: The cerebellomedullary fissure approach. *Pediatric Neurosurgery*, 27(1), 28-33.

Khong, P. L., Leung, L. H. T., Fung, A. S. M., Fong, D. Y. T., Qui, D., Kwong, D. L. W., Ooi, G.-C., McAlanon, G., Cao, G., & Chan, G. C. F. (2006). White matter anisotropy in post-treatment childhood cancer survivors: Preliminary evidence of association with neurocognitive function. *Journal of Clinical Oncology*, 24, 884-890.

Kieffer-Renaux, V., Bulteau, C., Grill, J., Kalifa, C., Viguier, D., & Jambaque, I. (2000). Patterns of neuropsychological deficits in children with medulloblastoma according to craniospatial irradiation doses. *Developmental Medicine and Child Neurology*, 42(11), 741-745.

Kieffer-Renaux, V., Viguier, D., Raquin, M.A., Laurent-Vannier, A., Habrand, J.L., Dellatolas, G., Kalifa, C., Hartmann, O., & Grill, J. (2005). Therapeutic schedules influence the pattern of intellectual decline after irradiation of posterior fossa tumors. *Pediatric Blood and Cancer*, 45(6), 814-819.

Kimmings, E., Kleinlugtebeld, A. T., Casey, A., & Hayward, R. D. (1995). Medulloblastoma: Factors influencing the educational potential of surviving children. *British Journal of Neurosurgery*, 9(5), 611-617.

Kirkwood, M.W., & Kirk, J. W. (2010). The base rate of suboptimal performance in a pediatric mild TBI sample: Performance on the medical symptom validity test. *The Clinical Neuropsychologist, 24*, 860-872.

Kirkwood, M. W., Kirk, J. W., Blaha, R. Z., & Wilson, P. (2010). Noncredible effort during pediatric neuropsychological exam: A case series and literature review. *Child Neuropsychology, 16*(6), 604-618.

Kirsch, P., Schienle, A., Stark, R., Sammer, G., Blecker, C., Walter, B., Ott, U., Burkart, J., & Vaitl, D. (2003). Anticipation of reward in a nonaversive differential conditioning paradigm and the brain reward system: An event-related fMRI study. *NeuroImage*, 20, 1086-1095.

Knutson, B., Adams, C. M., Fong, G. W., & Hommer, D. (2001). Anticipation of increasing monetary reward selectively recruits nucleus accumbens. *Journal of Neuroscience*, 21, RC159.

Kobayashi, S., Lauwereyns, J., Koizumi, M., Sakagami, M., & Hikosaka, O. (2002). Influence of reward expectation on visuospatial processing in macaque lateral prefrontal cortex. *Journal of Neurophysiology*, 87, 1488-1498.

Kohls, G., Peltzer, J., Herpertz-Dahlmann, B., & Konrad, K. (2009). Differential effects of social and non-social rewards on response inhibition in children and adolescents. *Developmental Science*, 12(4), 614-625.

Kohn, A. (1996). By all available means: Cameron and Pierce's defense of extrinsic motivators. *Review of Educational Research*, 66, 1-4.

Kombogiorgas, D., Sgouros, S., Walsh, A. R., Hockley, A. D., Stevens, M., Grundy, R., Peet, A., English, M., & Spooner, D. (2007). Outcome of children with posterior fossa medulloblastoma: A single institution experience over the decade 1994-2003. Childs Nervous System, 23, 399-405.

Konczak, J., Schoch, B., Dimitrova, A., Gizewski, E., & Timmann, D. (2005). Functional recovery of children and adolescents after cerebellar tumour resection. *Brain*, 128, 1428-1441.

Kruglanski, A. W., Friedman, I., & Zeevi, G. (1971). The effects of extrinsic incentive on some qualitative aspects of task performance. *Journal of Personality*, 39, 606-617.

Lafay-Cousin, L., Bouffet, E., Hawkins, C., Amid, A., Huang, A., & Mabbott, D. J. (2009). Impact of radiation avoidance on survival and neurocognitive outcome in infant medulloblastoma. *Current Oncology*, 16(6), 21-28.

Latham, G. P., & Kinne, S. B. (1974). Improving job performance through training in goal setting. *Journal of Applied Psychology*, 59, 187-191.

Latham, G. P., & Yukl, G. A. (1975). Assigned versus participative goal setting with educated and uneducated wood workers. *Journal of Applied Psychology*, 60, 299-302.

Lazaroff, J., & Castro-Sierra, E. (1996). Preoperative and postoperative analysis of visual and auditory memory in children with cerebellar tumors. *Child's Nervous System*, 12, 81-86.

Lea, S. E., & Webley, P. (2006). Money as tool, money as drug: The biological psychology of a strong incentive. *The Behavioral and Brain Sciences*, 29(2), 161-176; Discussion 176-209.

Lee, T., Locke, E., & Phan, S. (1997). Explaining the assigned goal-incentive interaction: The role of self-efficacy and personal goals. *Journal of Management*, 23, 541-559.

Leiner, H. C., Leiner, A. L., & Dow, R. S. (1986). Does the cerebellum contribute to mental skills? *Behavioral Neuroscience*, 100, 443-454.

Leiner, H. C., Leiner, A. L., & Dow, R. S. (1989). Reappraising the cerebellum: What does the hindbrain contribute to the forebrain? *Behavioral Neuroscience*, 103(5), 998-1008.

Leonard, S., Bower, C., Petterson, B., & Leonard, H. (1999). Medical aspects of school-aged children with Down syndrome. *Developmental Medicine and Child Neurology*, 41, 683-688.

Lepper, M. R., Corpus, J. H., & Iyengar, S. S. (2005). Intrinsic and extrinsic motivational orientations in the classroom: Age differences and academic correlates. *Journal of Educational Psychology*, 97(2), 184-196.

Lepper, M. R., Greene, D., & Nisbett, R. E. (1973). Undermining children's intrinsic interest with extrinsic reward: A test of the "overjustification" hypothesis. *Journal of Personality and Social Psychology*, 28, 129-137.

Lepper, M. R., Henderlong, J., & Gingras, I. (1999). Understanding the effects of extrinsic reward on intrinsic motivation—Uses and abuses of meta-analysis: Comment on Deci, Koestner, and Ryan (1999). *Psychological Bulletin*, 125, 669-676.

Lepper, M. R., Keavney, M., & Drake, M. (1996). Intrinsic motivation and extrinsic rewards: A commentary on Cameron and Pierce's meta-analysis. Review of Educational Research, 66(1), 5-32.

Lepper, M. R., Sethi, S., Dialdin, D., & Drake, M. (1997). Intrinsic and extrinsic motivation: A developmental perspective. In S. S. Luthar, J. A. Burack, D. Ciccheti, & J. R. Weisz, Eds., *Developmental Psychopathology: Perspectives on Adjustment, Risk, and Disorder*. New York: Cambridge University Press.

Levisohn, L., Cronin-Golomb, A., & Schmahmann, J. (2000). Neuropsychological consequences of cerebellar tumour resection in children: Cerebellar cognitive affective syndrome in a paediatric population. *Brain*, 123, 1041-1050.

Lincoln, A., & Chazan, S. (1979). Perceived competence and intrinsic motivation in learning disability children. *Journal of Clinical Child Psychology*, 8, 213-216.

Linke, J., Kirsch, P., King, A. V., Gass, A., Hennerici, M. G., Bongers, A., & Wessa, M. (2010). Motivational orientation modulates the neural response to reward. *Neuroimage*, 49(3), 2618-2625.

Locke, H. S. & Braver, T. S. (2008). Motivational influences on cognitive control: Behavior, brain activation, and individual differences. *Cognitive Affective Behavioral Neuroscience*, 8, 99-112.

Locke, E. A., & Latham, G. P. (1990). *A theory of goal setting and task performance*. Englewood Cliffs, NJ: Prentice Hall.

Locke, E. A., & Latham, G. P. (2002). Building a practically useful theory of goal setting and task motivation: A 35-year odyssey. *American Psychologist*, 57(9), 705-717.

Mabbott, D. J., Noseworthy, M. D., Bouffet, E., Rockel, C., & Laughlin, S. (2006). Diffusion tensor imaging of white matter after cranial irradiation in children for medulloblastoma: Correlation with IQ. *Neuro-Oncology*, 8(3), 244-252.

Mabbott, D. J., Penkman, L., Witol, A., Strother, D., & Bouffet, E. (2008). Core neurocognitive functions in children treated for posterior fossa tumors. *Neuropsychology*, 22(2), 159-168.

Mabbott, D. J., Spiegler, B. J., Greenberg, M. L., Rutka, J. T., Hyder, D. J., Bouffet, E. (2005). Serial evaluation of academic and behavioral outcome after treatment with cranial radiation in childhood. *Journal of Clinical Oncology*, 23(10), 2256-2263.

Maddrey, A. M., Bergeron, J. A., Lombardo, E. R., McDonald, N. K., Mulne, A. F., Barenberg, P. D., & Bowers, D. C. (2005). Neuropsychological performance and quality of life of 10 year survivors of childhood medulloblastoma. *Journal of Neuro-Oncology*, 72, 245-253.

Malm, J., Kristensen, B., Karlsson, T., Carlberg, B., Fagerlund, M., & Olsson, T. (1998). Cognitive impairment in young adults with infratentorial infarcts. *Neurology*, 51(2), 433-440.

Marin, R. S. (1996). Apathy: Concept, syndrome, neural mechanisms, and treatment. *Seminars in Clinical Neuropsychiatry*, 1, 304-314.

Marin, R. S. & Wilkosz, P. A. (2005). Disorders of diminished motivation. *Journal of Head Trauma Rehabilitation*, 20(4), 377-388.

Maslow, A. H. (1954). *Motivation and personality*. New York: Harper.

Mather, N., & Woodcock, R. W. (2001). Examiner's Manual. *Woodcock-Johnson III Tests of Cognitive Abilities*. Itasca, IL: Riverside Publishing.

Maxwell, J. S., Shackman, A. J., & Davidson, R. J. (2005). Unattended facial expressions asymmetrically bias the concurrent processing of nonemotional information. *Journal of Cognitive Neuroscience*, 17(9), 1386-1395.

McCabe, M. A., Getson, P., Brasseux, C. & Johnson, D. L. (1995). Survivors of medulloblastoma: Implications for program planning. *Cancer Practice*, 3, 47-53.

McCauley, S. R., McDaniel, M. A., Pedroza, C., Chapman, S. B., & Levin, H. S. (2009). Incentive effects on event-based prospective memory performance in children and adolescents with traumatic brain injury. *Neuropsychology*, 23(2), 201-209.

McClure, S. M., Berns, G. S., & Montague, P. R. (2003). Temporal prediction errors in a passive learning task activate human striatum. *Neuron*, 38, 339-346.

McClure, S. M., York, M. K., & Montague, P. R. (2004). The neural substrates of reward processing in humans: The modern role of fMRI. *Neuroscientist*, 10, 260-268.

McGrew, K. S., & Woodcock, R. W. (2001). Technical Manual. *Woodcock-Johnson III Tests of Cognitive Abilities*. Itasca, IL: Riverside Publishing.

McSweeny, A. J., Naugle, R. I., Chelune, G. J., & Luders, H. (1993). "T scores for change": An illustration of a regression approach to depicting change in clinical neuropsychology. *The Clinical Neuropsychologist*, 7, 300-312.

Medina, D., Detoledo-Morrell, L., Urresta, F., Gabrieli, J. D., Moseley, M., Fleischman, D., Bennett, D. A., Leurgans, S., Turner, D. A., & Stebbins, G. T. (2006). White matter changes in mild cognitive impairment and AD: A diffusion tensor imaging study. *Neurobiology of Aging*, 27, 663-672.

Meece, J. L., Anderman, E. M., & Anderman, L. H. (2006). Classroom goal structure, student motivation, and academic achievement. *Annual Review of Psychology*, 57, 487-503.

Merchant, T. E., Kiehna, E. N., Miles, M. A., Zhu, J., Xiong, X., & Mulhern, R. K. (2002a). Acute effects of irradiation on cognition: Changes in attention on a computerized continuous performance test during radiotherapy in pediatric patients with localized primary brain tumors. *Journal of Radiation Oncology, Biology, Physics*, 53(5), 1271-1278.

Merchant, T. E., Lee, H., Zhu, J., Ziong, X., Wheeler, G., Phipps, S., Boop, F. A., & Sanford, R. A. (2004). The effects of hydrocephalus on intelligent quotient in children with localized infratentorial ependymoma before and after focal radiation therapy. *Journal of Neurosurgery (Pediatrics 2)*, 101, 159-168.

Merchant, T. E., Williams, T., Smith, J. M., Rose, S. R., Danish, R. K., Burghen, G. A., Kun, L. E., & Lustig, R. H. (2002b). Preirradiation endocrinopathies in pediatric brain tumor patients determined by dynamic tests of endocrine function. *International Journal of Radiation Oncology, Biology, Physics*, 54(1), 45-50.

Meyer, E. A. & Kieran, M. W. (2002). Psychological adjustment of 'surgery-only' pediatric neuro-oncology patients: A retrospective analysis. *Psychooncology*, 11(1), 74-79.

Meyers, J. E. & Diep, A. (2000). Assessment of malingering in chronic pain patients using neuropsychological tests. *Applied Neuropsychology*, 7, 133-139.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, 24, 167-202.

Mitby, P. A., Robison, L. L., Whitton, J. A., Zevon, M. A., Gibbs, I. C., Tersak, J. M., Meadows, A. T., Stovall, M., Zeltzer, L. K., & Mertens, A. C. (2003). Utilization of special education services and educational attainment among long-term survivors of childhood cancer. *Cancer*, 97(4), 1115-1126.

Mizuno, K., Tanaka, M., Ishii, A., Tanabe, H. C., Onoe, H., Sadato, N., & Watanabe, Y. (2008). *NeuroImage*, 42, 369-378.

Monje, M. L., Mizumatsu, S., Fike, J. R., & Palmer, T. D. (2002). Irradiation induces neural precursor-cell dysfunction. *Nature Medicine*, 8, 955-962.

Muirhead, S. E., Hsu, E., Grimard, L., & Keene, D. (2002). Endocrine complications of pediatric brain tumors: Case series and literature review. *Pediatric Neurology*, 27(3), 165-170.

Mulhern, R. K., Armstrong, F. D. & Thompson, S. J. (1998). Function-specific neuropsychological assessment. *Medical and Pediatric Oncology Supplement*, 1, 34-40.

Mulhern, R. K., Kepner, J. L., Thomas, P. R., Armstrong, F. D., Friedman, H. S., & Kun, L. E. (1998). Neuropsychologic functioning of survivors of childhood medulloblastoma randomized to receive conventional or reduced-dose craniospinal irradiation: A Pediatric Oncology Group study. *Journal of Clinical Oncology*, 16, 1723-1728.

Mulhern, R. K., Palmer, S. L., Reddick, W. E., Glass, J. O., Kun, L. E., Taylor, J., et al. (2001). Risks of young age for selected neurocognitive deficits in medulloblastoma are associated with white matter loss. *Journal of Clinical Oncology*, 2, 472-479.

Mulhern, R. K., Reddick, W. E., Palmer, S. L., Glass, J., Elkin, D., Kun, L. E., Taylor, J., Langston, J., & Gajjar, A. (1999). Neurocognitive deficits in medulloblastoma survivors and white matter loss. *Annals of Neurology*, 46, 834-841.

Mulhern, R. K., White, H. A., Glass, J. O., Kun, L. E., Leigh, L., Thompson, S. J., et al. (2004). Attentional functioning and white matter integrity among survivors of malignant brain tumors of childhood. *Journal of the International Neuropsychological Society, 10*, 180–189.

Mulhern, R. K., Palmer, S. L., Merchant, T. E., Wallace, D., Kocack, M., Brouwers, P., et al. (2005). Neurocognitive consequences of risk-adapted therapy for childhood medulloblastoma. *Journal of Clinical Oncology*, 23, 5511-5519.

Nagel, B. J., Delis, D. C., Palmer, S. L., Reeves, C., Gajjar, A., & Mulhern, R. K. (2006). Early patterns of verbal memory impairment in children treated for medulloblastoma. *Neuropsychology*, 20(1), 105-112.

Nagel, B. J., Palmer, S. L., Reddick, W. E., Glass, J. O., Helton, K. J., Wu, S., Xiong, X., Kun, L. E., Gajjar, A., & Mulhern, R. K. (2004). Abnormal hippocampal development in children with medulloblastoma treated with risk-adapted irradiation. *American Journal of Neuroradiology*, 25(9), 1575-1582.

Newman, R. S. (1990). Children's help-seeking in the classroom: The role of motivational factors and attitudes. *Journal of Educational Psychology*, 82, 71-80.

Noell, G. H., Freeland, J. T., Witt, J. C., & Gansle, K. A. (2001). Using brief assessments to identify effective interventions for individual students. *Journal of School Psychology*, 39, 335-355.

Nordeson, A., Engstrom, B., & Norberg, A. (1998). Self-reported quality of life for patients with progressive neurological diseases. *Quality of Life Research*, 7, 257-266.

O'Doherty, J. P. (2004). Reward representations and reward-related learning in the human brain: Insights from neuroimaging. *Current Opinion in Neurobiology*, 14, 769-776.

O'Doherty, J., Critchley, H., Deichmann, R., & Dolan, R. J. (2003). Dissociating valence of outcome from behavioral control in human orbital and ventral prefrontal cortices. *Journal of Neuroscience*, 23, 7931-7939.

O'Doherty, J. P., Deichmann, R., Critchley, H. D., & Dolan, R. J. (2002). Neural responses during anticipation of a primary taste reward. *Neuron*, 33, 815-826.

O'Donnell, W. E., De Soto, C. B., & De Soto, J. L. (1993). Validity and reliability of the revised Neuropsychological Impairment Scale (NIS). *Journal of Clinical Psychology*, 49, 372-382.

Oyharcabal-Bourden, V., Kalifa, C., Gentet, J. C., Frappaz, D., Edan, C., Chastagner, P., Sariban, E., Pagnier, A., Babin, A., Pichon, F., Neuenschwander, S., Vinchon, M., Bours, D., Mosseri, V., Le Gales, C., Ruchoux, M., Carrie, C., & Doz, F. (2005). Standard-risk medulloblastoma treated by adjuvant chemotherapy followed by reduce-dose craniospinal radiation therapy: A French Society of Pediatric Oncology study. *Journal of Clinical Oncology*, 23, 4726-4734.

Packer, R. J., Goldwein, J., Nicholson, H. S., Vezina, L. G., Allen, J. C., Ris, M. D., Muraszko, K., Rorke, L. B., Wara, W. M., Cohen, B. H., & Boyett, J. M. (1999). Treatment of children with medulloblastomas with reduc-eddose radiation therapy and adjuvant chemotherapy: A Children's Cancer Group Study. *J ournal of Clinical Oncology*, 17(7), 2127-2136.

Packer, R. J., Sutton, L. N., Atkins, T. E., Radcliffe, J., Bunin, G. R., D'Angio, G., Siegel, K. R., & Schut, L. (1989). A prospective study of cognitive function in children receiving whole-brain radiotherapy and chemotherapy: 2-year results. *Journal of Neurosurgery*, 70, 707-713.

Packer, R. J., Sutton, L. N., Elterman, R., Lange, B., Goldwein, J., Nicholsen, H. S., et al. (1994). Outcome for children with medulloblastoma treated with radiation and cisplatin, CCNU, and vincristine chemotherapy. *Journal of Neurosurgery*, 81, 690-698.

Padmala, S. & Pessoa, L. (2009). Interactions between cognition and motivation during response inhibition. *Neuropsychologia*, 48(2), 558-565.

Pailing, P. E. & Segalowitz, S. J. (2004). The error-related negativity as a state and trait measure: motivation, personality, and ERPs in response to errors. *Psychophysiology*, 41(1), 84-95.

Palmer, S. L. (2008). Neurodevelopmental impact on children treated for medulloblastoma: A review and proposed conceptual model. *Developmental Disabilities Research Reviews*, 14, 203-210.

Palmer, S. L., Goloubeva, O., Reddick, W. E., Glass, J. O., Gajjar, A., Kun, L., Merchant, T. E., Mulhern, R. K. (2001). Patterns of intellectual development among survivors of pediatric medulloblastoma: A longitudinal analysis. *Journal of Clinical Oncology*, 19(8), 2302-2308.

Palmer, S. L., Reddick, W. E., & Gajjar, A. (2007). Understanding the cognitive impact on children who are treated for medulloblastoma. *Journal of Pediatric Psychology*, 32(9), 1040-1049.

Pessoa, L. (2008). On the relationship between emotion and cognition. *Nature Reviews Neuroscience*, 9, 148-158.

Pessoa, L. (2009). How do emotion and motivation direct executive function? *Trends in Cognitive Sciences*, 13, 160-166.

Pochon, J. B., Levy, R., Fossati, P., Lehericy, S., Poline, J. B., Pillon, B., et al. (2002). The neural system that bridges reward and cognition in humans: An fMRI study. *Proceedings of the National Academy of Sciences*, 99, 5669-5674.

Pollack, I. F. (2001). Neurobehavioural abnormalities after posterior fossa surgery in children. *International Review of Psychiatry*, 13, 302-312.

Pollack, I. F., Polinko, P., Albright, A. L., Towbin, R., & Fitz, C. (1995). Mutism and pseudobulbar symptoms after resection of posterior fossa tumors in children: Incidence and pathophysiology. *Neurosurgery*, 37, 885-893.

Pompili, A., Caperle, M., Pace, A., et al. (2002). Quality-of-life assessment in patients who had been surgically treated for a cerebellar pilocytic astrocytoma in childhood. *Journal of Neurosurgery*, 96, 229-234.

Quastoff, S., & Hartung, H. P. (2002). Chemotherapy-induced peripheral neuropathy. *Journal of Neurology*, 249, 9-17.

Radcliffe, J., Bunin, G. R., Sutton, L. N., Goldwein, J. W., & Phillips, P. C. (1994). Cognitive deficits in long-term survivors of childhood medulloblastoma and other noncortical tumors: Age-dependent effects of whole-brain radiation. *International Journal of Developmental Neuroscience*, 12(4), 327-334.

Reddick, W. E., Glass, J., Helton, K., Langston, J., Xiong, X., Wu, S., & Pui, C. (2005). Prevalence of leukoencephalopathy in children treated for acute lymphoblastic leukemia with high-dose methotrexate. *American Journal of Neuroradiology*, 26, 1263-1269.

Reddick, W. E., Russell, J. M., Glass, J. O., Xiong, X., Mulhern, R. K., Langston, J. W., Merchant, T. E., Kun, L. E., & Gajjar, A. (2000). Subtle white matter volume differences in children treated for medulloblastoma with conventional or reduced dose craniospinal irradiation. *Magnetic Resonance Imaging*, 18(7), 787-793.

Reddick, W. E., White, H. A., Glass, J. O., Wheeler, G. C., Thompson, S. J., Gajjar, A., Leigh, L., & Mulhern, R. K. (2003). Developmental model relating white matter volume to neurocognitive deficits in pediatric brain tumor survivors. *Cancer*, 97, 2512-2519.

Reeves, C. B., Palmer, S. L., Reddick, W. E., Merchant, T. E., Buchanan, G. M., Gajjar, A., & Mulhern, R. K. (2006). Attention and memory functioning among pediatric patients with medulloblastoma. *Journal of Pediatric Psychology*, 31(3), 272-280.

Reimers, T. S., Ehrenfels, S., Mortensen, E. L., Schmiegelow, M., Sonderkaer, S., Carstensen, H., Schmiegelow, K., & Muller, J. (2003). Cognitive deficits in long-term survivors of childhood brain tumors: Identification of predictive factors. *Medical and Pediatric Oncology*, 40, 26-34.

Reitan, R., & Wolfson, D. (1985). *The Halstead-Reitan Neuropsychological Test Battery: Theory and clinical interpretation*. Tucson, AZ: Neuropsychology Press.

Reynolds, C. R., & Kamphaus, R. W. (2004). *BASC-2: Behavior Assessment System for Children, Second Edition.* Circle Pines, MN: American Guidance Service.

Riestra, A., Spaan, P. E. J., & Schmand, B. (2010). Validation of symptom validity tests using a "child-model" of adult cognitive impairments. *Archives of Clinical Neuropsychology, 25*(5), 371-382.

Rigby, C. S., Deci, E. L., Patrick, B. C., & Ryan, R. M. (1992). Beyond the intrinsic extrinsic dichotomy: Self-determination in motivation and learning. *Motivation and Emotion*, 16(3), 165-185.

Ris, M. D., & Abbey, R. (2010). Pediatric brain tumors. In K. O. Yeates, M. D. Ris, H. G. Taylor, & B. F. Pennington (Eds.), *Pediatric Neuropsychology* (2nd Ed., pp. 92-111). New York: Guilford Press.

Ris, M. D., Packer, R., Goldwein, J., Jones-Wallace, D., & Boyett, J. M. (2001). Intellectual outcome after reduced-dose radiation therapy plus adjuvant chemotherapy for medulloblastoma: A Children's Cancer Group study. *Journal of Clinical Oncology*, 19(15), 3470-3476.

Riva, D. (2000a). Cerebellar contributions to behavior and cognition in children. *Journal of Neurolinguistics*, 13, 215-225.

Riva, D. (2000b). The cerebellum contributes to higher functions during development: Evidence from acquired cerebellar lesions. In D. Riva & A. Benton (Eds.), *Localization of brain lesions and developmental functions*. New Barnet, UK: John Libbey & Company.

Riva, D. & Giorgi, C. (2000). The cerebellum contributes to higher functions during development: Evidence from a series of children surgically treated for posterior fossa tumours. *Brain*, 123, 1051-1061.

Riva, D., Milani, N., Pantaleoni, C., Ballerini, E., & Giorgi, C. (1991). Combine treatment modality for medulloblastoma in childhood: Effects on neuropsychological functioning. *Neuropediatrics*, 22, 36-42.

Riva, D., Pantaleoni, C., Milani, N., & Belani, F. F. (1989). Impairment of neuropsychological functions in children with medulloblastomas and astrocytomas in the posterior fossa. *Child's Nervous System*, 5, 107-110.

Robbins, T. W., Cador, M., Taylor, J. R., & Everitt, B. J. (1989). Limbic-striatal interactions in reward-related processes. *Neuroscience and Biobehavioral Reviews*, 13, 155-162.

Roesch, M. R., & Olson, C. R. (2005). Neuronal activity dependent on anticipated and elapsed delay in macaque prefrontal cortex, frontal and supplementary eye fields, and premotor cortex. *Journal of Neurophysiology*, 94, 1469-1497.

Roman, D. D., & Sperduto, P. W. (1995). Neuropsychological effects of cranial radiation: Current knowledge and future directions. *International Journal of Radiation Oncology*Biology*Physics*, 31(4), 983-998.

Roncadin, C., Dennis, M., Greenberg, M. L., & Spiegler, B. J. (2008). Adverse medical events associated with childhood cerebellar astrocytomas and medulloblastomas: Natural history and relation to very long-term neurobehavioral outcome. *Childs Nervous System*, 24, 995-1002.

Ronning, C., Sundet, K., Due-Tonnessen, B., Lundar, T., & Helseth, E. (2005). Persistent cognitive dysfunction secondary to cerebellar injury in patients treated for posterior fossa tumors in childhood. *Pediatric Neurosurgery*, 41, 15-21.

Rutkowski, S., Bode, U., Deinlein, F., Ottensmeier, H., Warmuth-Metz, M., Soerensen, N., et al. (2005). Treatment of early childhood medulloblastoma by postoperative chemotherapy alone. *New England Journal of Medicine*, 352, 978-986.

Ryan, R. M., & Deci, E. L. (1996). When paradigms clash: Comments on Cameron and Pierce's claim that rewards do not undermine intrinsic motivation. *Review of Educational Research*, 66, 33-38.

Ryan, R. M., & Deci, E. L. (2000). Intrinsic and extrinsic motivations: Classic definitions and new directions. *Contemporary Educational Psychology*, 25, 54-67.

Rypma, B., Berger, J. S., Genova, H. M., Rebbechi, D., & D'Esposito, M. (2005). Dissociating age-related changes in cognitive strategy and neural efficiency using event-related fMRI. *Cortex*, 41, 582-594.

Rypma, B., Berger, J. S., Prabhakaran, V., Bly, B. M., Kimberg, D. Y., Biswal, B. B., & D'Esposito, M. (2006). Neural correlates of cognitive efficiency. *NeuroImage*, 33, 969-979.

Rypma, B., & D'Esposito, M. (1999). The roles of prefrontal brain regions in components of working memory: Effects of memory load and individual differences. *Proceedings of the National Academy of Sciences*, 96, 6558.

Rypma, B., Prabhakaran, V., Desmond, J. E., & Gabrieli, J. D. E. (2001). Age differences in prefrontal cortical activity in working memory. *Psychology of Aging*, 16, 371-384.

Sansone, C., & Morgan, C. (1992). Intrinsic motivation and education: Competence in context. *Motivation and Emotion*, 16, 249-270.

Salthouse, T. A. (1992). What do adult age differences in the Digit Symbol Substitution Test reflect? *Journal of Gerontology*, 47, 121-128.

Sawamoto, N., Honda, M., Hanakawa, T., Fukuyama, H., & Shibasaki, H. (2002). Cognitive slowing in Parkinson's Disease: A behavioral evaluation independent of motor slowing. *Journal of Neuroscience*, 22, 5198-5203.

Schatz, F., Kramer, J. H., Ablin, A., & Matthay, K. K. (2000). Processing speed, working memory, and IQ: A developmental model of cognitive deficits following cranial radiation therapy. *Neuropsychology*, 14, 189-200.

Schmahmann, J. D. & Sherman, J. C. (1998). The cerebellar cognitive affective syndrome. *Brain*, 121, 1051-1061.

Schmidt, M. M., Copeland, D., & Fletcher, J. M. (1989). Effects of cranial radiation therapy (CRT) and age at diagnosis (DA) on neuropsychological performance in pediatric brain tumor patients. *Journal of Clinical and Experimental Neuropsychology*, 11, 30.

Schmithorst, V. J., Wilke, M., Dadzinski, B. J., & Holland, S. K. (2002). Correlation of white matter diffusivity and anisotropy with age during childhood and adolescence: A cross-sectional diffusion-tensor MR imaging study. *Radiology*, 222, 212-218.

Schultz, W. (2000). Multiple reward signals in the brain. *Nature Reviews Neuroscience*, 1, 199-207.

Schultz, W. (2004). Neural coding of basic reward terms of animal learning theory, game theory, microeconomics, and behavioural ecology. *Current Opinion in Neurobiology*, 14(2), 139-147.

Schultz, G. F., & Switzky, H. N. (1993). The academic achievement of elementary and junior high school students with behavior disorders and their nonhandicapped peers as a function of motivational orientation. *Learning and Individual Differences*, 5(1), 31-42.

Schutter, D., & Van Honk, J. (2005). The cerebellum on the rise in human emotion. *The Cerebellum*, 4, 290-294.

Siffert, J., Young Poussaint, T., Goumnerova, L. C., Scott, R. M., LaVally, B., Tarbell, N. J., Carpentieri, S., & Pomeroy, S. L. (2000). Neurological dysfunction associated with postoperative cerebellar mutism. *Journal of Neuro-Oncology*, 48, 75-81.

Silverman, C. L., Palkes, H., Talent, B., Kovnar, E., Clouse, J. W., & Thomas, P. R. (1984). Late effects of radiotherapy on patients with cerebellar medulloblastoma. *Cancer*, 54(5), 825-829.

Small, J. A., Kemper, S., & Lyons, K. (2000). Sentence repetition and processing resources in Alzheimer's disease. *Brain and Language*, 75, 232.

Sorce, J. F., Emde, R. N., Campos, J., & Klinnert, M. D. (1985). Maternal emotional signaling: Its effects on the visual cliff behavior of 1-year-olds. *Developmental Psychology*, 21(1), 195-200.

Sowell, E. R., Thompson, P. M., Holmes, C. J., Jernigan, T. L., & Toga, A. W. (1999). *In vivo* evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*, 2(10), 859-861.

Spiegler, B. J., Bouffet, E., Greenberg, M. L., Rutka, J. T., & Mabbott, D. J. (2004). Change in neurocognitive functioning after treatment with cranial radiation in childhood. *Journal of Clinical Oncology*, 22(4), 706-713.

Stavinoha, P. L. & Burrows, F. (2004). Academic speed in childhood brain tumor survivors treated with cranial radiation, *Brain Impairment*, 5, 99.

Steinlin, M., Imfeld, S., Zulauf, P., Boltshauser, E., Lovblad, K. O., Ridolfi Luthy, A., Perrig, W., & Kaufmann, F. (2003). Neuropsychological long-term sequelae after posterior fossa tumour resection during childhood. *Brain*, 126, 1998-2008.

Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (Fifth ed.). Boston: Pearson Education, Inc.

Tang, S., & Hall, V. C. (1995). The overjustification effect: A meta-analysis. *Applied Cognitive Psychology*, 9, 365-404.

Taylor, S. F., Welsh, R. C., Wager, T. D., Phan, K. L., Fitzgerald, K. D., & Gehring, W. J. (2004). A functional neuroimaging study of motivation and executive function. *NeuroImage*, 21, 1045-1054.

Thornton, A. E., Boudreau, V. G., Griffiths, S. Y., Woodward, T. S., Fawkes-Kirby, T., & Honer, W. G. (2007). The impact of monetary reward on memory in schizophrenia spectrum disorder. *Neuropsychology*, 21(5), 631-645.

Thut, G., Schultz, W., Roelcke, U., Nienhusmeier, M., Missimer, J., Maguire, R. P., & Leenders, K. L. (1997). Activation of the human brain by monetary reward. *NeuroReport*, 8, 1225-1228.

Timmann, D. & Daum, I. (2007). Cerebellar contributions to cognitive functions: A progress report after two decades of research. *The Cerebellum*, 6, 159-162.

Todd, A., & Ruge, J. (1999). Medulloblastoma – Late outcome. *Critical Reviews in Neurosurgery*, 9(3), 174-179.

Tsujimoto, S., & Sawaguchi, T. (2005). Neuronal activity representing temporal prediction of reward in the primate prefrontal cortex. *Journal of Neurophysiology*, 93, 3687-3692.

Tzuriel, D. (1989). Development of motivational and cognitive-informational orientations from third to ninth grades. *Journal of Applied Developmental Psychology*, 10, 107-121.

Van Dongen, H. R., Catsman-Berrevoets, C. E., Van Mourik, M. (1994). The syndrome of "cerebellar" mutism and subsequent dysarthria. *Neurology*, 44, 2040-2046.

Vaquero, E., Gomez, C. M., Quintero, E. A., Gonzalez-Rosa, J. J., & Marques, J. (2008). Differential prefrontal-like deficit in children after cerebellar astrocytoma and medulloblastoma tumor. *Behavioral and Brain Functions*, 4(18), **PAGES**.

Vernon, P. A. (1983). Speed of information processing and general intelligence. *Intelligence*, 7, 53-70.

Vohs, K. D., Mead, N. L., & Goode, M. R. (2006). The psychological consequences of money. *Science*, 314(5802), 1154-1156.

Watanabe, M. (1990). Prefrontal unit activity during associative learning in monkey. *Experimental Brain Research*, 80, 296-309.

Watanabe, M. (1996). Reward expectancy in primate prefrontal neurons. *Nature*, 382, 629-632.

Watanabe, M. (2002). Integration across multiple cognitive and motivational domains in monkey prefrontal cortex. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 326-337). New York: Oxford University Press.

Watanabe, M. (2007). Role of anticipated reward in cognitive behavioral control. *Current Opinion in Neurobiology*, 17, 213-219.

Weaver, A. H. (2005). Reciprocal evolution of the cerebellum and neocortex in fossil humans. *Proceedings of the National Academy of Sciences*, 102(10), 3576-3580.

Wechsler, D. (1999). *Wechsler Abbreviated Scale of Intelligence.* San Antonio, TX: The Psychological Corporation.

Wechsler, D. (2003). Wechsler Intelligence Scale for Children—Fourth Edition. San Antonio, TX: The Psychological Corporation.

Wechsler, D. (2008). Wechsler Adult Intelligence Scale—Fourth Edition. San Antonio, TX: Pearson.

Weich, K., Seymour, B., Kalisch, R., Enno Stephan, K., Koltzenburg, M., Driver, J., & Dolan, R. J. (2005). Modulation of pain processing in hyperalgesia by cognitive demand. *NeuroImage*, 27, 59-69.

West, R. L. (1996). An application of prefrontal cortex function theory to cognitive aging. *Psychological Bulletin*, 2, 272-292.

Wong, C. S., & Van der Kogel, A. J. (2004). Mechanisms of radiation injury to the central nervous system: Implications for neuroprotection. *Molecular Interventions*, 4, 273-284.

Woodcock, R. W., McGrew, K. S., & Mather, N. (2001a). *Examiner's manual: Woodcock-Johnson III Tests of Achievement*. Itasca, IL: Riverside Publishing.

Woodcock, R. W., McGrew, K. S., & Mather, N. (2001b). *Examiner's manual: Woodcock-Johnson III Tests of Cognitive Ability*. Itasca, IL: Riverside Publishing.

Zuzak, T. J., Poretti, A., Drexel, B., Zehnder, D., Boltshauser, E., & Grotzer, M. A. (2008). Outcome of children with low-grade cerebellar astrocytoma: long-term complications and quality of life. *Childs Nervous System*, 24, 1447-145.