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# Relationship between Executive Functioning and Parental Demographics in Autistic Probands

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# Relationship between Executive Functioning and Parental Demographics in Autistic Probands

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RELATIONSHIPS BETWEEN EXECUTIVE FUNCTIONING AND PARENTAL  
DEMOGRAPHICS IN AUTISTIC PROBANDS

By

Philip Menard

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Approved at Ypsilanti, Michigan, on this date \_\_\_\_\_

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## **Background and Significance**

Autism is a developmental disorder characterized by abnormalities in language, social relationships, and a repetitive, restricted behavioral repertoire involving reactions to the environment (Koczat, Rogers, Pennington, & Ross, 2002). It has been suggested that since the core symptom of autism is its social element, that this causes the accompanying language, communication, and unusual behavior often associated with it, but not always (Klin, Jones, Schultz, Volkmar, & Cohen, 2002). However, this appears to oversimplify these interactions by implying a directional cause between symptoms.

Autism is extremely prevalent with rates around 7.1 in 10,000 to 18.7 in 10,000 when including all pervasive developmental disorders (Fombonne, 1999). The male to female ratio for autism is around 3.8 to 1 (Fombonne, 1999) with this ratio remaining consistent through the Full IQ range (Starr et al., 2001).

Autism's etiology remains complex and difficult to piece together. With such multi-dimensional components and diversity in autism, these disorders require a multifaceted approach with genetic studies narrowing in on interacting genes and chromosomes (Volkmar, 2003). To guide research, a broader autism phenotype has slowly been developing to help isolate and understand the triad of impairments in autism. The broad autism phenotype (BAP) is a wider range of social and communicative deficits associated with restricted, repetitive behavioral patterns of a quality similar to the triad found in autism but to a milder degree (Starr et al., 2001). Variants are described as mild, in which abnormality is evident in only one area of the social, communicative, or repetitive behavioral components, and a severe variant in which abnormality is evident in

at least two of the three areas (Fombonne, Bolton, Prior, Jordon, & Rutter, 1997). This suggests a dimensional perspective unlike the categorical perspective used when diagnosing probands (Murphy et al., 2000). Hughes, Plummet, and LeBoyer (1999) have attributed variability to include genetic heterogeneity, genetic instability, and gene-environment interactions. Pickles et al., (2000) published evidence of an increased risk among relatives of autistic probands for the BAP with sex ratios remaining similar to that of AU diagnosis where an excess of males over females are affected by these disorders.

Following the triad found in probands, the communication component may be an easy first empirical indicator of the broader phenotype. This includes language delay, reading retardation, articulation disorder, spelling difficulties, and unusual conversation style. Folstein et al., (1999) suggested that the language component appears to be separate from the social component. In addition, Fombonne et al., (1997) suggests that a deficit in reading/spelling is not an index of the broad phenotype when occurring in isolation, however, it is strongly associated with a history of language difficulties and often exists in concordance with other dimensions of the BAP. The familial loading of the communication component of the BAP seems to be different among families of probands with and without speech. Strong associations have been found with a proband's symptom severity and with obstetric optimality which were not found among families of probands without speech (Pickles et al., 2000). Parents who reported a history of early language problems performed lower on verbal IQ, spelling, and pseudo word reading tests and were worse with pragmatic language than parents who did not report a history of early language problems (Folstein et al., 1999). In addition, parents without this history also showed a verbal IQ exceeding their performance IQ (Folstein et al.,

1999). Folstein et al.'s same study also examined siblings, finding similar results. Siblings who had a reported history of early language problems had worse verbal IQ, spelling, and reading than siblings without a reported history. Hughes et al.'s, (1999) study found that siblings showed superior verbal span but poor performance on verbal fluency tasks. Siblings with the BAP had lower IQ scores and performed poorly in reading and spelling (Fombonne et al., 1997). Though a deficit in reading/spelling may not be an index of the BAP, it appears that the proband's speech abilities and symptom severity is associated with parents and siblings who have histories of language difficulties performing lower on verbal IQ, spelling, and verbal fluency tasks.

Cognitive abnormalities associated with autism and its broader phenotype follow a triad made of weak central coherence, poor theory of mind, and executive dysfunction. Central coherence is the normal cognitive tendency to put a premium on the extraction of meaning, gist, and gestalt in information processing (Happe, Briskman, & Frith, 2001). Theory of mind is defined as the ability to think about mental states (one's own or another person's), and to reason about behavior in terms of underlying mental states (Baron-Cohen & Hammer, 1997). Executive functions are problem solving behaviors including the ability to form abstract concepts, have a flexible sequenced plan of action, focus, and sustain attention and mental effort, utilizing working memory, self-monitoring, self-correctability as a task is performed, and inhibit impulsive responses (Liss et al., 2001).

Since mental retardation is found in 2/3 of autistic probands (Rutter, Bailey, Simonoff, & Pickles, 1994) it is important to recognize this influence on cognitive abnormalities and its influence on the BAP. Mental retardation does not appear to affect

familial loading of the BAP but may contribute to cognitive disabilities associated with the BAP. Boutin et al., (1997) stated mental retardation may influence the etiological heterogeneity in autism. Starr et al., (2001) found that probands with profound mental retardation don't appear to increase the familial loading for the BAP. Using Wechsler scales, Fombonne et al., (1997) found no specific profile and no increased incidence of mental retardation among first degree relatives of autism. However, low IQ and female autistic probands have more first degree relatives with cognitive disabilities (Boutin et al., 1997). In addition, Starr et al., (2001) found that familial loading for scholastic achievement difficulty was significantly slightly higher when the autistic proband was severely retarded.

A weakness in central coherence may contribute to behavior becoming locked in detail dictating interests (Baron-Cohen & Hammer, 1997). Though Happe et al., (2001) did not find evidence of weak central coherence amongst siblings of probands, parents (especially fathers) were detail-focused across visual and verbal tasks with faster, more accurate performance on the block design and embedded figures tasks. In addition, fathers of Asperger's probands were significantly faster on embedded figures tasks (Baron-Cohen & Hammer, 1997). This suggests that fathers are more likely to exhibit behavior involving attention to detail.

Theory of Mind has been investigated with Baron-Cohen's 'Reading of the Mind's Eye' tasks. This requires the participant to guess the mental state of a human with only the eye region being presented in the form of a photograph (Baron-Cohen, Wheelwright, Hill, Rase, & Plumb, 2001). Using this measure, high functioning autism and Asperger's probands have been found to be slightly impaired as well as parents of



Asperger's probands (Baron-Cohen et al., 2001; Baron-Cohen & Hammer, 1997).

The possible link between central coherence and Theory of Mind has recently been examined. Scores of verbal weak central coherence measure and Theory of Mind measure are associated suggesting language processing of contextual information and social cognitive impairment are related with language ability being the common link (Burnette et al., 2005). When Verbal IQ was statistically controlled this became marginally significant suggesting language is not the only link (Burnette, et al., 2005).

Both central coherence and Theory of Mind offer possible influences of stereotyped behaviors. Since a weak central coherence seems to be offset with a strong attention to detail while having a deficit in the ability to empathize and utilize theory of mind, repetitive behaviors may be the result of success in these areas with the least amount of interaction. Central coherence and Theory of Mind are considered and tested in the exhibit of social situations but have not been dissected as to which components make up central coherence and Theory of Mind. In addition, central coherence and Theory of Mind have not been analyzed for exclusivity amongst disorders. This creates an unbalanced triad in which Theory of Mind has the least component analysis, followed by central coherence, and finally, executive functioning which has been dissected into many components and continues to with developing research.

Executive dysfunction (ED) is found in Autism, ADHD, Conduct Disorder, early treated phenylketonuria, OCD, Tourette's syndrome, and schizophrenia but different components of EF's are associated with different disorders (Ozonoff, South, & Provencal, 2005). For AU, inhibition remains intact but not in schizophrenia; AU's sustaining attention remains intact but not in ADHD; Tourette's, and schizophrenia, and

AU have deficits in shifting attention which is not found in ADHD (Ozonoff et al., 2005). Liss et al., (2001) suggests that executive dysfunction is unlikely to cause autistic behaviors or deficits in adaptive function because it is not universal in autism. However, it is predicted that executive dysfunctions do cause difficulty in novel or ambiguous situations while maintaining performance in routine or well-learned situations (Hughes, LeBoyer, & Bouvard, 1997).

Though EF cannot completely account for social disabilities, it appears that social cognition and EF are not completely separable. Following Frith's first mention of executive functioning in 1972, Boucher (1977) tested EF with a maze task and found AU are more likely to stick to one solution strategy suggesting shifting abnormalities. When using the Wisconsin Card Sorting Task (WCST) on high-functioning AU (HFAU) adult men, Rumsey (1985) found AU had deficits in the number of categories completed, total errors, perseverative responses, and perseverative errors. Two years later, Schneider and Asarnow (1987) used the same test on AU individuals finding no executive dysfunction. But in one year Rumsey and Hamburger (1988) reinforced Rumsey's 1985 finding when they used the WCST on AU with age matched norms and severely dyslexic individuals to find AU sorted fewer WCST categories which was not a general result of learning or development disorders. Simplified WCST's have been developed for AU children. Prior and Hoffman (1990) found AU children had more errors, perseverated responses, and performed worse on Milner's Maze Test against matched controls suggesting AU children had difficulty learning from mistakes. That same year, Szatmari, Tuff, Finlayson, & Batolucci (1990) used the WCST on HFAU individuals finding the HFAU had more perseverated errors and completed fewer categories against controls made up of

80% of ADHD and conduct disorders. To begin pulling apart all the different components used in the WCST, Ozonoff, et al., (1991) used the Tower of Hanoi on AU individuals which had fewer failures to maintain set than controls suggesting the opposite of perseveration. Minshew and Goldstein's (1992) use of the WCST found no executive dysfunction in AU but used the Goldstein-Shearer Object Sorting Test to show that AU were less able to shift set. Ozonoff and McEvoy (1994) tested the Tower of Hanoi and WCST showing its consistency over a two year period.

As research continued probing executive functions, tests like the WCST were criticized for being too vague and not narrowing observations of specific components of executive functioning. Though the WCST tests cognitive flexibility it also requires attribute identification, categorization, working memory, inhibition, selective attention, and encoding of verbal feedback (Ozonoff et al., 2005). To tease these components apart, tests have been developed and manipulated to focus on certain areas. To study flexibility and inhibition, Ozonoff & McEvoy developed a Go-NoGo task. This requires a response to a neutral stimulus while inhibiting responses to other neutral stimuli requiring no set shifting, a condition requiring inhibition of a previously reinforced response, and a condition requiring frequent shifting from one response pattern to another testing flexibility (Ozonoff & McEvoy, 1994). The Stop-Signal measure was developed to measure the control of a voluntary motor response without requiring flexibility by having subjects perform categorizing tasks while subsets included auditory signals to indicate responses should be inhibited (Ozonoff & Strayer, 1997). The Negative Priming Task has participants view a five letter string and asked whether the second and fourth letters were the same or different. This process is immediately followed with the same test in

which letters one, three and five are the focus. Slowed performance after the shift in stimuli focus is thought to be due to the subject's ability to actively inhibit attention to the distracting stimulus (Ozonoff, et al., 1997). A computerized set-shifting task is the Intradimensional-Extradimensional Shift Task of the Cambridge Neuropsychological Test Automated Battery (CANTAB ID/ED). This task requires set shifting by introducing new shapes to shapes already giving feedback as well as shifting to different stimuli (shapes) to receive feedback (Hughes, Russell, & Robins, 1994).

These tests of flexibility and inhibition suggest that there may not be an impairment involving inhibition. Ozonoff et al.'s (1994) use of the Go-NoGo task with high functioning autistic individuals suggested no impairment in inhibition compared to controls. That same year, Hughes et al.'s (1994) use of the CANTAB ID/ED task suggested that autistic individuals had deficits in extra-dimensional shifts (Hughes et al., 1994). Ozonoff and Strayer (1997) used the Stop-Signal and Negative Priming Task on high functioning autistic individuals finding no difference to age and IQ matched controls (Ozonoff et al., 1997) with these results being replicated in 2003 by Brian, Tipper, Weaver, & Bryson (Brian et al., 2003). 1997 also saw the replication of Hughes et al.'s 1994 finding of an extra-dimensional shift in autistic individuals except they also found this in retarded individuals and not autistics of normal IQ suggesting the extra-dimensional shift deficit may be linked to IQ and not autism. This was again replicated in Ozonoff et al.'s (2004) use of the CANTAB ID/ED on 79 autistic individuals and 70 'well matched' controls (Ozonoff, et al., 2004).

Another form of flexibility is attention shifting. To test sensory modalities, Courchesne, Akshoomoff, and Ciesielski (1990) had subjects monitor one modality until

an oddball target was detected and then shift to the other modality to find targets. ‘False alarms’ are when subjects fail to disengage from a modality while ‘misses’ are when subjects fail to switch modality attention quickly. Another task is the visuospatial orienting task of Posner (1980) in which two boxes are on either side of a central fixation cross on a computer screen. Targets appear in one of the boxes as subjects are to respond as quickly as possible. A visuospatial cue is presented just before the target appears indicating where attention should be directed as well as neutral and invalid cues in which it is not given or given in the wrong area. The Global-Local task, in which stimuli appear at either global (large stimuli) or local (smaller stimuli which construct global stimuli when put, together, requires shifting attention between stimulus levels on trial by trial basis (Rinehart, Bradshaw, Moss, Brereton, & Tonge 2001).

These tests of attention shifting suggest that autistic individuals have a deficit in disengaging as well as shifting between processing levels. Courchesne et al’s tests of sensory attention shifting found autistic individuals well below controls in both their 1990 and 1994 studies suggesting a problem at the disengaging operation (Courchesne et al., 1990; Courchesne et al., 1994). The visuospatial orientating task of Posner suggested through two studies that autistic individuals took longer to disengage attention (Wainwright-Sharp & Bryson, 1993; Casey, Gordon, Mannheim, and Rumsey, 1993). The Global-local task suggests autistic individuals are slower at shifting between processing levels and Berger, et al., have found that performance on set-shifting tasks are better able to predict social understanding and social competence than other cognitive domains in high functioning and adult autistic individuals (Reinhart, et al., 2001; Berger, Aerts, van Spaendonck, Cools, & Teunisse, 2003).

Working memory is the component of executive function that keeps information in an activated, online state to perform cognitive processing (Baddeley, 1986). To test verbal working memory, tests of counting and sentence span tasks are compared with tests of rote short-term memory, verbal long-term, and recognition memory (Bennetto, Pennington, & Rogers, 1996). Other tasks testing working memory are the dice-counting task and an odd-man-out task (Russell, Jarrold, & Henry, 1996), an n-back task, and a box search task (Ozonoff & Strayer, 2001). The n-back task has participants identify whether a digit on a computer screen is the same or different from the digit of one or two previous trials. The box-search task penalizes a return to locations already examined.

These tests of working memory suggest that autistic individuals have no working memory deficits. Though Bennetto, et al.'s (1996) use of sentence and counting span tasks suggest that autistic individuals are the same as controls concerning declarative memory (rote, verbal long term & recognition), they appear significantly impaired in working memory to age and IQ matched controls, Russell et al.'s use of dice-counting, odd-man-out, and sentence span test that same year suggested no difference from matched controls (Bennetto, et al., 1996; Russell et al, 1996). Mottron, Peretz, Belleville, & Rouleau's (1999) use of a case study of an autistic individual with mental retardation suggested deficit in flexibility but not working memory while Ozonoff and Strayer (2001) used three tests on high functioning autism, Tourette's, and matched controls finding no group differences. Most recently, Hala, Rasmussen, and Henderson (2005) found intact working memory in AU during source monitoring tasks and suggest that this overall impairment may be due to broader impairments in executive functioning.

Since these cognitive constructs are developmental it is important to know the

development of executive functioning from very early on when these skills first become acquired. Identifying when these deficits emerge requires a comparison with very young aged norms. Adapting tasks of executive functioning for use with young autistic individuals began with the use of measures of prefrontal function developed for nonhuman primates and human infants (Diamond & Goldman-Rakic, 1986). McEvoy, Rogers and Pennington (1993) found that autistic individuals had more perseverated errors than age and IQ matched norms with a mean age of 5yrs 4mo. When comparing younger autistic individuals with developmentally delayed individuals matched on age and both verbal and nonverbal mental age, Griffith, Pennington, Whener, & Rogers (1999) suggests that both groups performed worse than expected suggesting that executive dysfunction is associated with general developmental delay. However, Dawson et al. (2002) found no significant differences between even younger autistic individuals, developmentally delayed individuals, and controls. These findings suggest inconsistencies through the developing of executive functions during early preschool which may be due to different tests for different ages (Ozonoff, et al., 2005)

A recently developed instrument for a comprehensive neuropsychological assessment of children ages 3-12 years is A Developmental Neuropsychological Assessment (NEPSY) (Korkman, Kirk, & Kemp, 1998). It utilizes child-oriented materials and procedures to measure attention/executive function, language, memory, sensorimotor, and visuospatial skills (Korkman, et al., 1998). It is purported to have “developmental sensitivity” with neurocognitive development occurring more rapidly between 5 and 8 years than 9 to 12 years (Korkman, Kemp, and Kirk, 2001). Findings support concurrent validity though scores do not always consistently produce associations

with other tests (Schmitt & Wodrich, 2004). Schmitt and Wodrich's (2004) validity study of the NEPSY found data that supports the contention that NEPSY scores are distinct and justifies using the instrument to assess children with neurological or scholastic concerns when a diagnostic battery contains no IQ tests. Even when IQ differences were statistically controlled, the data continued to reveal group differences on the NEPSY (Schmitt & Wodrich, 2004).

How executive functioning affects social processes is essential to our understanding of possible determinants of the core symptoms associated with autism. Bennetto et al. (1996) offer a model of how social interaction depends on the ability to hold variable streams of context-specific information including subtle verbal and nonverbal cues, then plan and respond to the ever-changing stream appropriately. McEvoy et al. (1993) correlated executive functioning with measures of social interaction and joint attention in preschool autistic and control groups. The correlation between joint attention and social interaction were replicated by Griffith et al. (1999). Interestingly, Dawson, Meltzoff, Osterling, and Rinaldi (1998) did not replicate the same findings but found a correlation between executive performance and memory tasks. That same year Swettenham et al. (1998) found that young autistic individuals have more difficulty shifting attention between social and nonsocial stimuli suggesting the nature of the stimulus is involved in the impairment. Further research including locomotion and balance control in AU children found that the main component affected in AU children during locomotion is movement planning having a negative affect on the goal of the action, the orientation toward the goal and the definition of trajectory (Vernazza-Martin et al., 2005). Executive function has been found as the best predictor of restricted,



repetitive behavior in AU with cognitive flexibility, working memory, and response inhibition being highly related to the behavior (Lopez, Lincoln, Ozonoff, & Lai, 2005).

The relationship between executive functioning and theory of mind has produced three dominant theories. Theory of mind is the ability to think about mental states, both one's own or another person's, and to reason about behavior in terms of underlying mental states (Baron-Cohen & Hammer, 1997). First, it is thought that executive functions are necessary for exercising theory of mind. Second is the idea of executive functions and theory of mind sharing cognitive underpinnings and third is the idea that these two components are subserved by neural networks in the same brain region. Ozonoff, Pennington, & Rogers (1991) finding of correlations between executive functions and false belief tasks has been replicated many times (Perner & Lang, 1999). Hughes and Russell (1991, 1993) used tasks of strategic deception ability to find that autistic individuals had a deficit in disengaging from an object. However, evidence of young children at risk of ADHD having intact theory of mind but deficient executive functions suggests there is no clear explanation to the correlations between executive functions and theory of mind (Perner, Kain, & Barchfeld, 2002). Yet, when examining executive functions and social awareness, Ozonoff et al.'s (1995) report of the computerized WCST being easier for autistic individuals has been replicated by Pascualvaca, Fantie, Papageorgiou, & Mirsky (1998) and Griffith (2002). When examining executive functioning and theory of mind on the processing level, both appear to require recursive or sequential analysis of information and embedded rule use (Frye, Zelzo, & Palfai, 1995; Hughes, et al., 1993). Baker et al., (1996) and Dias, Robbins, & Roberts (1996) used imaging studies to show the role of the frontal cortex in executive

functions as well as during social-cognition tasks (Baron-Cohen et al., 1999; Fletcher et al., 1995; Happe, et al., 1996; Stone, Baron-Cohen, & Knight, 1998). In addition, Carlson, Mandell, and Williams (2004) analysis of Theory of Mind and executive functioning in 2-3 year olds address a process analysis of what is required for successful reasoning about mental states would suggest that some level of executive skill is needed. Russell (1996) suggests children must be able to disengage from salient but misleading stimuli in the environment and suppress their own potent representation of events before accurate reflections on the mental states of self and others can be achieved. Carlson, et al.'s longitudinal study found that executive functioning performance in children as young as 24 months significantly predicted performance on Theory of Mind tasks one year later as an asymmetrical relationship concluding that executive functioning is a potent contributor to developing a Theory of Mind. Children with better executive functioning skills are likely to have better social and communication skills and therefore more opportunity for observation and interaction that develops Theory of Mind (Carlson, et al., 2004).

The relationship of executive functioning to language and IQ is important for understanding how executive functioning can affect an individual's performance in social settings. Hughes (1996) developed a test using hand gestures with preschool autistic individuals suggests that the autistic individuals appear to have a failure to use language to control thought and behaviors because they could not imitate a simple hand gesture after being primed with a different one. With the comparison of tasks involving arbitrary or novel rules and others requiring only a verbal response, Russell, et al (1999) found no differences between autism, mixed developmental delay, and typical development

groups. They suggested that the autism group had a failure to verbally encode rules and use them to drive behavior. This has been reinforced with the findings of Joseph, McGrath, and Tager-Flusberg (2005) of no specific association between language ability and executive performance in AU that does exist in positive correlation in their nonautistic group. Compounding these interactions, Ozonoff et al. have repeatedly identified significant contributions of IQ to EF performance in autistic individuals (Miller & Ozonoff, 2000; Ozonoff & McEvoy, 1994,; Ozonoff & Strayer, 2001).

It appears executive dysfunction of the type within autism consists of difficulty in extra-dimensional shifting (related to retardation), in disengaging, shifting between processing levels, and shifting between social and nonsocial stimuli. Areas that appear intact include inhibition, working memory, and declarative memory. Executive functioning has been found to correlate with social interaction and joint attention (McEvoy et al., 1993; Griffith et al., 1999) as well as memory tasks (Dawson et al., 1998) and appears to be intertwined with the ability to exercise theory of mind by possibly requiring executive functions, having the same cognitive underpinnings, or utilizing the same neural networks.

Looking for executive dysfunctioning within the broader phenotype, siblings have been found to mimic the findings in probands performing poorly on set-shifting, planning, and verbal fluency tasks (Hughes, et al., 1999), and a significant proportion of parents, especially fathers, showed impaired executive functioning with poor planning skills (Hughes, et al., 1997). Attentional flexibility was evenly impaired between mothers and fathers (Hughes, et al., 1997). This supports executive dysfunction being partially under genetic control. Parents shared deficits of spatial working memory and

response inhibition components on delayed oculomotor response tasks (Koczat, et al., 2002). Scores on planning tasks correlated with scores for spatial short-term memory, rather than working memory, suggesting unusual strategies were being used to solve tasks requiring these functions (Hughes, et al., 1997).

The social component of the broader autism phenotype has been explored further through the use of personality testing and examination of real-life skills. For school age boys in the general population, reciprocal social behavior is highly heritable (Constantino & Todd, 2000). Among relatives of autistic probands, Murphy et al., (2000) found increased expressions of the traits anxious, impulsive, aloof, shy, over-sensitive, irritable, and eccentric. Social elements involved in the broad phenotype include the tendency to be withdrawn and difficult, with males being more withdrawn than female relatives (Murphy, et al., 2000). Tenseness, as being anxious and sensitive, was thought to possibly relate to the stress of caring for an autistic proband and therefore not included in the broader phenotype. Parents, especially fathers, preferred solitary past times that are detailed and factual with less interest or ability in social interaction, (Briskman, Happe, & Frith, 2001; Murphy, et al., 2000). The tendency toward certain types of activities may be the influence of weak central coherence, the lack of social interest, and relatively stronger abilities for more detail-focused interests (Briskman, et al., 2001). Though causality cannot be determined with these studies, the interplay of the components studied clearly interact within the broader phenotype of autism.

The BAP consists of communication disorders including a delay in onset of speech, trouble learning to read and spell, and articulation problems. Cognitive disorders include executive dysfunctions of poor planning skills and attentional flexibility, weak

central coherence, and possibly a lack of ‘theory of mind’. Social disorders include being withdrawn and difficult with preferences for detailed, non-social interests. The broader autism phenotype appears to be stronger in males coinciding with the rates of autism amongst genders.

The broad autism phenotype includes traits that serve as an advantage in different situations. The weak central coherence allows for focus like that which is required in science, math, and computations (DeLong, 2004). These tasks are based on our understanding of the physical world, and can allow for a weakness in the understanding of our social world (Wheelwright & Baron-Cohen, 2001). A combination of this with preferences for parts over whole, or local processing, attracts detail-focused work with little social interaction. This could suggest that occupations geared for this type of work like engineering would be more successful for those with the broader autism phenotype. Wheelwright and Baron-Cohen (2001) have found fathers and grandfathers of probands over-represented in engineering, accounting, and science. Reinforcing this is a negative correlation between fathers and skilled manual labor, under-representation in teaching, and suggestions of possible attractions to physics and computer science as well (Wheelwright & Baron-Cohen, 2001). How these components interact to form social understanding and interaction is still unclear, however, as executive functioning appears to contain the components necessary for these actions, executive functioning underlies the behavioral interactions in society. The ability to separate these components is difficult and much research is still needed.

Many elements may be contributing to the severity of the broad phenotype. The degree of the relative to the proband, and possibly the proband’s APGAR birth optimality

score, may contribute to severity (Pickles, et al., 2000). For probands with speech, there also appears to be familial loading of the broad phenotype related to the severity of autism (Pickles, et al., 2000). However, samples invariably do not include parents with the most severe component difficulties because of their unlikeliness to become parents. In addition, difficulties of measurement may contribute to the severity of the broad phenotype.

Outside influences may also compound the complexity of autism and its associated components. The research of major mood disorders and autism has offered many interesting and valuable findings. Lauritsen, et al., (2004) reported the relative risk of autism in the child was about twice as high if the mother had been diagnosed with a psychiatric disorder.

Depression is the most common psychiatric disorder accompanying autism (DeLong, 2004). Depression, anxiety, compulsive, attention, hyperactivity, and sleep problems are common in autistic individuals (Lainhart, 1999). Relatives have shown high rates of major depression and social phobia that is not associated with the broad phenotype (Piven & Palmer, 1999), with expression in twenty to thirty six percent of first degree relatives (Lainhart, 1999). Increased rates of depression are not confined to post-birth (Lainhart, 1999). Piven and Palmer (1999) suggest a possible association of parents with the broad phenotype being more likely to mate with someone with major depressive disorder. Neuroimaging has shown a lateralization of serotonin synthesis in autistic individuals that appears similar to depression and both show abnormal function in congruent areas in the brain (prefrontal cortex, left dominant, left amygdale, hippocampus, cingulated cortex, and cerebellum) (DeLong, 2004).

Murphy, et al., (2000) reported increased rates of generalized anxiety disorder among relatives of autistic individuals, but findings have been inconsistent (Lainhart, 1999). Bolton, Pickles, Murphy, and Rutter (1998) suggest the possibility of obsessive-compulsive disorder as an index for underlying liability to autism. Individuals with obsessive-compulsive disorder are more likely to exhibit autistic like social and communication impairments while motor tics and obsessive compulsive disorder are significantly more common in relatives (Bolton, et al., 1998). There is also a correlation between the autistic trait of ‘insistence of sameness’ and a family history of obsessive-compulsive disorder (DeLong, 2004).

In DeLong’s review of autism and familial major mood disorders, the sub-type ‘childhood bipolar autistic disorder’ is presented possibly changing the treatment diagnosis, shifting from autism to Asperger’s to bipolar disorder. Treatment results suggest neurotransmitter and receptor characteristics of autism must be similar to mood disorder (DeLong, 2004). Treatment for mood disorders can improve core symptoms of autism. Divalproex sodium and lithium seem to act as mood stabilizers, and SSRIs may diminish mutism, free utterances, and improve syntax, semantics, and pragmatics (DeLong, 2004). Atypical antipsychotics improve behavioral problems involved in autism, such as severe tantrums, aggression, and self-injury (DeLong, 2004). However, bipolar cannot be considered an element of the broad autism phenotype because it is not significantly prevalent in autistic relatives (Lainhart, 1999).

In an attempt to further our understanding of the broader autism phenotype, this study examined the relationship between executive functioning and parents demographics of autistic probands. Since it is unclear as to how cognitive components within autism

interact and are exhibited in social contexts, this study offers the comparison of specific executive functions and any possible relationship to behaviors exhibit by the parents. This may offer insight to which components of executive function may be correlated with parental education, occupations, and familial history of disorders. This may help in identifying more specific sub-types of AU as well as quicken the implantation of remediacy. A control population of children with learning disability will be compared to children with autistic spectrum disorder (Autism, Asperger's, PDDNOS). This may help differentiate between possible cognitive profiles of each group as well as identify parental traits that may suggest exclusivity to each group. With this analysis, we may be able to more clearly identify what components underlie social processes. Since executive functioning, central coherence, and theory of mind are so closely intertwined but address different areas of behavior, this could further our understanding of the etiology of the social deficits defining autism.



## **Specific Aims and Hypothesis:**

This study will expand on previous research by examining the broader phenotype of PDD in relation to parental demographics. Current suggestions of Baron-Cohen's 'extreme male mind' theory will also be considered as gender differences of probands and their executive functioning will be compared. Through furthering our understanding of the broader phenotype and characteristics found in practical social environments (occupation, education) we will improve our diagnostic specificity and be better able to formulate specific diagnostic interventions based on 'real world' data.

### **1. Executive dysfunction in PDD**

*Examine executive functioning of PDD in relation to LD. Examine how a comorbid diagnosis may influence executive functioning. Explore whether executive functioning may be a component of Baron-Cohen's 'extreme male mind' hypothesis.*

- A. PDD probands will perform poorly in executive functioning tasks in relation to LD
- B. Individuals with comorbid diagnosis will perform worse on tasks of executive functioning.
- C. Male probands will perform poorly in executive functioning tasks in relation to female probands.

### **2. Parental demographics and PDD**

*Examine whether PDD's implication of the broader phenotype is found in characteristics of the parents. Examining level of education, occupation, speech/language disorders, PDD, and mood disorders in parents of PDD*

*probands in relation to LD.*

- A. Mothers and fathers of the PDD group will have more education than LD.
- B. Mothers and fathers of the PDD group will have more engineering/science occupations than LD (especially fathers).
- C. Parents of the PDD group will report more history of PDD than LD.
- D. Parents of the PDD group will report more history of speech/language disorders than parents of LD.
- E. Parents of PDD will report more history of the mother having prior mood disorders than the LD.

**3. Parental demographics of PDD children and executive functioning**

*Examine the relationship between demographics of parents of PDD and the executive functioning of the probands.*

- A. Parents of PDD probands in engineering/science occupation have children who perform worse on tasks of executive functioning than PDD children of parents in different occupations and than parents of LD in engineering/science occupations.
- B. Parents of PDD probands with higher education have children who perform worse on tasks of executive functioning than the PDD children of parents with less education and than parents of LD with less education.
- C. Parents of PDD children with histories of speech/language disorders have children who perform worse on tasks of executive functioning than PDD children of parents without a history and than LD and Norm children of parents without a history.

- D. Parents of PDD children with histories of prior mood disorders have children who perform worse on tasks of executive functioning than PDD children of parents without a history and than parents of LD without a history.
- E. Parents of PDD children with higher education and in engineering/science occupations have children who perform worse on tasks of executive functioning than all other groups.

## **Method**

Participants. Participants included children with PDD, including Autism and Asperger's (N=27, 22 males; Mean age=7.74 yrs) and LD children (N=14, 11 males; Mean Age=9.57 yrs) who presented for neuropsychological evaluation at a large metropolitan hospital based clinic.

Neuropsychological Procedures/Protocol. Each subject underwent neuropsychological/developmental examinations assessing intellectual, pre-academic functioning, motor, perceptual-motor, visuospatial, speech/language, verbal and visual memory, attention, and executive functioning abilities. This data reflects results from the NEPSY neuropsychological exam and was performed by a clinical neuropsychologist, postdoctoral fellow in neuropsychology, or master's level psychologist. PDD diagnosis was based on DSM-IV criteria and evaluation with the Childhood Autism Rating Scale (CARS). LD diagnosis was based on DSM-IV criteria. A comprehensive interview was conducted with the parents in which demographic and family history data was obtained.

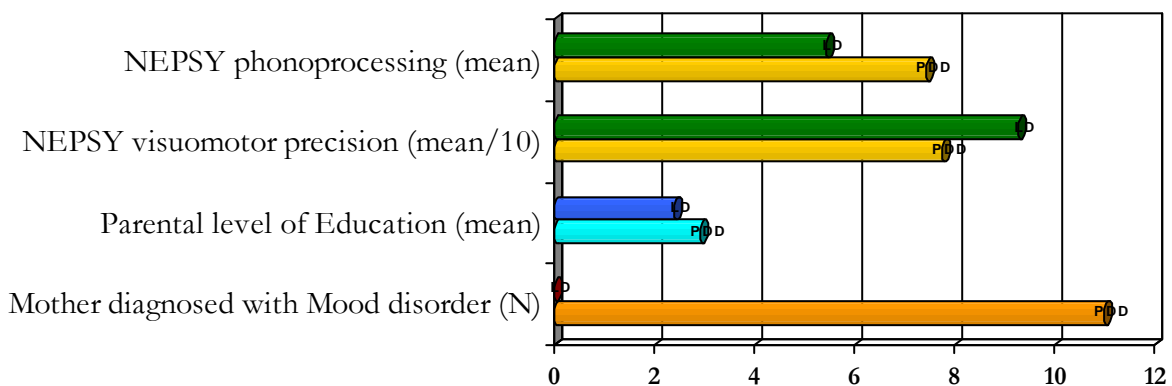
Data Analysis. Distributional qualities (age, gender, and intellectual functioning) of the participants were examined with either a t statistic or chi square. A t-statistic was used to examine group differences in all subtests of the NEPSY consisting of attention/executive domain, phono-processing, naming, comprehension, language domain, imitation of hands, visuomotor precision, sensorimotor domain, design copy, arrows, visual-spatial domain, memory faces, names, narrative memory, and memory domain.

## Results

PDD and LD groups did not differ significantly in Full Scale IQ or gender. The groups differed significantly on age ( $t=-2.296$ ;  $p=.027$ ), phono processing ( $t=2.157$ ,  $p=.037$ ), and visuomotor precision ( $t=-2.287$ ;  $p=.028$ ). When compared by gender no significant differences were found.

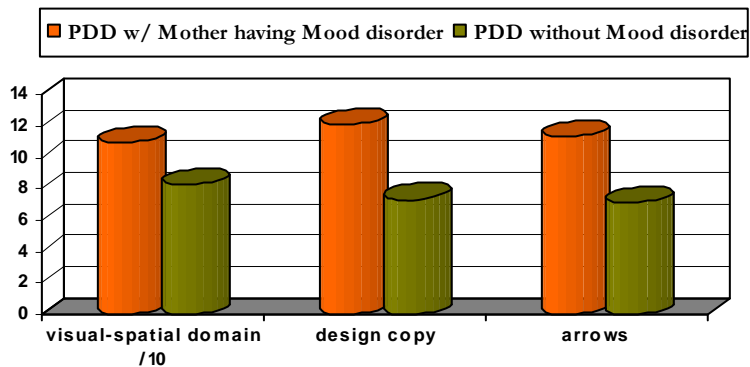
Comparing PDD and LD parental demographics, significant differences were found in parents level of education ( $t=2.171$ ;  $p=.036$ ) and mother's history of mood disorder ( $\chi^2=.038$ ). No significant differences were found between the groups in parental occupation, history of PDD, or history of speech/language disorder.

	PDD (N=29)	LD (N= 14)	<i>t</i>	<i>p</i>
Age (years)	7.48	9.57	2.575	.014
FSIQ	76.56	87.58	.906	.371
Male/Female ratio	3.83/1	3.67/1		



	PDD	LD		<i>p</i>
NEPSY visuomotor (mean)	5.83	8.07	$t=2.519$	.016
NEPSY phonoprocess (mean)	7.44	5.43	$t=2.157$	.037
Parental level of Education (mean)	2.9	2.4	$t=2.171$	.036
Mother Diagnosed with Mood Disorder (N)	11	0	$\chi^2=4.31$	.038

Comparing NEPSY scores of PDD probands whose mother has a mood disorder with PDD probands whose mother does not have a mood disorder found significant differences on the visual-spatial domain ( $t=3.245$ ;  $p=.004$ ) and in subtests of design copy ( $t=2.705$ ;  $p=.015$ ) and arrows ( $t=3.024$ ;  $p=.009$ ).



	PDD with Mother having Mood disorder	PDD without Mood disorder	<i>t</i>	<i>p</i>
NEPSY visual-spatial domain (mean)	109.86	82.31	3.245	.004
NEPSY design copy (mean)	12.14	7.33	2.705	.015
NEPSY arrows	11.29	7.1	3.024	.009

## **Discussion**

The first hypothesis that PDD probands will perform more poorly than LD probands on tasks of executive functioning was not confirmed. Comparing the additional domains included in the NEPSY battery, the PDD sensorimotor domain was significantly lower than the LD. Oculomotor tests suggest that individuals with PDD experience slowed visual pursuit which increases with age caused by intact processing mediated by a mechanism with weak central coherence (Takarai, Yukari, 2004). Phonological processing was significantly higher in the PDD group which is consistent with accelerated word recognition which may be the result of exquisite decoding and sight word reading.

The second hypothesis found that PDD parents have higher levels of education and that the mothers have significantly more Prior Mood disorders. Finding more mothers with Mood disorders amongst the PDD group was consistent with Lauritsen, et al.'s finding a higher risk for autism if a mother had been diagnosed with a psychiatric disorder and Delong's (2004) finding of depression being the most common. Though Piven and Palmer (1999) report higher rates not associated with the broad phenotype and even suggest parents with the broad phenotype being more likely to mate with someone with a depressive disorder, this has not been examined further. Delong reports of the lateralization of serotonin synthesis in autistic individuals appearing similar to depression with dysfunction in congruent areas in the brain (2004). This suggests that further analysis needs to be done comparing the neuropsychological battery performance of the individuals with PDD and their parents against others without diagnosed mood disorders and their parents. This study found that those individuals with PDD and a mother

diagnosed with a prior mood disorder have significantly higher scores than PDD individuals whose mother wasn't diagnosed with a prior mood disorder in all categories of the NEPSY visual-spatial domain, possibly identifying a sub-type existing within the PDD, autistic spectrum. This suggests that the mother's mood disorder serves as a moderating variable for this subtype.

In order to address internal validity and differentiate a visual-spatial subtype within PDD, further research needs to be completed with a control group of age matched norms. Utilizing effect size and logistical regression is needed to demonstrate model fit and predictive accuracy. In order to utilize a medium effect size of a  $\beta:\alpha$  ratio of 4:1 (.20:.05) the sample size of each group needs to exceed 87 ( $N>87$ ). Then Mean differences, significant  $r$ ,  $\chi^2$  (1 $df$ ), ANOVA (2 $ga$ ), and Multiple Regression analysis (4 $kb$ ), can determine the strength in difference while accounting for 4 independent variables.



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