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NEUROBIOLOGY, PRENATAL DEVELOPMENT, AND PRODIGIOUSNESS

The Neurobiological Foundations of Giftedness

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Case studies of extremely gifted individuals often reveal unique patterns of intellectual precocity and associated abnormalities in development and behavior. This article begins with a review of current neurophysiological and neuroanatomical findings related to the gifted population. The bulk of scientific inquiries provide evidence of unique patterns of right prefrontal cortex and inferior frontal activation implicated in gifted intelligence, although additional studies suggest enhanced neural processing and cerebral bilateralism. Geschwind, Behan, and Galaburda (GBG) first hypothesized the possible neurodevelopmental factors that account for unique brain development. This article explores more recent findings taken from the prenatal exposure literature and offers a proposed model for explaining aberrant developmental forces that may be at work in precocious individuals.

Keywords: prenatal exposures, gifted, biology, development, brain, neurology, intelligence, theory, intellectual precocity, prenatal neurodevelopment, genius

Educational and mental health professions have been pervasively interested in the deviant, those that represent the extreme ends of psychopathology, behavior, and ability. Individuals classified as "gifted" present with a unique set of abilities that set them apart from their peers, often from an early age. History is replete with countless examples of creative individuals whose unique talents and capabilities led to extraordinary accomplishments. The intrigue for the scientist is to determine what variables and characteristics enable eminently gifted persons to effortlessly achieve what others struggle to master.

The nature–nurture debate assumes a center role in the arguments surrounding the origins of giftedness. Common folklore tells us that highly gifted and creative individuals have some innate capabilities that facilitate their brilliance (i.e., genius is born, not made). Recent research also implicates the role of heredity in certain aspects of gifted cognitive ability (Posthuma, DeGeus, & Boomsma, 2001; Thompson, Cannon, & Toga, 2002). The opposing view challenges this assumption (i.e., Ericsson, Krampe, & Tesch-Romer, 1993) and argues that gifted abilities are more a product of effortful and deliberate practice. This view suggests that it is the individual who spends extraordinary time repeating, refining, and perfecting their skills who becomes exceptional (Bloom, 1985; Ericsson et al.). A more balanced perspective acknowledges that giftedness is likely a manifestation of a reciprocal relationship between genes and environment (LaBuda, DeFries & Fulker, 1987; Scarr & McCartney, 1974). Such discussions are not uncommon in the neuroscience literature regarding the heritability of disorders of the brain like schizophrenia and Alzheimer's disease (Saunders et al., 2003). The purpose of this article is to sidestep this exhaustive debate to explore the perspective that considers the neurological underpinnings of giftedness. Based upon evidence from neuroanatomical, neurophysiological, and neuropsychological dimensions of giftedness, we ask the question: What are the biological forces that may account for the emergence of the eminently gifted brain? Much has been written about the definitions and descriptors of giftedness, yet surprisingly few papers have sought to delineate the possible underlying neurological aspects and etiologies of gifted individuals. Our article begins with defining the scope of our thesis followed by a review of important historical theories and findings.

Articulating an accurate yet succinct definition of giftedness and talent has challenged theorists, researchers, and practitioners for decades (Kalbfleisch, 2004; Lubinski, Webb, Morelock, & Perrson Benbow, 2001; Robinson &

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Clinkenbeard, 1998). Furthermore, the terms *giftedness*, *talent*, *creativity*, and *genius* have often been used interchangeably in the literature to refer to exceptional individuals. Lewis Terman (1925) originally identified gifted individuals as those falling in the top 1% of general intellectual ability on standardized psychometric tests. Since then, general beliefs have evolved from narrow and mechanistic reliance on IQ performance (Ambrose, 2000) and moved toward broader definitions including those that involve traits, specific cognitive abilities, creativity, task commitment, achievement motivation, leadership potential, and even psychomotor ability (Feldhusen, 1986; Lubinski et al.; Robinson & Clinkenbeard; Winner, 2000). The U.S. Federal definition of gifted and talented individuals in the No Child Left Behind (NCLB) public law act of 2002 refers to individuals who give evidence of high achievement capability in such areas as intellectual, creative, artistic, leadership capacity, or specific academic fields. Thus, though multiple variables are used to describe and define giftedness, our focus will consider high cognitive performance as measured by psychometric scales. Our reference to giftedness is consistent with Kalbfleisch's definition of talent to extend beyond global IQ or psychometric *g* and include optimal cognitive performance and extraordinary ability in a domain-specific area. It is our contention that the tenets of our thesis apply to the highly and profoundly gifted populations using Gross's (2000) categorization, although we leave open the possibility of suitability for other populations of gifted individuals.

The idea that giftedness is associated with unusual or unique brain development can be highlighted by considering eminent historical figures. Leonardo Da Vinci, Sigmund Freud, Albert Einstein, and Pablo Picasso illustrate exceptional individuals whose extraordinary accomplishments will forever stand out in history. Yet the autobiographical and biographical accounts of these figures reveal patterns of aberrant behavior that also stood well outside the normal range of psychological functioning. These creative geniuses were plagued by pervasive affective and mood disturbances that are well documented (Ehrenwald, 1984). This interesting combination of extreme creativity and psychopathology has often led to the belief that creativity and madness may be invariably associated (Ehrenwald). Though scientific evidence supporting this link has not been substantive, there is reasonable evidence that aberrances of brain development would likely account for the atypical yet highly gifted accomplishments of these individuals.

Perhaps Albert Einstein represents the best example of how atypical brain functioning may influence giftedness. Much has been said about Einstein's aberrant development. He did not learn to talk until the age of 3 and his speech was not fluent until approximately age 10. He was not viewed as demonstrating early precocious behavior and, in fact, much has been made of his Greek teacher's comments that Einstein would not amount to anything. However, in time his precocious talent and originality emerged and he has often been called the greatest scientist of our time. Of interest, Einstein's accomplishments were not always immediately recognized because of his limitations with language. Einstein admitted in his autobiography that he thought with visual images rather than words (Hadamard, 1949). After his death, published postmortem investigations of Einstein's brain revealed a higher percentage of glial cells in select regions of the brain (Cardoso, 1997), greater neuronal density in the right cerebral cortex (Anderson & Harvey, 1996), and a larger corpus collosum (Witelson & Goldsmith, 1991). In addition, it was shown that Einstein's brain had extensive development of the inferior parietal region (Cardoso). This latter finding is important because this region of the brain is often associated with visuospatial cognition and advanced mathematical reasoning (O'Boyle et al., 2005). Thus, atypical brain development, although more commonly implicated in pathology, appears to play a role in giftedness and Einstein's anecdotal history represents a good example of this.

HISTORY

Early scientists postulated that there was a correlation between an individual's intellectual capabilities and the size of the brain. Franz Gall, the Father of Phrenology, initially suggested a link between external signs on the skull (protuberances) with size of the underlying portions of the cerebral hemispheres. Later, others hypothesized that the larger the brain, the greater number of neurons and neural connections and, hence, the greater the intellectual capacity. For instance, Karl Lashley's (1950) principle of "mass action" suggested that the brain worked as a unitary system. Lashley's experiments led him to observe that the amount of brain matter removed from a rat was correlated with a corresponding decrease in performance. This reinforced his belief that brain size played a determining role in overall capacity of the organism. Though Lashley's conclusions were subsequently determined to be erroneous due to imperfect lesioning methods, the idea that differences in brain morphology could be implicated in giftedness continues to be revisited, reconceptualized, and investigated (Fingelkurts & Fingelkurts, 2002; Geschwind & Galaburda, 1987, Winner, 2000).

In more recent times, beliefs about the origins of giftedness progressed to theories of atypical development and organization of the brain. Russell Brain (1960) stressed that genius was related to superior integration of perceptual and motor skill. He felt that higher organization of neurons in the brain formed sophisticated brain "schemas" that contributed to higher abilities. Brain had almost no empirical evidence to support his theory given the unavailability of modern technology. Yet his presumptions that gifted individuals had unique brain structure and functioning would later be supported by research.

Perhaps the most significant, though debated, contributions to the neurobiology of the intelligent brain came from

the writings of Normand Geschwind and Albert Galaburda (1984). Though both authors primarily investigated patterns of asymmetry in brain morphology and physiology, many of their findings had direct implications for understanding giftedness. In their book, *Cerebral Lateralization: Biological Mechanisms, Associations, & Pathology* (1987), the authors argued that mild abnormalities of neural migration are not only implicated in disorders of the nervous system but also could manifest themselves into superior abilities. They explained, "It may seem bizarre to speak of the neuropathology of superior intellectual functions; yet we suggest that a superior outcome, with or without accompanying problems in other areas, is not at all unusual" (p. 65). Subsequently the Geschwind, Behan, Galaburda (GBG; 1987) model of cerebral dominance argued that higher than normal concentrations of testosterone in utero may inhibit aspects of the brain development (typically aspects of left-hemisphere functioning) while enhancing other areas (typically right-brain development). In these extreme cases, individuals with enhanced right-brain development will show patterns of precocity, although they are also more likely to present with disabilities of verbal-language development and health concerns traced to elevated levels of testosterone in utero. In support of this hypothesis, Geschwind et al. (1987) referred to studies that demonstrated a link between a higher incidence of autoimmune disorders, asthma, allergies, and myopia among individuals with left or mixed handedness (Geschwind & Behan, 1982). Other studies (Benbow, 1986; Butterworth, 1999; Dehaene, 1997) investigating extremely mathematically precocious youth also found a much higher prevalence of these disorders in gifted populations. This theory has been criticized. Bryden, McManus, and Bulman-Fleming (1994) cited empirical evidence that does not support this theory. Berenbaum and Denburg (1995) found only marginal links between handedness and autoimmune disorders.

Yet the question of atypical brain development and organization in gifted children would not be abandoned. Fingelkurts and Fingelkurts (2002) furthered the argument of the existence of a connection between high intelligence and high testosterone levels in the prenatal fetus in a review of monozygotic male twins. Newer physiological studies also appeared to support the GBG hypothesis of atypical brain development and its explanation of giftedness. For instance, O'Boyle and colleagues (2005) found unique activation patterns using functional magnetical resonance imaging (fMRI) among mathematically gifted youths during mental rotation tasks. Specifically, gifted adolescents demonstrated enhanced right frontal development and increased bilateral brain activation during three-dimensional rotation tasks compared to controls. O'Boyle and colleagues targeted younger adolescents to reduce the effect of specialized learning environments on the brain. O'Boyle et al. felt that these findings reinforced the tenets of GBG model, citing that exposure to testosterone during the second trimester accounted for the much higher ratio of males to females in mathematically gifted youths.

NEUROPHYSIOLOGY

There is a growing body of research investigating the physiological underpinnings of intelligence and various dimensions of cognitive functioning. These findings have been summarized and expounded in specific papers (Blair, 2006; Kalbfleisch, 2004; Kalbfleisch, Van Meter, & Zeffiro, 2007). Though the link between intelligence, cognitive functioning, and underlying anatomy and physiology is a related topic, we look to more specific evidence from research involving gifted subjects. A common problem with this literature is the relative scarcity of subjects at the extreme end of the gifted spectrum (Lubinski et al., 2001), so not all fields of evidence will be covered with equal depth.

Initially, Terman (1925) argued that the unevenness of a gifted child's profile was no different than found in the general population. However, more recent research indicates that Terman was wrong and that unevenness between verbal and mathematical abilities may, in fact, be the rule and not the exception (Winner, 2000). Additional recent research yielded intraindividual differences and asymmetry between the cerebral hemispheres with patterns of disability and giftedness (Geake & Hanson, 2005). In a review of exceptional students, Detterman and Daniel (1989) found that mathematical abilities were much higher than verbal ability in high-IQ children than in children with lower IQ scores. Other studies (Benbow & Minor, 1990; Wilkinson, 1993) have found similar results in mathematically gifted students. The reverse has also been identified for students with verbal giftedness (Casey & Brabeck, 1989; Dark & Benbow, 1991). The tracking of profoundly gifted individuals from adolescence into adulthood also identified atypical differences between verbal and mathematical performance on standardized tests (Lubinski et al., 2001). Uneven patterns in intellectual profiles have also been identified in children who are gifted in music and art (Gardner, 1983; Winner).

Other evidence appears to support atypical brain organization in gifted children. Specifically, there is a trend toward increased right-hemisphere involvement in this population. Winner (2000) summarized these findings with five trends often noted among precocious youngsters. First, children who are gifted in math, the arts, and music demonstrate enhanced right-brain activity compared to normal children on tasks specific to the right hemisphere. Second, gifted children are disproportionately not right-handed. Third, musically and mathematically gifted children have more bilateral, symmetrical brain organization where the right hemisphere appears to be more involved in tasks ordinarily reserved for the left hemisphere. Fourth, giftedness in spatial activities is accompanied by a disproportionate

incidence of language-related disorders including dyslexia (Craggs, Sanchez, Kibby, Gilger, & Hynd, 2006). Fifth, children with higher IQs have a higher incidence of autoimmune problems and myopia.

Recent neurophysiological research has provided the most convincing evidence of neurological uniqueness among gifted individuals. The outcome of these investigations yielded greater right-hemisphere activity. For instance, Alexander, O'Boyle, and Benbow (1996) compared gifted adolescents with adolescents of normal intelligence and college students. Results suggested that comparable electroencephalography (EEG) activation between gifted adolescents and college students, but gifted adolescents tended to show greater right-hemisphere to left-hemisphere alpha activity. Another study by Jin, Kim, Park, and Lee (2007) compared EEG activation between 18 gifted Korean students with average students during neuropsychological tasks involving visuospatial construction. Results also reflected a dominance of right-hemisphere activity in the gifted students that correlated with higher performance on neuropsychological testing. The authors concluded that the results were consistent with the belief that right-hemispheric dominance was associated with superior coordination and allocation of cortical resources within the brains of gifted individuals. This is consistent with other research suggesting that frontal asymmetry within the right cortical area could be a physiological marker of the gifted brain (Fingelkurts & Fingelkurts, 2002). Case studies of math prodigies also implicate the right prefrontal and medial temporal areas (Pensenti et al., 2001).

In addition to the O'Boyle et al. (2005) study cited previously, other fMRI research has replicated atypical functional imaging activation patterns. Several studies identified regions in the brain including the prefrontal cortex (PFC), the anterior cingulate, and posterior parietal regions to be more highly involved on tasks with increased *g*-loadings (Geake, 2008). For instance, Geake and Hanson (2005) identified a network of regions within the PFC to be active during fluid reasoning tasks. Other studies also found increased activation in these regions when individuals were involved with reasoning and working memory tasks (Gray, Chabris, & Braver, 2003; Haier, Nathan, & Alkire, et al., 2003). However, these findings were typically conducted on individuals with IQs in the normal range. Lee et al. (2006) sought to overcome this limitation by investigating fMRI functioning differences by comparing individuals with average IQs with those classified as gifted. Results yielded somewhat unexpected results. Surprisingly, differences between groups were not characterized by engagement of extra network components unique to the gifted group. However, there was a greater level of activation of the entire frontal–parietal network, particularly in the PFC and posterior parietal cortex. Thus, the brains of gifted students did not use more or unique brain structures, but their brains showed signs of increased activation and likely reflected stronger interconnections than the average brain. These findings provided support for Brain's (1960) earlier contention that the brains of gifted individuals have more sophisticated brain schemas that become active during higher-level cognitive tasks.

The evidence suggesting that gifted individuals typically demonstrate increased brain activity is not widely accepted and some evidence points to the opposite finding. Early studies utilized positron emission tomography (PET) to measure brain activity during various tasks related to intellectual processing. The outcome suggested that performance on higher *g*-loaded tasks was associated with lower brain activity (Haier & Benbow, 1995; Haier et al., 1988). The belief was that high-ability subjects seemed to spend less time performing tasks than lower-ability subjects. Dubbed the "neural efficiency hypothesis" the implication was that more efficient brains (i.e., gifted brain) required less time and hence demonstrated less overall cortical activation. O'Boyle's (2008) most recent research is consistent with this hypothesis. O'Boyle suggested that mathematically gifted individuals have a more integrated and cooperative brain structure where the two hemispheres of the mathematically gifted individual are better able to efficiently share processing resources. The neural efficiency hypothesis makes intuitive sense because gifted individuals typically perform at a much higher level on measures of processing speed and other tasks related to speed embedded in intelligence tests.

Perhaps these discrepant findings can be reconciled by considering the developmental factors and maturation of the brain. Earlier PET research generally focused on the PFC as the key region of the brain involved in increased cognitive activity (Haier et al., 1988). Subsequent research (Jin et al., 2007; Lee et al., 2006; O'Boyle et al., 2005) also demonstrated the involvement of this region of the brain. However, there appears to be a shift in brain activity not only depending on the task but also depending on the age of the individual. Klingberg, Forssberg, and Westerberg (2002) conducted a unique study comparing the brain functioning with fMRI between children with a mean age of 9 with older adolescents with a mean age of 18 on working memory tasks. A higher level of ability, both between and within age groups, was associated with increased parietal activity and corresponding decrease in PFC activity. Regardless of age, the subjects who performed well on this task demonstrated increased parietal activity. The conclusions suggested a shift to more parietal activity with older subjects but also with those who performed at a higher level on the task itself. A limitation for this study was that the subjects were not gifted students, so direct inferences to the shift in brain activity could not be made.

There are other possible interpretations accounting for the seeming contradictory findings of brain activity in gifted individuals. A unique attribute of gifted individuals is the capacity for creative thinking. Intellectually creative individuals are typically highly task motivated (Lykken, 1998)

and able to consider a problem from many different perspectives. These individuals are sometimes referred to as "outside-the-box" thinkers because they can generate perspectives that most others do not consider. Some have suggested that there is a link between intellectual creativity and reduced latent inhibition (Carson, Peterson, & Higgins, 2003; Geake & Dodson, 2005). That is, highly intelligent individuals can cope with a relatively larger number of ideas and possibilities simultaneously. To do this, an individual's working memory capacity must be exceptional and, indeed, fMRI studies have suggested increased activation in the working memory regions of the brain (Rypma, Berger, & D'Esposito, 2002). A recent study by Rypma and colleagues (2006) noted that in some PFC regions, faster performers (likely individuals with higher cognitive abilities) on measures of processing speed showed less cortical activity in some regions, whereas in other PFC and parietal regions they showed greater activity than slower processors. Further analysis indicated that PFC exerted more influence over other brain regions for the slower performers. It is as if the faster performers were able to effectively "tune down" the inhibitory regulation of the brain on itself. Perhaps this is an important component to creative and intelligent thinking such that gifted individuals are able to escape the typical restraints of reasoning. There is evidence to link decreased latent inhibition with increased creative thinking in gifted individuals. However, this has yet to be delineated in neurophysiological research with gifted populations.

Perhaps the uniqueness of brain functioning in gifted individuals is related to their capacity to persevere on tasks and repetitively improve and enhance their ability well beyond what others typically do. And perhaps this ability fosters less involvement of the PFC and increased involvement of the parietal regions of the brain. Recall the O'Boyle study (2005) that identified young adolescents to have higher levels of activity in the PFC during fluid reasoning tasks. The subjects in this study were adolescents approximately 12 to 15 years of age. In contrast, the Lee et al. (2006) study mentioned previously, in addition to several others (Rypma et al., 2006), used older subjects (age 18 and higher) and found increased parietal activity and decreased PFC activity. Yet this same pattern did not hold true to other studies (Haier et al., 1988; Rypma et al.) for older yet lower-ability subjects. To date the functional brain imaging research has yet to answer this interesting question. Aside from the Klingberg et al. (2002) study, there is little research investigating change in brain functioning over time. Longitudinal studies measuring the changes in brain functioning for gifted versus nongifted individuals may provide stronger evidence of unique patterns of brain activity across the developmental lifespan. These studies may also unlock the mysteries of how certain cognitive processes that come naturally to gifted individuals evolve. The capacity of functional imaging, such as those described by Kalbfleisch (2008), has opened the door to better understanding the mysteries of gifted individuals and will no doubt lead to greater understanding in the future.

THE PRENATAL ORIGIN OF GIFTEDNESS: A HYPOTHESIS

Consistent with efforts to uncover gifted etiology, we would like to present a biologically plausible hypothesis regarding the etiology of giftedness. Our hypothesis is guided by the growing and substantive body of prenatal exposure research that investigates the relationship between a prenatal exposure and a later psychological, educational, or behavioral disability in offspring. This literature will provide a backdrop for our hypothesis, although a more in-depth analysis of the prenatal exposures literature is available elsewhere (e.g., Dombrowski & Martin, 2007, 2009; Martin & Dombrowski, 2008).

There is an abundance of research documenting severely adverse physical and neurological outcomes following a first-trimester exposure to a virus such as rubella or chemical agent such as alcohol or thalidomide (Persaud, 1985; South & Sever, 1985). Although this is an important and ubiquitous body of research, we are not interested in describing prenatal exposures that cause severe physical and neurological damage. Rather, we discuss a nascent yet growing multidisciplinary research agenda that links certain prenatal exposures with subtle changes in the central nervous system (CNS) of the fetus. These CNS perturbations do not produce observable physical anomalies but remain clinically silent until later in development when a child faces the complex demands of life. Resulting outcomes might include attention deficits, learning disabilities, speech–language delays, mood disorders, or reduced cognitive capacity (Dombrowski & Martin, 2007). These outcomes are often associated with a second- and/or third-trimester exposure (Cordero, 2003; Dombrowski & Martin, 2009; Dombrowski, Martin, & Huttunen, 2003).

What is important about the second and third trimesters of gestation and why do scientists increasingly emphasize this period rather than the first trimester of pregnancy for a relationship with psychological and behavioral outcomes? Neuroanatomical research indicates that the first trimester is responsible for forming the shell of the CNS, and the second and third trimesters encompass the commencing and refining of fine-grain neurological processes (Nowakowski & Hayes, 1999). A prenatal exposure during the first trimester of gestation typically leads to obvious and often severe physical (e.g., facial anomalies; cleft palate; missing arms) or neurological abnormalities (e.g., spina bifida; cerebral palsy; mental retardation), if not fetal death (Persaud, 1985). An exposure during the second or third trimester of gestation tends to be less harmful, depending upon the type exposure, and typically produces less deleterious outcomes.

The metaphor of the construction of a house might be useful in conceptualizing a difference between a first-trimester and middle- to late-trimester exposure. The first trimester is akin to the foundation and frame of the home. A disruption to construction of the foundation and frame of a home will affect all subsequent development and perhaps compromise the integrity of the home. A disruption or alteration at a later point in the home's construction, such as to interior flooring or wall covering, might be disruptive to a particular area but will not compromise the overall home. This is similar to second- and third-trimester disruptions. An exposure during these time periods may have an adverse impact but will be less likely to damage the overall integrity of the organism.

The second and third trimesters are linked to subtle outcomes via a disruption to important neurological processes. Throughout the second and third trimesters of gestation, brain development occurs more rapidly than at any other period in the human life span (see Martin & Dombrowski, 2008, chapter 2). Because of this accelerated period of growth, the brain is most vulnerable to insult. This position is consistent with the Dobbing hypothesis, a significant yet parsimonious developmental concept that emerged out of medical research over three decades ago. The Dobbing hypothesis (Dobbing & Sands, 1974) simply states that periods of most rapid development are most vulnerable to adverse impact. Additionally, it is important to recognize that the timing, magnitude, and duration of a prenatal exposure can influence important CNS developmental processes. Specifically, the processes of neuronal proliferation, migration, differentiation, myelination, and cell death all occur at precisely specified time periods during gestational development and are vulnerable to alteration or disruption (Aylward, 1997; Martin & Dombrowski, 2008). Depending upon its magnitude, timing, and duration, a prenatal exposure at a critical stage could adversely impact these important processes, contributing to psychological, behavioral, and educational pathology (Rosen, Waters, Galaburda, & Deneberg, 1995).

A substantial body of research has investigated the plausibility of this hypothesis via the link between in utero second- and third-trimester exposure to influenza and later onset of schizophrenia in offspring (McGrath & Castle, 1995; Mednick, Machon, Huttunen, & Bonnet, 1988). A smaller body of research has investigated other prenatal exposures such as maternal infection, fever, malnutrition, and stress as they relate to psychological and behavioral outcomes such as autism, bipolar disorder, schizophrenia, and learning disabilities, among others (Dombrowski et al., 2003; Martin & Dombrowski, 2008).

With the understanding that there are numerous prenatal factors (e.g., maternal stress, smoking, fever; see Dombrowski & Martin, 2009; Dombrowski et al., 2003; Dombrowski, Martin, & Huttunen, 2005; Huizank, Mulder, & Buitelaar, 2004; Martin & Dombrowski, 2008; Martin, Dombrowski, Mullis, Wisenbaker, & Huttunen, 2006) that could explicate the relationship between a prenatal exposure and adverse psychological/behavioral outcomes, we will furnish an overview of the prenatal influenza–schizophrenia literature because of its fairly extensive research base. The putative association between prenatal influenza and schizophrenia has a 20-year research history with more than three dozen studies in regard to outcomes and etiological processes (Dombrowski & Martin, 2009; Martin & Dombrowski, 2008). The preponderance of these investigations indicates that gestational influenza exposure during the second or third trimester of pregnancy is associated with later onset of schizophrenia in adulthood. It has also been linked in a small number of studies to affective disorders including depression and bipolar disorder (Machon, Mednick, & Huttunen, 1997). What is it about the influenza–schizophrenia connection that may lead to the association with psychiatric outcomes? This association is explained within the context of the neurodevelopmental hypothesis of schizophrenia. The neurodevelopmental hypothesis suggests that a disruption to brain development at an earlier stage (i.e., the prenatal time period) creates a vulnerability to psychopathology in offspring at a later stage of development (Waddington et al., 1999). The vast majority of the prenatal influenza studies have found an association when the exposure occurs during the second and third trimesters of pregnancy (McGrath & Castle, 1995). The second and third trimesters of gestation are important for several neurological events that are critical to the development of the human central nervous system, including neuronal proliferation, migration, differentiation, myelination, and cell death (apoptosis; Nowakowski and Hayes, 1999; Sidman & Rakic, 1982). Gestational exposure to influenza at the critical late second or early third trimester has been implicated in disrupting CNS organization, particularly the process of neuronal migration from the periventricular area to a variety of sites on the cortex (Barr, Mednick, & Munnk-Jorgensen, 1990). This disruption, among others, has been conjectured to contribute to the etiology of schizophrenia in offspring (Gilmore & Jarskog, 1997).

The influenza–schizophrenia literature (and all prenatal exposure–psychological consequences literature) has relevance for giftedness because it follows a similar model of neuropathology. We posit that a disruption or alteration via a prenatal exposure to one or more of the brain developmental processes of proliferation, migration, differentiation, myelination, and apoptosis may be important for the etiology of giftedness. Among the first to suggest a link between a prenatal event and later giftedness were Geschwind and Galaburda (1987). Geschwind and Galaburda presented a prenatal testosterone model in which an exposure to an increased level of testosterone alters neuronal migration, leading to more intensive right-hemisphere development (Geschwind & Behan, 1982). Additionally, high levels of testosterone (or greater sensitivity in utero) have been linked to greater coordination within and between the hemispheres (Alexander et al., 1996) via an unusually developed corpus

collosum (Habib et al., 1991). It is acknowledged that testosterone exposure in utero is a normally occurring process and all fetuses are exposed to testosterone during intrauterine development. Male fetuses experience a surge during the eighth week of gestation that in part is responsible for phenotypical differentiation. However, it appears that higher than normal levels of or greater sensitivity to testosterone appears to play a role in altering the organism-typical neurodevelopmental trajectory (Baron-Cohen, Lutchmaya, & Knickmayer, 2004). Thus, we posit that Geschwind and Galaburda (1987) prenatal testosterone model, which has been augmented by others (e.g., Alexander et al.; Benbow, 1986; Fingelkurts & Fingelkurts, 2002), represents a positive first step toward conceptualizing the relationship between a prenatal event, altered neurological development, and later giftedness. However, there are additional exposures beyond testosterone that can potentially alter and/or redirect central nervous system development during the fetal period (Dombrowki & Martin, 2007, 2009; Dombrowski et al., 2003; Martin & Dombrowski, 2008). Therefore, any proposed hypothesis must be broader in scope.

We contend that a prenatal exposure may be an etiological factor in giftedness. This represents a novel research hypothesis for the myriad psychologically and medically oriented disciplines that investigate human behavioral teratology. Despite its novelty, our research hypothesis is biologically plausible. The following depiction illustrates how a prenatal exposure might mediate not only the relationship with psychological/behavioral pathology but also giftedness.

As noted in Figure 1, the same prenatal neuropathological mechanisms implicated in producing psychological and behavioral outcomes might also contribute to giftedness. This includes an alteration or a disruption to microscopic brain developmental processes of neuronal proliferation, migration, differentiation, and apoptosis. For example, consider the prospect of enhanced neuronal proliferation in one part of the cortex that leads to unusually high densities. Or, perhaps neuronal apoptosis (i.e., neuronal pruning and axonal retraction) fails to occur in a specific location of the cortex. Or, suppose that neurons destined for one area of the brain partly responsible for language are diverted to another area (i.e., the cortex's inferior parietal region). These alterations might, as an example, contribute to macroscopic alterations in the structures of the brain, such as an overdevelopment in the inferior parietal region of the cerebral cortex, the area responsible for visual–spatial, musical, and mathematical reasoning. This extends Brain's (1960) earlier contention that the gifted brain may form more sophisticated networks setting the condition for higher abilities. Conversely, the redirection of neuronal migration away from areas responsible for language in favor of the inferior parietal region of the cortex might also lead to apparent language based disability such as dyslexia.

As mentioned previously, Albert Einstein was an example of an individual who experienced an overdeveloped inferior parietal region, conjectured to contribute to his vast mathematical capabilities (Anderson & Harvey, 1996; Witleson & Goldsmith, 1991). On the other hand, Einstein did not speak until age 3 and struggled with language early in life. We are not suggesting that both Einstein's gifts and apparent disability were related to a prenatal exposure but rather that a prenatal exposure paradigm is sufficiently broad that it could plausibly explain not only his genius but also his disability. Finally, perhaps certain neurons destined to differentiate into dopamine, serotonin, or glutamate neurotransmitter systems are instead altered. Because these neurotransmitter systems are implicated in perception and behavior, this alteration might contribute to eccentric or psychotic behavior on the one hand and exceptionally creative behavior and perceptive abilities on the other (e.g., Carl Jung, Mozart, Picasso).

Consistent with this hypothesis, it is interesting to note that homogeneous neurological characteristics produce heterogeneous functional outcomes. For instance, volume reductions in the left hemisphere and left cerebral cortex have been associated with not only giftedness but also Asperger's disorder, schizophrenia spectrum symptoms, and dyslexia (Gilger & Hynd, 2008; McGuire & Frith, 1996; Post, 1994; Ross & Pearlson, 1996; Weinberger, 1995). Yet, there is also overlap in characteristics. Individuals with schizophrenia as well as individuals who are gifted tend to be left handed (Nasrallah, McCalley, & Kuperman, 1982), and individuals who are gifted and individuals with autism spectrum disorders such as Asperger's have greater prevalence of allergies and autoimmune disorders (Sweeten, Bowyer, Posey, Halberstadt, & McDougle, 2003). These overlapping neurological and behavioral sequelae suggest the possibility of a common neuropathological mechanism.

In totality, our prenatal exposures hypothesis will need to be tested, debated, and replicated before being considered reified. And, it would be prudent to place our hypothesis within the context of other findings including those of Rosenzweig and Bennet (1996) and Diamond (1991) out of UC Berkeley who have empirically examined the relationship among heredity, environmental enrichment, and brain development (e.g., Diamond, 1991; Rosenzweig & Bennett, 1996) and that of Brizendine (2006), who discussed the in utero testosterone flood. Despite these caveats, our prenatal exposures hypothesis furnishes an intriguing model that might be useful in understanding the etiology of giftedness, particularly the eminently gifted, and provides further support for the increasingly recognized, yet scarcely investigated, dual exceptionality model (e.g., Geschwind & Galaburda, 1987; Gilger & Hynd, 2008; Kalbfleisch & Iguchi, 2007).

CONCLUSION

There is substantive evidence that gifted individuals have atypical brains and atypical brain functioning. Historical viewpoints argued that precocious brains were unique in size and function but based these theories largely on

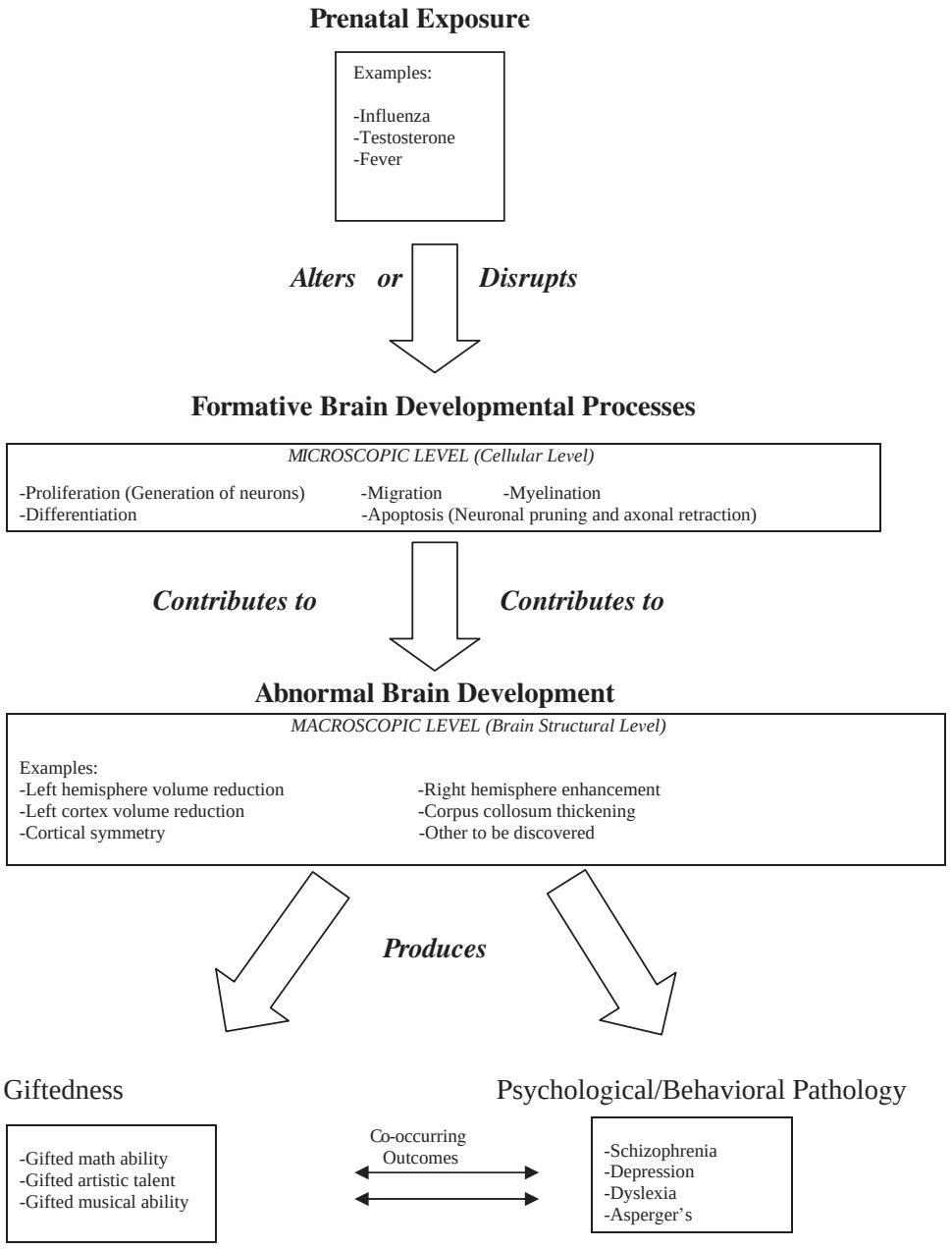

FIGURE 1 Prenatal exposures model of giftedness.

speculative evidence. Today the arguments appear to have come full circle as patterns of dysmorphology and unusual brain activity seem to be more the norm than the exception. There are arguments both supporting and refuting the GBG model of cerebral dominance and the possibility that high levels of testosterone in utero contribute to gifted mathematical and spatial abilities. Perhaps other prenatal exposures contribute to the etiology of giftedness via a disruption to the important brain developmental processes of neuronal proliferation, migration, differentiation, myelination, and apoptosis. We have presented a biologically plausible prenatal exposures paradigm that should serve as a framework for future research and perhaps move the field to a greater understanding of the etiology of high giftedness. Regardless of the cause of neuropathology, there is evidence that highly gifted brains appear more at risk for medical and psychological disorders. We are just in the infancy of research on the causes and correlates of giftedness. The transdisciplinary field of cognitive neuroscience holds promise for discovering new insights into the brain–exceptionality relationship in large measures because of recent advances in neuroimaging techniques. It is quite possible and—as we have asserted—biologically plausible that the same neurobiological factors that contribute to psychological and behavioral pathology also

contribute to giftedness. Future resources should be directed toward investigating the prenatal origins of giftedness.

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