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Neurodevelopmental Variation as a Framework for Thinking About the Twice Exceptional

Jeffrey W. Gilger and George W. Hynd

Developmental exceptionalities span the range of learning abilities and encompass children with both learning disorders and learning gifts. The purpose of this article is to stimulate thinking about these exceptionalities, particularly the complexities and variations within and across people. Investigators tend to view learning disabilities or abilities, and gifts or high-end exceptionalities, as if they were necessarily and completely independent. This approach has led many in the field to look upon only limited aspects of the exceptional child, culminating in an inability to resolve the great variation and covariation that exists within and across children. Although there are a number of cognitive differences models that correctly advocate for an appreciation of profiles of strengths and weaknesses in the exceptional child, there remains a need for a neuroscientific approach that can help us better understand and accommodate the twice-exceptional individual—one with developmental disorders but also with high skills in the talent, creativity, or intellectual domains. We propose a model that will help us to fully appreciate that the brain that produces developmental learning abilities across the spectrum must be viewed as an integrated and multifaceted organ that is more than a simple reflection of its separate parts or domain-specific symptoms. We use developmental reading disability or dyslexia and the twice-exceptional individual as a means to illustrate how this model can aid in our thinking about these conditions.

At times it seems as if those of us in the general field of child development and developmental disorders are like blind men looking at elephants: we are ostensibly studying the same pachyderm yet we often come up with quite different impressions or highlight quite disparate facets. Indeed, the study of the "exceptional child" is very broad in scope and diverse in disciplines, and exceptionalities can bridge both ends of the ability continuum. Because of this there has been an abundance of intellectual creativity and fine science, but unfortunately there has also been a lack of cohesion.

The purpose of this article is to stimulate some thinking about these exceptionalities, particularly the complexities and variations within and across people. The model we present is in reaction to a long-standing predilection in the field to talk of learning disabilities (LD) or abilities, and gifts or high-end exceptionalities, as if they were completely phenotypically, etiologically, and statistically independent. Traditional approaches have, over the years, led many "blind men" to look upon only limited aspects of the exceptional animal, culminating in an inability to resolve the great variation that exists or explain the larger beast in its entirety. Although there are a number of cognitive differences models that correctly advocate for an appreciation of profiles of strengths and weaknesses in exceptional children (see multiple intelligences or other approaches summarized in Gardner, 1999; Levine, 1992; Sternberg, 2000), there remains a need for a neuroscientific approach that can help us better understand and accommodate the twice-exceptional individual—one with developmental LD but also with high skills in the talent, creativity, or intellectual domains.

It is our proposal that we need to combine current approaches with some new ways of thinking. This will allow us to fully appreciate that the brain that produces developmental learning abilities across the spectrum must be viewed as an integrated and multifaceted organ that is more than a simple reflection of its separate parts or domain-specific symptoms. We will describe a *thinking tool* that focuses on developmental reading disability (RD) or dyslexia and the twice-exceptional individual. While we use RD as an exemplary disorder, what we will talk about may also be applicable to other developmental LDs as well.

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Address correspondence to Jeffrey W. Gilger, PhD, Purdue University, Beering Hall of Liberal Arts and Education, Room 6114, 100 N. University Street, W. Lafayette, IN 47907-2098. E-mail: jgilger@purdue.edu

THE NEED FOR A THINKING TOOL[1]

While the field of the study of learning disabilities, and particularly RD, has had a long and successful track record of sustained and focused neuroscientific research, the systematic study of giftedness and of populations of the twice-exceptional student (e.g., RD plus gifted; Craggs, Sanchez, Kibby, Gilger, & Hund, 2006; Kalbfleisch, 2004) is seriously lacking.[2] There are several key reasons why the neuroscience of the twice exceptional (TE) is limited.

First, the relative lack of empirical and neuroscientific study of the TE (or purely gifted) population is, in part, due to some long-standing traditions in the study of child development and the funding focus or preference that adopted the "disease model" of abnormal learning in certain populations of children. The concept of RD, for instance, grew out of a medical model and the people with a neurological, genetic, or remedial bent naturally approached poor readers as having unique etiologies and as being "disordered" or "diseased" (e.g., Clements & Peters, 1962; Orton, 1928; summarized Fletcher, Lyon, Fuchs, & Barnes, 2007). The major scientific machines and federal funding sources like NIH also favored a "disease model" approach, and money for the neuroscientific study of the "other end of the continuum" in and of itself, or along with an LD, is hard to find.[3] However, some federal and state funds have been available for the study of giftedness such as the Javitz monies and other resources, and these have yielded some prolific centers and involved nationally respected researchers. For examples see the National Research Center for Gifted and Talented at the University of Connecticut, the Belin-Blank Center for Gifted Education and Talent Development at the University of Iowa, and the Gifted Education Resource Institute at Purdue University.

Second, while it has been politically charged at times to define and study disordered populations as being biologically different than the rest of the people along the normal curve, it also has been difficult to talk about groups at the high end of the distribution as being different. A neuroscientific focus on the former group relative to the latter has been favored, however, because of what was seen as the obvious economic and emotional benefits of finding ways to treat people with a reading or learning disorder.

Third, the fairly limited empirical neuroscience research on the TE reflects the history of this field: it has been dominated by professionals less interested in cause and more interested in educational issues. Although the classification of TE is tied to special education, few basic neuroscience researchers live in the field and, instead, curriculum specialists and educational and clinician psychologists have tended to hold key posts and have chosen to focus more on treatment and identification. In other words, studies in the area of giftedness and of the TE student have tended to focus on curriculum, cognitive theory, learning styles, and definitional debates (e.g., McCoach, Kehle, Bray, & Siegle, 2004; Newman, 2004).

Finally, our historical and sometimes unconscious manner of approaching the study and understanding of the brain has helped limit basic research of the twice exceptional. Simply put, it has been difficult to reconcile TE brains with what we now know about the expression and causation of learning disabilities (see Footnote 2). For example, studies of RDs often are careful to control for ADHD, normal IQ, and psychiatric problems, but giftedness is not even considered as an important or potentially related variable. Learning disorders are explained as separate units with specialized neurological and genetic substrates, and to consider gifts in the same "disordered" brain is not part of these models. Indeed many fine-grained reading-specific neuropsychological and biological models have been proposed as have some models for giftedness in domains of cognition (e.g., Démonet, Taylor, & Chaix, 2004; Fletcher et al., 2007; Mody, 2004; Nicolson & Fawcett, in press; Simonton, 2005). But as these models of ability evolve, it is implied that "disabilities" must have a different etiology than "gifts" and the two shall never meet! Our grasp of these apparent disparate conditions would be well served if there were a unifying model that would help organize and conceptualize the central research issues in the areas of giftedness, LD, and TE.

We are not the first to call for such work, although the yield of prior calls has been limited (Kalbfleisch, 2004). Among the first such calls was by Norman Geschwind and colleagues, who spoke about the relationships between giftedness and developmental reading disorders over 20 years ago (Geschwind & Galaburda, 1987). They proposed that genetics and in utero hormonal activity modified neurodevelopment and hemispheric specialization such that a person could be born with a brain wired to be at risk for RD and superior nonverbal abilities as well. They and others have proposed that the setting of the left hemisphere language areas to be prone to language-based impairments

[1]In this article we focus on reading disability as our example learning disorder. This is because of our familiarity with the disorder as well as the fact that it is perhaps the most common and well-studied disorder in the current learning disability classification scheme (Fletcher et al., 2007). Furthermore, while our focus is on the twice-exceptional RD individual, much of what we say may be incorporated into research and practice in the area of pure giftedness as well.

[2]It is important to note that when we speak of the twice exceptional in this article we are not including conditions like the rare and talented autistic, savant, or prodigy (Butterworth, 2001; Casanova, Buxhoeveden, Switala, & Roy, 2002; Deutsch & Joseph, 2003). Instead, we are addressing the developmentally intact and normal child, who has a specific learning disorder along with a superior splinter skill or IQ in the "gifted" range.

[3]At the time of this writing it is noteworthy that the National Institute of Child Health and Development (NICHD) has begun to consider the issue of TE more formally and from this may come some important funding initiatives. See also the American Psychological Foundation (www.apa.org) and the Templeton Foundation (www.templeton.org) for some other nonfederal funding mechanisms in this area.

could in fact effect the growth of portions of the right hemisphere such that there might be an overrepresentation of nonverbal gifts in RD samples. In this way, the etiology of high and low abilities can be related at a basic biological level early in fetal development, and postnatal etiologic factors are considered less important (Craggs et al., 2006). Interested readers might also look at hormonal and immune system neurodevelopmental models that also attempt to explain high and low skills in the same person (Benbow, 1988; Butterworth, 2001; Schopler & Mesibov, 1992; Singh & Boyle, 2004). Still, these models tend to be domain specific and often deal with severely disabling conditions like autism and mental retardation (see Footnote 2), and all these models are in need of further validation.

Our purpose here is not to advocate for one model or another. We would say, however, that our understanding of what it is that makes someone TE (or gifted) is really paramount to our understanding of the etiology of the complete range of human abilities and no harm will come from grasping the neuroscience of the other side of the human continuum. More importantly to us is that there is a growing call for such research, be it directly or indirectly. For example, there is the fairly recent emphasis at the national level that our economic and intellectual future may lie in great part in the production of students in the science, technology, engineering, and mathematics (STEM) disciplines and the importance of identifying national talent in these areas, some of whom may be LD as well. Indeed, some federal funding agencies are responding to this need through programs tied to talent identification and STEM education (e.g., the Institute of Educational Sciences [IES]; and see the National Academies [n.d.] summary reports on this issue).

It is also becoming increasingly apparent that certain populations of children and adults are not well served by existing nosologies and models of special education for LD, TE, and giftedness (Eide & Eide, 2006; McCoach et al., 2004; Newman, 2004). Teachers, for instance, often are confused by conflicting abilities in the same child, and parents often are unable to get services for their child because the child does not fit any clear diagnostic scheme or qualification criteria. More well-designed neuroscientific study of the gifted and TE would help ameliorate some of these difficulties.

THE CONCEPT OF ATYPICAL BRAIN DEVELOPMENT APPLIED TO TWICE EXCEPTIONALITY: THE GIFTED RD INDIVIDUAL AS AN EXAMPLE

The Atypical Brain Development (ABD) model originally was designed in response to perceived needs in the field of LD, and especially RD (e.g., Bonifacci, 2004; Davis, 2004; Fadjukoff, Ahonen, & Lyytinen, 2001; Frith, 2001; Gilger & Kaplan, 2001; Gilger & Wilkins, 2008; Gilger & Wise, 2004; Goldstein & Schwebach, 2004; Jeffries & Everatt, 2004; Kaplan, Dewey, Crawford, & Wilson, 2001; Kaplan & Gilger, 2001; Kreuger & Markon, 2006; Lyon, Fletcher, & Barnes, 2003; Lyytinen et al., 2000; Missiuna, Gaines, & Pollock, 2002; Ping & Zu-wen, 2005; Rice & Brooks, 2004; Sonuga-Barke, 2003; Valtonen, Ahonen, P. Lyytinen, & H. Lyytinen, 2004; Zoia, Barnett, Wilson, & Hill, 2006). The ABD concept evolved primarily from thinking about the ecological validity of diagnostic categories. There also were issues being raised by ongoing genetic and neurological research at that time, especially in the area of RD, that were challenging to the assumptions behind the concept of a neurologically specific reading disability. The ABD approach was designed to provide some cohesion and reconcile research with some of the belief systems of many clinicians and educators who had extensive exposure to individuals with developmental problems like RD or other learning exceptionalities. Namely, practitioners were becoming dissatisfied with the practical utility of the dominant contemporary theories and guidelines, special education criteria, and how to deal with children who fail to fit molds constrained by these theories and related federal laws.

ABD Fundamentals

There are three fundamental assumptions behind the ABD concept. By and large the validity of assumption one is obvious. Assumptions two and three are likely also to be obvious, but some general references are provided (see Bartley, Jones, & Weinberger, 1997; J. W. Gilger, 1995; Pennington, 2002; Scerif & Karmiloff-Smith, 2005; P. Thompson, Cannon, & Toga, 2002; P. M. Thompson et al., 2001). The three assumptions are

1. The brain is the basis of behavior.
2. Individual differences in behavior are due to variable brain structure and function.
3. Ultimately, individual differences are the result of the complex effects of genes and the environment on the developing and learning brain.

It is important to realize that the concept of ABD is a neurological or neuropsychological model, but it does not speak directly to how the brain is organized or how specific areas or functions of the brain explain specific abilities as do other theories or models (e.g., see Eden & Zeffiro, 1998; Hynd & Orbrzut, 1986; Lieberman, 1984; Luria, 1973; Nicolson & Fawcett, in press; Pennington, 1991, 1999; Ramus, 2001, 2004; Rice & Brooks, 2004; S. E. Shaywitz et al., 1998; Sternberg, 2000). The purpose of the ABD perspective is not to supplant these other models; rather, it serves as a frame of reference that is able to draw together these models and provide some coherence, understanding, and new directions for research.

We want to also emphasize that the term *atypical* in ABD does not denote dysfunction or damage. A positive facet of ABD is that it is nonevaluative, and it encompasses phenomena at both ends of the ability continuum. It is thereby not invalidated by multiple, apparently disparate deficits in the same person or by seeing people with deficits and gifts at the same time. Furthermore, the term *development* in ABD accurately describes our current understanding that developmental learning disorders and TE are probably the result of prenatal and, to a lesser extent, postnatal brain growth and elaboration. This is not meant to devalue experience as a very important pre- and postnatal variable in brain development and learning. Nonetheless, ABD applies primarily to atypical abilities (disorders and gifts) that result from gene-regulated or gene-moderated developmental processes, and it does not apply to acquired disorders, disorders due mainly to adverse or teratogenic intrauterine environmental factors, or special talents that can be linked to specific practices in life (e.g., Frith, 2001; Greenough, Black, & Wallace, 2002; Huttenlocher, 2002; Ramus, 2003, 2004b; Solso, 2001; Toga & Thompson, 2005; see Footnote 2). In summary:

> ABD does not itself represent a specific disorder or syndrome; it does not pertain to brain injury, trauma, or disease in the classic medical sense. Rather, ABD describes the developmental variation of the brain and subsequent brain-based skills on either side of the real or hypothetical norm. (Gilger & Kaplan, 2001, p. 468)

ABD Conceptualization for RD and the Twice Exceptional

Dyslexia serves as a good illustrative disorder in that it has been well researched with regard to epidemiology, definition, genetics, and neurology (Fletcher et al., 2007). We present some data in this section that focus on RD as a way to familiarize the reader with the ABD model history and application. Throughout this discussion we also address TE as part of the ABD spectrum.

There are four research-based characteristics of RD that lend themselves to the ABD model. The first three of these aspects likely make ABD applicable to other LDs as well, since research on some other LDs is beginning to show trends similar to that for RD (e.g., developmental coordination disorder, ADHD, math disability, language disorder, among others). The fourth aspect, that RD is etiologically part of the normal range of reading rather than a disease may be RD specific, but here we have chosen to make this point salient as certain forms of giftedness and TE may be of a similar nature. That is, they are reflections of normal human genetic and neurological variation, although often unrecognized as such.

Variants in the RD profile and the frequency of comorbidity. One of the strongest arguments for supplementing current thinking in the field of learning exceptionalities with an ABD framework is the research that demonstrates the presence of a high degree of comorbidity, as well as intraindividual and interindividual variation in cognitive and behavioral profiles (Brody & Mills, 1997; Fuchs & Fuchs, 2002; Gilger & Kaplan, 2001; Kaplan et al., 2001; Pennington, 1999; Shapiro et al., 2002). Therefore, in some proportion of cases the distinctions between disorders (e.g., RD, ADHD, developmental coordination disorder, and so on) may, at times, be more artificial than real and may also muddle a complete understanding of the individual presenting a complicated symptom profile.

Given the well-documented correlation of RD with other low-end traits and the possible correlation with high-end traits, it would seem that maintaining a blanket approach to RD as if it were a completely independent and domain-specific disorder seems ill advised. Indeed, psychometric (e.g., multivariate factor analytic studies), genetic, and neurological research support at least some degree of non-independence, often showing correlated cognitive abilities or cognitive factors, overlapping or multiple neurological substrates, and shared genes for phenotypes, brain morphology, and brain function (e.g., Bartley et al., 1997; Butcher et al., 2006; DeFries & Alarcon, 1996; LaBuda, DeFries, & Fulker, 1987; Olson, Foresberg, & Wise, 1994; Ramus, 2001, 2004b; Rice & Brooks, 2004; Simonton, 2005; Sonuga-Barke, 2003; Thompson et al., 2001, 2002; Voeller, 1999; Willcut et al., 2002; Wood & Flowers, 1999). Therefore, when an individual exhibits characteristics of dyslexia, memory problems, motor skills deficits, or gifts, it seems an open question as to whether that child is displaying comorbid unitary abilities and disabilities or variable manifestations of one underlying impairment, several underlying impairments, or etiologic substrates that may or may not be independent (Gilger & Kaplan, 2001; Kaplan et al., 2001). The commonly seen co-occurrence of apparently disparate symptoms causes problems in both diagnosis and treatment, especially if they are on opposing ends of the continuum and, at the same time, it complicates an understanding of contemporary etiological models (Bergman & Magnussen, 1997; Cloninger, 2002; Gilger & Kaplan, 2001; Jeffries & Everatt, 2004; Kaplan et al., 2001; Lyytinen, Leinonen, Nikula, Aro, & Leiwo, 1995; Narhi & Ahonen, 1995; Sonuga-Barke, 2003).

The term *comorbidity* refers to multiple diseases in the same individual but in a less pathological way is analogous to the term *TE*. Thus, many of the methods of studying the neuroscience of comorbidity could be extended to the study of TE. Indeed, there are data indicating that RDs (and other LDs) can be represented significantly in gifted populations (e.g., Brody & Mills, 1997; Geschwind & Galaburda, 1987; Ruban & Reis, 2005; Schneps et al., 2007; von Karolyi & Winner, 2004; Geschwind, 1982). Some estimates, though fraught with definitional and sampling problems, have placed the prevalence rates of TE as low as 1% but with an upper end of 3%, 5%, or even 36% (Baum & Owen, 1988;

McCoach et al., 2004; Ruban & Reis, 2005). If severe cases of disabilities are removed from these data (e.g., autism) and the focus is on developmentally normal but reading disordered children, the rates may be on the low end, say roughly 1–3%. That is still a relatively high rate in practice or in the classroom.

For discussion purposes, a simple test of the RD-gifted association, if the two conditions are independent, can be conducted using the law of independent probabilities and the multiplicative rule of probabilities. If you assume that, say, RD has a base rate of around 7% and is truly independent from giftedness, and if you further, arbitrarily, choose a base rate of gifted IQ to be around 5%, the expected base rate of the co-occurrence of the two conditions can be obtained by multiplying the two rates: 7% * 5% = .0035%.[4] This .0035% is significantly lower that the observed 3–5% cited above. Thus, this simple model (albeit fraught with methodological problems) suggests some support for the folklore that exists that giftedness is overrepresented in RD populations (e.g., Geschwind, 1982; Geschwind & Galaburda, 1987; West, 1999) and more research of better design and control is needed to properly address this belief.

Percentages aside, focusing only on the learning deficit in TE individuals limits our understanding of their neurocognitive profile, the etiology of this profile, and what approaches to treatment or educational settings may be best applied. Current neuroscientific theoretical approaches to the study and treatment of RD do not address twice exceptionality in any explicit or meaningful way. At best, contemporary thinking views RD and giftedness as separate and unrelated conditions that just happen to cooccur. In the majority of cases this may be true, but we suggest that some RD-gifted cases may represent subtypes in which the learning problem shares etiology with the learning gift.

One way to help relax the research and application "tensions" raised in this section is to view individuals with, say, RD, motor deficits, inattention, and/or gifts as expressing symptoms of a diffusely atypical brain affecting multiple areas of behavior simultaneously. Thus, by evoking the concept of ABD the problematic issues of symptom independence, TE, and comorbidity are lessened. As researchers and clinicians, ABD conceptualizations can help us avoid a limited focus on a specific learning-related deficit while neglecting the entire profile of neurocognitive strengths and weaknesses because they do not fit well into the preferred diagnostic or categorical scheme. These cognitive strengths and weakness are, in an ABD perspective, correlated etiologically as they derive from the same coherent and integrated organ—the brain.

[4]Broader definitions of poor reading that do not require a significant discrepancy with nonreading abilities may yield prevalences as high as 20% or more, and in other linguistic populations where written language is more phonetically consistent than English, such as Italian, the frequency of RD can be significantly lower (Paulesu et al., 2001).

While we are recommending that the notion of distinct LDs or certain exceptionality categories be considered cautiously, we do not believe that research into the etiology and manifestations of specific conditions should be discontinued. Quite the contrary, such research continues to refine the nosology, symptomatology, and educational applications for exceptionalities in positive ways (Fletcher, Denton, Fuchs, & Vaughn, 2005; Fletcher et al., 2007; Fletcher, S. E. Shaywitze, & B. A. Shaywitz, 1999). We do believe that certain exceptionalities have their own neurological roots and processing components, but ABD concepts explicitly remind us that because there is variation within and between people in neurodevelopment, their symptoms, profiles, and trajectories will necessarily vary as well. The next section will help clarify this assumption.

Etiologic variability in learning disorders. It is a virtual certainty that genes play a significant role in RD-related symptoms or phenotypes and, therefore, logically, in the development of the brain that regulates them (Gayan et al., 1999; Hannula-Jouppi et al., 2005; Meng et al., 2005; Pennington, 1997, 2002; Petryshen et al., 2001; Regehr & Kaplan, 1988; Smith & Gilger, 2006). Although not as extensively studied, the research that exists on high-end abilities makes similar conclusions: superior skills have a genetic component and brain morphologies and functions have been linked to such traits (Simonton, 2005; Toga & Thompson, 2005).

At the molecular level, 10 or more tentative genes or susceptibility alleles already have been identified as contributors to RD risk (reviewed in Pennington, 2002; Smith & Gilger, 2007), and research points out that the genes putting individuals at risk for RD do not necessarily correspond to specific or independent cognitive aspects of reading ability such as memory, orthographic coding, or phoneme processing (Fisher et al., 2002, 1999; Gayán & Olson, 2003; Gayán et al., 1999; Grigorenko et al., 1997; Olson et al., 1994; Schulte-Körne, 2001; Smith & Gilger, 2007). In other words, there probably are multiple heterogeneous effects of the RD risk genes that act alone or together to give rise to multiple profiles of reading-related skills (Frith, 2001; Gilger & Kaplan, 2001; Ramus, 2001). Research suggests that genes may affect multiple brain areas and contribute to the variance in learning in a complex manner rather than in a focused, singular, and direct manner as predicted by single gene-single disorder models or by models of simple neural modularity (e.g., Aaron, Joshi, & Ocker, 2004).

Even if we consider the effects of a single gene variant for traits as complex as human learning, such a gene may yield multiple typical and atypical behaviors, especially if this gene is influential during the early stages of neural development and brain organization, or if it affects lower levels of neural organization upon which higher levels depend (Conn, 1992; Gerlai, 1996; Greenough et al., 2002; Huttenlocher, 2002; Luria, 1973; Rondi-Reig et al., 1999;

Scerif & Karmiloff-Smith, 2005). Examples relevant to this point include reports finding genes on chromosomes 3 and 6 that are linked to RD risk (Hannula-Jouppi et al., 2005; Meng et al., 2005). These genes are thought to be active in early neurodevelopment, such as neuronal migration. In one case (Meng et al., 2005), variants of the DCDC2 gene on chromosome 6 were shown to cause neuronal migration errors, although it is not known whether these errors show up preferentially in the left hemisphere as would be expected given current preferred theories.

Yet it is very possible that a developmental gene important to cortical cell migration or connection, for instance, would affect more than one brain area to varying degrees and thus have the potential to influence multiple behavioral areas (see further discussion on this topic in the next section). Even if the discrimination of primary LD subtypes (RD vs. math disability vs. ADHD, etc.) were a function of several distinct major genes influencing a different primary brain area for each disorder, the enormous co-occurrence of these conditions argues for at least some degree of multifocal action of the pertinent gene(s) and/or multifocal neurodevelopmental effects of single genes that originally operated on only specific brain areas (see also Marcus, 2004; Scerif & Karmiloff-Smith, 2005). Similarly, it is possible that through their broad neurodevelopmental effects, the genes for risk for RD may also yield some unique brains with above average capabilities to process, encode, and produce information (Craggs et al., 2006).[5]

The variable neuroanatomy of developmental learning disorders. As mentioned, it is unlikely that there is a one-to-one correspondence between a single finite brain area and type of developmental LD. More likely there would be a collection of specific brain areas, circuits or systems that act together to put an individual at risk for a certain type of LD, but these systems do not operate in isolation from the rest of the brain and other circuits. The structural or activational anomalies in the brain of an individual with a learning disability probably are numerous, although they may be more heavily focused in one region or another (e.g., Scerif & Karmiloff-Smith, 2005; Shaywitz et al., 1998, 2002). Perhaps it is the area with the heavier focus that gives rise to a person's primary diagnosis, simply because it results in the most salient profile features such as a problem with reading as opposed to math.

According to this perspective, the symptoms exhibited by people (e.g., deficits in reading, math, spelling, motor skills, attention, visual-spatial talents, or some combination) will depend on the relative amount of atypical development in primary ability areas of the brain and which of the many other brain areas also are affected. Moreover, we would expect that a complete "whole brain" study of an RD/LD individual to show peaks and valleys in abilities along with correlated variations in brain morphology and function. While it may be an over simplification, the ABD concept reminds us that every individual is a product of the neurodevelopmental variations he or she carries, and that a person's integrated and fully functioning brain is kind of an "average" of these variations across time. Within the same person, certain daily experiences will at times call upon aspects of brain functioning that may tap his ABD at the low end of the continuum (e.g., RD when asked to read) or at the high end of the continuum (e.g., high spatial abilities when asked to solve a puzzle).

Multiple brain areas have in fact been shown to be atypical in RD populations. Beyond the most commonly cited language areas of the left hemisphere, studies also have found morphological and/or functional differences in the occipital, parietal, and frontal lobes, the cerebellum, corpus collosum, and cortical thickness, among others (Démonet et al., 2004; Kibby, Fancher, Markanen, & Hynd, 2008; Mody, 2004; Nicolson & Fawcett, in press). These findings need to be reconciled with a focused left-hemisphere theory of RD. Thus, again, the brains of dyslexics may be diffusely atypical. For this reason there remains some question as to the specificity of neurodevelopmental processes underlying RD perhaps accounting for the large amount of correlated cognitive and behavioral traits typically observed in dyslexic individuals, whether they be other disorders, giftedness, or a variety of other subtle neuropsychological traits. There is an important point here particularly relevant to twice exceptionalities: that the neuroscience studies of RD-related conditions or symptoms have essentially focused on lower end skills (see Craggs et al., 2006), and while giftedness is a possible correlated symptom it has been largely neglected.

On the other hand, some researchers have discussed the idea that the atypical neuroanatomy noted in some exceptional populations could be the basis for deficits but may also be related to special skills (see Footnote 2). Although tending to focus on severely disabled samples such as those with autism and mental retardation, these researchers have suggested that differences in special populations, such as the size of the corpus callosum, cortical wiring and micorarchirecure, atypical parietal volume, and hemispheric asymmetry, may in fact be linked to superior skills (e.g., music, spatial abilities, memory; see Footnote 2 and Benbow, 1988). Thus, there is some empirical evidence that genes that put people at neurodevelopmental risk for disorders may at times also put people at risk for neurodevelopmental gifts. This is what ABD in fact suggests should occur.

This said, it is important to reemphasize that not all genes have broad neurodevelopmental effects. Some will have comparatively constrained or focused effects on the development of the brain (see other discussions on primary

[5]While this article focuses on early, prenatal neurodevelopment, there is accumulating evidence that postnatal neurodevelopment also is important to the expression and course of LD.

vs. secondary or correlated neural systems and functional units in this article). Neuroanatomical differences across species and similarities within species largely are driven by these genetics. Although brains can differ, all "normal" brains have essentially the same structures in the same place, and these structures, or parts of these structures, often are built in a way to serve specialized purposes. For example, in the human hand, normal variants have five fingers, a palm, and specialized bone placements and functions. But the size of appendages, their flexibility, dexterity, and so on can be quite different across people.

Similarly, for instance, the cerebellum is structurally and functionally different from the cortex, and portions of the temporal lobe seem prewired to acquire and mediate spoken and written language. Therefore, it is not surprising that functional MRI studies show a left temporal lobe profile common for dyslexics and it is likely that among the RD risk genes some will appear to have effects primarily in this area of the brain. In this scenario we are still faced with the finding of a diffusely atypical brain in RD individuals, we propose that even specialized genes may initiate secondary or reactive neurdevelopmental effects in other areas of the brain or functional units of the brain (see also Conn, 1992; Gerlai, 1996; Greenough et al., 2002; Huttenlocher, 2002; Kolb & Whishaw, 1998; Luria, 1973; Marcus, 2004; Rondi-Reig, Caston, Delhaye-Bouchaud, & Mariani, 1999; Scerif & Karmiloff-Smith, 2005; Thompson et al., 2002; Toga & Thompson, 2005).

RD is part of the normal continuum of reading. A thorough discussion on the genetics of this matter appears in Gilger, Borecki, Smith, DeFries, and Pennington (1996). Suffice it to say that RD does not appear to be a classic "disease." In the past there was some debate as to whether dyslexia actually exists as a learning disorder separate from the continuum of normality and whether it is an expression of unique etiologic factors (Fletcher, 1992; Pennington, 1991; Rutter & Yule, 1975; Siegel, 1989). Some researchers maintained that, if dyslexia were etiologically distinct, it should produce a "hump" at the lower end of the otherwise continuous distribution of reading scores in children as do certain forms of mental retardation defined as low IQ scores (Stevenson, 1988; Yule, Rutter, Berger, & Thompson, 1974). But several genetic and epidemiologic studies did not detect such a hump and RD appears to represent the effects of etiologic factors that are similar across the range of reading skills (Gilger, Borecki, DeFries, & Pennington, 1994; Gilger et al., 1996; Rodgers, 1983; S. E. Shaywitz, Escobar, B. A. Shaywitz, Fletcher, & Makuch, 1992; Smith & Gilger, 2007).

Moreover, reading itself was not instrumental to our development and survival as a species, although correlated traits may have been. If reading ability was somehow indirectly influenced by selection pressures on some other correlated trait, we might expect to see low-end reading abilities significantly represented in the population just as we do (Geary, 2005). There might be some characteristic that on average is more frequent in people now identified as RD that in our evolutionary past gave them some advantage. What this advantage might have been is pure speculation. For example, today's dyslexic progenitors may have had better interpersonal skills or spatial orientation abilities that gave them some sort of procreative edge (e.g., Geary, 2005; West, 1999).

What of the evolutionary and genetic history of *gifts*? Again, basic science is lacking in this area, although several authors have talked about the IQ distribution, special talents, and higher-order skills as being necessary considerations of models of human evolution (Geary, 2005; Marcus, 2004; Schneps, Rose, & Fischer, 2007). As these writers have suggested, and in alignment with an ABD concept, such variation is expected given the nature of the developing brain as it has interacted with its environment over time. So, everyday, humans necessarily produce other humans with the potential to be on both sides of the continuum as a matter-of-course and population variability. Whether or not these natural variations are manifested as LDs or gifts, or both, depends on the environment, experiential opportunities, focus of culture, and relevant current diagnostic schemes.

SUMMARY

First, the ABD concept suggests that variation occurs first at the neurological level and that this variation occurs within and between people in the population. Further, this variation can lead to correlated symptoms or traits, and the interpretation and identification of this variation will depend on the level of analysis applied: from the level of the neuron or neurological structure, to the level of neurological function, and, at the most removed level, that of behavior. Politics and a researcher's area of interest have been the determining factors in what has been looked at. However, brain variation and ABD often will go unnoticed in the laboratory unless the "whole brain" is assessed appropriately and with sensitive enough instruments. Similarly, the consequences of atypical brain development may go unnoticed in a person's everyday life unless his experience or life demands tap into his personal developmental peaks and/or valleys (i.e., a dyslexic when asked to read or a talented dyslexic when asked to create art).

Second, it is unlikely that there is a simple one-to-one mapping of single genes onto brain structures, or brain areas onto abilities, and this is especially true when the concern is complex cognitive traits, the effects of developmental genes, and the development of cognitive abilities across the lifespan (Changeaux, 1985; Hahn, van Ness, & Maxwell, 1978; Johnson, Munakata, & Gilmore, 2002; Jones & Murray, 1991; Noctor, Flint, Weissman, Dammerman, &

Kriegstein, 2001; Scerif & Karmiloff-Smith, 2005). Therefore, we suggest that molecular gene mapping and brain imaging studies need to take a multivariate and whole-brain approach (Butcher, Kennedy, & Plomin, 2006; Gilger & Kaplan, 2001; Toga & Thompson, 2005). Too often such research is limited to specific disorders or univariate phenotypes. Consequently, while supposedly finding genes or brain mechanisms for RD, the scientists also may have found that the same genes or brain areas highlighted also influence, say, other cognitive deficits, giftedness, or spatial skills, had they run such analyses.

A Pictorial Summary of the ABD Concept

Figure 1 presents the way that the ABD model conceptualizes the role of individual and group differences in neurodevelopment, TE, and RD. For the sake of clarity and simplification we will assume that all distributions discussed are fairly normal. Of course, Figure 1 simplifies many complexities and confounds, and it is meant to serve only as a point for discussion.

In view of Figure 1, a domain-specific learning ability, like reading, depends on multiple brain-based subabilities (e.g., memory, orthographic coding, phoneme awareness, etc.; Figure 1A) that in combination average to yield the normal (multivariate) distribution of global reading ability in the population (e.g., a general reading score on a standardized test; Figure 1B). The range of these subabilities is due to variation in neurodevelopmental structures, all of which are influenced to some extent by genetics and experience. If we had the correct measurement instruments, these structures might look different across people at the very microscopic level (e.g., arborization, cell number, neuronal memory nets, etc.) and at a more macroscopic level (e.g., size of structures or asymmetries).

At whatever level of analysis, it is hypothesized that structures correlate with internal brain function, which in turn correlates with externally measured behaviors. As predicted by a normal distribution, the fewest number of people will have brains with "superior" structure and concomitant superior reading ability, or "inferior" structures with inferior ability.

The characteristics of some of the most important structures, such as cell number and cortical layering or connectivity due to migrational effects, were set during the prenatal period. Other characteristics come from later brain elaborations such as myelination and neuronal connections due to learning. ABD recognizes that there is variation in these structures affecting internal brain function and therefore the external behavioral abilities shown across people and within

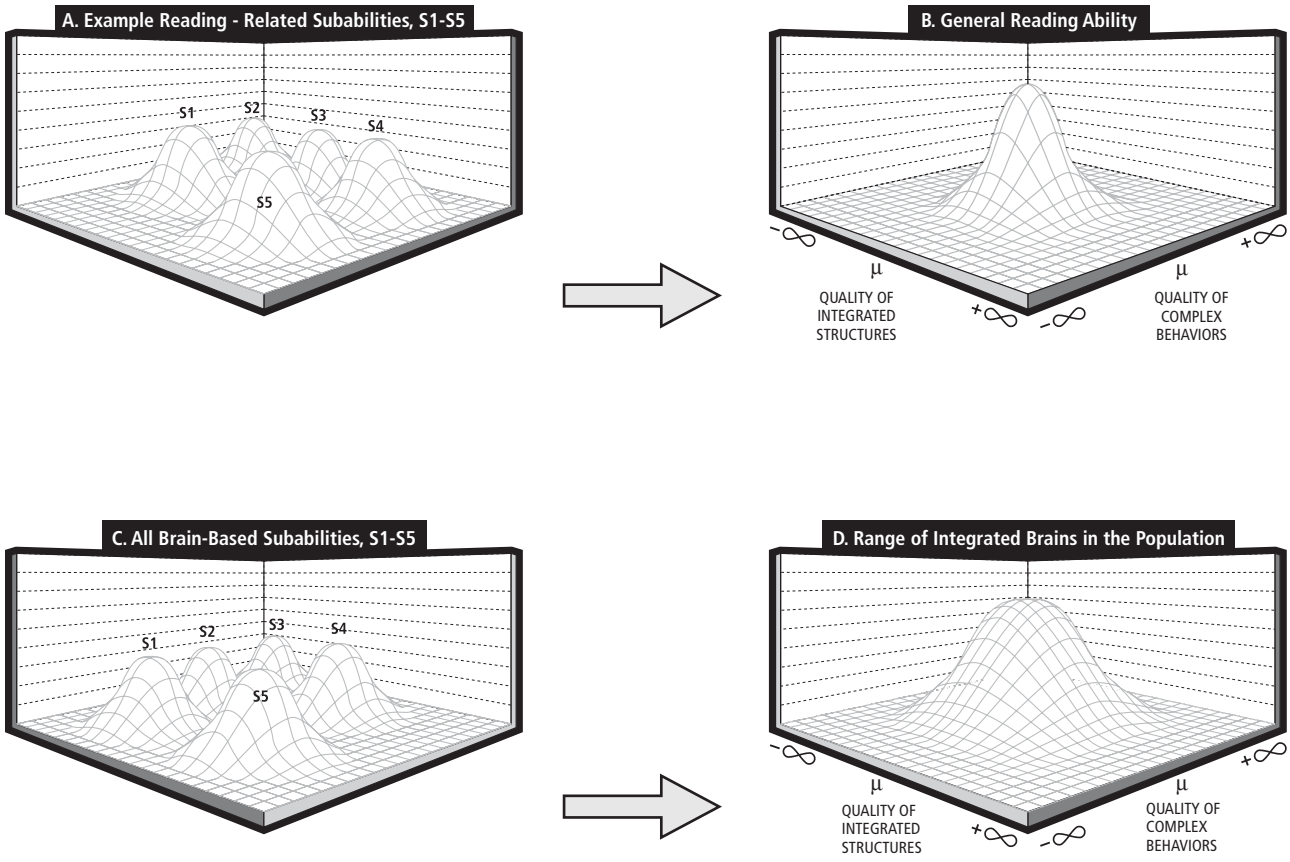

FIGURE 1 The ABD model. See text for explanation. Figure modified from Gilger and Wilkins (2008); printed here with permission from Guilford Press.

people. It is primarily through these effects that we see variations in global reading scores, which simply mirror average brain functioning for reading-related subabilities. These effects also lead to profiles of strengths and weaknesses within people for reading-related subskills (e.g., memory, phoneme awareness, and others).

Figure 1 further expands the example for reading to all brain-based cognitive skills (five subskills shown; e.g., spatial, verbal, speed, etc.). Again, ABD proposes that there is natural within- and between-person variability in the structures relevant to these skills and therefore the correlated behaviors (Figure 1C). The average of the functional effects of these structures essentially yields a "continuum of integrated brains" and general cognitive ability in the population (Figure 1D). Thus, the average integrated brain represents the "mythical normal brain." But like the mean in a theoretical normalized z-distribution, the mythical normal brain probably exists less in reality than in imagination. Again, within-person variation in brain-based structures and correlated functions determines an individual's behavioral profile of strengths and weaknesses.

APPLICATIONS AND IMPLICATIONS OF THE ABD CONCEPT

The conceptual tool of the ABD model should be used in conjunction with other perspectives in the field of exceptionality. Recognition of the utility of the ABD concept does not require dispensing with efforts to differentiate or refine exceptionality categories. In fact, it is our hope that the implications of the ABD model will become part of the contemporary psychology of the exceptional or special education field, sharing space with (not replacing) the more molecular approaches to research and treatment (see also Kalbfleisch, 2004). Here, specifically, is a summary of what is offered by thinking about ABD in practice and basic research:

Remember to Use a Whole-Brain Perspective

ABD as a concept strongly advocates for developmental and experimental studies of the whole brain. By this we mean that the commonplace methods of study that focus on distinct categories or the specific and microanalytical cognitive processing approaches to a behavior should be supplemented with an explicit awareness of the brain as a whole organ. An illustration of this point can be found in the typical brain imaging or genetic study of children with RD, which typically assesses, and then considers, mainly verbal skills but not (for instance) motor coordination, attention, or spatial gifts. This approach has been common in the past, but the ABD model would suggest expanding the assessment into realms other than verbal skills. Interestingly, when other, even nonlanguage areas of the RD brain are examined, they are often found to be atypical as well and multivariate behavioral genetic research suggests that many specific cognitive traits have genes in common (Butcher et al., 2006; Light, DeFries, & Olson, 1998; Mody, 2004).

The call for a broader approach is important, as is the recognition that the search for the genes that influence learning abilities, disabilities, or specific cognitive processes is really a search for the genes that determine atypical brain development or population variance for neuronal structures (Gilger, 1995; Gilger & Kaplan, 2001; Jones & Murray, 1991). We are looking for genes that cause brain variants, such that some people fare better or worse than others when it comes to learning. Consequently, future genetic research would be strengthened if it were to include multiple measures of brain integrity and function, including, but not limited to, tests of general reading, word recognition, math, processing speed, motoric processes, attention, visual-spatial abilities, and higher-order skills. Research employing a more limited phenotype may miss much of the complexity of an individual's biology and skills.

Learning Ability Variation, Across the Continuum, Can Be "Normal"

The ABD concept considers some "deficits" and "gifts" to be "normal" variation. It is one of the main tenets of the ABD approach that there is variation in brain structures resulting in functional variation that can be identified at the behavioral level—sometimes as apparently unitary traits such as RD or high-spatial ability. But the compartmentalization of a collection of traits into supposedly independent categories may be misleading. Rather, the ABD concept suggests that such traits also could be viewed simultaneously as simple symptoms of brain variation and that the best method for understanding etiology and treatment approaches is to consider both the category as well as the broader presentation of traits as reflections of a variable brain or a diffusely atypical brain. We recommend that with ABD as a sort of general perspective, more detailed and specific theories of brain-behavior relationships, networks, brain-processing components, etc. (e.g., see Eden & Zeffiro, 1998; Fletcher et al., 2007; Mody, 2004; Ramus, in press) can be tested and applied where valid, thus expanding our understanding even further.

It is worth reemphasizing that ABD considers it perfectly normal to have both significant deficits and strengths in the same person. This is more than simple within-person ability variation: it means that there is a possibility that a child who cannot read also can show spectacular gifts in art or math, where both abilities may reflect different aspects of the same atypical brain development mechanism that may have been operating in utero. The factors (e.g., cell migration

genes) causing ABD that yield reading deficits may be the same factors that yield a brain with a propensity for specific intellectual gifts as well. Given the assumption that this sort of ABD is normal, we expect certain proportions of twice-exceptional, but otherwise typical, people to be born every-day, although they may go unnoticed.

Reminder That Behavioral Phenotypes Can Be Considered in Terms of Neurology

The concept of ABD helps us redefine behavioral phenotypes into terms dealing with their ultimate basis: the brain. This is important, as it often appears that behavioral phenotypes are driving brain research rather than brain research driving phenotypes. This is perhaps a necessary state-of-the-art, as our ability to view the brain in situ has only recently become possible (Kennedy, Haselgrove, & McInerney, 2003) and we are still very heavily reliant on psychometrics. However, along with the common methods of brain study, additional and unforeseen information may be obtained by approaching research from a bottom-up alongside a top-down methodology. Specifically, we suggest the need for large-scale multivariate analyses where common brain areas are sought that show up in atypical and typical behavioral phenotypes as well as concomitant studies that begin with brain-imaging results and then look for their behavioral expression. Reconciliation of the findings from this sort of work is a first step toward a more fully developed and reliable picture of the brain, brain-behavior relationships, and individual variation in the population.

Developmental Models are Important

ABD makes a general statement that a developmental perspective must be maintained when talking about genetic and neurologic effects on the learning or cognitive system. At different points in time the surface phenotype of, say, a reading or spatial talent test may reflect genetic and environmental effects that occurred when the brain was just starting to form and/or genetic and environmental effects on the brain that continue to affect reading and spatial skills throughout life. Certain aspects of brain development, like cell migration and differentiation, occur very early after conception and then more or less stop, and the key genes moderating these processes "turn off" or serve other functions (Galaburda, 1992, 1993; Greenough et al., 2002; Huttenlocher, 2002;). This fact reminds us that when we are looking for genes that have affected brain structure to put a person "at risk" for RD or gifts we may be looking for genes that are no longer active (Craggs et al., 2006; Gilger, 1995; see Note 5). Genetic effects at this basic level of neurodevelopment may account for the relatively high degree of stability across time for reading deficits and related research that shows that much of this behavioral stability is due to genetics (Butcher et al., 2006; Wadsworth, Corley, Plomin, Hewitt, & DeFries, 2006).

Avoidance of One-Size-Fits-All Ways of Thinking

The ABD concept helps us avoid single etiology or one-size-fits-all schemes of thinking. It is again worth emphasizing that the ABD approach does not advocate against ongoing research or treatment approaches based on subtypes or diagnostic categories for LD or TE. Instead, we hope that the implications of ABD can be incorporated into these current models of study and treatment. The ABD concept should not be taken as anticategorization or antilabeling. Perhaps the ABD concept can best be thought of as a "thinking tool" that when used conjointly with other tools will result in a broader and more accurate picture of the etiology of complex human behavior. Simple diagnostic schemes or treatments that focus only on one aspect of the behaviors or symptoms of the atypical brain (e.g., RD, phonemic awareness), especially when intellectual gifts and deficits can coexist, do not do justice to the underlying causes of what is observed at the surface, and they increase the likelihood of treatment failure because of a limited focus on one behavioral category. Appreciating the complexities of within-person abilities has value in diagnostic and treatment domains and also has implications for prevention (see also Bergman & Magnussen, 1997; Shapiro, Church, & Lewis, 2002).

How to Deal with LD People with Multiple Cognitive Weaknesses and Strengths

The ABD concept may be especially useful in understanding the child or adult who does not fit the usual or simple diagnostic schemes. At all ages, there are individuals who appear to have a variety of symptoms without clear etiology or coherence. These cases can be very confusing and frustrating to clinicians and educators, often requiring a huge amount of management effort, diagnostic tests, and failed treatments. Often such individuals do not receive appropriate help and, especially as children, can end up receiving placements in special programs better suited to other forms of LD when they have so many other issues ongoing.

Complexities also are likely to be seen in adults seeking assessments as so many life experiences, skills, coping mechanisms, and learned behaviors can decrease the clarity with which deficits are manifest (Goldstein & Kennemer, 2005). We have seen such cases with a diffuse clinical picture, often exhibiting traits spanning nonverbal LD, reading, coordination and math problems, social skill deficits, anxiety, talents, and so on, while exhibiting basically normal IQs and an ability to function fairly well with guidance and practice. Applying the suggestions of the ABD concept does

not require that such individuals be diagnosed per se, but rather that they be recognized as having a diffusely atypical brain, of whatever etiology, that does not yield itself to simple classifications. Such individuals require broad-based assessments and treatments that focus on the many symptoms exhibited as well as the strengths displayed.

More on ABD's Diagnostic Utility

We certainly are not the first to question the value of diagnostic categories (e.g., Rapin 2002). Yet there are many pressures to assign diagnoses to people, even while the concept of individual differences is being acknowledged, and it is possible that these pressures sometimes prevent thorough educational assessments of various skills. Recognition that children with ABD represent an enormously heterogeneous group at the neurological level can have important beneficial effects on educational assessment (and treatment) strategies. In our opinion, for educational purposes, people need to be assessed for their individual strengths and weaknesses, and treatment plans need to be developed to address both. But financial, cultural, and other pressures often exist to distill a person's complex pattern of strengths and weaknesses into a few words describing a categorical diagnostic label with all of the implications and beliefs thereof. While diagnoses tell us something important about the person and they can provide research-based information and guidance, the ABD concept emphasizes a thorough, broad-spectrum analysis of each individual with the explicit aim to identify and track irregularities on *both* ends of the continuum.

We recognize that the financially driven pressure to categorize for service funding is not likely to disappear quickly under any new conceptual framework. But there is good reason to believe that this pressure to categorize is more than financially driven and only an open-minded reconsideration of the available data and approaches to research will allow a different perspective leading to different therapeutic and diagnostic techniques. Today, for instance, there is a movement to initiate a multistep "three-tiered model" for remediation in the schools (e.g., Fletcher et al., 2007; Vaughn & Fuchs, 2003). The focus of such models is not so much diagnosis per se but rather a careful assessment and tracking of a student with a possible LD and a responsive and evolving research-based remediation plan.

In this model, at the first two levels of intervention a child need not necessarily be diagnosed as a certain LD type to receive services in the regular education classroom. Instead, the teacher and other staff identify a student struggling with, say, reading, in the regular curriculum and then initiate a form of intervention with monitoring. The teacher is to modify his teaching methods according to how the student responds. This multilevel approach is just a beginning of what are some major changes in the LD field to come and it fits in well with the ABD concept, where diagnosis is less important and the focus on symptoms is key. It may be wise to institute an analogous plan for the fairly neglected gifted child as well.

FINAL WORDS

The history of the study of developmental disorders and exceptionalities is replete with debates between *lumpers* and *splitters*. Lumpers are people who develop large theories that tend to encompass many aspects of an issue; splitters are those who tend to be more concrete and who attempt to categorize and subtype a phenomenon into smaller parts. What we propose is that both approaches can live side-by-side harmoniously (Gilger & Kaplan, 2008).

Our proposed approach of employing the concept of ABD does not presume to exclude the definition of a circumscribed phenotype, but rather it suggests that there is value in more broadly characterizing the skill sets, neurology, and biology of the individuals being studied. The coexistence of the lumping and splitting approaches likely would offer an improved approach in both clinical and research applications, particularly with adults. Figure 2 shows one way of thinking about the ABD model, perhaps a bit on the lumping end, and how it might help the field better include considerations of LD in general as well as gifted and twice exceptionalities. As shown, ABD can serve as a framework alongside other more specialized and focused theories or approaches.

As Figure 2 suggests, all neurological/neuropsychological research essentially deals with the atypical or typical brain, depending on its focus. Hence, for developmental disorders and exceptionalities we show in brackets that ABD can serve as the broader concept under which would fall more specialized research. As the picture implies, the specialized type of research should not occur in isolation but should take place in a bigger context. To put it succinctly, this figure highlights the fact that as researchers and educators we might do well to acknowledge the role of the whole brain, lest we lose sight of the forest for the trees.

AUTHOR NOTES

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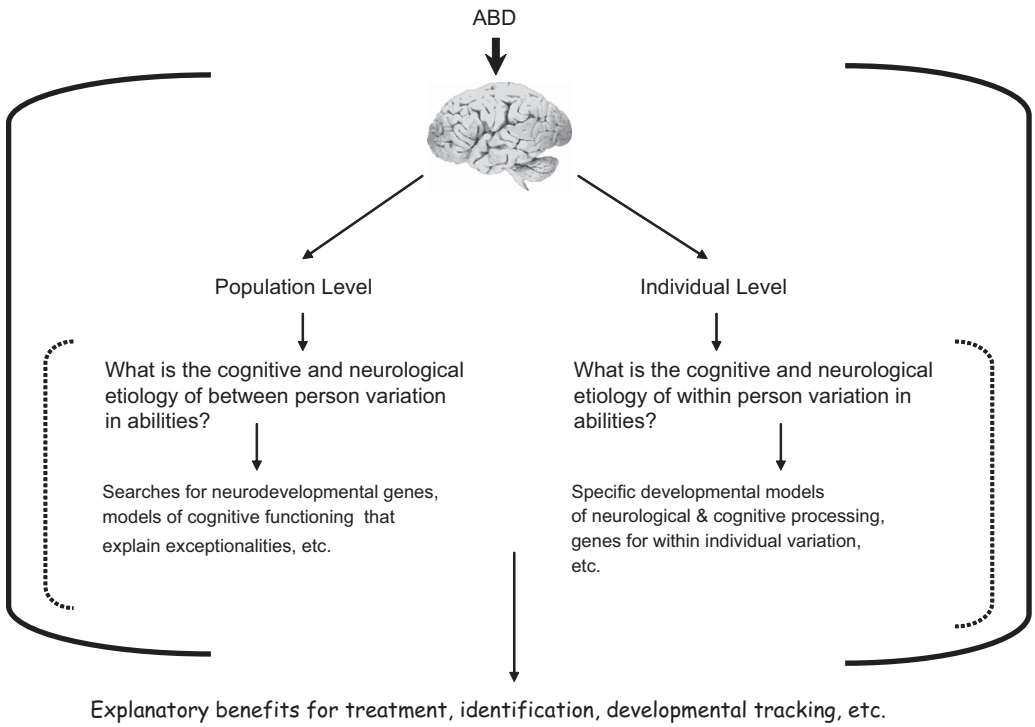

FIGURE 2 ABD as a collaborative model towards conceptualizing learning disorders, twice exceptionalities and inter/intraindividual differences. Figure modified from Gilger and Wilkins (2008); printed here with permission from Guilford Press.

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AUTHOR BIOS

Jeffrey W. Gilger, PhD, is currently the College of Education's associate dean for discovery and faculty development and professor of special education and psychological sciences (courtesy) at Purdue University. His background includes an MS and certification in clinical child/school psychology and an MA and PhD in experimental/developmental psychology with a specialty in behavioral genetics. His teaching and research has tended to focus on normal and abnormal neuropsychological development, genetics, and the etiology of learning-language disorders, especially dyslexia. Recent research projects include the neurology/genetics of the gifted-learning disabled individual and a multisite project on the efficacy of reading remediation programs. E-mail: jgilger@purdue.edu

George W. Hynd, PhD, is senior vice provost for education and innovation and dean, Mary Lou Fulton College of Education at Arizona State. He is a Fellow of Division 16 (School Psychology) and 40 (Clinical Neuropsychology) in the American Psychological Association (APA) and is a Fellow in the National Academy of Clinical Neuropsychology (NAN). His research has been published in 11 authored or edited books (e.g., *Pediatric Neuropsychology, Neurological Basis of Childhood Psychopathology, Neuropsychological Assessment in Clinical Child Psychology*, etc.), in 57 book chapters, in 153 refereed journal articles, and includes gaining a better understanding of the neurobiological basis of childhood behavior and learning problems. E-mail: George.Hynd@asu.edu