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Autism and Dyslexia: A Glance Over 25 Years of Research

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Abstract
Autism and dyslexia are wrongly classified as childhood disorders: They are lifelong and therefore have to be studied in adults as well as in children. Individual variability is enormous, and, as a result, behavioral diagnosis remains problematic. The study of the underlying cognitive abilities in autism and dyslexia has acted as a gateway for the emergence of developmental cognitive neuroscience.

Keywords
developmental disorders, Asperger syndrome, cognitive neuroscience

A Fundamental Truth About Developmental Disorders

A big turning point in my research was the realization that the developmental disorders I studied, autism and dyslexia, are lifelong conditions and should also be studied in adults. Twenty-five years ago, it was odd to see a developmental psychologist recruit dyslexic and autistic adults as participants in experiments. There were no diagnostic instruments for adults at that time. Now that we have these instruments, some deeper truths have emerged: First, diagnosis based on behavioral criteria will always be problematic, as behavior is strongly influenced by comorbidity, motivation, age, and ability. Second, compensatory learning can circumvent or cover up, but not eliminate, some basic difficulties in cognitive processing. Third, comorbidity and low general ability tend to reduce opportunities for compensation.

Twenty-five years ago, it became apparent that many dyslexic adults had learned to read accurately and could achieve an acceptable reading age. Later, it also became apparent that, in Italy, dyslexic children read perfectly accurately. This success is due to the transparent and regular Italian writing system, which I have suggested can act as a therapeutic environment for dyslexia (U. Frith, 1999). However, we were able to demonstrate the presence of dyslexia in Italy and the persistence of reading problems in dyslexic adults using measures of reading speed and tests designed to tap phonological processing. These measures revealed underlying neurocognitive problems, indicating that they are universal, regardless of language, and persistent, regardless of compensatory learning (Paulesu et al., 2001).

In the case of autism, the huge range of clinical manifestations is a major problem. This can be observed not only by comparing different individuals, but also by tracing changes during the lifetime of a single individual. Thus, since the 1980s, it has been known that some individuals start off being aloof and withdrawn and later become socially active but odd. Repetitive behaviors often lessen with age; special interests can vanish and savant talents can change. It is still not clear what causes these changes and wide variations, but comorbidity increases severity whereas compensation makes it possible to disguise persisting problems (U. Frith, 2008).

Adults with dyslexia and adults with autism who show good compensation can sail through standard diagnostic tests. Have they ceased to be dyslexic or autistic? Of course not. They themselves tell us about their persisting problems, and we can reveal these problems with more subtle tests. We now know that dyslexics read well at a cost of time and effort. The same is true for autistic people. They can use well-rehearsed social scripts that work

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in familiar situations. However, stress and unfamiliar situations can produce catastrophic collapse. To visualize the differences between surface behavior and underlying cognitive mechanisms, I have found it useful to apply a framework representing three levels of explanation: behavior, cognition, and brain (U. Frith, 2001). These do not map onto each other in one-to-one fashion. Rather, a large number of biological risk factors may lead to difficulties in a few specific cognitive processes. These few difficulties in turn may give rise to a large number of behavioral signs and symptoms (U. Frith, 2012).

A Fundamental Change in Methods: Looking Inside the Brain/Mind

It is a little known fact that the development of diagnostic tests for adults gained impetus from the desire to make use of the emerging neuroimaging techniques. This advance occurred because it seemed wrong to scan children. In fact, when we scanned the first dyslexic adults in the mid 1990s, we had known them from when they had been first diagnosed as children. The same was true for the first autistic adults. The diagnostic tests now available have been a great boon in many ways, but especially for those people who had slipped through the net and were not diagnosed as children. They had unjustly been labeled slow, lazy, or unintelligent. Having a name for your problems and learning that there are others like you can be liberating.

It is remarkable how quickly research on disorders of cognitive development has transformed into cognitive developmental neuroscience. This advance was due to the generous collaboration of researchers who pioneered neuroimaging methods and volunteers who braved the still novel scanners. In those heady days, we naively hoped that we would be able to immediately identify the malfunctioning brain circuit that gave rise to critical difficulties in cognitive processing, such as difficulty in phonological processing in dyslexia or difficulty in social processing in autism. Things turned out to be far more complex, particularly as we are still largely ignorant of how the brain works. Which brain circuits are necessary for distinct cognitive processing mechanisms? How do they function in neurotypical brains? We had, and still have, a lot to learn.

Today, when researchers have reassured themselves that MRI methods are safe also for children, the field is changing again. There are now several longitudinal studies in progress in which individuals are scanned repeatedly over many years. This work is giving us information about the developing brain and mind that we simply never had before. For instance, there is a dramatic waxing and waning restructuring of gray and white matter throughout childhood and adolescence. This work should get us closer to understand which cognitive processes might go wrong in neurodevelopmental disorders and when.

The Rise and Fall of Asperger Syndrome

Autism has always been with us, but it has been recognized only since the mid 20th century, and then only very slowly. The prevalence is now thought to be around 1%, which is a staggering increase from the 0.1% estimate from 25 years ago. One major reason for the increase is the widening of the diagnostic criteria (U. Frith, 2008)—the spectrum of autism is now incredibly broad. At one extreme is the individual who does not use speech and has severely repetitive and restricted behaviors; at the other extreme is the individual with Asperger syndrome who is highly articulate and has superior analytic abilities and fascinating special interests. Despite these differences, there is a common denominator: a highly recognizable lack of reciprocal social interaction. Even the very able individual on the milder part of the autism spectrum cannot compensate for a peculiar void in social interaction: engaging in the ordinary to and fro of everyday human communication (Frith, 2004).

The widespread public recognition of Asperger syndrome was impossible to foresee when I translated Asperger's landmark paper (U. Frith, 1991) at a time when most people had never heard of the name. But the rise over the last 20 years may now be followed by a fall—the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* has abandoned the label. It was removed because it is widely agreed that the critical features distinguishing Asperger syndrome from other neurodevelopmental disorders are the same as those seen in autism. In addition, there is a largely unrecognized downside to the rise of Asperger syndrome: Researchers have become less interested in studying those who are intellectually disabled. These individuals are much more difficult to study. Yet they have so much to tell us, if we only knew the right questions to ask.

Mechanisms of Social Cognition

Autism provided the inspiration for the idea that social information might be processed differently from other information and given priority in neurotypical minds (U. Frith & Frith, 1999). Of course, we still don't know what the processes are like and how the prioritizing works. Autism throws the spotlight on social communication, and furthermore, it reverses the tables: What is it that makes us insatiable in our craving for social communication?

Many different mechanisms in our mind are dedicated to processing social stimuli (C. Frith & Frith, 2012). One of these, known as Theory of Mind, or *mentalizing*,

entered psychologist's jargon as shorthand for the tendency to attribute mental states to each other. Amazingly, this tendency has a circumscribed basis in the brain (U. Frith & Frith, 1999) and has been operationalized by tasks such as the Sally-Ann test. In this test, children above the age of 4 can attribute a false belief to Sally, but autistic children cannot do so. However, it turned out that this is not the whole story—able autistic adolescents and adults can pass the Sally-Ann test and similar tasks.

There is also a nonverbal type of intuitive mentalizing, which can be assessed via eye gaze patterns, and here we get a different story: Even young infants engage in this intuitive mentalizing, but able autistic adults do not (Senju, Southgate, White, & Frith, 2009). Perhaps this is why they struggle with everyday fast moving reciprocal communication even when they have learned to attribute mental states explicitly (U. Frith, 2012). Many questions are now being raised about mentalizing and its likely presence in other species, such as birds and dolphins. What is uniquely human in social communication? Why have only humans invented governments and schools, and how do they manage their reputation in the age of the World Wide Web (Tennie, Frith, & Frith, 2010)? So much remains to be explored in the next 25 years.

Declaration of Conflicting Interests

The author declared no conflicts of interest with respect to the authorship or the publication of this article.

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