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Introduction

Seeking Coherence in Autism: From fMRI to Intervention

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Introduction

In Kanner's classic paper describing the developmental phenomenon now known as autism, two statements in particular stand out as distilling the essence of the condition. The first statement describes the quality of interpersonal and social relating, while acknowledging its natural or biological origin:

> The outstanding "pathognomic," fundamental disorder is the children's inability to relate themselves in the ordinary way to people and situations from the beginning of life (Kanner, 1943, p. 242).

The second statement, urging the reader to think about how the person with autism experiences the world, captures the remarkable struggle to cope with change in routine or context:

> Their world must seem to them to be made up of elements that, once they have been experienced in a certain setting or sequence, cannot be tolerated in any other setting or sequence; nor can the setting or sequence be tolerated without all the original ingredients in the identical spatial or chronological order. Hence the obsessive repetitiousness (p. 249).

The descriptions of these apparently disparate behaviors, so elegantly and meticulously expressed, create compelling images of what autism is. They note in the process its origins from the beginning of life—that is, its biological root—and the

sense of a fixed, yet fragmented world that is unnervingly unpredictable. Kanner's influence has been profound and enduring: These two aspects of behavior are still represented today in the diagnostic criteria for autism—the first in the impairments in social interaction and communication and the second in the repetitive behaviors and desire for sameness.

Autism is a newly discovered syndrome, relatively speaking, and the contribution of Kanner and of other outstanding researchers and clinicians notwithstanding, it is marked as much by what professionals do not know as by what they do know. Despite rapid advances in neuropsychology, epidemiology, etiology, diagnosis, cognition, and, recently, genetics, the research community continues to wrestle to identify biological causes and developmental processes, at the neural, cognitive, and affective levels. It is, however, an exceptionally vibrant research environment, which is producing an outstanding number of new findings and approaches within its individual specialisms. The challenge is to develop a fuller understanding within these highly productive, specialized, research areas while, increasingly, considering how new findings fit into and even alter the whole knowledge base. The ultimate goal is to integrate research findings fully in order to map out the boundaries and the landscape of autism. This would be a substantial aid to early and precise diagnosis, provide a guide to the most effective remediation, and, in the process, add considerably to our understanding of both atypical and typical development at the levels of brain and behavior across the lifespan.

Autism is not a unitary concept. It consists of a miscellany of features, some of which have no obvious relationship. It appears to varying degrees in different individuals and coexists with a variety of medical conditions. Academic researchers have made continuing efforts in the last 25 years to establish whether these apparently disparate features can be the outcome of a set of simpler core features underlying the condition (Leslie, 1991; Morton & Frith, 1995). Much effort has been made to discover a pattern of cause and effect or association, initially at a relatively simple level, either by demonstrating how features might be linked directly (for example, atypical affect leading to difficulty in social cognition (Hobson, 1993)), or by showing how they could be incidentally related at the neurological level (for example, through linked pathways in the brain (Baron-Cohen & Ring, 1994)). Recent hypotheses are more complex. One posits that the two dominant characteristics of autism, the social and non-social behaviors, may be coexistent but not genetically co-dependent (Ronald, Happé, & Plomin, 2005). Another identifies no fewer than six candidate broader phenotype autism traits (Dawson et al., 2002), while functional magnetic resonance imaging (fMRI) studies propose connectionist models of brain function that indicate atypical modulation of activity and incorporate compensatory mechanisms (see Wicker, Chapter 2). How to rationalize all these? Considerable progress has been made, but it seems that,

paradoxically, still further expansion is needed, both within and between research fields, before a fully coherent account can be achieved.

The Challenge of Integration

The reference to seeking coherence in the title of this chapter relates first to the longstanding goal of seeking a coherent explanation for the constellation of behavioral features that comprise autism. However, of no less importance, it relates to the process by which that will eventually be achieved, by referring more specifically to the need for coherence among or across different fields of inquiry. Over time, research areas have become increasingly specialized, with very limited inter-area collaboration or "cross-talk." Yet, ultimately, a coherent account of autism can be created only by a process of integration of research perspectives and findings. It is tempting to use the analogy of a jigsaw puzzle in stressing the need to integrate across research fields, but this would be simplistic, as the task is not merely to join up information from different specialisms, but to merge it. This is no small challenge and there are two obvious difficulties inherent in that process. First, the sheer volume of published work makes the task of keeping pace with all aspects of research daunting. In the years since Kanner's publication, and particularly in the past decade, research on autism has grown exponentially. Volkmar, Lord, Bailey, Schultz, and Klin (2004) observe that almost 3,000 articles have been published on autism between 1994 and 2004. This is, they note, equal to the number published between 1943 and 1989. High-quality reviews of specific research areas at key points do reduce the problem, though they do not remove it. To review the complete literature is now impossible, although two systematic overviews have eased the situation by providing highlights and main achievements across research fields (e.g., Bailey, Phillips, & Rutter, 1996; Volkmar et al., 2004).

The second difficulty is the greater of the two. As recognition of the complexity of the condition has increased, the growth of research has not followed a narrowly defined route in the predictable areas of medicine and clinical psychology, but has expanded into many others. Autism research is now represented in several areas of psychology, including developmental and biological psychology, ethology, and comparative cognition, cognitive neuroscience, and, of course, clinical and educational psychology. It is also researched within medicine, in neurology, genetics, and child psychiatry, and in other professional areas such as education and speech and language, music, and art therapy. Beyond the obvious medical/psychological areas it has attracted the interest of academic researchers in philosophy, computer science, and linguistics (Carruthers & Smith, 1996; Moore & Taylor, 2000; Stirling & Barrington, in press). Each specialism has an understanding of the condition

within a certain model of functioning, with its own circumscribed knowledge base and terminology and even its own academic culture (in particular, the medical model and the educational model are very different and not always compatible: see Chapter 16). These have resulted in a range of perspectives on autism and could produce quite different answers to the questions "How should autism be defined?" and "What are the fundamental processes through which it develops?" To merge these perspectives would require a decision or agreement about employing a broadly comprehensible terminology, a willingness to explain one's own field, while striving to comprehend another, and a consideration of autism as it is represented in two or more research contexts. Moreover, the answers might be multilayered. After all, it is some time since Goodman (1989) observed that there might not be one overarching mechanism that explains autism. The temptation is to ask, "Is it unrealistic to talk about integrating perspectives?" Is there another way?

The response to these concerns is a growing recognition that effective progress from now on will, in fact, rely on integration. A number of major contributors to research in autism have considered the issue and reached similar conclusions on the need for integration, despite its potential complexity. More than a decade ago, Bailey et al. (1996) first mooted the idea that integration of perspectives should be the way forward, at the same time acknowledging that any attempt needs to recognize that a range of causal models has to be considered. More recently, Dawson et al. (2002), reviewing the cognitive neuroscience of social and language impairments in autism, made a compelling case for integration across genetic, cognitive neuroscience, animal, and clinical studies, of not only research findings, but also approaches and concepts. They maintained that this is a requirement if there is to be progress in understanding. Volkmar et al. (2004) have observed that the full benefit of isolated breakthroughs will actually depend on connections between different fields of inquiry. In fact, they go further, proposing that mandates for future research should include the building of an integrated model of the constructs and mechanisms involved in the pathogenesis of autism. There is little dispute, then, that this is the next rational research step, and that there is a need to merge.

We have outlined the two most obvious difficulties. But there is also a less obvious, although very important problem nested within the second. Any reference to integration usually occurs within the context of integrating aspects of research within medicine (or the clinical diagnostic area), within psychology (or social and neurocognition), or between these two. However, the third main strand of research, that of education/intervention is almost invariably overlooked. Rarely has an explicit reference been made to incorporating this aspect within the integrated frame, though some authors have broached the matter briefly (see, e.g., Sigman, Spence, & Wang, 2006).

Yet one would expect that intervention should be highly dependent on the

other two areas, and, in addition, that the outcome of research on intervention would offer considerable insights into the nature of autism. With regard to the first point, Volkmar et al. (2004) comment:

> A major concern is the large, and possibly growing, gap between what science can show is effective, on the one hand, and what treatments parents actually pursue. Another concern is the extent to which the full benefit of scientific research is translated into best practices in actual classroom settings (p. 155).

Sigman et al. (2006) endorse that view. There is, therefore, support for the argument that research and intervention programs should be connected; however, it is not in the context of a move toward integration more broadly. A restricted conception of the role of intervention within any explanatory model has implications for understanding autism: It is undervaluing the contribution that research in intervention might make; additionally, it is distancing the work and potential contributions of the practitioner from those of the researcher: a loss to both. Consequently, the outcomes of both research and practice in intervention could and should be part of any integration framework. In Chapter 16, Leekam and McGregor propose an explanation for this gap: partly an historical outcome of the way in which research in autism evolved such that the general framework has emphasized the causal rather than the developmental; partly a consequence of the dominant role of medicine in the initial characterization of autism.

Those advocating integration have been responding to a perceived need for a common framework to explain autism focused predominantly on causation—or at least causal mechanisms. However, there is a risk that considering theoretical models only in terms of causal mechanisms might underplay the essential contribution of development. Yet autism is defined as a developmental disorder; there is no question that the developmental aspect is intrinsic to the condition, and any explanatory framework must account for this fact. If it has been overlooked in earlier models, an integration of perspectives provides the opportunity needed to incorporate development as it deserves.

There has, certainly, been an increase in awareness of the need for longitudinal data in order to track developmental trajectories, an encouraging shift (Hale & Tager-Flusberg, 2005; Hazlett et al. 2005; Landa & Garret-Mayer, 2006; Lord, Shulman, & DiLavore, 2004; Munson et al., 2006). These studies are predominantly diagnostic, however, and generally lookat change over time on one or two measures. Some do refer to developmental trajectories, though they are not designed for any elaborated modeling of interactive developmental processes. A developmental perspective is certainly emerging. For example, Klin, Jones, Schultz, and Volkmar (2003) locate their discussion of the "enactive mind" in autism within a clear developmental context; and although they do not explicitly advocate a wider-ranging,

integrated approach to autism research, they do indicate a need to increase the range of methodologies to study social adaptation (see also Jones & Klin, Chapter 4). During the past decade, a developmental approach has been proposed for other developmental disorders, arguing that the necessary frame of reference should be a dynamic construct that encompasses the process of change (Bishop, 1997; Goswami, 2003; Karmiloff-Smith, 1997). The ground, therefore, is being prepared. Starting from such fertilized ground, Leekam and McGregor expand on these issues in the final chapter; throughout this volume, the argument for integration is developed whenever there is an opportunity to cite, draw together, and elaborate on examples from the other chapters of the book

Toward Achieving Integration

The next question for this chapter is, how might the process of integration of research in autism be achieved? There are two possible ways: first, by applying or overlaying models of autism, or aspects of autism from one area to that of another, and assessing, or at least speculating about, the degree of "fit." In this way, reassessment can be made about the likely accuracy of the model and scope offered for the construction of a more complex representation. Second, cross-area collaboration can offer a direct way of testing the degree of fit, or of filling gaps in an incomplete or simple model. With regard to the first, there has always been some interest in, for example, mapping a cognitive model onto a neurological one, matching behaviors characteristic of autism to specific brain areas (e.g., associating impairments in social cognition with atypical functioning of the frontal cortex (Baron-Cohen & Ring, 1994), and this continues at a more sophisticated level with improving technology (see Wicker, Chapter 2). Ideas have been incorporated from developmental psychology that have helped broaden the range of diagnostic instruments, some of which now incorporate the assessment of joint attention and imitation (e.g., the ADOS-G, Lord et al., 2000). These are natural spill-over effects of a subject area that is researched in different disciplines where useful links will naturally be made from time to time. Nevertheless, they do not constitute integration.

With regard to the second way of achieving integration, cross-area collaboration, a growing number of studies are taking an interdisciplinary approach to answering research questions. For example, genetics and cognition have combined in phenotype studies of cognitive profiling in families with autism (e.g., Nurmi et al., 2003). Advances in neurological technology have opened up new possibilities, as performance on cognitive tasks is highly suitable for combination with fMRI scanning studies. These do indicate a distinct move toward a more collaborative approach to research in autism; but they, too, still fall far short of a truly integrated

approach. In addition, collaboration has rarely involved more than two topic areas. This method of integrating would provide a very limited and presumably rather protracted merging of models in a prolonged process of pairing collaborations.

The Three Areas of This Book's Focus

This section gives an outline and brief history of each of the three areas of the book's title: neurocognitive, clinical/diagnostic, and intervention. The three areas identified have generally followed strongly independent research paths. Historically, autism was first the preserve of medicine, originally located within psychiatry and later extending into pediatrics, neurology, and genetics. Education/intervention has operated in parallel, responding to behavioral difficulties rather than underlying cognitive anomalies, although, gradually, it has acknowledged the diagnostic criteria in its rationale for intervention (see Jones & Jordan, Chapter 14), and cognitive interventions have been explored (see Golan & Baron-Cohen, Chapter 12). Psychology's interest in autism emerged in the 1960s, initially at a low level, then exploding into a dominant topic of research interest from the 1980s. In one sense psychology occupies the space between the medical and the educational, and has the potential to serve as a powerful facilitator in the process of integrating research in these three areas. Each content chapter in this book covers a major topic from at least one of the three main areas of the book title, and all chapters elaborate on one or more aspects of the core information that follows here.

Clinical/diagnostic area

Diagnostic markers

As yet, there is no identifiable biological marker for autism although there are thought to be atypical patterns of development in certain brain areas (Courchesne, Townsend, & Saitoh, 1994; DeLong, 1992; and see Chapter 2); abnormalities of neurotransmitter function (McDougle, 1997; and see Chapter 3); genetic predispositions (Bailey et al., 1995; Curran et al., 2006; Ronald, Happé, Price, Baron-Cohen, & Plomin, 2006); and co-morbid medical conditions such as epilepsy (Danielsson, Gillberg, Billstedt, Gillberg, & Olsson, 2005). Consequently, autism is identified at the level of behavior, so that diagnosis relies on interpretation and professional judgment. Within medical research, there are two main sub-areas: in diagnosis and in epidemiology (Volkmar et al., 2004). These sub-areas are interdependent, in that epidemiological study requires some kind of diagnostic instrument. At the

same time, screening programs have resulted in refinements to instruments as questions were raised about milder or borderline cases.

Diagnosis

Clinicians assess a child by interviewing parents and observing the child's behavior. Current diagnostic criteria are the product of international efforts (Gillberg, 1992; Rutter & Schopler, 1992; Volkmar, et al., 1994 and development of diagnostic tools continues. Williams (Chapter 10) provides more detail on these. Autism was recognized as an official class of disorder in 1980 and is listed in the American Psychiatric Association's *Diagnostic and statistical manual* (1994) and in the World Health Organization's ICD-10 (1993) classification of mental and behavioral disorders (under pervasive developmental disorders). To be given a diagnosis of autism, an individual has to have displayed: (a) a number of proscribed qualitative impairments in social interaction; (b) a number of qualitative impairments in communication; and (c) restricted repetitive and stereotyped patterns of behavior, interests, and activities. Beyond the listed criteria, some children have abnormal eating behavior, disrupted sleeping patterns, or abnormalities of mood or affect. Around 50% have a coexistent general intellectual disability.

Epidemiology

More than 30 epidemiological studies have been conducted (e.g., Fombonne, 2003; Lotter, 1966; Wing & Potter, 2002). Reported prevalence varies substantially (see Chapter 10 for rates and an explanation for variation) but does not appear to vary geographically or ethnically. Genetic influences are considered to have a dominant role in etiology (Bailey et al., 1996). Twin and family studies indicate an autism phenotype, with some family members of normal intelligence showing mild characteristics (Bacchelli et al., 2003; Nurmi et al., 2003; Parr, Lamb, Bailey, & Monaco, 2006). Although autism criteria are listed in psychiatric manuals, it is neither a disease nor a psychiatric condition. It is characterized by disordered or atypical development of the behaviors listed above.

Educational/interventionist area

With the recognition of the condition in the 1940s and 1950s came an accompanying understanding that there was a need for intervention or support. Intervention could take a number of routes. First, it might seek to reduce or remove the behaviors that were considered inappropriate or excessive, such as repetitious behaviors

or echolalia. Second, it might aim to introduce or enhance behaviors that seemed to be missing or underdeveloped, such as social interaction or understanding of affect. Third, it might seek to identify the fundamental cause, and by intervening alter the behavioral consequences (see Williams, Chapter 10; Jones & Jordan, Chapter 14). Although a great diversity of interventions is available for both children and adults with autism spectrum disorder (ASD), a common challenge lies in enabling participants to generalize the skills learnt to other contexts (see Golan & Baron-Cohen, Chapter, 12; Dunlop, Knott, & MacKay, Chapter 13). Volkmar et al. (2004) report that "even with the most ecologically valid treatments, generalization needs to be specifically addressed or it will very rarely happen" (p. 151), an echo of Kanner's observation on the need for elements to be re-experienced in the same setting or sequence with all the original features in the same order.

Neurocognitive research areas

As in medicine, psychology too is exploring autism from a range of perspectives. Its interest was slower and later to emerge but has made up for that in the past two decades. It had focused initially on linguistic and symbolic aspects, helping shift the perception of autism from the psychiatric to the developmental (Hermelin & O'Connor, 1970). From the mid-1980s, research in developmental psychology mushroomed as three dominant cognitive theories were proposed in quick succession, replicated, and refined: the theory of mind (ToM) theory (Baron-Cohen, 1993; Baron-Cohen, Leslie, & Frith, 1985; Leslie, 1987), the weak central coherence deficit theory (Happé & Frith, 1996) and the executive function deficit theory (Ozonoff, Pennington, & Rogers, 1991; Pennington & Ozonoff, 1996).

Theory of mind theory

The theory of mind (ToM) theory proposed that people with autism have a deficit or delay in understanding mental states. This was elaborated to include difficulties in emotion recognition (Boucher, 1996; Davies, Bishop, Manstead, & Tautama, 1994; Mitchell, 1997). Precursors were identified, including joint attention, imitation, the ability to pretend, and the ability to relate oneself to others (Charman, 1997; Hobson, 1993; Jarrold, Smith, Boucher, & Harris, 1994; Libby, Powell, Messer, & Jordan, 1997). Theory of mind abilities are often tested using short experimental tasks in story form which most typical 4-year-olds can pass but the majority of people with autism fail. The minority who pass are likely to fail more complex tasks and/or tests of subtler mentalizing phenomena, such as irony, sarcasm, or double bluff (Happé, 1994), or tests of reading subtle signs of complex emotions in the eyes

(Baron-Cohen, Jolliffe, Mortimore, & Robinson, 1997). Task success is associated with verbal mental age (Yirmiya, Solomonica-Levy, Shulman, & Pilowski, 1996) and chronological age (Steele, Joseph, & Tager-Flusberg, 2003). Even those who pass these formal tasks are still challenged in social functioning in daily life (e.g., Klin, Jones, Schultz, Volkmar, & Cohen, 2002 a & b; see Chapters 4 and 5). Thus theory of mind tasks are a useful indicator, but not a comprehensive measure of social understanding or even the everyday understanding of mental states.

Weak central coherence theory

Chapters 6 and 7 together give a comprehensive account of the literature on weak central coherence (WCC) theory, so it is sufficient here merely to give a definition. The original theory, proposed by Frith (1989), states that people with autism "do not tend to integrate incoming information in its context, but instead, preferentially attend to local information" (López, Chapter 6). Thus, people with autism would demonstrate strength in processing detail but weakness in global processing. Refinements to the original theory make a distinction between processing of perceptual and conceptual information and explore the assumption of dissociation between local and global processing abilities implicit in the original theory. One offshoot of the research in weak central coherence, building on research on understanding mind, face configural processing, has been an important sub-topic (see also Wicker, Chapter 2, and López, Chapter 6).

Executive function theory

Within the cognitive-developmental field, the impact of atypical cognitive profiles in attention, memory, planning, and reasoning has been another area of interest (Shah & Frith, 1993; Russell, Jarrold, & Henry, 1996). Ozonoff et al. (1991) first proposed that the central difficulty in social cognition in autism might be due to more general problems in executive functions, opening the way to a wave of research into executive deficits themselves and their potential impact on the understanding of all areas of impairment in autism. Chapter 8 provides a full account of this aspect of functioning in autism and its links to theory of mind.

Language

From the time of its first description (Kanner, 1943), the unusual use of language has been recognized in autism and language, and communication impairments are a diagnostic feature. There is considerable variety in language use across the spectrum, so the tendency to echolalia and pronoun reversal is commonly observed

in less able people, but for more able people, a pedantic style and atypical prosody are more characteristic. The area of language may have been somewhat neglected by research in recent years (though see Tager-Flusberg, 2000), with a tendency to contemplate language impairments as a sub-product of more central impairment (such as theory of mind). However, as McCann et al. (Chapter 11) show, there are important aspects of language, such as prosody, that continue to deserve attention, and an appropriate consideration of language should be part of an integrated view on autism.

Neuroscience

At the outset, neuroscience was expected to have powerful explanatory power, as there was an expectation of identifiable atypicalities. However, it initially provided an inconsistent picture of differences in size of specific brain areas in autism relative to the typical population. Although anatomical differences were evident, it was not easy to map such information onto what was known from other research areas. The only clear message was that there was no obvious, simple anomaly that would offer an explanation. But advances in brain imaging techniques and, recently, electrophysiological studies are enabling researchers to adopt more sophisticated approaches, such that neuropsychology should, after all, ultimately make a major contribution to explaining autism. fMRI studies are the most predominant (Bock & Goode, 2004; McAlonan et al., 2005; Salmond, De Haan, Friston, Gadian, & Vargha-Khadem, 2003), conducted more commonly with adult and adolescent participants than children. For the study of brain function in young children or those with limited verbal ability the use of electroencephalograms (EEGs) and measurement of event-related brain potentials (ERPs) may be a simpler option methodologically, and perhaps ethically (e.g., Chantal & van Engeland, 2006; Lepisto et al., 2005; McPartland, Dawson, Webb, Panagiotides, & Carver, 2004). Recent studies in brain voluming offer potential clues to developmental puzzles, indicating that the brain in autism may be subject to an increase in neuronal growth or a lack of neuronal "pruning," which would have an impact on the specialization of the developing brain (Courchesne, 2002; Frith, 2004). However, longitudinal studies are needed to confirm or disconfirm this hypothesis (see Wicker, Chapter 2, for a fuller account).

Volkmar et al. (2004) observe that the recent neuroimaging studies, suggesting that broader neural systems rather than discrete brain areas underlie autism, challenge the notion of discrete core deficits proposed in the cognitive theories of the 1980s and 1990s. Instead, they prompt a reconsideration of an earlier model involving social motivation processes that in turn influence orientation preference (see Jones & Klin, Chapter 4).

The Perspective of Those With Autism

Those who have a severe coexistent intellectual disability are not usually aware that they fall into a clinical category, that they are different from the norm. However, able adolescents and adults with autism are aware of this categorization. As autism has gained increasing attention—clinical, educational, and from the media, in recent years—able people with autism have begun to comment on their status and how others see them. They may be very sensitive to the repeated use of terms such as "disorder," "abnormality," or "deficit," conscious of being "objectified" by the research process.

As a counteraction to this tendency, the growing community of able people with a diagnosis of ASD have asserted that they are not deficient, but neurologically atypical. In a reverse of the usual diagnostic process, they label the rest of the population "neurotypicals" or NTs. This nicely reminds researchers and clinicians that, certainly for able people with ASD, the impairment exists only in the context of social functioning in the general population. A further counteraction has come from some members of the research community (e.g., the Autism Research Centre in Cambridge) who prefer the term Autism Spectrum Condition (ASC) to Disorder, indicating difference rather than pure deficit. Some of the chapters of this book may refer to "deficits" or "impairments" or "disorder" as this is the conventional language of clinical writing. However, it should be recognized that the terms are context-dependent and relative to "neurotypical" behavior.

The Origins of This Book

In this chapter we have identified the difficulties in integrating research in autism, but pointed out that there is a general consensus that it is nonetheless the only way to proceed. We have then proposed that there are two methods by which we could proceed: first, through mapping findings from one area with those of another and assessing the degree of fit; second, through active collaboration across research areas to test any elaborated models. The latter option would, in general, rely on first carrying out the former. The starting point, however, would be the creation of opportunities to share perspectives across diverse areas in order to encourage cross-talk, not only between two, but among three or more areas. This book is the product of such an opportunity, created by the inter-university seminar series. The seminars encouraged dialogue across a growing network of members with the goal of promoting a coherent program of future research integrating psychological, clinical, and educational perspectives. Following the presentation of

research perspectives from different areas, the next stage in the integration process was to produce a book that would provide a representative sample of such perspectives with a view to supporting their integration. The chapters collectively offer a balanced representation of topical research, from fMRI studies to naturalistic intervention, reflecting the range of current research output from teams of international repute. Contributors provide contents based upon their own expertise, but shape their chapters for an interdisciplinary audience. Earlier efforts at integration elsewhere have taken the form of pairing across two research areas. We considered that such an approach is too limited. A more elaborate method of exploring the fit of different models would be to map out a network of connections or, indeed, disconnections and implications of findings across a number of research areas. The book makes a starting point in this direction. It goes beyond a series of independent presentations of recent research, by explicitly cross-referencing chapter findings, noting the implications of one for another. Each chapter ends with an integration section in which authors were asked to identify links with other chapters, not only in their own but also in other areas and discuss the theoretical, conceptual, and practical implications. Finally, in a broader debate about approaches to research in autism and the role of development, a concluding chapter draws examples from these chapter sections, considering theoretical links and, in addition, the practical opportunities and potential challenges to integrating research perspectives. Thus, the book follows the first method or stage toward integration identified in this chapter, by mapping findings, theories, and concepts. It is to be hoped that through this process, the ground has been prepared for the second stage—that of active collaboration to test elaborated and integrated models.

Contents

Content chapters are divided into two main sections—a neurocognitive section and a clinical and intervention section.

Part I Neurocognitive research

Part I begins with two chapters covering aspects of the brain basis of behaviors that characterize autism (Chapters 2 and 3). The first of these reviews the literature on the neural basis of social understanding, argues for a connectionist model, and looks at atypical patterns of connectivity among brain areas in autism during social processing. It identifies novel patterns of spared and affected behaviors and

areas. The following chapter presents a theoretical argument for allocentrism as an explanation of the atypical social processing of autism. Evidence from research on neurotransmitter function in autism is presented to support the proposition.

The next two chapters (4 and 5) explore social understanding at the cognitive level. The starting point for both is the "theory of mind" impairment in autism, but they present aspects of processing that are not accounted for by the main theories of the impairment. In Chapter 4, eye-tracking studies identify the relative salience of the mouth and eye regions and of non-social stimuli for people with autism when viewing others engaged in social interaction. Adding a developmental dimension, it takes a microanalytic approach to the topic, using data on physical and social salience from a toddler with autism. Chapter 5 links theory of mind and the theory of Central Coherence to the domain of cultural knowledge, highlighting challenges for individuals with ASD in the flexible processing of social scripts.

Chapters 6 and 7 in this section continue the theme of Central Coherence, proposing theoretical refinements based on the identification of areas of strength and weakness in visuo-spatial abilities with regard to perceptual and conceptual knowledge. The former chapter differentiates also between global and contextual processing in autism, noting the implications for social processing. The final chapter of this grouping reviews executive function in autism, how it relates to autistic symptomatology, adaptive behavior and theory of mind accounts of ASD, and integrates these into a causal modeling framework of the condition.

Part II Clinical and intervention research

Part II begins with three chapters relating to diagnosis (Chapters 9–11). A number of tools have now been established which reliably diagnose classical Kanner's autism in children aged 3–5 years, but much has still to be done to expand the diagnostic boundaries, encompassing younger children and milder cases. The chapters on diagnosis explore less obvious aspects of identification of ASD. The first chapter in this section outlines research that uses home movies to reveal early indicators of autism in the first year of life, the findings bridging social and non-social aspects of development. The second chapter focuses on mild manifestations of autism that may be identified through primary school screening. This chapter provides information on the diagnostic process and range of tools as well as a summary of evaluated intervention studies, offering insights into the current obstacles to implementing screening programs for ASD. The third chapter considers language development in ASD, focusing in particular on receptive and expressive prosody.

The next two chapters (12 and 13) offer contrasting methods of intervention to aid social interaction in autism. The first is based on neurocognitive research find-

ings suggesting emotion-processing difficulties in ASD and explores the use of a multi-media tool to teach emotion recognition. The second aims to address some of the restrictions to generalization of learning in cognitive interventions, using a naturalistic group learning approach. Chapter 14 has a wider remit, examining the influence of theory and research on interventions in ASD. The authors argue for the need to make use of neurocognitive findings in devising interventions. The final content chapter (15) moves beyond the individual with ASD to tackle the broader issue of the impact of autism on family functioning. This chapter also explores the effects on the family when parents implement home-based intervention programs, such as early intensive behavioral intervention.

Conclusion

We have no illusions about the complexity of any process of integrating research perspectives in this highly varied research area. However, there is a consensus from some of the most prestigious research teams in the field that integration is the only way to proceed. What we do offer in this book is a starting point, in which fourteen contributors or author groups have shown a real willingness to think about the theoretical perspectives and practical applications of their work within a much broader research and practitioner context. We hope that it encourages further dialogue and efforts to see the "bigger picture" of autism. The Scottish Autism Research Group continues to build on this initial effort.

References

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: APA.

Bacchelli, E., Blasi, F., Biondolillo, M., Lamb, J. A., Bonora, E., Barnby, G., et al. (2003). Screening of nine candidate genes for autism on chromosome 2q reveals rare nonsynonymous variants in the cAMP-GEFII gene. *Molecular Psychiatry*, *8*(11), 916–924.

Bailey, A., Le Couteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., & Rutter, M. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine*, *25*, 63–78.

Bailey, A., Phillips, W., & Rutter, M. (1996). Autism: Towards an integration of clinical, genetic, neuropsychological and neurobiological perspectives. *Journal of Child Psychology and Psychiatry*, *37*(1), 89–126.

Baron-Cohen, S. (1993). From attention-goal psychology to belief-desire psychology: The development of a theory of mind, and its dysfunction. In S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding other minds: Perspectives from autism* (pp. 59–82). Oxford, UK: Oxford University Press.

Baron-Cohen, S., Jolliffe, T., Mortimore, C., & Robinson, M. (1997). Another advanced test of theory of mind: Evidence from very high functioning adults with autism or Asperger Syndrome. *Journal of Child Psychology and Psychiatry*, *38*(7), 813–822.

Baron-Cohen, S., Leslie, A., & Frith, U. (1985). Does the autistic child have a "theory of mind"? *Cognition*, *21*, 37–46.

Baron-Cohen, S., & Ring, H. (1994). A model of the mindreading system: Neuropsychological and neurobiolocal perspectives. In C. Lewis & P. Mitchell (Eds.), *Children's early understanding of mind* (pp. 183–207). Hove, UK: Lawrence Erlbaum Associates.

Bishop, D. V. M. (1997). Cognitive neuropsychology and developmental disorders: Uncomfortable bedfellows. *Quarterly Journal of Experimental Psychology Section A., 50*, 899–923.

Bock, G., & Goode, J. (Eds.) (2003). *Autism: Neural basis and treatment possibilities*. Novartis Foundation Symposium 251. Chichester, UK: John Wiley. Online publication available at http://www.netlibrary.com/Details.aspx

Boucher, J. (1996). What could possibly explain autism? In P. Carruthers & P. Smith (Eds.), *Theories of theories of mind* (pp. 223–241). Cambridge, UK: Cambridge University Press.

Carruthers, P., & Smith, P. (Eds.) (1996). *Theories of theories of mind.* Cambridge, UK: Cambridge University Press.

Chantal, K., & van Engeland, H. (2006). ERPs and eye movements reflect atypical visual perception in Pervasive Developmental Disorder. *Journal of Autism and Developmental Disorders*, *36*, 45–54.

Charman, T. (1997). The relationship between joint attention and pretend play in autism. *Development and Psychopathology*, *9*(1), 1–16.

Courchesne, E. (2002, August). Abnormal early brain development in autism. *Molecular Psychiatry*, *7* (Suppl. 2), S21–S23.

Courchesne, E., Townsend, J., & Saitoh, O. (1994). The brain in infantile autism: Posterior fossa structures are abnormal. *Neurology*, *44*, 214–223.

Curran, S., Powell, J., Neale, B. M., Dworzynski, K., Li, T., Murphy, D., et al. (2006). An association analysis of candidate genes on chromosome 15 q11–13 and autism spectrum disorder. *Molecular Psychiatry*, *11*(8), 709–713.

Danielsson, S., Gillberg, I. C., Billstedt, E., Gillberg, C., & Olsson, I. (2005). Epilepsy in young adults with autism: A prospective population-based follow-up study of 120 individuals diagnosed in childhood. *Epilepsia*, *46*(6), 918–923.

Davies, S., Bishop, D., Manstead, A. S., & Tantam, D. (1994). Face perception in children with autism and Asperger's syndrome. *Journal of Child Psychology and Psychiatry*, *35*(6), 1033–1057.

Dawson, G., Webb, S., Schellengerg, G. D., Dager, S., Friedman, S., Aylward, E., et al. (2002). Defining the broader phenotype of autism: Genetic, brain and behavioural perspectives. *Development and Psychopathology*, 14, 581–611.

DeLong, F. G. (1992). Autism, amnesia, hippocampus, and learning. *Neuroscience and Biobehavioral Reviews*, *16*, 63–70.

Fombonne, E. (2003). Epidemiological surveys of autism and other pervasive developmental disorders: An update. *Journal of Autism and Developmental Disorders*, *33*, 365–382.

Frith, C. (2004). Is autism a disconnection disorder? *Lancet Neurology*, *3*(10), 577

Frith, U. (1989*). Autism: Explaining the enigma*. Oxford, UK: Basil Blackwell.

Gillberg, C. (1992). The Emmanuel Miller Memorial Lecture 1991. Autism and autistic-like conditions: Sub-classes among disorders of empathy. *Journal of Child Psychology and Psychiatry*, *33*, 813–842.

Goodman, R. (1989). Infantile autism: A syndrome of multiple primary deficits. *Journal of Autism and Developmental Disorders*, *19*, 409–424.

Goswami, U. (2003). Why theories about developmental dyslexia require developmental designs. *Trends in Cognitive Sciences*, *7*(12), 534–540.

Hale, C. M., & Tager-Flusberg, H. (2005). Social communication in children with autism: The relationship between theory of mind and discourse development. *Autism*, *9*(2), 157–178.

Happé, F. (1994). An advanced test of theory of mind: Understanding of story characters' thoughts and feelings by able autistic, mentally handicapped and normal children and adults. *Journal of Autism and Developmental Disorders*, *24*, 129–154.

Happé, F., & Frith, U. (1996). The neuropsychology of autism. *Brain*, *119*, 1377–1400.

Hazlett, H. C., Poe, M., Gerig, G., Smith, R. G., Provenzale, J., Ross, A., et al. (2005). Magnetic resonance imaging and head circumference study of brain size in autism. *Archives of General Psychiatry*, *62*(12), 1366–1376.

Hermelin, B., & O'Connor, N. (1970). *Psychological experiments with autistic children.* Oxford, UK: Pergamon Press.

Hobson, R. P. (1993). Understanding persons: The role of affect. In S. Baron-Cohen, H. Tager-Flusberg, & D. Cohen (Eds.), *Understanding other minds: Perspectives from autism* (pp. 204–227). Oxford, UK: Oxford University Press.

Jarrold, C., Smith, P., Boucher, J., & Harris, P. (1994). Comprehension of pretense in children with autism. *Journal of Autism and Developmental Disorders*, *24*, 433–455.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, *2*, 217–250.

Karmiloff-Smith, A. (1997). Crucial differences between developmental cognitive neuroscience and adult neuropsychology. *Developmental Neuropsychology*, *13*(4), 513–524.

Klin, A., Jones, W., Schultz, R., & Volkmar, F. (2003). The enactive mind, or from actions to cognition: Lessons from autism. *Philosophical Transactions of the Royal Society, London*, *358*, 345–336.

Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002a). Defining and quantifying the social phenotype in autism. *American Journal of Psychiatry*, *159*(6), 895–908.

Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002b). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, *59*(9), 809–816.

Landa, R., & Garrett-Mayer, E. (2006). Development in infants with autism spectrum disorders: A prospective study. *Journal of Child Psychology and Psychiatry*, *47*(6), 629–638.

Lepisto, T., Kujala, T., Vanhala, R., Alku, P., Huotilainen, M., & Naatanen, R. (2005). The discrimination of and orienting to speech and non-speech sounds in children with autism. *Brain Research, 1066*, 147–157.

Leslie, A. (1987). Pretense and representation: The origins of "theory of mind." *Psychological Review*, *94*, 412–426.

Leslie, A. (1991). The theory of mind impairment in autism: Evidence for a modular mechanism of development? In A. Whiten (Ed.), *Natural theories of* mind (pp. 63–78). Oxford, UK, and Cambridge, MA: Basil Blackwell.

Libby, S., Powell, S., Messer, D., & Jordan, R. (1997). Imitation of pretend play acts by children with autism and Down syndrome. *Journal of Autism and Developmental Disorders*, *27*(4), 365–383.

Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Jr., Leventhal, B. L., DiLavore, P. C., et al. (2000). The autism diagnostic observation schedule-generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorder*, *30*, 205–223.

Lord, C., Shulman, C., & DiLavore, P. (2004). Regression and word loss in autistic spectrum disorders. *Journal of Child Psychology and Psychiatry*, *45*(5), 936–955.

Lotter, V. (1966). Epidemiology of autistic conditions in young children. I: Prevalence, *Social Psychiatry*, *1,* 124–137.

McAlonan, M., Cheung, V., Cheung, C., Suckling, J., Lam, G. Y., Tai, K. S., et al. (2005). Mapping the brain in autism. A voxel-based MRI study of volumetric differences and intercorrelations in autism. *Brain*, *1*(28), 268–276.

McDougle, C. J. (1997). Psychopharmacology. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd edn., pp. 707–729). New York: Wiley.

McPartland, J., Dawson, G., Webb, S. J., Panagiotides, H., & Carver, L. J. (2004). Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, *45*, 1235–1245.

Mitchell, P. (1997). *Introduction to theory of mind: Children, autism and apes*. London, UK: Edward Arnold Publishers.

Moore, D. J., & Taylor, J. (2000). Interactive multimedia systems for people with autism, *Journal of Educational Media*, *25*(3), 169–177.

Morton, J., & Frith, U. (1995). Causal modelling: A structural approach to developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Manual of developmental psychopathology* (pp. 357–390). New York: John Wiley.

Munson, J., Dawson, G., Abbott, R., Faja, S., Webb, S. J., Friedman, S. D., et al. (2006). Amygdalar volume and behavioural development in autism. *Archives of General Psychiatry*, *63*(6), 686–693.

Nurmi, E. L., Dowd, M., Tadevosyan, L. O., Haines, J. L., Folstein, S. E., & Sutcliffe, J. S. O. (2003). Explorary subsetting of autism families based on savant skills improves evidence of genetic linkage to 15q11-q13. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*(7), 856–863.

Ozonoff, S., Penington, B., & Rogers, S. (1991). Executive function deficits in high-functioning autistic children: Relationship to theory of mind. *Journal of Child Psychology and Psychiatry*, *32*, 1081–1106.

Parr. J. R., Lamb, J. A., Bailey, A. J., & Monaco, A. P. (2006). Response to paper by Molloy et al.: Linkage on 21q and 7q in autism subset with regression. *Molecular Psychiatry*, *11*(7), 617–619.

Pennington, B., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, *37*(1), 51–87.

Ronald, A., Happé, F., & Plomin, R. (2005). The genetic relationship between individual differences in social and nonsocial behaviours characteristic of autism. *Developmental Science*, *8*(5), 444–458.

Ronald, A., Happé, F., Price, T. S., Baron-Cohen, S., & Plomin, R. (2006). Phenotypic and genetic overlap between autistic traits at the extremes of the general population. *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*(10), 1206–1214.

Russell, J., Jarrold, C., & Henry, L. (1996). Working memory in children with autism and moderate learning difficulties. *Journal of Child Psychology and Psychiatry*, *37*(6), 903–910.

Rutter, M., & Schopler, E. (1992). Classification of pervasive developmental disorders: Some concepts and practical considerations, *Journal of Autism and Developmental Disorders* [Special issue on classification and diagnosis], *22*(4), 459–482.

Salmond, C., De Haan, M., Friston, K. J., Gadian, D. G., & Vargha-Khadem, F. (2003). Investigating individual differences in brain abnormalities in autism. *Philosophical Transactions of the Royal Society*, *B., 358*, 405–413.

Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, *34*, 1351–1364.

Sigman, M., Spence, S. J., & Wang, A. T. (2006). Autism from developmental and neuropsychological perspectives. *Annual Review of Clinical Psychology*, *2*, 327–355.

Steele, S., Joseph, R., & Tager-Flusberg, H. (2003). Brief report: Developmental change in theory of mind abilities in children with autism. *Journal of Autism and Developmental Disorders*, *33*(4), 461–467.

Stirling, L., & Barrington, G. (in press) "Then I'll huff and I'll puff or I'll go on the roff!" thinks the wolf: Spontaneous written narratives by a child with autism. In: A. C. Schalley, & D. Khlentzos (Eds.), *Mental states: Language and cognitive structure.* Amsterdam/ Philadelphia: John Benjamins.

Tager-Flusberg, H. (2000). The challenge of studying language development in children with autism. In L. Menn & N. Bernstein Ratner (Eds.), *Methods for studying language production* (pp. 313–332). Mahwah, NJ: Lawrence Erlbaum.

Volkmar, F., Klin, A., Siegal, B., Szatmari, P., Lord, C., Campbell, M., et al. (1994). Field trial for autistic disorder in DSM-IV. *American Journal of Psychiatry, 151*(9)*,* 1361–1367.

Volkmar, F., Lord, C., Bailey, A., Schultz, R., & Klin, A. (2004). Autism and pervasive developmental disorders. *Journal of Child Psychology and Psychiatry*, *45*(1), 135–170.

Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: Is the prevalence rising? *Mental Retardation and Developmental Disabilities Research Review*, *8*, 151–161.

World Health Organization (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva: World Health Organization.

Yirmiya, N., Solomonica-Levy, D., Shulman, C., & Pilowski, T. (1996). Theory of mind abilities in individuals with autism, Down's syndrome and mental retardation of unknown aetiology: The role of age and intelligence. *Journal of Child Psychology and Psychiatry*, *37*(8), 1003–1014.

Part I

Neurocognitive Research