Adults with ASD

Digby Tantam

Published online: 22 January 2014
© Springer International Publishing AG 2014

Abstract ASD in adults is still under-researched, and under-provided by services. I have chosen what I consider to be the key areas for future research, often because these are the areas of most concern to people with ASD and their carers. They include the impact of diagnosis, still the single most sought after intervention, the association of ASD with other disorders, and whether or not people can get better from ASD (some can, I conclude).

Keywords Autism · Diagnosis · Intervention · DSM5 · Executive dysfunction · Co-morbidity · Asperger-type ASD

Introduction

I have been providing a clinical assessment service for adults with an ASD since 1980, but I believe that this was a unique innovation at the time. Children who had been diagnosed with an ASD normally passed out of the ken of child and adolescent psychiatry into intellectual disability services (even these names are new), often with a strong focus on in-patient beds. Most adults with an ASD who had not been diagnosed as children either fitted in as best they could, or were passed off as eccentric, or diagnosed with schizophrenia. There was an emphasis on humane management, and a focus on challenging behaviour in these settings, and little need, therefore, to consider the specific cognitive impairments that distinguished adults with an ASD from other people with intellectual disability. A consultant colleague of mine taxed me at the time with my interest, saying to me: “Asperger syndrome, isn’t that what pushy middle class parents say their mentally handicapped children have?” The notion that there might be

multiple cognitive impairments, that mental handicap was not a unitary impairment, that improvement and not just deterioration could be expected in many people with an ASD, and that people with an intellectual disability were prone to psychiatric disorders that might worsen their situation—none of this was widely thought about, or even thought to matter. Protective care was the main priority. One of my research students wanted to do a study of the prevalence of psychiatric disorders in people with intellectual disability. It was turned down flat because the chair of the research ethics committee, a local councillor, thought that it was absolutely wrong to increase the burden on poor people with an intellectual disability by suggesting that they might also be mentally ill.

Much has changed since then, but perhaps not all of these attitudes have disappeared. What has most markedly changed is the one size fits all approach. The frontal lobes are no longer the “inert” areas of the brain that they were, unbelievably, thought to be. Neuroimaging coupled with new paradigms in social cognition have begun to tease apart some of the distinct functions that make up the generalization “intellectual disability”.

Diagnosis

Developmental trajectories diverge from infancy to old age and, we must assume, the brain’s connections change accordingly [1]. Intra-uterine factors, early care, peer relations, familial socialization and acculturation have already shaped the infant’s capacity for socialization and thought and possibly speech by the end of infancy. The genetic endowment—the transcriptome, as it is sometimes termed—that the infant brings to their part in these interactions accounts for an apparently large part, judging by estimates of heritability, but the impact of social factors increases with age (although genes may still be turned on or off as a result of environmental influence), and the heritability accordingly diminishes.

D. Tantam (✉)
University of Sheffield, Sheffield, UK
e-mail: digby.tantam@gmail.com

Clinical attempts to find distinct syndromes, i.e., recognizable patterns, have largely failed and this has led to the abandonment of the Asperger sub-type in DSM-5 despite universal recognition that services for more able people with an ASD need to be very differently configured than for those who are less able.

This variation means that the diagnosis of ASD in adulthood can rely less on prototypes or recognizable patterns. Although looking at genotypes rather than phenotypes has been proposed as a way of finding more defined patterns, a possibility opened up by the availability of more and larger chips, patterns have not so far been found. Looking at patterns of connection, the so-called connectomes, is another possibility too, which may be more likely to be successful since connections quite possibly reflect experience as well as genotype. Lacking clearly identifiable patterns, many clinicians fall back on exhaustive symptoms inventories, usually including developmental information, and then apply an algorithm supplied by the authors of the inventory. The clinical validity of this approach has not been tested and, in my opinion, stultifies the clinical, intuitive skill that has been so successful in other areas of medicine.

It is particularly useful in adulthood to be able to identify significant functional impairments since these can be used as a focus for “work arounds” by the person with an ASD, their carers, or their employers. DSM-5 has taken a step forward in this respect, as its two dimensions correspond well to two important functional impairments [2]. The first, impaired social communication, is a less mixed bag than it was in DSM-IV. The second, a repetitive/restrictive behaviours (RRBs) dimension remains quite heterogeneous, but research justification for commonality exists [3] for this group of symptoms that are linked to the commonsense term, rigidity, to the neurological terms, perseveration, to Lhermitte’s utilization behaviour, but probably not to Uta Frith’s concept of weak central coherence [4]. The field trials conducted as part of the DSM-5 development showed good test-retest reliability for ASD, and also for attention deficit hyperactivity disorder in children [5], but no tests were conducted for agreement on these dimensions, and none in adults—a continuing reflection of the bias towards the 16 years of life that people with an ASD will spend in childhood and away from the nearly 60 years that life expectancy in the general population would indicate that they will spend as adults or near adults.

There have been few studies of how clinicians make diagnoses of ASD in adults, with the notable exception of studies by Matson and his co-workers [6], which have concentrated on diagnosis in people with additional significant intellectual disability who probably constitute at most half of the population of people with ASD. My own practice is to use non-verbal communicative ability, whether the ability to express feelings and thoughts non-verbally, or to interpret them in other people, as the most important single criterion for

diagnosis. But the systematic observation of non-verbal communication gets little attention in clinical training, and in Matson et al’s study was one of the least reliable ratings. Training markedly improves both the sensitivity and specificity of three observations of nonverbal communication in diagnosing children with an ASD [7] and in my clinical experience can have a similar effect in improving the diagnostic acuity of psychiatrists assessing adults. Structured observation, using, for example, the Autism Diagnostic Observation Scale, should in theory increase reliability and validity, but at least in adults with intellectual disability and ASD, the ADOS is over-sensitive [8].

It may still be possible to interview the parent of a young adult, and, therefore, use the tried and tested methods of assessing developmental history that are useful in children, but as adults get older, there is greater likelihood of their parents being frail, dead, or alienated. The reliance on developmental information may be one reason that so little is known about older people with an ASD or how to diagnose the condition in older people.

The Impact of DSM5

It is too early to tell what the impact of DSM5 will be, and how closely the next revision of the International Classification of Disease (ICD-11) will mirror it. The last minute addition of “Social communication disorder” (SCD), which is effectively indistinguishable from the first dimension of autistic disorder in DSM5 but without the second, RRBs, dimension, may be the most problematic consequence in the long run, given that the the protests about the disappearance of the Asperger epithet have died down now that people realize that this term can still be used in descriptive fashion. Having a diagnosis of SCD will not, for example, entitle a person or their carers to receive services from organizations for people with autism, or benefit from the Autism Act 2009 in the UK, which mandates particular services for people with autism. The problem is exacerbated by the differential developmental trajectory of the two dimensions of ASD: in some people the RRBs may dwindle as they go through adolescence, perhaps in response to being exposed to changes that they have to accommodate. Should these people, who may have been eligible to receive a diagnosis of ASD as children, be “disenfranchised”, as some people might call it, as adults because their RRBs are less obtrusive but their fundamental difficulty, the social communication disorder, has persisted?

Psychological Research and Diagnosis

Psychological research in ASD is often driven by the intrinsic interest of the field, and its relevance to human nature rather

than by clinical priorities. There may be little correlation between deficits defined by researchers and the symptoms that are important to people with ASD, their families, and their professional carers [9]. The measures that psychologists use to characterize ASD-related impairment, for example, measures of theory of mind, of executive function, or of empathy, are rarely applied in clinical practice, but executive function and empathy are obviously relevant to adult functioning and more use could be made of the research tools developed to study them.

Executive Dysfunction

Executive dysfunction is itself a heterogeneous dimension. Although there are conventions about how it might be estimated, and studies often present a score of executive function, there is considerable variation in the results when different batteries are applied in practice. This may be one reason that clinicians do not formally assess executive function. Yet, the often mysterious reasons that able and intelligent adults with an ASD are unable to live independently can often be traced back to failures to plan, to persist with frustrating tasks, and to be able to switch from one task to another without losing one's place, i.e., to executive dysfunction. One recent computer-based method of ascertaining executive function did not show an impairment in adults with Asperger-type ASD [10] but another battery did [11]. The difference may be due to different competences being tested, but it might also be explicable if, as my clinical experience suggests, executive function impairment is most marked in people with both ASD and ADHD. Nyden et al compared adults with ASD, adults with ADHD, and adults with both, and found that the latter were more distractible than adults with ASD alone. But the groups were not well-matched for IQ, and there were few differences in executive function between any of them, and fewer differences from population norms than might be expected. Interestingly, there were no differences between the groups of theory of mind tasks either [12]. Gargaro et al [13] concluded from a literature review that there were theoretical reasons for expecting executive difficulties to be worse in the combined ASD/ ADHD group, but the results of empirical studies were unclear.

Empathy

The explosion of interest in empathy in the field, and to some degree in society more generally, can be dated back to Uta Frith's decision to encourage a promising young PhD student to apply to ASD a primate paradigm developed by Premack and Woodruff for chimpanzee research [14]. The result has been a burgeoning theory of mind (ToM) theory that has

annexed the concept of empathy put forward by Lipps at the end of the nineteenth century. It is clear that there are four distinct ways that people use the term "empathy", both generally, and specifically in neurodevelopmental research: for contagious emotion, gaze interaction (also linked to shared attention), narrative accounts of motivations and emotions, and compassion or lack of it. Each of these may be impaired in adults. The first two are most likely interlinked since reading facial expression requires gazing at another person's eye area and nasolabial fold, but the others are distinct. Unfortunately almost all of the psychological tests that have been used to test these different elements of empathy do not correspond to useful distinctions in clinical practice, and almost all of them use self-report in the assessment. Self-report introduces the ability to name emotions as a confounding variable, and this may be just as much of a difficulty for a socially inexperienced adult with ASD, as it would be for a child.

One particular unfortunate result has been that a lack of compassion has become confounded with a lack of empathy. Cleckley proposed many years ago that the German term for "personality disorder", *psychopathie* (incidentally, Asperger's own term for Asperger disorder was "*autistische Psychopathie*") corresponded to a valid clinical entity. Although there is no formal diagnosis of "psychopathy" in DSM5, it has been used by politicians, judges, and psychologists as if it is a defined syndrome, characterized by a lack of empathy.

"Psychopathy" is not the only diagnosis that has added confusion to the evaluation of empathy. Gillberg, in an influential lecture [15], suggested that there was a class of empathy disorders that included anorexia nervosa as well as ASD. Abstaining and underweight people with anorexia have been found to have ToM impairments but these disappear when weight returns to normal [16]. This is one of many findings that points towards social and emotional narrative being important in many disorders, and that this is, at least in human primates, what underlies so-called theory of mind deficits in ASD and in other conditions [17].

Adults with ASD show impaired emotion contagion in naturalistic circumstances [18•] and impaired gaze interaction [19], but narrative competence and, therefore, theory of mind performance in adults is variable [20•]. Theory of mind—more often now called "mentalizing" as it becomes a vaguer, popularized epithet—is linked to other functions associated with speech processing, such as working memory, and it is not surprising therefore that ToM impairment has been found in ADHD, itself associated with working memory disorder.

A consequence of impaired theory of mind may be a reduced ability to create and present an identity to the outside world. This may be evident in reduced autobiographical ability [21].

A lack of empathy is thought by many people to be a cause of dangerousness, although it could equally be argued that

dangerous people suppress their empathy in order to maintain a level of dangerousness required for survival, profit, or social acceptance. Little is known about either emotional contagion or theory of mind in ordinary life, but what there is suggests that it is of less importance than is assumed [22]. Although, in clinical practice, empathy deficits do seem to be associated with vulnerability and, therefore, a risk of the unempathic person being abused. One as yet not understood observation is that people with an ASD can undertake most empathy tasks with prompting, but do not do so spontaneously [23••].

Self-Report Methods of Assessment and the Impact of the Developmental Trajectory

Diagnosis of mental health conditions in adulthood relies on self-report, and as adults generally have more developed narrative abilities than children, this is a good strategy. The recent UK NICE guidelines on diagnosis in adults placed heavy emphasis on the use of one self-report scale, the Autism Quotient. A recent epidemiological study did not support the value of the AQ in adults even in screening, and this should not be surprising. The core symptoms of ASD are not readily accessible to self-inspection. How many of us know, for example, how discriminating we are in our social judgements? The AQ and other self-report scales, therefore, rely on symptoms that are common consequences of ASD, such as attitudes and preferences. Emotional maturity does apparently develop less in people with ASD [24] and this probably contributes to the difficulties that adults with ASD have in their social relations. But is this slower development a core symptom of ASD, or is it a consequence of social isolation and, therefore, lack of practice in dealing with emotions? Similar questions apply to other conditions suspected recently of being a component of ASD, for example, “theory of mind” [20••] or “alexithymia” [25] both of which may simply be a consequence of a lack of practice, particularly a lack of engagement in conversations about emotions and motivation [26]. This is not to say that these are not useful guides to the consequences for many adults of having an ASD [27] which may give clues about how people with an ASD can tackle some of their difficulties [28]. Nor that they may not be used as alerts, only that they should not be used as diagnostic criteria in self-report scales. Piling on the blankets and shivering might appropriately prompt further investigation of a possible infection, but does not usefully discriminate a cold from pneumonia, and its absence does not rule out pneumonia, either.

Neurological Co-morbidity

The unwillingness of doctors to take account of neurodevelopmental disorders is strange given our willingness

to embrace diagnoses [29] that have been taken over by politicians and have been found to have unsatisfactory reliability in the field [5]. Attention deficit disorder in children remains a disputed diagnosis, and its impact on adult life is as yet ignored by many mental health services even though it may persist throughout adulthood [30]. ADHD is the neurodevelopmental disorder most often associated with ASD, and its presence may alter the presentation, for example, by increasing impulsivity [31]. Other associated neurodevelopmental disorders include epilepsy, developmental coordination disorder, Tourette’s syndrome, topographical agnosia, prosopagnosia, synaesthesia, dyslexia, writing apraxia, and a variety of ill-defined speech and language disorders. These may be of little significance in adults, or of great significance depending on the person and their situation. They are often unrecognized or misidentified [32].

Psychological Co-morbidity

Few adults reach maturity without accumulating some degree of stress, and without having acquired some degree of resilience. Adolescence may be a particularly important stage for both, not only because of the rapid social changes with which it is associated, but because of not well understood neurological changes [33]. Some of these are associated with an increased risk of psychosis. Anxiety and depression originate earlier in childhood, possibly from the age of 7 years in the case of anxiety, when the presentation is largely psychosomatic or obsessional, but become more prevalent in adolescence. Social phobia typically has its onset in adolescence and is so common in adults with ASD that the differential diagnosis may create clinical difficulties. The presence of a nonverbal impairment is the most reliable method of distinguishing ASD from social phobia [34].

There have been a number of studies of the prevalence of psychiatric disorders in adults with ASD, but most of them are either clinic samples or samples restricted to people with ASD and intellectual disability (see Tantam, 2012 for a review [35]). A recent study [36] using a community sample, a registry, relied on parental diagnoses but probably gives a more unbiased picture. Although it was of children, it showed an increasing risk of anxiety from age 5 years to age 18 years; that anxiety affected nearly half: the same estimate I reached (ref), and that anxiety affected the more-able group much more than the less-able group.

Diagnostic uncertainty about the distinction between generalized anxiety and depression, which remains a problem in DSM5, makes the association with depression unclear. It is my own clinical impression that non-psychotic depression is no more common in people with an ASD. I would though agree with Rosenberg et al’s (2011) findings that there is an increased risk of bipolar disorder, and, therefore, psychotic

depression, in ASD. Anxiety may be disabling and also worsens performance in those areas, such as social skills, that are impaired by the ASD itself. Perhaps for this reason, or perhaps because of an older hierarchical model of mental health diagnosis in which one disorder “trumps” another, anxiety disorder may be less often diagnosed in people with an ASD than it would be in the general population.

Emotional disorders respond best to counselling in the general population, and this is true also for people with an ASD. Evaluation of which psychotherapy or counselling interventions are most effective, and how conventional interventions might need to be adapted, has only just started. Assumptions may be misleading. Group counselling, which might seem to be contra-indicated in adults with an ASD, may be as effective as individual counselling for ASD [37].

Bullying

Mouse models suggest that there may be genetic links between anxiety proneness and ASD [38], although no genetic links are likely to extend across the whole population of people with an ASD, since this is genetically polymorphous. Parents are more likely to identify bullying as a cause of anxiety and low self-confidence in children with an ASD [39] and bullying continues into adulthood, as do its effects. Adults with ASD avoid situations where they might have to interact with others, and these are often situations where they have been bullied in the past [40]. Bullying in ASD has been underestimated until recently, but in one study we found that young adolescents with ASD were seven times more likely to be bullied than their neurotypical peers [41]. Bullying, or rather the social anxiety that it causes, may increase hostility towards other people in adulthood, and, therefore, increase the risk of aggression [42].

Sensitivity to bullying, associated anxiety related symptoms of ASD, like sensory hypersensitivity [43] and rigidity, may all affect employability, and require a special kind of employer [44] or support in employment [45]. In the longer term, a corrective experience of fitting in at college or in the work place may reverse the damaging effects of school bullying.

Prevalence

One recent epidemiological study concluded that the prevalence of ASD does not change through the life span [46] but this was not the conclusion of another more, fine-grained, regional study [47] in which the apparent prevalence of ASD fell from early adolescence into adulthood. The presence of a small group of improvers has also been identified in a study of children going into adolescence [48]. It has for a long

time been accepted that some children “grow out of” ADHD—perhaps, indeed, accepted too uncritically as many do not. Why then should children with an ASD not sometimes “grow out of” ASD? There is certainly evidence of ongoing change in the brain that would support this [49]. This is not to say that everyone will grow out of ASD, or that therapeutic passivity should be the rule. If anything, it indicates to me that therapeutic activity is indicated since the brain is responsive to social influence, and that therapy can be used to influence the final outcome of maturational development.

There are other reasons for a reduction in prevalence. One of the most likely is that some of the symptoms of ASD are the result of a mis-match between the social competencies of the person with ASD and the social demands of the environment. There are likely to be cases where the social demands, not the competencies, evolve, leading to a reduction of symptoms, perhaps below the level at which a person evaluates themselves to have a disorder. There may also be increased mortality. What happens to people with an ASD as they age is not known [50]. Less is known about those who are out of contact with family, friends, and services but they might be most at risk of neglect or inanition.

The excess of men over women persists in adults, but how much does this reflect the greater ability, or willingness, of women to try to fit in, and the greater willingness of men to adopt and defend eccentricity? We will only know, and will only be able to provide accurate prevalence rates for ASD once the fundamental, age- and gender-invariant, determinants of the syndrome are known.

Conclusions

ASD in children is being increasingly well characterized, and research funding has increased substantially. This research has fertilized the previously somewhat arid fields of social psychology, and has given an impetus to the new field of social neuroscience. ASD in adulthood has been under-funded and rarely studied in its own right. The situation is changing gradually, but needs to change more quickly. We need to know, for example, what happens to unsupported people with an ASD as they age. ASD in adults may be as rich a source of models of normal development as ASD in childhood has been [51]. How, for example, does early experience influence the final shape of the brain? Is there indeed a final shape or is the connectivity of the brain so important in its function, that apparent structural stability of the brain does not represent a final stage in functional capacity, at all. Is the success of people with ASD in working with machines and in IT an indication that theirs is the brain organization of the future? How much does face to face interaction matter, if one is closely connected via social media? So many unanswered questions: if there is one final conclusion of this article, it is

that we have just started to see the questions, and have quite some way to go before we find the answers.

Compliance with Ethics Guidelines

Conflict of Interest Digby Tantam declares that he has no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Tehrani JJ. The Phylogeny of Little Red Riding Hood. *PLoS ONE*. 2013;8(11):e78871.
2. Shuster J, et al. Review of factor analytic studies examining symptoms of autism spectrum disorders. *J Autism Dev Disord*. 2014;44:90–110.
3. Happe F, Ronald A. The 'Fractionable autism triad': a review of evidence from behavioural, genetic, cognitive and neural research. [Article]. *Neuropsychol Rev*. 2008;18(4):287–304.
4. South M, Ozonoff S, McMahon WM. The relationship between executive functioning, central coherence, and repetitive behaviors in the high-functioning autism spectrum. *Autism*. 2007;11(5):437–51.
5. Regier DA et al. DSM-5 field trials in the United States and Canada, Part II: test-retest reliability of selected categorical diagnoses. *Am J Psychiatr*. 2013;170(1):59–70.
6. Matson J, Wilkins J, González M. Reliability and factor structure of the autism spectrum disorders—diagnosis scale for intellectually disabled adults (ASD—DA). *J Dev Phys Disabil*. 2007;19(6):565–77.
7. Oner P, Oner O, Munir K. Three-item Direct Observation Screen (TIDOS) for autism spectrum disorder. *Autism*. 2013. doi:10.1177/1362361313407028
8. Sappok T et al. Diagnosing autism in a clinical sample of adults with intellectual disabilities: how useful are the ADOS and the ADI-R? *Res Dev Disabil*. 2013;34(5):1642–55.
9. Brunsdon VEA, Happé F. Exploring the 'fractionation' of autism at the cognitive level. *Autism*. 2014;18:17–30.
10. Sachse M et al. Executive and Visuo-motor function in adolescents and adults with autism spectrum disorder. *J Autism Dev Disord*. 2013;43(5):1222–35.
11. Brady DI et al. Conceptual and perceptual set-shifting executive abilities in young adults with Asperger's syndrome. *Res Autism Spectr Disord*. 2013;7(12):1631–7.
12. Nydén A et al. Adults with autism spectrum disorders and ADHD neuropsychological aspects. *Res Dev Disabil*. 2010;31(6):1659–68.
13. Gargaro BA et al. Autism and ADHD: how far have we come in the comorbidity debate? *Neurosci Biobehav Rev*. 2011;35(5):1081–8.
14. Premack D, Woodruff G. Does the chimpanzee have a theory of mind? *Behav Brain Sci*. 1978;1:515–26.
15. Gillberg CL. The Emanuel Miller Memorial Lecture 1991. Autism and autistic-like conditions: subclasses among disorders of empathy. *J Child Psychol Psychiatry*. 1992;33:813–842.
16. Oldershaw A et al. Emotional theory of mind and emotional awareness in recovered anorexia nervosa patients. *Psychosom Med*. 2010;72(1):73–9.
17. Mason RA et al. Theory of Mind disruption and recruitment of the right hemisphere during narrative comprehension in autism. *Neuropsychologia*. 2008;46(1):269–80.
18. Sato W et al. Impaired social brain network for processing dynamic facial expressions in autism spectrum disorders. *BMC Neurosci*. 2012;13:99. *For some time, it has been speculated that dynamic faces are a more life-like test than static images, and this study used both kinds of stimuli although the dynamic faces were synthesized. There may have been some differences in matching on nonverbal IQ, common in studies of adults, although the difference was not statistically significant, but it is at least a study of adults. The equipment used is an example of the increasingly sophisticated tests that can be given to people actually in an MRI scanner. The study confirms the differences in face processing that have been a constant source of debate and repeated studies but helps to explain why these studies have been inconclusive by showing evidence of alternative routes for face processing in people with Asperger-type ASD. These and other studies are moving the field forward by showing that ability is constantly evolving and not a fixed capacity, in line with the likelihood that some people overcome childhood ASD. It also provides further evidence for dysconnectivity in ASD.*
19. Tantam D, Holmes D, Cordess C. Nonverbal expression in autism of Asperger type. *J Autism Dev Disord*. 1993;23(1):111–33.
20. Scheeren AM et al. Rethinking theory of mind in high-functioning autism spectrum disorder. *J Child Psychol Psychiatry*. 2013;54(6):628–35. *This study is one of several that indicate that experimental testing may not give a good picture of naturalistic performance in people with an ASD. In this study, it is theory of mind that is being considered, but the same finding applies to other cognitive skills. Unfortunately the authors do not directly test the naturalistic function of the children, adolescents, and young adults in their study who passed the advanced theory of mind tests that they gave them, but they conclude from a selective literature review that narrative ability linked to theory of mind is restricted in people with Asperger type ASD in real life and that it is necessary to go beyond a purely cognitive model of ToM to understand why the skills is not more successfully applied. It is therefore a counterpart of the neuroimaging study of Senju et al.*
21. Crane L, Goddard L, Pring L. Autobiographical memory in adults with autism spectrum disorder: the role of depressed mood, rumination, working memory and theory of mind. *Autism*. 2013;17(2):205–19.
22. Bryant L et al. Theory of mind experience sampling in typical adults. *Conscious Cogn*. 2013;22(3):697–707.
23. Senju A. Atypical development of spontaneous social cognition in autism spectrum disorders. *Brain Dev*. 2013;35(2):96–101. *A review paper that provides a good starting point for future research into the area discussed by Scheeren et al: that some capacities for social interaction may be intact when accessed by the left brain and deliberate intention, but are not spontaneously evoked in social interactions. The fundamental deficit in ASD may be in what causes this triggering. My own hypothesis is that this is the 'interbrain' and I discuss this in two recent books [49, 50].*
24. Sappok T et al. Emotional development in adults with Autism and intellectual disabilities: a retrospective, clinical analysis. *PLoS ONE*. 2013;8(9):e74036.
25. Heaton P, et al. Measuring the effects of alexithymia on perception of emotional vocalizations in autistic spectrum disorder and typical development. *Psychol Med*. 2012;4:1–7.
26. Allen R, Davis R, Hill E. The effects of autism and alexithymia on physiological and verbal responsiveness to music. *J Autism Dev Disord*. 2013;43(2):432–44.

27. Cook R et al. Alexithymia, not autism, predicts poor recognition of emotional facial expressions. *Psychol Sci*. 2013;24(5):723–32.
28. Bird G, Press C, Richardson DC. The role of alexithymia in reduced eye-fixation in Autism Spectrum Conditions. *J Autism Dev Disord*. 2011;41(11):1556–64.
29. Gask L, Evans M, Kessler D. Personality disorder. *BMJ* 2013;347.
30. Michielsen M et al. Prevalence of attention-deficit hyperactivity disorder in older adults in The Netherlands. *Br J Psychiatry*. 2012;201(4):298–305.
31. Buhler E, et al. Differential diagnosis of Autism Spectrum Disorder and attention deficit hyperactivity disorder by means of inhibitory control and 'Theory of Mind'. *J Autism Dev Disord*. 2011;41:1718–26.
32. Hoglund Carlsson L et al. Coexisting disorders and problems in preschool children with autism spectrum disorders. *Sci World J*. 2013;213979.
33. Blakemore S-J, Mills KL. Is adolescence a sensitive period for sociocultural processing? *Annu Rev Psychol*. 2013. In press.
34. Tyson K, Cruess D. Differentiating high-functioning autism and social phobia. *J Autism Dev Disord*. 2012;42(7):1477–90.
35. Tantam D. Autism spectrum disorders through the life span. London: Jessica Kingsley; 2012.
36. Rosenberg RE, et al. Parent report of community psychiatric comorbid diagnoses in autism spectrum disorders. *Autism Res Treat*. 2011;405–408.
37. Hesselmark E, Plenty S, Bejerot S. Group cognitive behavioural therapy and group recreational activity for adults with autism spectrum disorders: a preliminary randomized controlled trial. *Autism*. 2013. doi:10.1177/1362361313493681.
38. Pobbe RL et al. General and social anxiety in the BTBR T+ tf/J mouse strain. *Behav Brain Res*. 2011;216(1):446–51.
39. McPheeters ML et al. Family report of ASD concomitant with depression or anxiety among US children. *J Autism Dev Disord*. 2011;41(5):646–53.
40. Balfe M, Tantam D. A descriptive social and health profile of a community sample of adults and adolescents with Asperger syndrome. *BMC Res Notes*. 2010;3(1):300.
41. Naylor P et al. Prevalence of bullying secondary schools by SEN type. ESRC impact report, Economic and Social Research Council, Swindon, 2012.
42. White SW et al. Social anxiety mediates the effect of autism spectrum disorder characteristics on hostility in young adults. *Autism*. 2012;16(5):453–64.
43. Tavassoli T, et al. Sensory over-responsivity in adults with autism spectrum conditions. *Autism*. 2013 doi:10.1177/1362361313477246.
44. Parr AD, Hunter ST. Enhancing work outcomes of employees with autism spectrum disorder through leadership: Leadership for employees with autism spectrum disorder. *Autism*. 2013. doi:10.1177/1362361313483020.
45. Mavranzeouli I, et al. The cost-effectiveness of supported employment for adults with autism in the United Kingdom. *Autism*. 2013.
46. Brugha TS et al. Epidemiology of autism spectrum disorders in adults in the community in England. *Arch Gen Psychiatry*. 2011;68(5):459–65.
47. Balfe M, Tantam D, Campbell M. Possible evidence for a fall in the prevalence of high-functioning pervasive developmental disorder with age? *Autism Res Treat*. 2011;2011:325495.
48. Gotham K, Pickles A, Lord C. Trajectories of autism severity in children using standardized ADOS scores. *Pediatrics*. 2012;130(5):e1278–84.
49. Dickstein DP, et al. Developmental meta-analysis of the functional neural correlates of autism spectrum disorders. *J Am Acad Child Adolesc Psychiatry*. 2013;52(3): 279–289 e16.
50. Happe F, Charlton RA. Aging in autism spectrum disorders: a mini-review. *Gerontology*. 2012;58(1):70–8.
51. Tantam D. Can the world afford autistic spectrum disorder? non-verbal communication, Asperger Syndrome and the Interbrain. London: Jessica Kingsley; 2009.