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ORIGINAL PAPER

Progress in Understanding Autism: 2007–2010

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Abstract Scientific progress is discussed in relation to clinical issues; genetic issues; environmental issues; and the state of play on psychological treatments. It is concluded that substantial gains in knowledge have been achieved during the last 3 years, and there have been some unexpected findings, but major puzzles remain. We should be hopeful of ever greater gains in the years ahead, but both prevention and cure remain elusive.

Keywords Scientific advances · Research challenges

In this article, scientific progress will be discussed in relation to advances in our understanding of clinical features, advances in genetics, progress in environmental research, and the state of play on psychological treatments. Basic science, including animal models, is not reviewed here, although the findings on mirror neurons and immunology are obviously potentially very important. The emphasis is placed particularly on advances during the last 3 years, but attention is paid to earlier findings where they are relevant to the contemporary issues.

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Clinical Features

Given that there has been a huge investment in clinical research that goes back for well over half a century, it might be supposed that all that needed to be known is already well established and free of controversies. However, that is far from the case.

Developmental Regression

Despite the fact that the phenomenon of temporary developmental regression (especially of language and language-related skills) is noted from the very first reports of autism, until recently there had been surprisingly little systematic research into the phenomenon. That situation is now changing. At first, some people were sceptical of the reality of regression but carefully conducted studies of home videos (Werner and Dawson 2005) confirmed the validity of the phenomenon. The next question that needed tackling was whether such regression occurred in all neurodevelopmental disorders or whether it was in some way particularly characteristic of autism. Findings from studies by Baird et al. (2008a) and Pickles et al. (2009) showed that the period of regression was distinctly rare in other neurodevelopmental disorders, but seemed to be quite strongly associated with autism. Findings also suggested that it was misleading to think of regression as a categorical present/absent phenomenon; rather, even minor degrees of regression were pointers towards autism. Parr et al. (in press), using data from affected sibling pairs, found that the concordance rate of 18.9% was not significantly above that of 13.5% expected under independence. The overall rate of regression found (24%), was closely comparable to that reported in singleton and epidemiological samples. Several questions derive out of these findings. First, if even

minor degrees of regression are of diagnostic importance, what criteria should be used to identify it? Second, what neural processes underlie the occurrence of regression? The findings suggest that it is unlikely that regression is due to some factors that are exogenous to autism but, nevertheless, what neural processes are giving rise to the regression?

Savant Skills

The situation with respect to savant skills is somewhat comparable to that already noted for regression. That is, such skills have been observed from the very outset (Asperger 1944; Frith 1989; Kanner 1971; Treffert 2010). Our understanding of savant skills was greatly increased by the pioneering studies undertaken by O'Connor and Hermelin (1988; Hermelin 2001). Through innovative experimental designs and use of appropriate comparison groups they showed that savant skills represented real abilities and not simply tricks. It has usually been supposed that savant skills are quite uncommon in autism, but Howlin et al.'s systematic study (2010) showed that about a third of individuals with autism had either a savant skill based on parental report or an exceptional cognitive skill. Strikingly, however, no individual with a non-verbal IQ below 50 met the criteria for a savant skill and it is evident that the traditional general term of "Idiot Savant" is misleading and should be abandoned. Savant skills may sometimes occur in individuals with a very low non verbal IQ but this is not the usual situation. Numerous studies have shown that the range of skills is very large, spanning splinter skills at one end, prodigious savants at the other end, and talented savants in the middle. Systematic comparative studies between autism and other disorders have yet to be undertaken, but it would appear that savant skills are particularly commonly associated with autism. Psychological studies have suggested that a detail-focused cognitive style may predispose to talent in savant domains; others have argued that the excellent attention to detail has its origin in sensory hypersensitivity. The term "talent" would seem to imply an in-built capacity but it is also apparent from a range of different research designs that intensive prolonged practice is also involved. It is striking that autism is associated with both intellectual disability and superior talents, and the question is—what sort of neural functioning could account for both?

Epilepsy and New Psychiatric Disorders

It has long been recognized that about a quarter of individuals with autism develop epilepsy (Volkmar and Nelson 1990). However, Rutter's (1970) early follow-up study was striking in showing that in many cases epileptic attacks did not begin until adolescence. The recent larger-scale follow-up into adult life undertaken by Bolton et al. (in press) had findings that were important in several different respects. First, the rate of epilepsy (22%) in individuals with autism was substantially higher than the general population rate of 0.63% at the same age. Second, the proportion of autistic individuals with epilepsy who developed seizures for the first time after the age of ten (58%) was significantly higher than that in either a national general population study or that in a Scottish cohort of children with idiopathic mental retardation (Goulden et al. 1991). Epilepsy was significantly more common in individuals with either very limited language or a low non-verbal IQ but epilepsy occurred in autistic individuals at all levels of intelligence. Epilepsy was not related to the severity of the autism; nor was it associated with a family history of epilepsy. On the other hand, epilepsy was associated with the likelihood of a relative having the broader autism phenotype—suggesting that the epilepsy was associated with an overall familial liability to autism. It was not associated with regression and it was also not associated with the development of new medical conditions. The unusual late onset of epilepsy must have some neuropathological meaning, but what that might be remains obscure.

A separate study based on the same sample (Hutton et al. 2008) showed that about a fifth of autistic individuals developed a new psychiatric disorder by adult life. This was unassociated with the presence of epilepsy or with the timing of the onset of epileptic attacks. The most common disorders were affective in type but the presence of obsessive–compulsive behavior and of catatonia (which seemed usually to stem from obsessive–compulsive symptoms) seemed to be particularly characteristic of individuals with autism. Although individuals with autism may sometimes develop new disorders that appear to be closely related to their autism, many new disorders seem to arise relatively independently, albeit possibly precipitated by major life changes.

Increased Brain Size

In his first paper describing autism, Kanner (1971) noted that four out of the eleven individuals studied had an unusually large head. Little notice was taken of head size for many years. However, during the 1990s, there were several reports on different studies noting that increased head size was common in individuals with autism (Woodhouse et al. 1996). At about the same time, the first structural brain imaging studies similarly showed an increased brain size (Piven et al. 1995). Since then, numerous imaging studies have shown increased brain size in a substantial minority of individuals with autism (Palmen and van Engeland 2004). The findings mainly

suggest a global increased brain growth. The findings on the pattern of brain growth are inconclusive and it is not known whether it reflects an excess of neurons, and/or reduced synaptic pruning (Keller and Persico 2003). The most important recent finding is that the brain size is normal at birth but increases markedly during the early years—a time period that parallels that of the first obvious manifestations of autism (Courchesne et al. 2003, 2007; Redcay and Courchesne 2005). On the whole, the evidence suggests that increasing brain growth plateaus during middle childhood, but there are some reports that, possibly to a lesser degree, brain size can remain enlarged during adolescence and adult life. The major importance of the early increase in brain growth finding is that it indicates some kind of neural process that only comes on line in the toddler age period, even though the genetic liability has presumably been present from before birth. Quite what this process comprises remains unknown but, although adequate comparative studies have not yet been undertaken, it does appear that this increase may be peculiar to autism. The challenge that remains is to determine what that neural process might be.

Dimension or a Diagnostic Category

Throughout the whole of medicine, including the field of mental disorders, it has become evident that most conditions have a dimensional liability (Rutter 2003). The concept of a broader phenotype of autism (see below) implies that dimensional approaches are relevant for autism, as well as with most other multifactorial conditions. However, in recent years, there has been the additional claim that autism may not constitute a cohesive syndrome. Rather, the individual components of autism may not only be more separate than usually appreciated, but also they may reflect different genetic influences (Happé and Ronald 2008; Ronald et al. 2005). This suggestion is a reasonable one but the evidence so far is contradictory and inconclusive. What is needed in order to test the proposition properly, is a general population study in which the different components of autism are individually, adequately and independently measured (in a way that does not necessitate the diagnostic concepts), in order to answer the question as to what extent the three main domains of impairment co-occur. In tackling this question, it would be important to recognize that it cannot be assumed that there are three such domains. For example, much evidence suggests that the distinction between social reciprocity and social communication is artificial and that these two domains would be better combined (see Gotham et al. 2007). On the other hand, there is more uncertainty as to quite how to deal with the abnormal language features such as stereotyped utterances, verbal rituals, inappropriate questions, neologisms, and pronominal reversal. The factor analysis of the social communication questionnaire suggested that these needed to be dealt with as a separate domain (Rutter et al. 2003; Berument et al. 1999). Somewhat similar questions have been posed with respect to restricted repetitive behaviors. For example, Lam et al. (2008) suggested that the evidence indicated that circumscribed interests needed to be differentiated from repetitive motor behaviors. What all of this means is that an open mind must be maintained on the cohesiveness, or otherwise, of the various features of autism spectrum disorders.

Broader Phenotype

Folstein and Rutter's twin study provided the first clear-cut evidence that the genetic liability for autism extended beyond the traditional diagnosis (Folstein and Rutter 1977; Le Couteur et al. 1996). Since then, many studies confirmed the extension of the traditional diagnosis to a broader phenotype, using data from family studies as well as twin studies (Bailey et al. 1998; Bailey and Parr 2003). In recent years there have been various attempts to develop measures of this broad autism phenotype. Thus, Losh et al. (2008) used a mixture of measures to assess broader phenotype features. The findings showed that the features were significantly more common in multiple incidence autism families than in single incidence autism families and both of these had higher rates than Down syndrome families. The findings were surprising in that three-quarters of the individuals in multiplex families showed at least one such feature, half of those in single incidence families also showed the same, but even in the Down syndrome families the rate was 22%. If the assumption is that the Down syndrome families involved no predisposition to show the broader phenotype, the implication is that the false positive rate in the general population would be very high. Dawson et al. (2007) developed a new instrument that combined interview and observational measures and for which systematic training of the professionals using this measure was provided. The findings showed reasonable inter-rater reliability and internal consistency but only moderate correlations between the observational and interview measures. There was no measure of test–retest reliability to assess the temporal stability of the measures.

It may be concluded that some limited progress has been made in the measurement of the broader phenotype using informant report, self-report and observation but there is no agreed set of measures as yet. The available evidence suggests that broader phenotype differs from traditional autism in that it is not associated with either intellectual disability or with epilepsy. The existence of the broader phenotype raises the query of how it becomes transformed into 'autism proper'. Is this simply a measure of the

severity of the genetic liability or is there some kind of two-hit mechanism and, if there is, what is the other influence? We do not as yet know.

Prodromal Features in Infancy

About a third to a half of parents of a child with an autism spectrum disorder recall abnormalities dating back to the first year and, similarly, early home videos have also identified early manifestations of autism by 12–18 months although the indications are often quite subtle (Rutter 2005a; Yirmiya and Charman 2010). Screening questionnaires for autism work reasonably well at 18 months and above, but are not particularly useful below that age when parents have no clinical concerns (Dietz et al. 2006). It became clear that if there was to be early detection of autism in the infancy period, much more detailed observational measures would be required. The way forward arose from the recognition that siblings of a child with autism have a much increased risk of developing autism. This has led to multiple international 'baby-sibling' studies in which siblings are studied prospectively from early in life to identify and delineate precursors of autism (Bryson et al. 2007; Landa et al. 2007; Zwaigenbaum et al. 2005). In the best of these studies observational and clinical measures are being combined with biological assessments (Elsabbagh and Johnson 2010). Preliminary data suggests that these studies are going to yield important findings because differences have been found between the siblings of children with autism and controls but it remains uncertain how far the findings can be used for individual predictions. Possible preventive interventions have been suggested but, necessarily, they remain speculative at the moment.

Adult Functioning

Long term follow-up studies have all shown substantial variability in outcome among individuals with autism (Howlin et al. 2004). Two factors that have been consistently associated with prognosis are language development and IQ. Very few children who have not developed some useful communicative speech by the age of 5–6 years have a positive outcome and, conversely, individuals who were either cognitively untestable as children or who had non-verbal scores below 50 were almost invariably reported as highly dependent. Best outcomes have been found for individuals with an IQ of at least 70 in childhood. Nevertheless, even in this higher-functioning group (of whom a third had a good or very good outcome in the Howlin et al. (2004) study but just over two-fifths had a 'poor' or 'very poor' outcome) it remains quite unclear why that was the case. Did it reflect the inadequacy of services in childhood, the inadequacy of services in adult life, or did it reflect a basic biological handicap? We do not know. The other query with respect to adult outcome concerns the functioning of individuals with the Asperger syndrome or the broader phenotype. Howlin and her colleagues have a current ongoing study to investigate this further but the small number of participants with a 'broader phenotype' is likely to mean that we will still lack adequate understanding of how they fare in adult life.

Cognitive Patterns

There are well replicated findings on impairments in theory of mind (Frith 2003); joint attention (Mundy and Burnette 2005); central coherence (Happé 2005); and executive functions (Ozonoff et al. 2005). A range of queries remains (Rutter and Bailey 1993; Happé 2003). There is now no doubt that specific cognitive deficits play a major role in the liability to autism. Progress has come particularly from a greater use of experimental designs, the application of eye-tracking methodology (Klin et al. 2005), the use of functional brain imaging (Frith and Frith 2008) and the baby-sibling prospective studies. The hope had been that it would be possible to identify a single modular cognitive deficit that fully accounted for autism, but that now seems less likely. Rather, the imaging studies suggest atypical connectivity as the basic feature, although there is inconsistency across studies in the details (Frith and Frith 2008). It has to be added, too, that it is not yet clear what atypical connectivity means in terms of neural functioning.

Subclassification

Both DSM IV and ICD 10 subdivided autism spectrum disorders (previously called pervasive developmental disorders) into several subcategories. The DSM V Child and Adolescent Psychiatry Working Party has recently suggested that all subdivisions be removed, leaving a single broad category of autism spectrum disorders (see Rutter, in press). They rightly argued that the sub-classification has not worked in practice. However, the removal of all subcategories presents difficulties. First, no one doubts that Rett syndrome constitutes a distinct condition as a virtue of both its progressive course and its origin in a single genetic mutation. Although not clearly spelled out, it seems that what is supposed to happen is that the overall undivided ASD category should be used for the period when children with Rett syndrome show features similar to autism. The recognition of Rett syndrome as a specific cause would be picked up by its diagnosis as a type of neurological disorder.

There are two problems with that. First, it was included as a sub-category of ASD purely because the neurological

section of ICD 10 did not make any mention of Rett syndrome. It is not known what is happening with ICD 11. As far as DSM V is concerned, the difficulty is that, unlike ICD, it does not form part of an overall medical classification and hence the designation of Rett syndrome is more problematic. Second, there is the category of disintegrative disorder. The problem here is that it has been subject to so little research that we simply do not know whether it constitutes an unusual variant of autism or something quite different. It would seem important to keep it in the classification somewhere in order that it may be subject to further research. Third, there is the uncertainty as to whether Asperger syndrome does, or does not, differ meaningfully from high-functioning autism. The published studies comparing the two are quite unhelpful because Asperger syndrome has been dealt with in such varied ways. When, however, there has been a focus on the one key feature of the presence or absence of competence in language structure, developmental trajectory, although similar in shape to that found with autism, is different in being associated with a better outcome (Szatmari et al. 2009). There may be a dispute on whether or not that is most appropriately equated with the syndrome as outlined by Asperger but the distinction does seem worthwhile. It seems that the DSM V working party envisage the distinction being picked up by dimensional codings and, if those can be made to work, that may well be a suitable solution. All that can be concluded firmly at the moment is that it is highly likely that there are meaningful sub-categories of autism spectrum disorders but that these are not well identified by the behavioral diagnoses in the existing classification systems.

Quasi-Autism

The UK study of English and Romanian adoptees (Rutter and Sonuga-Barke 2010) showed that the profound institutional deprivation that lasted beyond the child's age of 6 months was associated in about one in six children with a clinical picture that was similar to autism, but atypical in some features. The mechanism is not well understood, but the implication is that autism may develop on the basis of an external environmentally imposed restriction of stimuli, as well as an internal genetically influenced impairment in the processing of stimuli. It remains to be determined whether abuse and neglect in the family can have the same effect, but the limited available evidence suggests not.

Lack of a Marked Response to Medication

Numerous studies have documented that autism stands out from almost all other psychiatric disorders in showing no marked benefits of psychotropic medication on core symptoms (such as impaired social reciprocity and social communication). (Buitelaar 2003; Scahill and Martin 2005). Why? One possible implication is that the basic deficit does not involve neurotransmitters; if not, what does it involve? It is important to pose the question, not so much because of the implications for treatment today, but rather because a satisfactory answer could have important implications for the neural basis of autism. For the moment, medication is of some value for associated problems, but the enigma is why that seems to be all.

Genetic Findings

Twin and family studies undertaken over a period of several decades have been consistent in showing that ASDs have an overall heritability of about 90% (Rutter 2005b). The falloff rate from MZ to DZ twins, together with that from first degree to second degree relatives, was used by Pickles et al. (1995) to estimate the number of genes that were likely to be involved (Pickles et al. 2000). The findings indicated that there were likely to be at least three or four genes involved in susceptibility to autism but the number could be substantially greater than that. On the other hand, a single-gene Mendelian disorder would not account for the bulk of the findings. The third important finding was that the genetic liability for autism extended to include a broader phenotype (Bailey et al. 1995; Le Couteur et al. 1996). Fourthly, examination of MZ pairs concordant for autism showed that there was enormous clinical heterogeneity even when pairs shared exactly the same segregating genetic alleles (Le Couteur et al. 1996). Over the same period of time, numerous studies showed that Autism Spectrum Disorders were associated with chromosomal abnormalities or genetically determined medical conditions in at least 10% of cases (Rutter et al. 1994). All of these findings still stand today but, during the last decade, there has been no particular progress with respect to these aspects of genetics. Rather, attention has shifted to molecular genetic studies; see Abrahams and Geschwind (2008), Geschwind and Levitt (2007), Folstein and Rosen-Sheidley (2001) and Bacchelli and Maestrini (2006) for reviews of findings.

Rare Pathogenic Gene Mutations

There are multiple replicated findings that autism is associated with rare pathogenic gene mutations such as neuroligins, neurexin and SHANK 3 (Persico and Bugeron 2006; Durand et al. 2007; Bourgeron 2007; Geschwind and Levitt 2007; Jamain et al. 2008). They account for a tiny proportion of cases (circa 1%) but it has been claimed that they are, nevertheless, true 'causes' of autism. The

dilemma is that, although the clinical picture associated with these genes includes autistic features, intellectual disability often dominates. Of course, genes do not code for specific psychiatric categories and pleiotropic effects are to be expected. Nevertheless, the lack of specificity raises doubts on the extent to which the findings are informative with respect to most cases of Autism Spectrum Disorder.

Copy Number Variations (CNVs)

It is now possible to detect tiny sub-microscopic chromosomal deletions or duplications (known as copy number variations). Several studies have shown that CNVs, especially those involving chromosomal deletions, are found in some 5% of cases of autism—a rate significantly higher than that in controls (Cook and Scherer 2008; Szatmari et al. 2007; Sebat et al. 2007; Marshall et al. 2008). The evidence indicates a causal role for CNVs in both autism and schizophrenia (International Schizophrenia Consortium 2008), and also ADHD (Williams et al. 2010) but important queries remain. Most CNVs arise de novo and, therefore, cannot account for familiality. Also, when inherited, the CNVs may be present in family members who are unaffected by autism; thus the causal effect is not necessarily determinative. It is also striking that the relevant CNVs seem frequently to be different in different families (Pinto et al. 2010). Also, it is necessary to ask what causes the raised frequency of CNVs; one possibility is raised parental age (see below). The same question applies to major chromosome anomalies, which are also more frequent in individuals with autism than in the general population.

Genome-Wide Association Studies (GWAS)

It is now possible to undertake genome-wide association studies instead of relying on candidate genes to direct the search for susceptibility genes. GWAS require huge samples and, inevitability, will give rise to many false positives (Dodge and Rutter, in press). GWAS have the important advantage of being able to detect novel genetic associations but the findings so far have been unimpressive in the field of multifactorial mental disorders. Moreover, because the identified susceptibility genes have been found to have very weak effects, it remains uncertain whether the findings will be very informative on biological causal pathways.

Epigenetics

There is now great interest in the possibility that many genetic effects derive from epigenetics rather than changes in gene sequence. Epigenetics refers to neuro-chemical changes that influence gene expression (Meaney 2010). Expression is both tissue-specific and developmental phase-specific. It involves multiple DNA elements, chance effects, and environmental influences. There is limited evidence that epigenetic mechanisms might be involved in autism but so far their role remains uncertain (Gregory et al. 2009).

Why Doesn't Autism Become Extinct?

It is known that both autism and schizophrenia are associated with a markedly reduced fecundity (ability to reproduce). Accordingly, why doesn't autism die out? What enables it to persist in the population? (Uher 2009). No convincing answer is available, but the findings suggest that the genetic influences on autism and on schizophrenia may well involve mechanisms that are different from those that apply to other mental disorders (possibly involving a greater role for rare pathogenic gene mutations or CNVs—see above).

Why Haven't the Susceptibility Genes for Autism Been Identified?

As noted above, twin and family studies have been consistent in indicating that autism has a very high heritability (circa 90%); why, therefore, has it proved so difficult to find the specific genes responsible? We do not really know what the answer should be but, in addition to the likelihood of genetic heterogeneity and the very small effects of individual genes, the explanation may lie in epigenetics, or in gene environment correlations and interactions, or in synergistic effects among genes.

Environmental Findings

MMR and Thimerosal

Claims have been made that either the Measles, Mumps, Rubella (MMR) vaccine or Thimerosal (a mercury preservative used in some vaccines), or both, were responsible for an epidemic of autism. So far as MMR is concerned, epidemiological research has been consistently negative with respect to that claim (Rutter 2008); most decisively, the evidence from Japan that when MMR was totally withdrawn, there was no effect on the overall rise in the rate of diagnosed autism (Honda et al. 2005). Moreover, well conducted studies have also shown that the measles virus in tissue claims were mistaken (Baird et al. 2008b; Hornig et al. 2008; D'Souza et al. 2006; Afzal et al. 2006). The situation with respect to Thimerosal is somewhat more complicated in that there is no doubt that mercury is a proven neurotoxin. Nevertheless, the same types of

epidemiological research have also failed to support the claim that this has led to an epidemic of autism. In particular, the withdrawal of Thimerosal from all vaccines in Scandinavia, at a time when such use was continuing in the rest of the world, provides convincing evidence against the 'epidemic' notion (Atladóttir et al. 2007). A broader range of research has examined the effects of mercury toxicity in humans and has also examined putative Thimerosal effects in various different ways. Findings are rather consistently negative so far as Thimerosal is concerned (particularly as a risk factor for autism) but it is clear that, in moderately raised dosages, there can be significant effects from mercury toxicity.

Raised Parental Age

There are now replicated findings that children born to older fathers have an increased rate of autism (Reichenberg et al. 2006; Croen et al. 2007; Cantor et al. 2007). Less certainly, this may also apply to older mothers. The likely explanation is that the older father effect reflects the increased likelihood of genetic mutations with an increasing number of cell divisions, but the association has been too little investigated in humans to be certain about this mechanism. Little is known on the effects of older fathers on the risk for other mental disorders, apart from schizophrenia for which a meta-analysis suggested that late fatherhood increased the risk (Wohl and Gorwood 2007).

Maternal Immigration

There are now several studies (Keen et al. 2010 for example), showing that maternal immigration is associated with an increased risk of autism in the children. Earlier studies had examined the possibility that rates of autism might be higher among immigrants (see Fombonne 2005) with results that were inconsistent, but largely unsupportive of the suggestion. Newer findings refer specifically to immigration of the mother and these appear to be sound. Nevertheless, the evidence remains scanty, the effect is weak, and it remains uncertain whether this association represents a causal effect and, if it does, the mechanism remains obscure.

Other Pre-Natal and Early Post-Natal Influences

The evidence that autism spectrum disorders (ASD) are multifactorial in nature means that some environmental factors are likely to be implicated in causation. Increase over time in the rate of diagnosis of ASD, if it reflects a true rise in incidence (which remains uncertain), would also point to some environmental effect. Prospective longitudinal studies of very large samples starting during pregnancy, and including good biological measures, are needed to test the possibility. The Norwegian mother and baby study (MoBa) of some 100,000 children is one such investigation (Magnus et al. 2006; Rønningen et al. 2006; Stoltenberg et al. 2010).

Psychological Treatments

There have been no major developments during the last 3 years in our understanding of autism derived from psychological treatments, but there is continuing controversial discussion over claims that very intense, very early behavioral treatment can lead to 'recovery'. That such treatment can bring worthwhile benefits is not in doubt (Medical Research Council 2001; National Research Council 2001). Equally, it is known that, even in the absence of such early treatment, huge gains in functioning can sometimes occur. Whether or not there is complete recovery is much less certain (Helt et al. 2008). Also, the claims of the necessity for very early treatment and high intensity for some 40 h a week for at least 2 years remain very questionable (Howlin 2003, 2005). What is new is the accumulation of better evidence deriving from well planned randomized controlled trials.

An important new randomized controlled trial of early intensive behavioural intervention provides possibly the best evidence to date (Dawson et al. 2009). Findings showed that there was a significant increase in IQ (albeit of modest size) in the treated group but not in controls. However, what this means is uncertain because there was no increase in social functioning as measured by the Vineland scale at 12 months. Also, the results showed that there was no effect of the treatment on core features of autism as assessed by the autism diagnostic observation schedule. Accordingly, the study certainly does not support the Lovaas claims (Lovaas 1987; McEachin et al. 1993) about the huge benefits of early treatment leading to recovery.

What is also very new is the introduction of methods of treatment focused on improving parental sensitivity and responsiveness. The randomized controlled trial undertaken by Green et al. (2010) provide an excellent model of how RCTs should be undertaken. The findings are encouraging in showing substantial significant positive changes in parental sensitivity/responsivity but disappointing in that there was only a very small improvement (relative to the control group) in the children's autistic features.

Conclusions

Looking back over the last 50 years (Feinstein 2010) it is clear that the understanding of autism has been transformed

in numerous different ways. The substantial gains in knowledge during the last few years have been equally impressive. There have been many important findings, some of which have been rather unexpected, but major puzzles remain. We should be hopeful of even greater gains in the years ahead, but both prevention and cure remain elusive.

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