

## REPORT

# Why specific developmental disorders are not specific: On-line and developmental effects in autism and dyslexia

Uta Frith<sup>1</sup> and Francesca Happé<sup>2</sup>

1. MRC Cognitive Development Unit, University College London, UK

2. Social, Genetic and Developmental Psychiatry Research Centre, Institute of Psychiatry, London, UK

### Abstract

*In this paper we will suggest that impairments in domain-specific functions may have wide-ranging developmental effects which mimic domain-general impairments. This may explain why 'pure' cases of developmental disorders are rare. The understanding of autism and dyslexia has been advanced by theories postulating failure of a specific mechanism against a background of unimpaired general processing. Impairment in the mechanisms underlying 'theory of mind' in autism, and 'phonology' in dyslexia, predict and explain the pattern of poor and good performance on a range of experimental and real-life tasks. However, existing accounts deal largely with the on-line effects of modular deficits, and can be criticised as not truly developmental. Here we attempt to trace developmental or down-stream effects of specific impairments in mentalising and phonological processing. We argue that these modules act as vital gatekeepers in development. Important in this process is the interaction of cognition with socio-cultural environment, an effect well-illustrated by cross-language differences in dyslexia. We claim that consideration of these developmental effects makes clear why postulating specific impairments may be sufficient for explaining more general deficits.*

### Specific cognitive deficit as a cause of developmental disorder

A psychologist studying normal children may find it quite natural to think of the mind as all of one piece. By contrast, a psychologist investigating developmental disorders may be inclined to view the mind as fractionated. Different developmentally disordered groups show different, and characteristic, patterns of cognitive abilities and disabilities. Explaining these is the task set for cognitive theories of disorders. Such theories also impact on our understanding of the normally developing mind, and may suggest a modular organisation based on a number of different computational mechanisms (but see Karmiloff-Smith, this issue, for an alternative perspective).

Autism and dyslexia are examples of specific developmental disorders for which cognitive theories of modular impairment have proved useful. Both dyslexia and autism have a genetic origin, an anatomical basis in the brain, and extremely variable behavioural manifesta-

tion. Diagnosis in both cases is based on behavioural criteria, but the core features of these disorders are best understood in terms of specific deficits at the cognitive level.

### Dyslexia

Figure 1 illustrates the phonological deficit hypothesis, using the causal modelling notation developed by Morton and Frith (1995). The relationship between biological and cognitive levels, and between cognitive and behavioural levels, is indicated by causal arrows. The relationship here is mediated by the component 'phoneme-grapheme system knowledge' (labelled g-p), which is influenced by the particular language and orthography that is being acquired. Some of the behavioural manifestations of a phonological deficit are poor acquisition of reading, poor performance on phoneme awareness tasks (Bradley & Bryant, 1978), slow naming speed (Denckla & Rudel, 1976), and impaired verbal short term memory (Nelson & Warrington,

Address for correspondence: Dr Francesca Happé, Senior Scientist in Cognitive Psychology, Institute of Psychiatry, SGDP Research Centre, 111 Denmark Hill, London SE5 8AF, UK.

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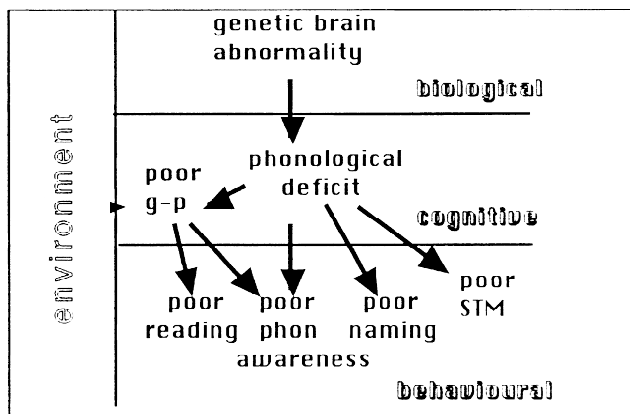


Figure 1 A schematic causal model for dyslexia

1980). The hypothesis that a phonological deficit can cause dyslexia has led to the following tested and confirmed predictions: a) problems predate the acquisition of literacy (Scarborough, 1990), b) they are specific, independent of IQ (Siegel, 1989), c) they persist over time (Pennington *et al.*, 1990), d) they are universal, regardless of language (Landerl, Wimmer & Frith, 1997), e) their neurological basis is found in the speech processing areas of the brain (Paulesu *et al.*, 1996).

Autism

Figure 2 illustrates the mentalising deficit hypothesis (Baron-Cohen, Leslie & Frith, 1985). A dedicated cognitive mechanism, which enables representation of own and others' thoughts, is postulated to be impaired in individuals with autism<sup>1</sup> (Leslie, 1987; Frith, Morton & Leslie, 1991). This hypothesis can explain impairments in imaginative pretend play, insightful social interaction, and ostensive-inferential communication – the core features of autistic spectrum disorders. Specific signs of mentalising deficit include the inability to understand psychological motivation (e.g. deception) and communicative intention (e.g. metaphor and irony; see chapters in Baron-Cohen, Tager-Flusberg & Cohen, 1993).

<sup>1</sup>The mentalising hypothesis does not explain the whole clinical picture of autism, and it is necessary to consider additional hypotheses to account for the non-social features of autism (see, for example, Bailey, Phillips & Rutter, 1996). Notable among these are weak central coherence and impaired executive functioning. These impairments are of a domain-general rather than specific nature and therefore do not belong in the present discussion of the effects of specific deficits. Likewise in dyslexia, arguments for more general deficits in, for example, temporal, auditory or visual processing (see chapters in Hulme & Snowling, 1997), are beyond the scope of the present discussion.

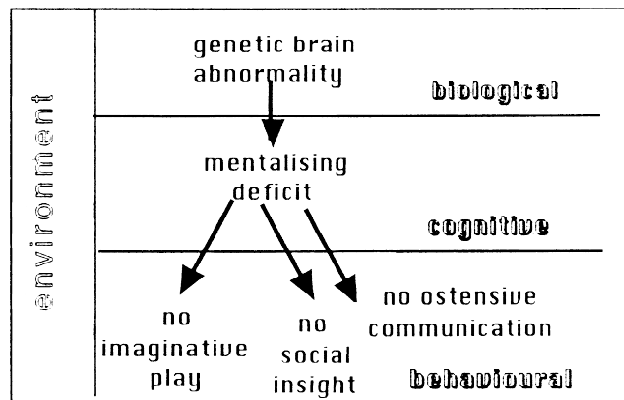


Figure 2 A schematic causal model for autism

Fine cuts to identify modular mental mechanisms

Proving the specificity of the postulated impairments in mentalising and phonology is a complex task. The 'fine cuts' method (Frith & Happé, 1994a) contrasts intact and impaired performance on closely matched tasks differing only in their demand for the key cognitive component. For instance, children with autism understand sabotage but not deception - the latter requiring manipulation of belief rather than behaviour (Sodian & Frith, 1992). In the same way, children with autism understand that a photograph (physical representation) may become out of date, but fail to understand that a belief (mental representation) may become false if the believer does not witness a critical change of affairs (Leslie & Thaiss, 1992). The method of fine cuts in the exploration of the phonological deficit in dyslexia has led to the contrast between good verbal fluency to a semantic cue (animals) versus poor fluency to a phonemic cue (words starting with /s/; Frith, Landerl & Frith, 1995). The demonstration of these fine cuts at otherwise hidden seams supports the idea of separable cognitive mechanisms which appear to contribute to the development of specific functions. It remains a possibility, however, that a domain-general system might, if lesioned, produce such a pattern of apparently specific deficits (e.g. Plaut, 1995).

The fine cuts method is well suited to functional brain imaging, which relies on subtraction of activation during contrasting conditions. To date, imaging studies have supported the notion that mental activity requiring, respectively, theory of mind (Fletcher *et al.*, 1995), and phonology (Démonet *et al.*, 1992), have distinct and isolable pathways in the normal brain, which are not activated when compensated adults with autism or

dyslexia engage in these tasks (Happé *et al.*, 1996; Paulesu *et al.*, 1996).

### Developmental versus on-line effects of cognitive deficits

While the postulated specific deficits in autism and dyslexia succeed in predicting the pattern of good and poor performance across a range of tasks in a stringent fine-cuts technique, the developmental effects of these modular deficits have not yet been fully considered. For example, a distinction needs to be made between the immediate effects of a mentalising deficit in tests tapping the on-line monitoring of mental states (e.g. predicting behaviour based on a false belief), and the long term effects of this deficit on the development of other abilities (e.g. language acquisition).

Language delay is the norm in autism, reflecting, we have argued, the role of mentalising in the acquisition of the agreed names for things (Frith & Happé, 1994b). Recent work has made it clear that the normal acquisition of referential meaning is facilitated by orienting towards ostension and following adult eye gaze, and that this process fails in autism (Baron-Cohen, Baldwin & Crowson, 1997). Slow and idiosyncratic vocabulary acquisition in autism is thus hypothesised to be the developmental effect of failure to recognise intentions.

In dyslexia too, developmental effects of phonological impairment should have an impact on vocabulary acquisition. This would be for a different reason, however, namely difficulties in forming/accessing phonological representations and hence in learning new words (Snowling, 1995). Thus scores on vocabulary tests tend to be impaired and verbal IQ tends to be lower than performance IQ in adults with dyslexia (Nelson & Warrington, 1980).

#### *Cognition in interaction with environment*

The developmental effects of specific modular deficits are the product of interaction with the socio-cultural environment. As regards literacy acquisition, the relevant environmental factors vary markedly between cultures. Phonological and orthographic complexity in the learning environment, as well as teaching method, may aggravate or ameliorate the effects of the postulated modular deficit in dyslexia. Landerl, Wimmer and Frith (1997), showed that dyslexic children being taught the transparent orthography of German had milder reading impairments than dyslexics learning the irregular orthography of English, even though they were equally impaired on pure phonological tasks (e.g. Spoonerisms).

At the extreme of cognitive-environment interaction dyslexia, defined as a specific deficit in phonological processing, should exist even in pre-literate cultures. Affected children would show a delay in learning names for things, but the impact over the lifetime might be slight. A child with dyslexia born into a pre-literate society would in effect be a 'hidden case'.

Children with autism are not so lucky. Here cross-cultural differences are not evident. Indeed the relevant cultural context is a universal mode of human social interaction based on mentalising. In fact, this 'culture' is so pervasive that the developmental effects of mentalising impairment may be extremely wide-ranging. As children with dyslexia miss out on learning based on the written word, so children with autism will miss out on learning based on social insight.

#### *Specific impairments and general deficits: the case of intelligence*

A strong piece of evidence for the modularity of the mentalising mechanism is the existence of otherwise intelligent children with autism who show a specific impairment in social reasoning. However, three quarters of children with autism also have mental retardation (IQ below 75). Is this inconsistent with the idea of a modular deficit? Earlier biological accounts have suggested a two-hit model, with one hit leading to autism and the second to mental retardation. However, as Rutter and Bailey (1993) point out, this leaves unexplained why autism and mental retardation should be so strongly associated, and why autism is not more frequent in other forms of mental handicap in which IQ is compromised (e.g. Down Syndrome).

Could consideration of the developmental effects of mentalising impairment shed light on the high incidence of mental retardation in autism? That is, can a domain-specific impairment have effects on domain-general processes through development? It has frequently been suggested that on-line effects of general social or motivational impairment in autism lead to underestimation of IQ on standard tests. However, this would lead one to expect a flat profile of depressed scores across all experimenter-administered tasks. This is not the case. The spiky IQ profile, with peak performance on Block Design and a dip in performance on the Comprehension subtest, has frequently been replicated (work reviewed in Happé, 1994). A general on-line effect of lack of social motivation or interest, then, will not explain the pattern seen. However, the more specific account from mentalising impairment takes us somewhat further. Attribution of mental states appears to be necessary on-line for some but not all IQ subtests. Indeed, poor

performance on the Comprehension subtest, which requires on-line pragmatic competence, relates to poor performance on standard false belief tasks (Happé, 1994).

If it is possible to explain the troughs in autistic IQ performance by reference to on-line effects of mentalising impairment, does this mean that the peaks of IQ performance represent the true ability level in autism? That would mean that even children who scored poorly on standard IQ tests, in fact possessed the potential for good intelligence. Is there any evidence for this? A task which may measure basic processing capacity without a social component (either on-line or developmental) is the Inspection Time (IT) task developed as a computer-presented task by Anderson (1992). In this task, the child makes a simple same-different discrimination between two lines of varying lengths, presented for varying intervals followed by a mask. The minimum time necessary for success in judging the display is calculated via psychophysical procedures. This measure has been shown to correlate significantly with standard measures of IQ in normal and mentally handicapped children (Kranzler & Jensen, 1989). If our speculations are correct, and children with autism are specifically rather than generally impaired, we should expect good performance on the IT task (better than that by children with general learning disability). We have compared IT performance in 18 children with autism, 21 non-autistic mentally handicapped and 13 normal children (Scheuffgen *et al.*, submitted). The autism group achieved IT scores as fast as those of normal children, even though their Wechsler IQ was, on average, 40 points lower. They also performed significantly better than an IQ-matched group of mentally handicapped children. We do not conclude from this result that children with autism are unimpaired in general intelligence, rather we suggest that their potential for normal acquisition of information and skills is critically compromised by the developmental sequelae of a modular deficit.

Why is mentalising important in the development of skills tapped by IQ assessments? We suggest that a great deal of information is acquired in normal development through interpersonal processes. From early years, tracking eye gaze, social referencing and joint attention shape normal children's interests and select for them those features of the world which are relevant and deserve processing. In autism, these processes are absent, leading to attention to and interest in idiosyncratic features of the world, and bizarre valence associations. The function of objects is normally learnt by reference to the maker's/user's intentions. Pedagogy requires that the teacher recognise the knowledge state (ignorance, false belief) of the pupil, and that the pupil

recognise the informative intent of the teacher. That may be why no non-human species deliberately teaches its young, just as no non-human species has as yet provided compelling evidence of mentalising (Heyes, 1993).

The claim that autism is commonly accompanied by low measured IQ as a consequence of modular impairments in social insight, forces us to predict an association between the severity of handicap in these two areas. This may not be the same thing as predicting that level of IQ and severity of autism are strongly related, for at least two reasons. Firstly, severity of autism is an ill-defined concept; it is often unclear to which symptoms, social or nonsocial, it applies. Secondly, we predict that autism will show a range in intelligence, as observed in the non-autistic population, including low normal intelligence, and mental retardation as a result of additional pathology.

In dyslexia the phonological impairment does not appear to have such broad effects on general ability, apart from the developmental consequences on language-related functions. Thus, verbal IQ as measured on the Wechsler scales is often lower than performance IQ, but most prominent are the dips on the Digit Span and Arithmetic subtests. The Digit Span test is subject to on-line effects of the phonological deficit: poor inner speech leads to poor verbal rehearsal which in turn leads to poor recall. In the Arithmetic subtest, verbal memory is also taxed by the verbally presented problems. However, developmental effects on learning number facts also play a role: the critical phonological mechanism appears to be involved in the rote learning of verbally coded information (e.g. multiplication tables; Miles, 1983).

## Conclusions

Teasing apart the on-line and developmental effects of specific cognitive deficits is clearly possible, but awaits empirical demonstration. However, it is already apparent that a puzzling phenomenon might be solved by applying this distinction. It is now clear that 'pure' cases, which one might expect to exist on the basis of a modular cognitive deficit, are in fact extremely unlikely. Only in acquired disorders will on-line effects be seen in isolation, unconfounded by developmental sequelae. The gist of our argument is that general impairments (e.g. low IQ) in developmental disorders need not be the result of primary damage to domain-general mechanisms. Rather, they may be the developmental consequence of damage to very specific, even modular, mechanisms which act as gatekeepers in development.

The examples given here of effects on language acquisition and measured intelligence in autism suggest that there may be no need to invoke additional cognitive deficits to account for the frequent occurrence of language delay and mental retardation. Likewise, in the case of dyslexia, language delay, memory and arithmetic difficulties can be seen as developmental sequelae of one and the same faulty phonological processing mechanism. Thus specific disorders may be specific only in their underlying cognitive cause, and not at all specific in their far reaching developmental consequences.

## References

- Anderson, M. (1992). *Intelligence and development: A cognitive theory*. Oxford: Blackwell.
- 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**, 89–126.
- Baron-Cohen, S., Leslie, A.M., & Frith, U. (1985) Does the autistic child have a 'theory of mind'? *Cognition*, **21**, 37–46.
- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D.J. (Eds.) (1993) *Understanding Other Minds: Perspectives from Autism*. Oxford: Oxford University Press.
- Baron-Cohen, S., Baldwin, D., & Crowson, M. (1997) Do children with autism use the speaker's direction of gaze strategy to crack the code of language? *Child Development*, **68**, 48–57.
- Bradley, L., & Bryant, P.E. (1978) Difficulties in auditory organization as a possible cause of reading backwardness. *Nature*, **271**, 746–747.
- Démonet, J.F., Chollet, F., Ramsay, S., Cardebat, D., Nespoulous, J., Wise, R. *et al.* (1992) The anatomy of phonological and semantic processing in normal subjects. *Brain*, **115**, 1753–1768.
- Denckla, M., & Rudel, R. (1976) Rapid 'automatized' naming (RAN): dyslexia differentiated from other learning difficulties. *Neuropsychologia*, **14**, 471–479.
- Fletcher, P.C., Happé, F., Frith, U., Baker, S.C., Dolan, R.J., Frackowiak, R.S.J., & Frith, C.D. (1995) Other minds in the brain: a functional imaging study of 'theory of mind' in story comprehension. *Cognition*, **57**, 109–128.
- Frith, U., & Happé, F. (1994a) Autism: beyond 'theory of mind'. *Cognition*, **50**, 115–132.
- Frith, U., & Happé, F. (1994b) Language and communication in the autistic disorders. *Philosophical Transactions of the Royal Society, series B*, **346**, 97–104.
- Frith, U., Landerl, K., & Frith, C. (1995) Dyslexia and verbal fluency: More evidence for a phonological deficit. *Dyslexia*, **1**, 2–11.
- Frith, U., Morton, J., & Leslie, A.M. (1991) The cognitive basis of a biological disorder: Autism. *Trends in Neuroscience*, **14**, 433–438.
- Happé, F.G.E. (1994). Wechsler IQ profile and theory of mind in autism: A research note. *Journal of Child Psychology and Psychiatry*, **35**, 1461–71.
- Happé, F., Ehlers, S., Fletcher, P., Frith, U., Johansson, M., Gillberg, C., Dolan, R., Frackowiak, R., & Frith, C. (1996) 'Theory of mind' in the brain. Evidence from a PET scan study of Asperger syndrome. *NeuroReport*, **8**, 197–201.
- Heyes, C.M. (1993) Anecdotes, training, trapping and triangulating: Do animals attribute mental states? *Animal Behaviour*, **46**, 177–188.
- Hulme, C., & Snowling, M. (Eds) (1997) *Dyslexia: Biology, cognition and intervention*. London: Whurr.
- Kranzler, J.H., & Jensen, A.R. (1989). Inspection time and intelligence: A meta-analysis. *Intelligence*, **13**, 329–47.
- Landerl, K., Wimmer, H., & Frith, U. (1997) The impact of orthographic consistency on dyslexia. A German-English comparison. *Cognition*, **63**, 315–334.
- Leslie, A.M. (1987) Pretence and representation: The origins of 'Theory of Mind'. *Psychological Review*, **94**, 412–426.
- Leslie, A.M., & Thaiss, L. (1992). Domain specificity in conceptual development: Neuropsychological evidence from autism. *Cognition*, **43**, 225–51.
- Miles, T.R. (1983) *Dyslexia: The pattern of difficulties*. London: Granada.
- Morton, J., & Frith, U. (1995) Causal modelling: a structural approach to developmental psychopathology. In, D. Cicchetti & D.J. Cohen (Eds), *Manual of Developmental Psychopathology*. Vol.1, chapter 13. New York; John Wiley & Sons. (pp.357–390)
- Nelson, H.E., & Warrington, E.K. (1980) An investigation of memory functions in dyslexic children. *British Journal of Psychology*, **71**, 487–503.
- Paulesu, E., Frith, U., Snowling, M., Gallagher, A., Morton, J., Frackowiak, R.S.J., & Frith, C.D. (1996). Is developmental dyslexia a disconnection syndrome? Evidence from PET scanning. *Brain*, **119**, 143–157.
- Pennington, B.F., VanOrden, G.C., Smith S.D., Grenn, P.A., & Haith, M.M. (1990) Phonological processing skills and deficits in adult dyslexics. *Child Development*, **61**, 1753–1778.
- Plaut, D. (1995) Double dissociation without modularity: evidence from connectionist neuropsychology. *Journal of Clinical and Experimental Neuropsychology*, **17**, 291–331.
- Rutter, M., & Bailey, A. (1993) Thinking and relationships: mind and brain (some reflections on theory of mind and autism). In, Baron-Cohen, S., Tager-Flusberg, H. & Cohen, D.J. (Eds.) (1993) *Understanding Other Minds: Perspectives from Autism*. Oxford: Oxford University Press. (pp.481–504)
- Scarborough, H.S. (1990) Very early language deficits in dyslexic children. *Child Development*, **61**, 1728–1743.
- Scheuffgen, K., Happé, F., Anderson, M., & Frith, U. (submitted) High 'intelligence', low 'IQ'? Speed of processing and measured IQ in children with autism.
- Siegel, L.S. (1989) IQ is irrelevant to the definition of learning disabilities. *Journal of Learning Disabilities*, **22**, 469–478.

- Snowling, M.J. (1995) Phonological processing and developmental dyslexia. *Journal of Research in Reading*, **18**, 132–138.
- Sodian, B., & Frith, U. (1992) Deception and sabotage in autistic, retarded and normal children. *Journal of Child Psychology and Psychiatry*, **33**, 591–605.

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