

Theories of autism

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The purpose of the present paper was to review psychological theories of autism, and to integrate these theories with neurobiological findings. Cognitive, theory of mind, language and coherence theories were identified, and briefly reviewed. Psychological theories were found not to account for the rigid/repetitive behaviours universally described in autistic subjects, and underlying neurobiological systems were identified. When the developing brain encounters constrained connectivity, it evolves an abnormal organization, the features of which may be best explained by a developmental failure of neural connectivity, where high local connectivity develops in tandem with low long-range connectivity, resulting in constricted repetitive behaviours.

Key words: autism, Asperger's disorder, theory of mind, coherence, neural connectivity.

Australian and New Zealand Journal of Psychiatry 2007; 41:859–868

The essential features of autistic disorder are the presence of markedly abnormal or impaired development in social interaction and communication, and a markedly restricted repertoire of activity and interests. The impairment in social interaction is gross and sustained, and there may be marked impairment in the use of multiple non-verbal behaviours (e.g. eye-to-eye gaze, facial expression, body postures and gestures). There may also be a lack of varied, spontaneous make-believe play, or social imitative play appropriate to developmental level. Speech development is delayed and the pitch, intonation, rate, rhythm or stress may be abnormal, with abnormal grammar and repetitive use of language or idiosyncratic language. Behavioural symptoms include odd responses to sensory stimuli (e.g. oversensitivity to sounds or being touched, and fascination to certain repetitive stimuli and a lack of fear in response to real danger), as well as overactivity, short attention span, impulsivity, aggression, and self-injurious behaviour.

Asperger's disorder is defined by similar criteria to autistic disorder, namely qualitative impairment in social interaction and restricted, repetitive and stereotyped patterns of behaviour, interests and activities, but specifies that there is no clinically significant general delay in language. Autistic spectrum disorders (ASD) include autistic disorder and Asperger's disorder (DSM-IV) [1]. According to Allen *et al.* these disorders exhibit significant repetitive behaviours and restricted interests, including ritualistic behaviours such as counting, tapping, flicking, or repeatedly restating information, and compulsive behaviours such as lining up objects requiring a rigid adherence to routine and a marked resistance to change [2]. While the onset of these disorders is believed to be prior to or at birth, they are poorly understood.

Theory of mind

The theory of mind (TOM) hypothesis seeks to explain pragmatic impairments of language and communication in terms of social deficits with some specific accompanying neurocognitive deficits. The cognitive theories of Boucher, and Rutter also consider as central the autistic child's difficulty in

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Received 6 May 2007; accepted 12 July 2007.

understanding other people's mental states [3,4]. According to Baron-Cohen, TOM starts from the premise that mental states are not directly observable, but have to be inferred, requiring a complex cognitive mechanism [5]. Thus the cognitive theory places more emphasis on the ability to infer mental states, such as 'beliefs about something' implying intentionality, rather than emotions. Baron-Cohen describes this ability as the attribution of mental states with content to others, as a TOM [6] because it involves the person postulating the existence of mental states, and using them to explain and predict another person's behaviour. 'Our beliefs about or concepts of the physical world may be called 'primary representations'. 'However our beliefs about other people's mental states (such as their beliefs and desires) are representations of other representations', which may be called 'second-order representations', or 'meta-representations' [7,8]. The cognitive theory points out that in autism the capacity for meta-representation is impaired. Thus according to Baron-Cohen the cognitive theory proposes that the observed pragmatic deficits in autism are those that would be expected if autistic children are using language without a TOM [5].

Tager-Flusberg has approached these issues by comparing studies of William's syndrome (WMS) and autism [9]. The TOM hypothesis has been thought to explain the failure of children with autism on tasks investigating TOM, and to also explain deficits in pretence, social functioning and communication. These deficits were taken as evidence in support of the modularity of the mind. In contrast, WMS is thought to be characterized by sparing in the TOM domain, because of relatively good language skills, excellent face processing ability, strong social interest and attention to faces and people. However, Tager-Flusberg points out that the TOM hypothesis of autism has been criticized on the basis that mentally retarded children and adolescents also show TOM deficits as reported by Zelazo *et al.* [9,10]. Also autistic symptoms emerge very early in infancy, before TOM is measurable or developed in normal children. Also, some autistic children pass TOM and false belief tasks.

Baron-Cohen and Tager-Flusberg have shown that adolescents with WMS performed no better than IQ-matched controls on TOM, false belief and explanation of action tests [5,9]. Tager-Flusberg has proposed a two-component model to help explain the aforementioned contradictory findings [9,11]. The model describes a primary social-perceptual and a higher-order social-cognitive capacity. The latter refers to the meta-representational capacity to make

more complex cognitive inferences about the content of mental states, requiring information across time and events. Tager-Flusberg describes the early social-perceptual deficits in autism as being associated with failure to perceive others as intentional as well as joint attention deficits [9].

Boucher reviewed the hypothesis that autistic people have impaired meta-representative ability, and as a result, lack a TOM [12]. According to Boucher, the term 'TOM' derives from a paper by Premack and Woodruff entitled 'Does a chimpanzee have a theory of mind?' [6]. Premack and Woodruff described experiments in which chimpanzees exhibited rudimentary understanding of the mind of others, although the question remains a matter of debate. Boucher points out that both Perner, and Leslie and Frith used the term 'theory of mind' in a narrow sense [12-14]. Perner suggested three levels of representational abilities. At the first level a child encodes perception (knowledge base). At the second level the child is able to manipulate his/her knowledge base to engage in pretend play and distinguish between things real and imaginary, while at the third level the child becomes able to reflect on his/her own representations. Perner calls the ability to reflect on alternative models or representations of the world 'meta-representation', and maintains that only at this stage can a child be said to have a TOM [13].

Leslie also maintained that pretence, like TOM, involves second-order representations and that pretence is a manifestation of a primitive TOM [15]. Leslie defined pretend play as requiring at least one of the following: (i) one object is substituted for another; (ii) non-existent properties are attributed to an object; and (iii) absent objects are imagined.

Leslie suggested that pretend play was dependent on second-order representations, requiring first a primary representation and then a secondary representation, which is a copy of the primary representation, but can be manipulated in play, without distorting the original primary representation, utilizing a decoupling mechanism [15].

According to Boucher, one of the difficulties in assessing the TOM hypothesis of autism is that there has been no clear statement of what is meant by key terms such as 'theory of mind' and 'meta-representation' [12]. She outlined a definition, which might be acceptable to its proponents as: (i) that autistic individuals have impaired ability to attribute mental states to others; (ii) that this is caused by some specific impairment of higher-order representational capacity; (iii) that this specific impairment, when fully understood, will be seen to be primary, in the sense of

not itself being in need of further behavioural explanation and being able to explain all the criterial features of autistic behaviour.

Thus impairment of higher-order representational capacity is consistent with a higher-order domain-general incapacity.

Boucher questions whether the fact that autistic children appear not to attribute mental states to others means that they are not able to attribute mental states to others [12]. She quotes her own work, which suggests that autistic children can pretend play as adequately as language-matched controls, but do not spontaneously do so. For example, Lewis and Boucher reported a study in which autistic children spent less time than controls in both pretend and non-pretend (functional) play [16]. Boucher suggests that autistic children's paucity of spontaneous imaginative play (both functional and pretend) is at least partly the result of a problem with motivation or initiative [12], and also points out that the TOM hypothesis is a circular argument in that autistic children's impaired ability to attribute mental states to others is caused by impaired meta-representative ability. Thus other tests than pretend play are required to break this circularity. DeGelder argues that it is likely that lower order biological functions are impaired in autism, because autism originates in infancy, long before there is evidence of an impaired TOM [17].

Thus while TOM describes aspects of the psychological functioning in autism, deficits are also observed in mental retardation, and autistic phenomena are described at an earlier developmental stage than that expected for TOM.

Language in autism

Walenski *et al.* reviewed language deficits in autism, ASD and Asperger's syndrome [18]. They pointed out that while 20% of children with autism are essentially non-verbal, others acquire functional language to varying extents, and that ASD may be associated with a particular pattern of both relatively spared and impaired language functions. The authors described two exploratory theories of language in ASD, which are complementary in that they focus on different sets of language functions.

The authors describe pragmatics as the use of language appropriately for the social and real-world contexts in which utterances are made, including interpreting a speaker's intended meaning across different social contexts. Widespread pragmatic impairments have been described in autism and ASD.

Prosody is described as the timing, rhythm and intonation of speech. The pragmatic functions relate to non-grammatical pauses, and the use of stress in language. Prosodic deficits are common in ASD, as are difficulties with metaphor, irony and jokes. According to Walenski *et al.* ASD involves pervasive impairments in the pragmatic aspects of language usage [18]. The theory hypothesizes 'that people with ASD are fundamentally impaired at causally linking their own and other people's behaviour to mental states'. This is thought to be evidenced by deficits in the understanding of false belief and emotion. The authors note that functional neuroimaging studies in ASD suggest abnormalities in the neural structures thought to underlie the processing of TOM. These include the medial frontal cortex (BA 8/9) bordering on the cingulate gyrus, lateral inferior frontal cortex (primarily Broca's area BA 44/45), and posterior superior temporal/temporo-parietal cortex. While neuroimaging studies of ASD suggest a tendency for decreased activation (relative to controls) in both frontal areas, they show activation (relative to controls) in the posterior superior temporo-parietal cortex.

According to Tager-Flusberg, language is integrally linked to the social-cognitive component of TOM [9]. Astington and Jenkins were able to show that language and, in particular, syntactic knowledge, predicted later TOM in a longitudinal study of preschoolers [19]. Tager-Flusberg describes studies that postulate that sentential or tensed compliments, which allow for the embedding of tensed propositions under a main verb, are a prerequisite of children's acquisition of a representational TOM. Thus although a main clause may be true, an embedded clause may be false [20]. Tager-Flusberg concluded that her claim for the distinction between perceptual and cognitive levels for representing mental states was consistent with other accounts of the hierarchical nature of representational systems [9].

Baron-Cohen reviewed the literature describing language and pragmatics (social use of language) in autism [5]. He described an affective theory and a cognitive theory. The affective theory proposes that the social and communicative deficits in autism are primarily affective. The theory articulated by Hobson starts from the assumption that normal infants are pre-wired to be sensitive to and comprehend another person's emotions, which are perceived 'directly' in their bodily expressions [21]. He then proposes that the development of a symbolic capacity and a conceptual role-taking ability are both directly

derived from the infant's affective relationships with others. The infant thus comes to appreciate another person's way of conceiving and seeing an object, and it is this that provides the infant with the notion of symbolic interpretation and other people's conceptual viewpoints.

Baron-Cohen points out that while Hobson's model may account for his 1986 experiment showing that autistic children have difficulty in matching facial, vocal, and gestural emotional expressions, it does not necessarily imply difficulty in understanding beliefs [5]. A second axiom of Hobson's theory is that a non-functional ability to perceive people's emotional states is an inability to abstract and symbolize, accounting for autistic children's deficits in pretend play. However, Baron-Cohen believes that the mechanism by which the development of a symbolic capacity occurs requires more clarification and empirical evidence.

The procedural deficit hypothesis (PDH) posits that grammatical impairments in the disorder, including syntax, morphology, and phonology are explained by neurocognitive deficits in the procedural memory system, whereas lexical knowledge, which depends on the declarative memory system, remains relatively spared. The PDH is, according to Walenski *et al.* implicated in the learning of new tasks [18], and in the control of long-established motor and cognitive skills, habits and other procedures such as typing, or game playing [22]. Walenski *et al.* describe the procedural system as being composed of a network of neural interconnected brain structures, particularly in the left cerebral hemisphere, encompassing frontal lobes, and basal ganglia (neostriatum) [18]. Within the frontal cortex, the supplementary motor area (SMA) and Broca's area are thought to be important. The anticipated effects in autistic spectrum disorder (ASD) depending on this system are deficits of grammar, syntax, morphology and phonology. However, because the circuits are composed of parallel and functionally segregated loops, related to a particular set of cortical/subcortical structures, deficits may be heterogeneous. The heterogeneity will relate to different pathways and also to 'direct' disinhibitory versus 'indirect' inhibitory influences from the basal ganglia.

In contrast to the procedural memory system, the declarative memory system is described as subserving long-term learning representation and the use of knowledge about facts (somatic memory). The medial temporal structures, that is, the hippocampus and parahippocampal gyrus, consolidate new memories, which eventually depend on neocortical areas, such as

the temporal lobes. The authors believe that declarative and procedural memory systems interact both cooperatively and competitively. However, dysfunction of one system can lead to enhancement of the other. Thus Walenski *et al.* posit that in ASD the declarative memory system will tend to take over certain grammatical functions from the dysfunctional procedural memory system [18]. Complex structures that are able to be composed by the procedural system (walk plus 'ed') are simply stored as chunks in lexical/declarative memory in individuals with ASD, particularly shorter higher frequency and less complex forms. When declarative memory is dysfunctional, such compensation is less available. The authors suggest that declarative memory is often but not always spared in ASD, resulting in relative sparing of lexical knowledge. However, rule-governed compositional aspects of grammar are largely abnormal in ASD. This results in dependence on the use of memorized complex representations. Levy has described the case of an autistic girl who was able to sing memorized tunes at preschool but unable to produce sentences and/or communicative language [23].

The language theory thus suggests a relative imbalance in neurophysiological aspects of language development, favouring a mechanical memory-based language system.

Central coherence theory

Frith proposed that autism is characterized by a 'specific imbalance in integration of information at different levels' [24]. She points out that a characteristic of normal information processing is the tendency to draw together diverse information to construct higher-level meaning in context, 'central coherence'. Frith proposed that central coherence is disturbed in autism, providing a parsimonious explanation of the aforementioned deficits and assets. Shah and Frith cite data from the embedded figures test, which demonstrated that autistic children picked out hidden figures more rapidly than controls [25]. The authors suggested that there was preliminary evidence that the central coherence hypothesis was a good candidate for explaining 'idiot savant' phenomena and that the central coherence hypothesis might explain executive function deficits in autism, in which inhibition of prepotent reactions was dependent on recognition of context-appropriate responses. An area for future definition was the level at which coherence is weak in autism. 'While Block Design and Embedded Figures

tests appear to tap processing characteristics at a fairly low or perceptual level, work on memory and verbal comprehension suggests higher-level coherence deficits'. The authors indicate that a way forward might be to contrast local coherence within modular systems, and global coherence across these systems in central processing. For example, they suggest that within text there may be the word-to-word effect of local association, as well as the effect of sentence context, and the longer effect of story structure [25].

Frith and Frith have pointed out that the success of human social interactions depends on the development of a 'social intelligence', whose components include knowing one's place in society, learning from others and teaching novel skills to others [26]. According to Frith and Frith, mental state attribution is demonstrated by age 4, for children, where deliberate deception is commonplace. Children orient towards persons and imitate observed actions by 18 months. However, autistic children appear not to orient towards other people's attention focus and do not engage in pretend play, or understand false belief (for example in the 'Sally-Anne' task most 4-year-olds recognize that Sally, who has not seen Anne transfer a ball from one hiding place to another, will think the ball is in its original hiding place, but this ability is lacking in autistic children).

According to Frith and Frith abilities such as recognition of subtle differences in emotional expression, recognition of other individuals, and recognition of their status and relationships developed in the monkey before the emergence of mentalizing [26]. These abilities are thought to depend on complex and sophisticated object recognition of the kind supported by the ventral system. Frith and Frith believe that the emergence of mentalizing requires the development of capabilities relating to the representation of actions and the intentions behind them [26]. While Frith and Frith relate central coherence deficits to the emergence of mentalizing ability, it is also possible that lack of central coherence relates to an underlying lack of integration of modular perceptions, as suggested by Frith [24].

Repetitive behaviour

Frith and Happe have pointed out that the mentalizing deficit theory of autism does not explain all features of autism [27], including: (i) restricted repertoire of interests [28]; (ii) obsessive desire for sameness [29]; (iii) islets of ability [30]; (iv) idiot

savant abilities [31]; (v) excellent rote memory [30]; and (vi) preoccupation with parts of objects [1].

Boucher argued that social and communicative impairment are unlikely to be completely explained by impaired meta-representation [12]. For example failure to use available language and repetitive use of language are not so easily explained in this way. Importantly, Frith and Happe state that repetitive behaviour, including restricted repertoire of interests and obsessive desire for sameness, cannot be explained in terms of impaired meta-representation [27]. Wing described a triad of impairments in autism consisting of (i) social impairment; (ii) communicative impairment; and (iii) impairment of imaginative activity, with substitution of repetitive activity [32]. The triad is similar to the DSM-IV, but significantly TOM explanations do not explain the restricted, repetitive and stereotyped patterns of behaviour in her triad (which may require a domain-general deficit to explain the control of behaviour by lower-level stereotyped routines).

The important omission of restricted repetitive behaviour patterns tends to strengthen Boucher's argument that the fundamental features of autistic disorder are not completely accounted for by impaired meta-representation or TOM. Turner noted that the importance of repetitive behaviour to the autistic syndrome has been emphasized since Kanner [30], although it was sometimes regarded as a marker of non-specific impairment [33]. A second reason for the apparent lack of interest in repetitive behaviour is the assumption that the behaviours function as a coping mechanism, and thus as a secondary feature of the disorder. However, such accounts fail to explain why repetitive behaviours are so ubiquitous, pervasive and enduring in autism.

Turner systematically investigated the various theories of repetitive behaviours in autism [33]. She first described a taxonomy of repetitive behaviours, including tics, stereotypic movements, self-injury, stereotyped manipulation of objects, abnormal object attachments and preoccupations, insistence on sameness of environment, rigid adherence to routines and rituals, repetitive use of language, circumscribed interests and obsessions and compulsions. These behaviours are characterized by (i) high frequency of repetition; (ii) the invariant way in which the behaviour or activity is pursued; and (iii) inappropriateness or oddness in its manifestation or display.

Turner compared 22 high-functioning autistic children and adults with a verbal IQ >75 and 22 learning-disabled individuals with autism and IQ <75, as well as two groups of age-, sex-, and ability-matched

controls [33]. She found that 98% of the autistic subjects displayed repetitive behaviours in three or more of the 11 classes of the aforementioned behaviours in comparison with 17% of controls, but showed little effect of age or ability on the repetitive behaviour. Turner suggested that the findings support Kanner's original assertion that repetitive behaviour is a core feature of autism [33]. Thus 'if we want to provide a full explanation of the pervasive tendency to repetitive behaviour that characterizes autism, and sets it apart from other clinical disorders, then we must look towards those hypotheses which provide a mechanism of autistic repetitions'.

Turner also investigated whether the TOM deficit is able to explain repetitive behaviour [33]. The theory predicts that levels of repetitive behaviour will be highest when the individual is in a novel or unpredictable social situation, and lowest in a highly familiar environment. However, most studies report that rates of stereotypic behaviour are lowest during periods of social interaction.

The theory of central coherence explains the performance of autistic subjects in terms of inability to integrate perceptual information, leading to a focus on seemingly insignificant details of the environment [24]. According to Turner this account is consistent with the common insistence that even minor features of the environment remain unchanged, including the narrowness of repetitive behaviour [33]. However, when Turner investigated the relationship between performance on the Children's Embedded Figures task and four domains of repetitive behaviour, she found no difference between high and low scorers on this task. Thus Turner is critical of the ability of the central coherence theory to explain the high degree of repetition and invariance characteristic of autistic behaviour.

Turner suggested that executive function, or the supervisory attentional system (SAS) is crucial to normal, flexible and adaptive regulation of behaviour [33]. Inability to inhibit actions is thought to result in perseverative behaviour. However, response perseveration does not account for inappropriate responding, although it may account for 'stuck in set'. Turner reports that those high-functioning autistic subjects who produced the lowest number of novel responses and highest number of immediate repetitions on a Sequence task, also showed the highest number of repetitive movements and extreme circumscribed interests. However, her argument appears somewhat circular in terms of low novel responses being associated with circumscribed interests. Finally Turner found that autistic subjects produced signifi-

cantly fewer responses than learning-disabled controls despite a mean IQ 40 points higher. She concluded that repetitive behaviour is associated with general disruption of a system such as the SAS, which is responsible for controlling volitional activity, but this does not explain the insistence on sameness of environment, or why repetitive behaviours are so constant across different individuals and different cultures.

Biological findings

Leckman *et al.* have suggested that consistent with emergent data from brain imaging studies, an evolutionary perspective suggests that obsessive-compulsive symptom dimensions are based on overlapping brain-based alarm systems that have the potential to become dysregulated due to genetic vulnerability, adverse environment during development, or brain injury [34]. This view is consistent with the emergence of repetitive and obsessional symptoms in conditions such as autism/Asperger's syndrome, in which social appraisal of potentially fearful stimuli is disturbed.

Belmonte *et al.* have noted that while people with autism have been described as suffering from a lack of 'central coherence', the field of autism itself suffers from a lack of integration of differing analytical and theoretical concepts, including executive function, complex information processing, TOM and empathy [35]. They point out that autism, defined and diagnosed by purely behavioural criteria, was first described and investigated using the tools of behavioural psychology, but more recent results from brain anatomy, physiology, genetics and biochemistry have not been fully integrated. The triad of deficits consisting of impaired social interaction, impaired communication, restricted interests, and repetitive behaviours are believed to be the extreme of a spectrum of abnormalities including Asperger's syndrome and the 'broader autistic phenotype'.

Face processing

Pierce *et al.* have investigated the development of face processing in autism [36]. They note that processing of the human face is the focal point of most social interactions, and that autism is one of the only disorders in which affected individuals spend reduced amounts of time engaged in face processing from birth. The investigators utilized functional magnetic resonance imaging (fMRI) haemodynamic

responses during face processing task to investigate four regions of interest: the fusiform gyrus (lateral to the parahippocampal gyrus in the temporal lobe); the inferior temporal gyrus; the medial temporal gyrus; and the amygdala in adults with autism and controls. They point out that unlike autistic subjects, 100% of normal subjects had maximal neural responsiveness to faces in the fusiform gyrus, and that two opposing explanations have been put forth to explain the invariance of the fusiform face area (FFA) activation in normals. The first interpretation suggests that this reflects an innately determined face module that is specific to and required for face processing, namely a domain-specific view. The second view posits that the FFA is an experience-dependent neural region, evolved to process subordinate levels of extremely familiar classes of objects: a domain-general view. The investigators found that while autistic subjects could perform the face perception task, none of the regions supporting face processing in normals were found to be significantly active in the subjects investigated. They found that in every autistic subject, faces maximally activated aberrant and individual-specific neural sites (e.g. frontal cortex, primary visual cortex and cerebellum).

Amygdala theory

Pierce *et al.* found decreased structural amygdala volume in adult autistic people [36]. They note that this finding is consistent with the idea that the amygdala is abnormal in autism, and that the amygdala plays a key role in establishing the social significance of a face, interpreting it as threatening or fearful, monitoring eye gaze, and assigning hedonic values to stimuli in general. The authors suggest that an absence of normal amygdala functioning would thus prevent many of the normal social perceptual activities of a newborn and young child, preventing activity-dependent development and refinement of the amygdala. Thus malfunction of the amygdala could represent an essential neural insult that initiates a cascade of social maldevelopments found in the disorder. They posit a critical period for the development of the FFA, not just for experience with faces per se, but between exemplars of a particular class of objects. The authors finally suggest that abnormal neural responding to faces in autism is probably the result of inefficient or faulty networks, extending beyond the FFA and amygdala, relating to top-down processes such as fronto-parietal networks involved in attention.

Baron-Cohen *et al.* have discussed the amygdala theory of autism [37]. They cite Brothers, who proposed a network of neural regions comprising the 'social brain' [38]. Evidence during the last two decades has shown that the amygdala is intricately connected with many brain regions, including neocortex, basal forebrain, the limbic striatum (nucleus accumbens and ventral pallidum), the neostriatal structures (the caudate nucleus and the putamen), the hippocampal formation and the claustrum. According to the authors, the amygdala receives considerable visceral inputs from olfactory input and the hypothalamus, as well as inputs from temporal and anterior cingulate cortex, while fibers leave the amygdala to reach many of the same areas that send efferents to it. Thus the amygdala is in a position to influence both drive-related behaviour and the related emotions.

Phillips *et al.* have reviewed the neurobiological basis of emotion perception in terms of three related processes [39]. These are described as (i) the identification of emotionally salient information; (ii) the production of affective states in response; and (iii) the regulation of the emotional state. Based on animal and human studies, the authors describe a ventral system, including the amygdala, insula, ventral striatum and ventral regions of the anterior cingulate gyrus and prefrontal cortex, predominantly important for processes (i) and (ii), and automatic regulation of emotional responses; and a dorsal system, including the hippocampus and dorsal regions of anterior cingulate gyrus and prefrontal cortex, predominantly important for process (iii). Thus the ventral system is important for the identification of the emotional significance of environmental stimuli, production of affective states, and automatic regulation of autonomic responses to emotional stimuli, while the dorsal system is important for executive functions, including selective attention, planning, and effortful rather than automatic regulation of affective states.

Herba discussed developmental studies of amygdala activation to fearful faces, and pointed out that with age, there is increased prefrontal and decreased subcortical activity [40]. Thus early in development, children may have difficulty labelling neutral faces, which may be interpreted as ambiguous, giving rise to amygdala activation, whereas adults show greater amygdala activation in response to fearful facial expressions. Skuse has described an important evolutionary connection between emotions, feelings and ability to interact appropriately in social situations [41]. He notes that the amygdala responds specifically

to eye contact in adults and is maximally activated by exaggerated wide-open eyes, associated with fearful expressions. Thus direct eye contact in primates can elicit an instinctive fear response of fight or flight. However, in humans, response to a stimulus that could be a threat is normally determined by a full evaluation of that stimulus by means of complex neocortical connections, allowing an appropriate social response. According to Skuse a crucial component of the modulating circuitry in humans is the recruitment of language centres, and the conscious processing of a 'feeling' response. Thus neural circuitry that evolved for the purpose of fear detection in others' faces are now associated with the development of social skills [41].

Neural connectivity

Belmonte *et al.* have attempted to integrate some of the aforementioned observations and theories of autism in terms of abnormal neural connectivity [42]. They differentiate local connectivity within neural assemblies from long-range connectivity between functional brain regions and distinguish physical connectivity associated with synapses and tracts from the computational connectivity associated with information transfer. The authors posit that in the autistic brain high local connectivity may develop in tandem with low long-range connectivity, perhaps as a consequence of widespread alterations in synapse elimination and/or formation. Furthermore, indiscriminately high physical connectivity and low computational connectivity may reinforce each other by failing to differentiate signal from noise. They describe the model as consistent not only with impairments in higher order cognition described by the autism diagnostic triad, but also impairments of motor coordination [43], and abnormal growth within regions of local, but not long-range white matter projections [44], and the substantial comorbidity with epilepsy [45].

Belmonte *et al.* utilized fMRI to investigate the aforescribed concepts in autistic subjects [42]. They postulated that in an over-connected network, sensory inputs should evoke abnormally large activation for attended and unattended stimuli alike, giving rise to an overall increase in activation but reduction in selectivity within sensory regions. Conversely 'brain regions subserving integrative functions will be cut off from their normal inputs and should therefore manifest reductions in activation and in functional correlations with sensory regions'. Thus

a combination of electroencephalogram and fMRI measures in a task of visual spatial attention demonstrated exactly this pattern in autistic subjects [46,47]. Belmonte and Baron-Cohen were able to demonstrate abnormally strong activation in parietal cortex during suppression of distractors at the same time as integrative regions in prefrontal and medial temporal cortices were abnormally quiescent [48].

Belmonte *et al.* note that because autism is a developmental disorder, primary dysfunction can be masked by the evolution of compensatory processing strategies, and by the induction of activity-dependent secondary dysfunctions that disrupt behaviour in new ways [35]. They note that a decrease in signal to noise can arise from abnormalities of neural connectivity in either direction: overconnection allows so much noise to pass that it swamps the signal, whereas an under-connected network passes so little signal that it becomes lost in noise. 'In either case, large segments of the network are constrained to either an all-on or an all-off state, and the network's information capacity is thereby reduced'. Thus a failure to delimit activation can give rise to hyperarousal in response to sensory input, a decreased ability to select among competing sensory inputs. Functional imaging has demonstrated heightened activity in autism in brain regions associated with stimulus-driven, sensory process and decreased activity in regions that normally subserve higher-order processing. Studies have shown heightened activity during face processing in peristriate cortex and inferior temporal gyrus, while fusiform activity is abnormally low [49,50]. Similarly heightened activity in the superior temporal gyrus during inference of mental state from pictures of eyes, and decreased connectivity between extrastriate visual areas and prefrontal and temporal areas were associated with inference of mental state, while prefrontal and temporal activations were again abnormally low.

Belmonte *et al.* have outlined a developmental theory of abnormalities of perceptual processing dependent on the idea of a developmental chain of abnormal function [35]. 'When a developing brain is confronted with an abnormal constraint on information processing, it will evolve an abnormal organization in order to accommodate that constraint, resulting in a succession of autistic behavioural abnormalities extending into sensory, motor and later developing cognitive functions.' 'From the earliest months of infancy, the flood of input generated by over-aroused, under-selective primary processing would overload nascent higher-order cognitive processes.' Faced with the bottleneck, Belmonte and Baron-Cohen believe that the brain would likely

evolve a cognitive style that avoids reliance on high-level integrative processing and instead emphasize low-level features [35]. This pattern of autistic perception has been characterized by Frith as weak central coherence, which results in a loss of identity of fragments once they are assembled into a single object, but can confer advantages in tasks that demand attention to detail [24]. Thus people with autism have an advantage in the Embedded Figures Test and process faces as collections of individual features rather than centrally coherent gestalts, while their rote memory is superior to normal.

According to Belmonte *et al.* weakened central coherence may be a secondary property emerging from the interaction of normal cognitive development with abnormal neural information processing, encouraging unusual cognitive dependence on low-level processing of individual details [35]. They suggest that this may impair the use of contextual information on complex perceptual and executive tasks and may impact on capacity to form a model of another person's mental state (TOM), impairing the development of joint attention and shared affect. Also failure to use context may, according to the authors, base learning on statistical associations, leading to a preference for ritualized, scripted repeatable interactions.

The work of Courchesne *et al.* on head circumference measurements suggests overgrowth and exuberant synaptogenesis and dendritic arborization within the first 6–14 months in the frontal lobes (essential for attention, social behaviour and language), but lesser effects in the precentral gyrus and orbital cortex [51]. Thus 'the cortical areas most affected are precisely those broadly projecting, phylogenetically and ontogenetically late-developing regions that are essential to complex cognitive functions such as attention, social behaviour, and language'.

Conclusion

While theories of language, mind, central coherence and meta-representation all describe features of autism, they fail to explain the central features of repetitive behaviour, including restricted repertoire of interests and obsessive desire for sameness. These features may be best explained by a developmental failure of neural connectivity in which high local connectivity develops in tandem with low long-range connectivity, perhaps as a consequence of widespread alterations in synapse elimination and/or formation. This gives rise to a localized modular style of information processing that may explain both

the psychological features and obsessive behaviour observed in autistic syndromes.

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