The role of emotional processes in the development of autism spectrum disorder

Sebastian B Gaigg

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Department of Psychology,

City University, London, UK

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PRELIMINARY NOTE

The research presented in this thesis includes four experiments that have either been published or are currently under review. These studies have been incorporated here in relatively unaltered form, which raises several issues that readers need to be aware of. First, the order in which the experiments are presented here does not correspond with the order in which the studies were submitted for publication. As a result some of the experimental chapters refer to work that is presented only at later stages of the thesis. In relation to this point, it is also important to acknowledge that the studies that have been published (or are under review) do not refer to any work that appeared in the literature after the manuscripts were accepted for publication. Thus, studies are mentioned in the Literature Review (Chapter 1) which, despite being relevant, are not acknowledged in some of the empirical chapters. A second point concerns the use of pronouns. Since the published and submitted manuscripts are all co-authored by my supervisor Prof. Dermot Bowler, the relevant sections in this thesis make use of the pronoun 'we' rather than 'I', whilst the latter is used in the remainder of this thesis.

ABSTRACT

The current thesis presents five experiments which suggest that Autism Spectrum Disorders (ASD) are characterised by abnormalities in the processes necessary for the acquisition of emotionally distinct representations of environmental stimuli. In the context of a long standing theoretical debate about the role of emotional disturbances in the development of ASD, this evidence supports the notion that abnormalities in emotional rather than socio-cognitive development lie at the root of the disorder. The findings also provide indirect support for the idea that abnormalities in amygdala functioning may play a central role in the neuropathology underlying ASD, since this limbic structure is well known to be involved in the mediation of emotional learning processes. Based on the evidence presented in this thesis and in the relevant literature, I develop the argument that developmental abnormalities in basic emotional learning processes may not only explain the socio-emotional disturbances characterising the disorder but also account for some of the non-social manifestations of the ASD phenotype.

CHAPTER 1: INTRODUCTION

Overview

Ever since Autism Spectrum Disorders (ASD) were first described more than six decades ago, atypicalities in affective behaviours have been regarded as hallmark features of their clinical manifestations. Both Kanner (1943) and Asperger (1944) concluded their seminal case descriptions with the suggestion that disturbances of affective development constituted an important element underlying the atypical social behaviour characterising the disorder and to this date abnormalities in emotional conduct continue to be considered diagnostically sensitive manifestations of ASD. At the neural level, many authors now agree that abnormalities in the functional integrity of the amygdala may be responsible for this facet of the disorder (e.g. Bachevalier & Loveland, 2006; Baron-Cohen, Ring, Bullmore, Wheelwright, Ashwin & Williams, 2000; Schultz, 2005) but in developmental terms the role of affective disturbances in the ontogenesis of ASD remains the matter of debate. More specifically, some authors continue to share the views of Kanner (1943) and Asperger (1944) in suggesting that abnormalities in emotional development lie at the heart of the condition (e.g. Hobson, 1993, 2002; Loveland, 2005, Mundy & Sigman, 1989). Others, by contrast, have adopted the view that abnormal emotional behaviours merely represent a consequence of disturbances in the development of socio-cognitive capacities (e.g. Baron-Cohen, 1995; Baron-Cohen, et al., 2000; Frith, 2003; Leslie & Frith, 1990).

The principal aim of the work presented in the subsequent chapters is to contribute to the formulation of a developmentally and neurologically plausible framework regarding the role of affective disturbances in ASD. To make such a contribution ultimately requires a consideration of how affective processes shape typical development and to what extent disturbances of these processes may give rise to the particular developmental trajectory that characterises ASD. Hobson (1993, 2002) offers a useful framework in this context. He argues that various facets of human cognition are anchored in the affectively patterned interpersonal relations that infants enjoy from the minute they are born. ASD, so this argument goes, is the

consequence of disturbances in such relatedness. The current thesis attempts to elaborate on Hobson's views by further specifying the cognitive and neural mechanisms that may underlie the ability to affectively relate to others. I will ultimately come to the conclusion that considering ASD as a disorder of these mechanisms holds far more promise for our endeavours to understand the disorder than nativist alternatives that view ASD as the result of dysfunctional socio-cognitive 'modules' (e.g. Baron-Cohen, 1995; Frith, 2003). Before I justify this position, however, it is important to review and furnish the empirical basis on which my arguments will rest.

Literature Review

In order to provide the relevant context for the subsequent chapters, the literature reviewed below will centre around the following issues. In the first section, a summary of the evidence pertaining to the emotional disturbances in ASD will demonstrate that it is now widely accepted that this spectrum of disorders is characterised by often very severe limitations in various aspects of affectively patterned communication. The second section will show that whilst most authors agree that abnormalities in the functioning of the amygdala most probably underlie this facet of the disorder, the developmental significance of abnormal emotional processes remains the matter of debate. More specifically, I will demonstrate that the currently available evidence can be accommodated within competing theoretical frameworks that consider abnormalities in emotional processes either as a cause for, or consequence of, the developmental trajectory of ASD. The third section will provide a closer examination of how the concept of emotion is currently operationalised and how current frameworks regarding the constitutional components of affective behaviours and processes may be exploited for the study of ASD. This section will focus on the longstanding consensus that emotional processes are the result of the interplay between primitive autonomic responses and cognition. Since this interplay is known to involve neural processes that are mediated by the amygdala and because the amygdala is widely acknowledged to play an important role in the neuropathology associated with ASD, I will argue that the distinction between autonomic and cognitive emotional processes offers a useful heuristic device for the

further exploration of the nature of emotional disturbances in this disorder. In the final section I will recapitulate some of the earlier points in order to formulate the principal hypothesis of the current thesis, namely that autistic atypicalities in emotional conduct are the result of abnormalities in how the amygdala mediates the integration of physiological and cognitive aspects of emotional experiences. A brief summary of research pertaining to the influence of stimulus induced arousal on learning, memory and perception will show how this hypothesis can be tested empirically thus setting the scene for the empirical chapters of this thesis. Before I turn to these issues, however, it is necessary to provide a more thorough description of what the autism spectrum actually is.

What is the Autism Spectrum?

The *Autism Spectrum* describes a set of related pervasive developmental disorders that all share a triad of impairments in the domains of communication, reciprocal social behaviour and imagination in the presence of a restricted and sometimes obsessive pattern of interests and activities (e.g. Wing & Gould, 1979). The disorders subsumed under this spectrum include *Autistic Disorder*, *Asperger's Disorder*, and *Pervasive Developmental Disorder Not Otherwise Specified* (PDD-NOS), which together affect approximately 1% of the population and are around three to four times more common in males than females (Baird, Simonoff, Pickles, Chandler, Loucas, Meldrum et al., 2006; Bertrand, Mars, Boyle, Bove, Yeargin-Allsopp & Decoufle, 2001). Current diagnostic criteria (DSM-IV-TR; American Psychiatric Association, 2000) specify that a necessary feature of all three disorders is a severe impairment in the development of reciprocal social behaviour, which includes abnormalities in developing peer relations, deploying non-verbal communicative acts to modulate social interaction (e.g. eye-contact, gestures, facial expressions, etc...), mastering pragmatic aspects of language (i.e. modulating language use according to the social context and the listener's needs) and developing joint attentional behaviours (e.g. showing, pointing, bringing, following gaze, etc...). Autistic Disorder and Asperger's Disorder furthermore share an early onset of symptoms (i.e. before age 3 years) and the presence of repetitive and stereotyped patterns of behaviour such as motor stereotypies (e.g. finger-flicking, rocking,

etc...), preoccupations with highly specific topics (e.g. acquiring an encyclopaedic knowledge of high-voltage power cables) and an inflexible adherence to non-functional routines and rituals (e.g. arranging food in a certain manner prior to eating it). What distinguishes these two disorders are language and general cognitive development, both of which are significantly compromised in Autistic Disorder but not Asperger's Disorder. The classification of PDD-NOS is used for individuals who present with the characteristic reciprocal social impairment but do not meet the full criteria for a specific pervasive developmental disorder, either because of sub-threshold manifestations or late onset of symptoms.

In addition to the clinically defining symptoms set out above, all subtypes of ASD also share a uniquely patterned cognitive profile, which includes difficulties in understanding behaviour in terms of mental states, such as beliefs and desires (e.g. Baron-Cohen, 1995; Baron-Cohen, Leslie & Frith, 1985; Frith, 2003), difficulties in deploying cognitive resources flexibly in order to plan and execute goal directed behaviours (e.g. Hill, 2004; Ozonoff, Pennington & Rogers, 1991a), and a tendency to process information in a piecemeal and perceptually driven rather than holistic and conceptually driven fashion (e.g. Frith, 2003; Mottron & Burack, 2001; Shah & Frith, 1993). The existence of this characteristic *endophenotype* (i.e. the heritable characteristics of a condition that are not necessarily direct symptoms) together with the absence of any consistent evidence supporting a differentiation of subtypes of autistic pathologies has led to the widely held view that all three disorders described above represent different instances of the same underlying syndrome (see Bowler, 2007; Prior, Eisenmajer, Leekam, Wing, Gould & Ong, 1998; Volkmar, Lord, Bailey, Schultz & Klin, 2004; Wing ,1993 for recent reviews; see also Wing & Gould, 1979). To reflect this shift in the conceptualisation of the disorder the current thesis will adopt the term *'Autism Spectrum Disorder'* (ASD) to cover all conditions and use the word *'autistic'* to describe the characteristics that defines them. If distinctions are necessary, only those provided by the current diagnostic classification system (see above) will be employed. Other terminologies such as *'Low-Functioning Autism'* (LFA) and *'High-Functioning Autism'* (HFA) will be avoided[1].

[1] This terminology originally distinguished between individuals whose intellectual abilities fell within the 'extremely low' range (i.e. IQ < 70) and those whose abilities fell above that. The spectrum view of ASD, now encompasses individuals with truly superior abilities (i.e. IQ > 130) rendering the term 'High-Functioning' nothing but misleading.

There is one more aspect of ASD worth noting before concluding the preliminaries and that is the extreme heterogeneity with which the syndrome manifests. In the domain of reciprocal social behaviour, for example, we find individuals who seem entirely oblivious to the existence of other people at one end of the spectrum, whilst at the other end we find those who make at least some attempts (even if awkward) to engage socially (Wing & Gould, 1979). Similarly, the domain of language development includes individuals who never acquire any linguistic competence at all and those who publish academic books or insightful autobiographies (e.g. Grandin, 2006; Mukhopadhyay, 2007). And the realm of repetitive and stereotyped behaviours ranges from manifestations such as the spinning of the wheels of toy-cars for hours on end to an almost obsessive acquisition of encyclopaedic knowledge about specific topics. No matter what domain or cognitive ability one focuses on, the autistic spectrum encompasses individuals who exhibit marked abnormalities and those who do not. In part this heterogeneity most likely reflects the underlying variability in the genetic, neurological and biochemical factors associated with the disorder (for reviews see Bachevalier & Loveland, 2006; Tuchman & Rapin, 2006; Schultz & Anderson, 2004). But only in part. A considerable proportion of the disorder's symptom inconsistency seems to be the result of the idiosyncratic way in which affected individuals acquire knowledge and organise their experiences (see Bowler, Gaigg & Gardiner, 2008a). This idiosyncrasy needs to be explained in developmental terms and an explanation in terms of emotional processes has much to offer in this respect. But more on this later. First, let me consider the evidence pertaining to the affective abnormalities characterising the autistic spectrum.

Autistic disturbances in affective conduct

When Kanner (1943) published his landmark clinical accounts of the 11 children who came to be considered the first officially identified cases of ASD he was not only the first to systematically describe the disorder but also to note that affective disturbances constituted a core feature underlying the condition. Kanner eloquently emphasised this point in the final paragraph of his publication where he wrote:

> "We must then assume that these children come into the world with innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical or intellectual handicaps. If this assumption is correct, a further study of our children may help to furnish concrete criteria regarding the still diffuse notions about the constitutional components of emotional reactivity. For here we seem to have pure-culture examples of *inborn autistic disturbances of affective contact.*" (p.250; italics appear as in original)

Unfortunately it was to take several years before Kanner's insights were fully appreciated. During the two decades immediately following his publication the influential psychodynamic approach to human behaviour identified early childhood experiences as the origin of emotional disturbances and so gave rise to the unfortunate idea that autistic abnormalities were the result of pathological parenting (e.g. Bettelheim, 1967). During the 1960s this erroneous view was finally abandoned (Rimland, 1964; cited in Dawson, 1989) and the focus returned to Kanner's original proposal that ASD was a biologically determined developmental disorder. Laboratory and field based experiments (e.g. Hermelin & O'Connor, 1967; O'Connor & Hermelin, 1967; Hermelin & O'Connor, 1970; Bartak, Rutter & Cox, 1975) as well as meticulous epidemiological studies (e.g. Rutter, 1968; Wing & Gould, 1979) replaced the psychoanalytic approach of the previous years and in 1980 ASD was finally recognised as a distinct developmental disorder and added, under the label of *'infantile autism'*, to the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; APA, 1980). From this point onwards research into the causes of autistic pathologies increased at an exponential rate and it was not long before Kanner's emphasis on affective disturbances found support in experimental studies.

Amongst the earliest investigations to address the affective capacities of individuals with ASD were a series of studies by Hobson and his colleagues who showed that children with ASD were limited in understanding the emotional expressions of others. Hobson (1986a, 1986b) and Hobson, Ouston and Lee (1988a), for example, showed that compared to children with moderate learning difficulties and typically developing children, those with a diagnosis of ASD experienced unusual difficulties in matching emotional expressions across different modalities. More specifically, these authors found that whilst children with and without ASD performed similarly on tasks in which drawings of objects (e.g. a bird, train) had to be paired with sound recordings or blurred video sequences of the same objects, children

with ASD performed significantly worse on matching facial, vocal and gestural expressions of emotions with one another. Subsequent studies suggested that this difficulty reflected a fundamental difference in how children with ASD actually perceived emotional expressions in the first place. Weeks and Hobson (1987), for instance, asked children simply to sort a series of photographs of faces into different groups. The photographs varied on several dimensions including the age and gender of the person depicted, the type of hat worn by that person and the facial expression of emotion portrayed. Whilst non-autistic children readily opted to sort these photographs according to the emotional dimension, children with ASD were more inclined to sort them according to one of the other features, suggesting that emotional expressions were not particularly salient to them. Hobson, Ouston and Lee (1988b) furthermore showed that even when children with ASD did sort faces according to their emotional expressions, they seemed to deploy qualitatively deviant perceptual processes in order to do so. In this rather complicated experiment, children were again asked to sort photographs of faces into different groups but this time they were explicitly instructed either to sort the photographs according to the identity of the individuals or the emotional expressions they portrayed. The photographs were either of whole faces, of faces with the mouth area blanked out, of faces with both the mouth and forehead areas blanked out or of whole faces presented upside down. The results revealed that with increasing degradation of the upright face photographs, performance in sorting according to identity steadily declined by a similar margin in both groups of children. When the photographs had to be sorted according to the emotional expressions, however, children with ASD exhibited a more marked decline in their performance. This, together with the observation that children with ASD were better than the comparison children at sorting upside down faces according to their emotional expressions, led the authors to suggest that children with ASD may rely on qualitatively different processes in order to identify expressions of emotion, a conclusion I will return to in more detail shortly.

Since the studies by Hobson and colleagues more than 40 investigations relevant to the emotion perception abilities of individuals with ASD have appeared in the literature. Some of these have failed to support the initial conclusions (Adolphs, Sears & Piven, 2001; Ashwin, Wheelwright & Baron-Cohen, 2006; Castelli, 2005; Hillier & Allinson, 2002; Loveland, Tunali-Kotoski, Chen, Ortegon, Pearson, Brelsford et al., 1997; Ozonoff, Pennington & Rogers,

1990; Prior, Dahlstrom & Squires, 1990; Serra, Minderaa, van Geert, & Jackson, 1999), but the vast majority of studies leaves little doubt that individuals with ASD perceive emotional expressions differently and often not as readily as comparison groups (Ashwin, Baron-Cohen, Wheelwright, O'Riordan & Bullmore, 2007; Ashwin, Chapman, Colle & Baron-Cohen, 2006; Begeer, Rieffe, Terwogt & Stockmann, 2006; Bormann-Kischkel, Vilsmeier & Baude, 1995; Braverman, Fein, Lucci & Waterhouse, 1989; Bölte & Poustka, 2003; Buitelaar, van der Wees, Swaab-Barneveld & van der Gaag, 1999; Capps, Yirmiya & Sigman, 1992; Celani, Battachi & Arcidiacono, 1999; Critchley, Daly, Bullmore, Williams, van Amelsvoort, Robertson, et al., 2000; Da Fonseca, Santos, Bastard-Rosset, Rondan, Poinso, & Deruelle, 2008; Davis, Bishop, Manstead & Tantam, 1994; Deruelle, Rondan, Gepner & Tardif, 2004; Dennis, Lockyer & Lazenby, 2000; Dziobek, Rogers, Fleck, Bahnemann, Heekeren, Wolf, et al., 2008; Fein, Lucci, Braverman & Waterhouse, 1992; Golan, Baron-Cohen & Hill, 2006; Gross, 2004; Gross, 2005; Grossman, Klin, Carter & Volkmar, 2000; Hubert, Wicker, Moore, Monfardine, Duverger, Da Fonséca, et al., 2007; Kamio, Wolf & Fein, 2006; Lindner & Rosén, 2006; Losh & Capps, 2003; Moore, Hobson & Lee, 1997; Macdonald, Rutter, Howlin, Rios, Le Couteur, Evered et al., 1989; Njiokiktjien, Vershoor, de Sonneville, Huyser, Op het Veld & Toorenaar, 2001; Ozonoff, et al., 1991a; Ozonoff, Rogers & Pennington, 1991b; Pelphrey, Sasson, Reznick, Paul, Goldman & Piven, 2002; Piggot, Kwon, Mobbs, Blasey, Lotspeich, Menon, et al., 2004; Rosset, Rondan, Da Fonséca, Santos, Assouline & Deruelle, 2008; Smalley & Asarnow, 1990; Tantam, Monaghan, Nicholson & Stirling, 1989; Teunisse & de Gelder, 2001; Yirmiya, Sigman, Kasari & Mundy, 1992). Table 1.1 below provides a brief summary of this literature and sets out information regarding the participant groups, experimental paradigms and emotional expressions studied in each of the relevant publications. First, Table 1.1 lists reports that have failed to show ASD-specific difficulties in the emotion perception domain (i.e. no difference between ASD and comparison groups). Next are listed studies that suggest that individuals with ASD experience difficulties not only with the perception of emotional expressions but also with non-emotional properties of complex visual stimuli, whilst the remaining entries concern reports that have demonstrated either quantitative or qualitative differences between ASD and comparison participants that seem relatively specific to the perception of emotional expressions.

Table 1.1

Summary of studies of emotion perception abilities in ASD

Reference	Participant Groups	*N*	Matching	Paradigm	Emotional expressions studied						
					Happy	Sad	Anger	Fear	Surprise	Disgust	Other
Studies failing to note *ASD-specific* atypicalities											
Braverman et al. (1989) Comparison 1	ASD/PDD; Typical	15; 15	VA	Match facial expressions with one another and identify facial expressions	x	x	x	x			
Ozonoff et al. (1990) Experiment 1	ASD; Typical	14; 14	VA	Sort photographs of faces according to emotional expression; Match facial expressions with one another, with vocalisations and with situational contexts	x	x	x				
Prior et al. (1990)	ASD; Typical; Dev. Delay	20; 20	VA	Match schematic facial expressions with vocalisations, gestures and contexts	x	x	x	x			
Loveland et al. (1997)	ASD; Typical; Dev. Delay	35; 23; 18	VA	Identify expressions from dynamic verbal and non-verbal signals	x	x	x		x		
Serra et al. (1999)	PDD-NOS; Typical	31; 31	CA; FSA	Explain how situational contexts influence a protagonist's actual and displayed emotions	Not specified						
Buitelaar et al. (1999)	ASD; Typical; Dev. Delay	40; 20; 20	CA; FSA, VA, NVA	Match facial expressions with one another and with situational contexts	x	x	x	x	x	x	x
Adolphs et al. (2001) Experiment 1	ASD; Typical; Amygdala lesion	6; 28; 3	FSA	Discriminate facial expressions of various intensities	x	x	x	x	x	x	
Adolphs et al. (2001) Experiment 2	ASD; Typical; Amygdala lesion	7, 18, 8	Not specified	Identify facial expressions	x	x	x	x	x	x	
Hillier & Allison (2002)	ASD; Typical; Dev. Delay	10; 20; 10	CA; VA, NVA	Influence of audience on judgements of embarrassment of a protagonist							x
Castelli (2005)	ASD; Typical	20; 20	VA	Match facial expressions with one another and identify facial expressions	x	x	x	x	x	x	
Ashwin et al. (2006)	ASD; Typical	18; 18	CA; FSA	Visual search for schematic facial expressions	x		x				
Begeer et al. (2006) Test 2	ASD; Typical	28; 32	CA	Select photographs of faces according to what person is most likely to offer a sweet or tell someone off	x		x				

Table 1.1 Continued

Reference	Participant Groups	*N*	Matching	Paradigm	Emotional expressions studied						
					Happy	Sad	Anger	Fear	Surprise	Disgust	Other
Studies failing to note *emotion-specific* difficulties in ASD											
Tantam et al. (1989) Experiment 1	ASD; Dev. Delay	10; 10	CA; NVA	Identify mismatching facial expressions	x	x	x	x	x	x	
Ozonoff et al. (1990): Experiment 2	ASD; Typical	14; 14	NVA	Match facial expressions with one another, with vocalisations and with situational contexts	x	x	x				
Davis et al. (1994) Experiment 1	ASD; Typical; Dev. Delay	20; 10; 10	CA; VA, NVA	Match face photographs varying on emotional and non-emotional dimensions according to a sample match		x	x		x		
Davis et al. (1994) Experiment 2	ASD; Typical; Dev. Delay	19; 11; 20	CA; VA, NVA	Match facial expressions with one another	x	x	x		x		
Moore et al. (1997)[2]	ASD; Typical; Dev. Delay	13; 13	CA; VA	Describe point-light displays of people enacting emotional and non-emotional behaviours	x	x	x	x	x		
Njiokiktjien et al. (2001)	ASD	3		Match facial expressions with one another and identify facial expressions	x	x	x	x			
Deruelle et al. (2004)	ASD; Typical	11; 22	CA; VA	Match facial expressions with one another	x				x	x	
Studies demonstrating specific emotion perception difficulties in ASD											
Hobson (1986a)	ASD; Typical; Dev. Delay	23; 38; 11	CA; VA, NVA	Match schematic facial expressions with gestures, vocalisations and situational contexts	x	x	x	x			
Hobson (1986b)	ASD; Dev. Delay	13; 13	CA; NVA	Match schematic drawings of gestures with videos of vocalisations and facial expressions	x	x	x	x			
Weeks & Hobson (1987)	ASD; Dev. Delay	15; 15	CA; VA	Sort face photographs varying on emotional and non-emotional dimensions according to preference	x	x					

[2] It is worth noting that in this study emotion perception difficulties in ASD were specific in relation to non-emotional actions (e.g. lifting) but not non-emotional body states (e.g. itchy)

Table 1.1 Continued

Reference	Participant Groups	*N*	Matching	Paradigm	Emotional expressions studied						
					Happy	Sad	Anger	Fear	Surprise	Disgust	Other
Studies demonstrating specific emotion perception difficulties in ASD											
Hobson et al. (1988a)	ASD; Dev. Delay	21; 21	CA; VA	Match vocal expressions with facial expressions	x	x	x	x	x	x	
Hobson et al. (1988b) Experiment 1	ASD; Dev. Delay	17; 17	CA; VA	Sort whole or partial photographs of facial expressions according to emotion	x	x	x	x			
Hobson et al. (1988b) Experiment 2	ASD; Dev. Delay	17; 17	CA; VA	Sort upright and inverted facial expressions according emotion or identity	x	x	x	x			
Braverman et al. (1989): Comparison 2	ASD/PDD; Typical	15; 15	NVA	Match facial expressions with one another and identify facial expressions	x	x	x	x			
Macdonald et al. (1989)	ASD; Typical	10; 10	CA; NVA	Identify emotion from situational contexts and vocal recordings	x	x	x	x			
Tantam et al. (1989) Experiment 2	ASD; Dev. Delay	10; 10	CA; NVA	Identify upright and inverted facial expressions	x	x	x	x	x	x	
Smalley & Asarnow (1990)	ASD; Typical	9; 9	CA; NVA	Match facial expressions with one another and identify facial expressions	Not specified						
Ozonoff et al. (1991a)	ASD; Dev. Delay	20; 20	CA; FSA, VA, NVA	Match facial expressions with one another	x	x	x	x	x	x	x
Ozonoff et al. (1991b)	ASD; Dev. Delay	23; 20	CA; FSA, VA, NVA	Match facial expressions with one another	x	x	x	x	x	x	x
Capps et al. (1992)	ASD; Typical	18; 14	CA; FSA, VA, NVA	Describe personal experiences of certain emotions	x	x					x
Fein et al. (1992)	ASD/PDD; Typical	15; 30	VA, NVA	Match facial expressions with situational contexts	x	x	x	x			
Yirmiya et al. (1992)	ASD; Typical	18; 14	CA; FSA, VA, NVA	Label emotion experienced by protagonists in video segments and report own emotional reaction to it	x	x	x	x			x

Table 1.1 Continued

Reference	Participant Groups	*N*	Matching	Paradigm	Emotional expressions studied						
					Happy	Sad	Anger	Fear	Surprise	Disgust	Other
Studies demonstrating specific emotion perception difficulties in ASD											
Baron-Cohen et al. (1993)	ASD; Typical, Dev. Delay	15; 15; 12	CA; VA	Match shematic and photographed facial expressions with one another	x	x			x		
Bormann-Kischkel et al. (1995)	ASD; Dev. Delay	41; 41	CA; NVA	Identify facial expressions	x	x	x	x	x	x	x
Celani et al. (1999)	ASD; Down; Typical	10; 10; 10	CA; VA	Match facial expressions with one another and select preferred expression	x	x					
Critchley et al. (2000)	ASD; Typical	9; 9	CA; FSA	Distinguish between emotionally expressive and neutral facial expressions	x		x				
Dennis et al. (2000)	ASD; Typical	8; 8	CA; VA	Identify the emotional expression of a story character's actual feeling and the emotion that would be expressed for the purpose of deception	x	x					
Grossman et al. (2000)	ASD; Typical	13; 13	CA; FSA, VA	Label facial expressions accompanied by no, congruent or incongruent verbal labels	x	x	x	x	x		
Teunisse & deGelder (2001)	ASD; Typical	17; 48	not specified	Match facial expressions with one another and identify facial expressions	x	x	x	x			
Pelphrey et al. (2002)	ASD; Typical	5; 5	CA	Identify facial expressions	x	x	x	x	x	x	
Bölte & Poustka (2003)	ASD; Typical; Schizophrenia	35; 22; 21	NVA	Label facial expressions	x	x	x	x	x	x	
Losh & Capps (2003)	ASD; Typical	28; 22	CA; VA	Label emotion experienced by protagonist in video segments and define emotions through verbal descriptions	x	x	x	x	x	x	x
Gross (2004) Experiment 1	ASD; Language Delay; Dev. Delay; Clinical	27; 28; 26; 27	CA; FSA	Identify facial expressions of humans, orangutans and canines	x	x	x		x		
Gross (2004) Experiment 2	ASD; Language Delay; Dev. Delay; Clinical	18; 9; 12; 9	CA; FSA	Identify facial expressions from whole, top-half or bottom-half of human, orangutan or canine faces	x	x	x		x		
Piggot et al. (2004)	ASD; Typical	14; 10	CA; FSA, VA, NVA	Match facial expressions with one another and label facial expressions			x	x	x		

Table 1.1 Continued

Reference	Participant Groups	*N*	Matching	Paradigm	Emotional expressions studied						
Studies demonstrating specific emotion perception difficulties in ASD					Happy	Sad	Anger	Fear	Surprise	Disgust	Other
Gross (2005)	ASD; Language Delay; Dev. Delay; Clinical	24; 20; 18; 21	CA; FSA	Match facial expressions of humans or canines with one another	x	x	x				
Ashwin et al. (2006)	ASD; Typical	26; 26	CA; FSA, VA	Identify facial expressions	x	x	x	x	x	x	
Begeer et al. (2006) Test 1	ASD; Typical	28; 31	CA	Match face photographs varying on emotional and non-emotional dimensions according to preference	x		x				
Golan et al. (2006)	ASD; Typical	21; 17	CA; FSA, VA, NVA	Label facial expressions and vocalisations	x	x	x	x	x	x	x
Kamio et al. (2006)	ASD; Typical	18; 18	CA; FSA	Rate likeability of japanese ideographs preceeded by subliminal or supraliminal facial expressions	x			x			
Lindner & Rosén (2006)	ASD; Typical	14; 16	CA; VA	Match facial expressions with static or dynamic facial expressions and with vocalisations	x	x	x				
Ashwin et al. (2007)	ASD; Typical	13; 13	CA; FSA	Label facial expressions		x	x	x	x	x	
Hubert et al. (2007)	ASD; Typical	19; 19	CA	Describe point-light displays of people enacting emotional and non-emotional behaviours	x	x	x	x	x		
Dziobek et al. (2008)	ASD; Typical	17; 18	CA; FSA	Label emotion experienced by protagonist in photographic scene and report own emotional reaction to it	Not specified						
Rosset et al. (2008)	ASD; Typical	20; 40	CA; FSA	Categorise emotional expressions of upright and inverted cartoon and human faces	x	x	x				
Da Fonseca et al. (2008)	ASD; Typical	19; 19	CA	Describe point-light displays of people enacting emotional and non-emotional behaviours	x	x	x	x	x		

Note: CA (Chronological Age), VA (Verbal Ability), NVA (Nonverbal Ability), FSA (Full-scale Ability).

The obvious point of Table 1.1 is that emotion perception difficulties in ASD are now well replicated. The Table also serves to highlight methodological difficulties that are inherent to the study of ASD, however, and it is worth dwelling on these for a moment. Inspection of Table 1.1 reveals that many of the earliest studies that failed to observe ASD specific difficulties in the perception of emotional expressions, involved comparisons of individuals with and without ASD who were matched on measures of verbal ability. During the 1990s, this pattern led some authors to suggest that the emotion perception difficulties associated with ASD may not be a specific manifestation of the disorder but rather a more general reflection of abnormalities in language development (e.g. Prior et al., 1990; Ozonoff et al., 1990). Although many subsequent studies demonstrated emotion perception difficulties in ASD even when participant groups were closely matched on verbal ability, the observation that performance on emotion perception tasks often correlates with performance on verbal ability tasks (e.g. Hobson, 1986b; Ozonoff, et al., 1990; Prior, et al., 1990; Smalley & Asarnow, 1990; Yirmiya, et al., 1992) lends some support to the 'language' hypothesis. More specifically, this association allows for the possibility that atypicalities in language development disproportionately alter the development of emotion perception abilities, such that, in some instances, individuals with ASD exhibit difficulties in the latter domain even when compared to language ability matched comparison groups. Whilst such an interpretation is to some extent legitimate, it is important to note that associations of the kind just described do not permit inferences of causality. That is, it would be a fallacy to conclude that abnormal language development is responsible for the atypical emotion perception abilities characterising ASD. Associations between domains reveal nothing about their causal relation in development and, in fact, do not even specify the ability or process that mediates the association. The point here is that the observation that individuals with ASD sometimes do not exhibit difficulties on emotion perception tasks *vis-à-vis* verbal ability tasks (or verbal ability matched comparison groups) does not preclude the possibility that atypical emotional processes are causally involved in the development of the disorder. In fact, one needs to consider this observation in relation to the finding that emotion perception difficulties in ASD are almost always evident when non-verbal ability tasks serve as the basis for comparison. This pattern suggests that verbal and non-verbal abilities relate to emotion perception abilities

in a qualitatively different way when an individual develops ASD rather than typically and this supports rather than contradicts the suggestion that there is something peculiar about the emotion perception abilities associated with ASD (as much as it confirms that there is something peculiar about the verbal and non-verbal abilities associated with the disorder).

The issue discussed above raises the important question of how to best match groups of participants in order to determine whether *specific* emotion perception difficulties exist in ASD. Answering this question is notoriously difficult and several authors have discussed this topic at length (see Bowler, 2007; Burack, Iarocci, Flanagan & Bowler, 2004; Hobson, 1991a; Mottron, 2004). The bottom line of these discussions is that one must not draw premature conclusions from a single pattern of observations but rather consider the available evidence in its' entirety. In addition, one must not place too much emphasis on comparing groups of participants with one another but also to compare how individuals within a group perform on one *vis-à-vis* another task and how individuals go about trying to solve the problem posed by the experimental tasks. Table 1.1 lists several studies that have demonstrated that individuals with ASD sometimes experience difficulties not only on tasks revolving around emotion perception but also on tasks relying on non-emotional perceptual abilities. This pattern has given rise to a 'socio-perceptual' theory of ASD that I will describe shortly. For the moment it is worth repeating that concurrent difficulties on tasks assessing different but overlapping abilities does not reveal anything about the causal relation between these abilities in development and that observations of non-specific emotion perception difficulties need to be considered in relation to demonstrations where such specificity is evident. The studies by Hobson and colleagues, outlined earlier, were chosen as examples for illustrating that emotion perception difficulties do exist in ASD, because they exemplify the kind of variable methodological approach that is needed in order to probe this domain of functioning effectively. These studies either attempted to employ carefully designed control tasks that aimed to rule out the possibility that general task demands, rather than emotion specific difficulties, could account for the diminished performance of individuals with ASD (e.g. Hobson, 1986a,b; Hobson, et al., 1988a). Or they created situations that allowed the authors to determine whether such individuals have the same natural tendency as non-autistic individuals to attend to, and act upon, the emotional expressions of others (e.g.

Weeks & Hobson, 1987). Because these studies, and many subsequent ones, suggest that individuals with ASD experience often disproportionate difficulties in their perception of emotional expressions, and that such individuals deploy qualitatively different processes in order to 'decode' certain configurations of facial features as emotional, it is now widely accepted that the disorder is associated with limitations in the perception of affect.

What further strengthens the foregoing conclusion is the fact that virtually all studies to date that have assessed the emotional competences of individuals with ASD within naturalistic settings have documented abnormalities[3]. In fact most available diagnostic instruments and screening tools (Lord, Rutter, Goode, Heemsbergen, Jordan, Mawhood et al., 1989; Lord, Rutter & Le Couteur, 1994; Robins, Fein, Barton & Green, 2001; Schopler, Reichler, DeVellis & Daly, 1980) recognise abnormalities in reciprocal affective behaviours as a diagnostically sensitive manifestation of the disorder. It is consistently reported that individuals with ASD are less likely than non-autistic individuals to direct emotional expressions at others during naturalistic interactions (Bieberich & Morgan, 1998; Dawson, Hill, Spence, Galpert & Watson, 1990; Dissanayake, Sigman & Kasari, 1996; Joseph & Tager-Flusberg, 1997; Kasari, Sigman, Baumgartner & Stipek, 1993; Kasari, Sigman, Mundy & Yirmiya, 1990; Macdonald et al., 1989; Mundy & Sigman, 1989; Sigman, Kasari, Kwon & Yirmiya, 1992; Snow, Herzig & Shapiro, 1987; Yirmiya, Kasari, Sigman & Mundy, 1989; Zwaigenbaum, Bryson, Rogers, Roberts, Brian & Szatmari, 2005) and Macdonald et al. (1989) found that individuals with ASD experience difficulties in expressing emotions that would typically be elicited by different situations (e.g. receiving a present). In addition, individuals with ASD tend not to automatically mimic the facial expressions of others even though they can do so when explicitly instructed (McIntosh, Reichmann-Decker, Winkielman & Wilbarger, 2006). What the association between affect perception and affect expression reminds us of, is that emotional expressions ultimately serve an important communicative function. Just as children who are born deaf experience difficulties in learning how to speak, so children with ASD seem to have problems in learning how to modulate reciprocal social interchanges through the use of emotional expressions. The real-life consequences of this

[3] To the best of my knowledge no studies actually report typical emotional conduct in naturalistic observations of individuals with ASD.

communicative limitation are nowhere as elegantly exposed as in the studies by Kasari and Sigman and their colleagues who observed children's reactions to the emotional displays of others during semi-structured naturalistic play periods. In one of their experiments (Kasari, et al., 1993), these authors set up a scenario during which ASD and non-ASD children were asked to complete two puzzles. After having done so the children were praised for their accomplishments and independent judges were asked to rate each child's reaction to the adult's praise. The ratings revealed that whilst all children expressed positive affect to a similar extent immediately after having completed the puzzles, children with ASD were less likely than children without such a diagnosis to reciprocate the adult's praise with an affective expression of their own. Not only that, children with ASD were also less likely than non-autistic children to actively encourage such praise by drawing the adult's attention to their accomplishment. In other words, whilst children with ASD *seemed* to experience pleasure in having completed their puzzles[4] (otherwise why would they express positive affect at all?), they appeared neither compelled to communicate this experience to others nor did they seem to realise that others were attempting to join them in this emotional episode. In a series of even more telling studies (Corona, Dissanayake, Arbelle, Wellington & Sigman, 1998; Dissanayake, et al., 1996; Sigman, et al., 1992; see also Bacon, Fein, Morris, Waterhouse & Allen, 1998; Loveland & Tunali, 1991) the authors extended these findings to situations in which adults displayed negative rather than positive affect. More specifically, children were videoed during interactions with an experimenter or parent who suddenly expressed distress, fear or discomfort and independent judges were again asked to rate each child's reactions to these emotional displays. Not surprisingly, children with learning difficulties and typical children responded to the adult's expressions by interrupting their play behaviour and orienting to the adult, often with marked concern. Children with ASD, on the other hand, were more inclined to keep playing with their toys and ignore the adult. In addition, the study by Corona and colleagues (1998) showed that children with ASD, unlike non-autistic children, did not even exhibit physiological orienting responses (i.e. changes in heart rate) to the adult's distress. In other words, children with ASD did not only appear to be *inattentive* to the emotional experiences of other individuals but they seemed to be generally less *responsive* to

[4] I stress the word *seemed* here because I do not believe that individuals with ASD subjectively experience emotional states in the same way as non-autistic individuals do. I will discuss this issue in more depth in the final chapter of this thesis.

them. What is also worth noting about these studies is that the authors were not only able to assess how children would react to the fearful or distressed adult but also how they would react to the object eliciting these emotions. Here too, children with ASD were found to react atypically in comparison to non-autistic children.

The evidence set out thus far suggests that individuals with ASD are often very markedly limited in multiple aspects of affectively patterned communication. Laboratory experiments and naturalistic observations converge in showing that emotional expressions are not particularly salient to them and although such individuals seem to be able to identify emotional expressions following explicit instructions, they often appear to deploy qualitatively atypical perceptual processes in order to do so. Together with the relatively infrequent attempts of individuals with ASD to initiate affective interchanges, these perceptual limitations severely compromise the foundations of emotional communication, affording autistic individuals fewer opportunities to share their emotional experiences with others and to learn about the emotional significance of environmental stimuli through them (see also Hobson, 1993; 2002). That these disturbances exist and that they have serious repercussions for the developmental trajectory of the disorder is no longer disputed. What continues to divide opinion is what causes these abnormalities in the first place and it is to this debate that the discussion will now turn.

The role of affective disturbances in the development of ASD

Currently, four major theories offer explanations for the affective disturbances associated with ASD. These include the idea that abnormalities in the development of a neural network mediating socio-perceptual capacities is responsible for the emotional disturbances described above (e.g. Schultz, 2005; Schultz, Grelotti, Klin, Kleinman, Van der Gaag, Marois, et al., 2003), the suggestion that abnormalities in the self-regulation of behaviour plays a critical role (e.g. Loveland, 2005; Bachevalier & Loveland, 2006), the view that a deficient mechanism for the understanding of mental states is to blame (e.g. Baron-Cohen, 1995; Baron-Cohen, et al., 2000; Frith, 2003; Leslie & Frith, 1990) and finally the idea that a disruption in the development of affectively patterned interpersonal relations lies at the

root of the problem (e.g. Hobson, 1993; Hobson, 2002; Loveland, 2005). It is beyond the scope of the current review to discuss the extensive literature that informs these theories in detail. Instead the principal aim of the following overview is to show 1) that these alternative viewpoints converge on the suggestion that abnormalities of the amygdala are most likely to blame for the emotional disturbances characterising ASD, 2) that the developmental significance of abnormal emotional processes in the emergence of the clinical manifestations of ASD remain the matter of debate and 3) that the evidence discussed thus far cannot distinguish between theoretical frameworks that consider emotional disturbances as either a cause or consequence of the development of ASD.

The socio-perceptual account and the amygdala

The idea that abnormalities in the development of socio-perceptual capacities are responsible for the affective disturbances associated with ASD originates primarily from the finding that such individuals tend to perceive faces through qualitatively deviant perceptual processes (Behrmann, Avidan, Leonard, Kimchi, Luna, Humphreys, et al., 2006; Behrmann, Thomas & Humphreys, 2006; Dawson, Webb & McPartland, 2005; Grelotti, Gauthier & Schultz, 2002; Jemel, Mottron & Dawson, 2006; Lahaie, Mottron, Arguin, Berthiaume, Jemel & Saumier, 2006; Schultz, 2005; Wallace, Coleman & Bailey, 2008). Studies, such as that by Hobson and colleagues (Hobson et al., 1988b) outlined earlier, suggest that individuals with ASD rely more on a piecemeal and featural rather than holistic and configural analysis of a face in order to identify emotional expressions (see also Davis et al., 1994; Deruelle, Rondan, Salle-Collemiche, Bastard-Rosset & Da Fonséca, 2008; Gross, 2005; Tantam et al., 1989; Teunisse & de Gelder, 2001). What has also become apparent, however, is that this atypical manner of analysing faces does not only interfere with the perception of affect but also with the perception of non-emotional information such as the identity (e.g. Boucher & Lewis, 1992; Boucher, Lewis & Collis, 1998; Davis, et al., 1994; Deruelle et al., 2008; Joseph & Tanaka, 2003; Wallace et al., 2008) age (Gross, 2002; Gross, 2005; Hobson, 1983) or gender (Deruelle et al., 2004) of an individual (see also section 2 of Table 1.1). In addition, eye-tracking studies suggest that individuals with ASD tend not to fixate on faces as much as non-

autistic individuals when viewing complex social scenes and that, even when they do, they look less at the eye-region and more at the mouth or non-feature regions of the face (Dalton, Nacewicz, Johnstone, Schaefer, Gernsbacher, Goldsmith, et al., 2005; Klin, Jones, Schultz, Volkmar & Cohen, 2002; Pelphrey, et al., 2002; Speer, Cook, McMahon & Clark, 2007; Spezio, Adolphs, Hurley & Piven, 2006). Together with an increasing number of recent neuroimaging and electrophysiological studies, which show that the neural correlates of face perception in ASD are also abnormal (Critchley, et al., 2000; Dalton et al., 2005;McPartland, Dawson, Webb, Panagiotides & Carver, 2004; Pierce, Müller, Ambrose, Allen & Courchesne, 2001; Piggot, et al., 2004), this evidence strongly suggests that general face processing abilities are compromised in ASD (see Bowler, 2007 and Jemel et al., 2006 for detailed overviews of this literature).

On the basis of the evidence just mentioned, and because it is well established that infants exhibit a strong preference to attend to social stimuli (particularly faces) from birth (see Walker-Andrews, 1997 for a review), Schultz and his colleagues (e.g. Schultz, 2005; Schultz et al., 2003) argue that the social and affective disturbances associated with ASD arise because of abnormalities in the development of a neural system that sub-serves the perception of social information. Central to this system is an area known as the Fusiform gyrus (FG), which is located on the inferior portion of the temporal lobes and widely believed to be functionally specialised for the perception of faces (see Kanwisher, 2000 for a review). Although the FG was originally thought to be innately hardwired for face perception, it is now thought to be more flexibly involved in the processing of visual expertise (Gauthier, Skudlarski, Core & Anderson, 2000). As such, the functional organisation (and face specialisation) of the FG is moderated by experience and it is in this experience driven neural specialisation that Schultz (2005) suspects the causes of the autistic socio-emotional abnormalities. More specifically, Schultz (2005) argues that an early emerging abnormality of the amygdala – a limbic structure known to be involved in the processing of emotionally salient stimuli (e.g. LeDoux, 1994) – results in individuals with ASD being relatively insensitive and inattentive to their social environment (see also Dawson et al., 2005). As a consequence, the functional organisation of systems such as the FG is compromised and the course of reciprocal social and emotional development altered.

What makes the account summarised above appealing is that it explains the affective disturbances associated with ASD within a broader neurodevelopmental framework of social cognition. As such, the theory is not only relevant to our understanding of ASD but also to our endeavour to comprehend the neural basis of the complex reciprocal socio-emotional capacities that uniquely define the human condition. The principal assumptions underlying this ambitious prospect are 1) that the amygdala and FG form part of a perceptual system that is necessary for the processing of socially relevant stimuli 2) that the maturation of this system necessitates attention to (and the perception of) social stimuli and 3) that a failure of such attention compromises the development of the system in such a way as to give rise to the clinical manifestations of ASD. A considerable amount of evidence validates all three of these assumptions. Studies of neurological patients who have suffered injury to the FG, for instance, show that such individuals are severely compromised in processing faces (Farah, Levinson & Klein, 1995) and several functional neuroimaging studies of typical individuals confirm that the FG is activated significantly more during face perception than object perception tasks (see Kanwisher, 2000 for a review). Similar neuropsychological evidence regarding the amygdala, confirms that also this structure is involved in processing socially relevant information, particularly in relation to identifying emotional expressions (see Adolphs, 2003 for a review). In relation to the 2nd and 3rd assumptions listed above, there is no direct evidence to support the idea that abnormalities in the perception of, or attention to, social stimuli compromises the neural maturation of the FG or other areas relevant to social behaviour. However, a considerable amount of work with congenitally blind children confirms that a lack of visual experience compromises the development of interpersonal skills (e.g. Cass, Sonksen & McConachie, 1994). In fact, several studies have noted a striking similarity between the reciprocal social abnormalities associated with congenital blindness and ASD (Bishop, Hobson & Lee, 2005; Brown, Hobson, Lee & Stevenson, 1997; Hobson & Bishiop, 2003; Hobson, Lee & Brown, 1999; Minter, Hobson & Bishop, 1998; Preisler, 1995). Together with the accumulating evidence documenting structural and functional abnormalities of the FG and amygdala in ASD (Bachevalier, 1994; Baron-Cohen, 1995; Bauman & Kemper, 2005; Critchley et al., 2000; Dawson, Webb, Schellenberg, Dager, Friedman, Aylward, et al., 2002; Schultz, 2005), this similarity between congenitally blind and ASD individuals lends strong

support to the suggestion that the clinically defining symptoms of ASD may be the result of abnormalities in early emerging socio-perceptual functioning.

In addition to offering a plausible account of the reciprocal social impairments associated with ASD, the socio-perceptual framework can also account for some of the sub-clinical manifestations of the disorder, such as the limitations in imitative behaviour (e.g. Hobson & Hobson, 2008; Rogers, Hepburn, Stackhouse & Wehner, 2003) and difficulties in understanding behaviour in terms of mental states (e.g. Baron-Cohen, 1995). Recent imaging studies suggest that the FG and amygdala play a role in such capacities (e.g. De Gelder, Snyder, Greve, Gerard & Hadjikhani, 2004; Lee, Josephs, Dolan & Critchley, 2006; Williams, Whiten, Waiter, Pechey & Perrett, 2007; Schultz et al., 2003) and congenitally blind individuals also exhibit developmental delays in these areas (Bishop et al., 2005; Minter et al., 1998). Another important asset of the socio-perceptual framework is that it allows room for the possibility that additional abnormalities outside the socio-perceptual brain network are responsible for atypicalities in domains other than the socio-emotional sphere. The account is for example compatible with evidence suggesting that perceptual functions operate differently in ASD (e.g. Mottron & Burack, 2001; Mottron, Dawso, Soulières, Hubert & Burack, 2006) and it may even be argued that abnormalities in socio-perceptual functioning constitute a specific instance of the autistic tendency to process information in a piecemeal and perceptually driven rather than holistic and conceptually driven manner (Behrman et al., 2006; Lahaie et al., 2006). Similarly, the account does not exclude the possibility that abnormalities of the frontal lobes contribute to the clinical manifestations of ASD (e.g. Bachevalier & Loveland, 2006) or that cerebellar or other brain-stem abnormalities are involved in the neuropathology underlying the disorder (e.g. Courchesne, 1997; Jou, Minshew, Melhem, Keshavan & Hardan, 2008). In other words, whilst the socio-perceptual account is specific enough to offer a neurologically plausible explanation for the clinically defining socio-emotional manifestations of the disorder, it is also sensitive to the fact that ASD is a complex multipathologic condition.

The role of behavioural self-regulation

The view put forward by Loveland and her colleagues (Loveland, 2001, 2005; Bachevalier & Loveland, 2006) in many ways complements the ideas put forward by Schultz (2005) and so requires little introduction. Loveland points out that ASD is characterised by abnormalities not only with respect to the perception of socio-emotional signals but also with regards to the regulation of behaviour in accordance with these signals. This may seem a trivial point to make because it should come as no surprise that an individual who experiences difficulties in perceiving certain properties of the world should also respond to these properties differently. Loveland's arguments are far from trivial, however, because they stress that perception is not merely a passive process but one that is intimately linked with action, particularly in development (see also Fogel, 1993). Perceiving the emotional significance of someone else's facial and postural expressions is of little use if one does not know how to respond appropriately, and not understanding the behavioural affordances of emotional signals may be reason enough not to attend to them. Thus the difficulty in ASD may not arise from a dysfunctional perceptual process but from an abnormality in a system that regulates behaviour accordingly. As outlined earlier, individuals with ASD are consistently found to be rather unresponsive to the emotional displays of others (e.g. Loveland & Tunali, 1991; Sigman et al., 1992; Yirmiya et al.,1992) and this passivity is often observed even when such individuals seem to be aware of the emotional displays in question (e.g. Bacon et al., 1998; Corona et al., 1998). Corona et al. (1998) for instance, found that although children with ASD were less likely than comparison children to look at an adult who simulated distress, both groups of children looked more often at the adult when s/he simulated distress than when s/he displayed an emotionally neutral expression. In other words, children with ASD differentiated between an adult's expressive displays but did not regulate their behaviour to the same extent as children without this disorder.

Within the context described above, it becomes apparent that any account of the socio-emotional difficulties characterising ASD must acknowledge the role of behavioural self-regulation, and Bachevalier and Loveland (2006) approach this challenge from a neural point of view. Based on evidence implicating aspects of the frontal cortex (in particular orbitofrontal

regions) in behavioural self-regulation, Bachevalier and Loveland (2006) argue that ASD may be characterised by abnormalities not only in relation to socio-perceptual functions of the amygdala but also in relation to orbitofrontal-amygdala interactions that may be involved in socio-emotional behavioural self-regulation. Although speculative at present, this framework not only complements Schultz's (2005) account, but also promises to address some of the heterogeneity of the symptom severity associated with ASD. More specifically, Bachevalier and Loveland (2006) suggest that the maturation of the amygdalo-orbitofrontal system may be compromised at different stages in development and either be associated with more widespread mediotemporal-frontal dysfunctions or not. Since the developmental time course of the amygdala and orbitofrontal cortex differ (e.g. Happaney, Zelazo & Stuss, 2004; Nikolić & Kostović, 1986), variations in the timing of the neuropathological onset in ASD would probably alter the nature and severity of the orbitofrontal-amygdala system's dysfunction and thus alter the symptom severity characterising the developing child. Abnormalities early in life may affect the amygdala disproportionately and result in the kind of developmental cascade described by Schultz (2005). Later emerging abnormalities may primarily affect the orbitofrontal cortex (with indirect effects on the amygdala) and result in socio-emotional difficulties that are less of a perceptual and more of a behavioural self-regulation nature. If abnormalities extend to other mediotemporal – frontal systems, such as the hippocampal – dorsolateral prefrontal circuit, ASD may be associated with more severe intellectual difficulties than when this system is spared (Bachevalier & Loveland, 2006). Although these suggestions await empirical testing, they pose interesting questions for future studies to address.

The Mentalising account

The socio-perceptual account of Schultz (2005) and the framework put forward by Bachevalier & Loveland (2006) offer well founded explanations for the socio-emotional abnormalities of the disorder in terms of neurodevelopmental processes. Whilst such neural accounts are perfectly acceptable, they reveal little about the cognitive and psychological processes that would allow a characterisation of ASD in terms of the mental experiences of those diagnosed with the disorder. This is the level of explanation that developmental

psychologists seek, and it is this level that remains the source of much controversy. According to advocates of the '*mentalising*' account of ASD (e.g. Baron-Cohen, 1995; Frith, 2001; Frith, 2003), the emotional disturbances associated with the disorder are a reflection of an underlying impairment in the capacity to understand other people's minds. In direct opposition to this view, Hobson (1993, 2002) argues that the autistic limitations in various cognitive faculties, including the ability to '*mentalise*', are the consequence of an infant failing to develop affectively patterned relations with others. I will outline this latter view in more detail in the next section. First, I will summarise the former.

Mentalising refers to the ability to understand, describe and explain behaviour in terms of mental states (e.g. beliefs, desires, intentions, etc...) and it is well established that individuals with ASD experience difficulties in this domain[5]. Such individuals often fail to understand that someone's actions are motivated by their beliefs about reality rather than the actual state of affairs (Baron-Cohen, 1989a; Baron-Cohen, et al., 1985; Happé, 1995; Leekam & Perner, 1991; Leslie & Thaiss, 1992; Perner, Frith, Leslie & Leekam, 1989; Surian & Leslie, 1999), for example, and they infrequently refer to mental states in order to describe social phenomena (Serra, et al., 1999; Tager-Flusberg, 1992) or the function of the brain (Baron-Cohen, 1989b). Individuals with ASD also have trouble comprehending that acts of deception can alter somebody's behaviour, and that somebody's behaviour may not necessarily reflect a person's inner thoughts (Russell, Mauthner, Sharpe & Tidswell, 1991; Sodian & Frith, 1992). When asked to sort pictures in order to form coherent stories, individuals with ASD perform worse on narratives that revolve around the thoughts and believes of story characters whilst they have little difficulty with stories concerning physical causality (Baron-Cohen, Leslie & Frith, 1986). They also find it hard to recognize stories in which a character commits a *faux pas* (Baron-Cohen, O'Riordan, Stone, Jones & Plaisted, 1999), and they frequently have trouble appreciating that the literal meaning of language is qualified by the intentions of the speaker and the listener (Happé, 1994; Jolliffe & Baron-Cohen, 1999). Finally, individuals with ASD perform poorly on a test battery in which mental

[5]Although the term 'Theory of Mind' is often employed to describe this ability, I will avoid this terminology throughout this thesis in order to avoid the implication that mental states are necessarily understood through a process of theorising. Although this view is endorsed by some (e.g. Astington & Gopnik, 1991; Gopnik, Meltzoff & Kuhl, 1999; Perner, 1991) it is not the only way of conceptualising the ability to understand behaviour in terms of mental states (e.g. Gordon, 1996; Hobson, 1991b; 1993; 2002).

states need to be inferred from video-clips of emotionally charged interactions (Golan, Baron-Cohen, Hill & Golan, 2006; Golan, Baron-Cohen & Golan, 2008), sound recordings of speech segments (Golan, Baron-Cohen & Hill, 2006) or photographed eye-regions of a face (Baron-Cohen, Jolliffe, Mortimore & Robertson, 1997; Baron-Cohen, Wheelwright & Jolliffe, 1997; Baron-Cohen, Ring, Wheelwright, Bullmore, Brammer, Simmons et al., 1999, Baron-Cohen, Wheelwright, Hill, Raste & Plumb, 2001). In short, individuals with ASD exhibit difficulties across a variety of experimental paradigms in which behaviours need to be understood, described or predicted in terms of unobservable mental phenomena (see Baron-Cohen, 2001; Bowler, 2007 and Frith, 2003 for more detailed reviews of this literature).

When evidence of a mentalising difficulty in ASD first emerged during the 1980s, it stimulated one of the most wide-ranging and long-lasting debates in the history of developmental psychology. Motivated by philosophical questions regarding the nature of the 'Mind' and the 'Self' and what it means to entertain mental representations of the world, different opinions have emerged about what it means to be able to mentalise, why and from what the ability has evolved, how mentalising can best be measured, when the ability emerges during childhood, what role language and culture might play in its development and why self awareness might be important for the ability to mature. It is far beyond the scope of the current review to delve into this extensive literature (see Baron-Cohen, 1995; Bowler, 2007; Heyes, 1998; Hobson, 1993; Hobson, 2002; Perner, 1991; Scholl & Leslie, 1999; Tirassa, Bosco & Colle, 2006; Tomasello, 1999; Tomasello & Rakoczy, 2003 for relevant discussions). Fortunately, a detailed discussion is not necessary in order to show that the affective disturbances associated with ASD can be plausibly explained in terms of a mentalising impairment. All one needs to be content with is a conceptualisation of mentalising that presupposes that the processes involved in this ability are a prerequisite for the kinds of socio-emotional capacities that are characteristically disrupted in ASD and this is precisely the assumption underlying the 'Mindreading' model proposed by Baron-Cohen (1995, 2005).

According to Baron-Cohen (1995, 2005), the ability to mentalise reflects the operation of a Mindreading System that consists of six neurocognitive mechanisms – The Intentionality Detector (ID), Eye Direction Detector (EDD), Emotion Detector (TED), Shared Attention

Mechanism (SAM), Theory of Mind Mechanism (ToMM) and The Empathising System (TESS). In typical development, the ID, EDD and TED components are the first to be functionally mature (between birth and 9 months of age) and allow infants to understand simple mental states by building dyadic representations of certain *Agent-Object* relations. More specifically, the ID represents self-propelled movements as goal-directed and volitional, EDD ascribes perceptual capacities to entities with eyes, and TED represents certain acts in terms of affective states such as anger and happiness. Thus, during the first 9 months of life, infants acquire an increasingly sophisticated understanding of how agents (including themselves) can relate to objects (including other agents) in their environment through unobservable mental processes such as 'wanting' something (ID) 'seeing' something (EDD) or being 'angry' about something (TED). According to Baron-Cohen (1995, 2005) these mechanisms are, in principle, functionally intact in ASD although their development may be delayed. Support for this suggestion stems from studies, which indicate 1) that individuals with ASD ascribe volitional mental states to agents (e.g. Baron-Cohen et al., 1986; Bowler & Thommen, 2000), 2) that they are able to detect what someone is looking at (e.g. Baron-Cohen, 1989c) or when someone is looking at them (Baron-Cohen, Campbell, Karmiloff-Smith, Grant & Walker, 1995)[6] and 3) that they sometimes do not seem to have any difficulty with the perception of non-mentalistic emotional expressions such as anger, fear or happiness (e.g. Baron-Cohen, Spitz & Cross, 1993; Castelli, 2005, Prior et al., 1990)[7].

The next mechanism to mature in typical development (between 9 and 18 months) is SAM, which uses the dyadic representations from ID, EDD and TED to build more complex triadic representations of *Self-Agent-Object* relations. These triadic representations allow young children to understand that the object of their own mental scrutiny may also be the object of another agent's mental scrutiny and since SAM provides a link between the ID, EDD and TED mechanisms, children also come to understand that the eyes (EDD) are particularly

[6]Some studies suggest that individuals with ASD may experience some difficulties in detecting gaze direction (e.g. Senju, Yaguchi, Tojo & Hasegawa, 2003). Such findings may reflect a developmental delay in the maturation of EDD to which some experimental paradigms may be more sensitive. The principal argument here is that the functional integrity of ID, EDD and TED are *relatively* preserved (relative to the function of the other mechanisms of the Mindreading System which are severely compromised).

[7]It is worth noting that the evidence for this last claim is rather scarce (see Table 1.1)

informative about an agent's desires (ID) and affective states (TED)[8]. Thus, the computational capacities of SAM equip the young child with a host of new behavioural skills such as gaze monitoring, protodeclarative pointing and social referencing that allow for a much richer interaction with others. The deficient emergence of these so called joint attention behaviours in ASD (see Bruinsma, Koegel & Koegel, 2004 for a review) is what prompts Baron-Cohen (1995, 2005) to suggest that the functioning of SAM is severely compromised in this disorder and that this has detrimental consequences for the subsequent maturation of the last two components of the Mindreading System – ToMM and TESS.

In order to understand what ToMM is needed for, it is useful to consider the limitations of SAM. According to the Mindreading System, SAM mediates an understanding of triadic relations that are limited to those that are perceptually anchored and thus rigidly fixed in reality. That is, SAM is unable to represent so called *epistemic* mental states (e.g. beliefs, pretence, imaginations, dreams, etc...) that can either stand in true or false relationship to the actual state of the world (e.g. one can *believe, pretend* or *imagine* that it is raining even if it is not). These mental state concepts can only be represented by ToMM, which is thought to gradually mature between 18 and 48 months of age. Following a theoretical framework developed by Leslie (1987), Baron-Cohen (1995) suggests that the function of ToMM is to compute the full range of epistemic mental states (believing, pretending, dreaming, etc...) by enabling representations of the world to be *decoupled* from reality. Without going into undue detail, the process of *decoupling* (see Leslie, 1987 for a detailed description) involves copying 'true' representations of objects into a working memory type store where they can be manipulated without the constraints of reality. This decoupling process provides the necessary context for children to understand that mental (or any other) representations can either reflect the world truthfully (e.g. he thinks it is raining when it is actually raining) or falsely (e.g. he thinks it is raining when it is actually sunny). The first evidence that young children comprehend this characteristic of mental phenomena is when they start to understand (and engage in) acts of pretence and symbolic play (e.g. use a banana as if it were a telephone) around 18 months of age (Leslie, 1987, see also Perner, 1991). Since the

[8]This proposed link forms the basis of mentalising tasks in which individuals are asked to infer mental states and certain emotions form photographed eye-regions of a face (e.g. Baron-Cohen et al., 1999).

operation of ToMM crucially depends on the input from SAM and because SAM is functionally impaired in ASD, Baron-Cohen (1995, 2005) suggests that virtually all aspects of ToMM should be compromised in this condition. Indeed, pretend and symbolic play are severely delayed or abnormal in children with ASD (see Jarrold, 2003 for a review of this literature) and as outlined above, there is an impressive amount of evidence to suggest that individuals with ASD have difficulties in understanding a wide range of epistemic mental states.

The final component of Baron-Cohen's (2005) Mindreading System is TESS, which enables children not only to describe and understand behaviour in terms of mental states but also to respond to such states with appropriate affect. In other words, TESS allows children to *react* to another's mental states with empathy and according to Baron-Cohen this ability does not mature until the age of about 14 months during typical development (see also Perner, 1991). Similar to SAM and ToMM, TESS accomplishes its function by computing triadic relations, this time of a rather special kind that Baron-Cohen (2005) calls E-Representations (i.e. Empathising Representations). Formally expressed, these representations take the form of $Self_{Affective\ State}$-$Other_{Affective\ State}$-*Object* relations, with the critical constraint that the affective state of the self must be *appropriate to* and *triggered by* the affective state of the other (Baron-Cohen, 2005). Again, the computational capacities of TESS (which are not specified) rely on the input from SAM and again this means that the behaviours supported by TESS should be severely compromised in ASD. In support of this suggestion, individuals with ASD seem to have particular difficulties understanding the concept of empathy (e.g. Dziobek et al., 2008; Yirmiya, et al., 1992) and there is evidence indicating that they struggle with the idea that beliefs can cause emotions even though they have no trouble comprehending that physical events can do so (Baron-Cohen, 1991; Baron-Cohen, et al., 1993; Dennis, et al., 2000). In addition, studies such as the one by Sigman and colleagues (Sigman et al., 1992) outlined earlier, demonstrate that individuals with ASD do not react empathically to the distress of others. Finally, there is also evidence to suggest that individuals with ASD have difficulties understanding particularly those kinds of emotions that rely on a greater degree of mentalising. Pride and embarrassment, for example, arise when one realises that one's behaviour is the focus of someone else's mental scrutiny and children with ASD have difficulties explaining these emotions even when they can describe simpler emotions such as

happiness or sadness (Baron-Cohen et al., 1999; Capps, et al., 1992 but see Hillier & Allinson, 2002). In short, the patterning of affective disturbances associated with ASD, together with their severe difficulties on tasks that require an understanding of other people's minds, is consistent with the suggestion that deficiencies in a Mindreading System are responsible for much of the socio-emotional atypicalities characterising the disorder.

Before leaving the topic of Mindreading, it is worth pointing out that this framework is compatible with the socio-perceptual account offered by Schultz (2005). As outlined earlier, Schultz (2005) suggests that the amygdala is essential for directing an infant's attention toward social stimuli (particularly the face), allowing the socio-perceptual brain network (e.g. the Fusiform Gyrus) to functionally specialise for the modulation of socio-emotional behaviours. According to this view, the socio-emotional deficiencies associated with ASD are the result of an early emerging amygdala abnormality and the resulting disruption of a socio-perceptual brain network that normally modulates socio-emotional behaviours. Baron-Cohen (1995, 2005), also suggests that the amygdala plays an important role in directing attention to social stimuli (particularly the eye region of a face; i.e. EDD) but unlike Schultz, he attempts to specify the developmental consequences of such attention in cognitive rather than neural terms. More specifically, he argues that attention to the eyes plays an important role in the maturation of a Mindreading System that mediates the understanding of how behaviour is guided by mental representations of the world. The socio-emotional abnormalities characterising ASD are therefore considered to reflect a disruption in the computational mechanisms subserving the ability to understand other people's minds.

ASD as a disorder of affectively patterned interpersonal relations

Whilst few authors would deny that socially oriented attention is important (and even necessary) for the development of affectively patterned interpersonal skills, not everyone is content with the idea that such attention promotes the maturation of interpersonal life by facilitating the development of computational mechanisms such as a Mindreading System. Hobson (1993, 2002) has a rather different opinion and argues that abilities such as 'mentalising' actually result from, rather than promote, reciprocal social development.

Hobson's (1993, 2002) approach is based on the assumption that much of an infant's cognitive development is anchored in an innate disposition to psychologically and emotionally relate to other individuals. He agrees that socially oriented attention constitutes an important facet of this disposition, but for Hobson the *attention to* and *perception of* other people is not the driving force in development. What matters the most is an infants' emotional *engagement with* and *relatedness to* others, which provides her with the means to learn about the world (and the people within it) *through* the attitudes and emotional orientations of others. On the basis of this view, the clinically defining socio-emotional abnormalities of ASD are therefore seen as the result of a failure to establish affective interpersonal relations with others.

In line with Hobson's theory is the observation that infants, soon after birth, do not simply observe and attend to other individuals. They also actively react to and engage with them. As mentioned earlier, it is now well established that infants exhibit a strong preference to attend to social stimuli (see Walker-Andrews, 1997 for a review) from birth. Neonates, for instance, preferentially look at faces and face-like stimuli rather than non-face stimuli (e.g. Cassia, Simion & Umiltà, 2001; Johnson, Dziurawiec, Ellis & Morton, 1991; Turati, Simion, Milani & Umiltà, 2002; Valenza, Simion, Cassia & Umiltà, 1996) and the human auditory system is so finely attuned to the perception of human speech (Ramus, Hauser, Miller, Morris & Mehler, 2000; Vouloumanos & Werker, 2004) that even before birth the foetus distinguishes between the mother's voice and that of a stranger (Kisilevsky, Hains, Lee, Xie, Huang, Ye, et al., 2003). Young infants, however, are not merely passive perceivers of their social surroundings. They are equipped with a variegated behavioural repertoire that allows them to actively control and synchronise with the behaviours of others. For instance, neonates have been found to synchronise their general motility levels with the patterning of adult speech (e.g. Condon, 1979; Kato, Takahashi, Sawada, Kobayashi, Watanabe & Ishii, 1983). This, so-called 'entrainment' is thought to facilitate a primitive, yet coordinated, interchange between the infant and her caretakers (Kato et al, 1983). In addition, it is now well established that infants, only a few days old, will imitate a range of facial and gestural behaviours (Meltzoff & Moore, 1977, 1989, 1997; Meltzoff, 2007), including certain expressions of emotions such as happiness, sadness and surprise (Field, Woodson, Greenberg & Cohen, 1982). This last finding is particularly important because it shows that,

even during this very early stage in development, infants are equipped with the basic motoric competences to engage in emotionally charged interactions. In fact, careful analyses of imitative behaviours have shown that neonates are emotionally expressive even when they are imitating non-emotional expressions such as mouth opening (Kugiumutzakis, Kokkinaki, Makrodimitraki & Vitalaki, 2005) and even outside the context of imitation, neonates are emotionally surprisingly expressive. Soon after birth, neonates spontaneously smile, even when asleep (see Messinger & Fogel, 2007 for a review), and three day old neonates express disgust in response to smelling unpleasant odours (see Soussignan & Schaal, 2005 for a review). Regardless of whether one considers these neonatal emotional expressions as intentional or reflexive, the fact that they are present from birth and that they are recognised as emotionally meaningful by adults means that infants are born with the necessary competences to send emotional signals to others and hence influence their behaviour.

The synchronised and emotionally expressive behavioural repertoire of neonates described above constitutes the earliest form of interpersonal engagement between an infant and her social environment (see also Trevarthen's concept of 'primary intersubjectivity'; e.g. Trevarthen, 1979). Although the synchronicity at this young age is not easy to detect, from the age of about 6 to 8 weeks, the reciprocal intent and communicative value of infant behaviour is no longer so elusive. At this age, infants start to use gaze patterns, social smiles (i.e. smiles directed at others) and vocalisations to modulate face-to-face interactions with others (e.g. Lavelli & Fogel, 2002; Messinger & Fogel, 2007; Trevarthen, 1979; Tronick, Als & Adamson, 1979) and over the course of the next few months these reciprocal social behaviours become increasingly well integrated, timed and co-ordinated. Most important for the current discussion is that infants from about 6 - 8 weeks become significantly distressed when the flow (i.e. the timing and synchronicity) of reciprocal social behaviour is disrupted. During the still-face paradigm, for instance, an infant's previously energetic and positive engagement with an adult becomes sober and negative when the adult ceases to react and simply maintains a blank expression (see Adamson & Frick, 2003 for a review of the still-face paradigm). Similarly, when the contingency between the infant's behaviour and that of a social partner is temporally distorted (through the use of delayed video-feedback), infants become visibly disconcerted (e.g. Soussignan, Nadel, Canet & Gerardin, 2006; Striano,

Henning & Stahl, 2005; Striano & Bertin, 2005). What these observations show, is that the increasingly sophisticated interchanges between an infant and her caretaker continue to be intensely emotional (see Nadel & Muir, 2005 for a collection of reviews). For Hobson (1993, 2002), this is the crucial point to emphasise because this emotional dimension leads to a transformation of an infant's understanding of other people. Initially (i.e. during the first 9 months or so) infants primarily understand other people as the kind of 'thing' that affords synchronous and co-ordinated interaction. What they do not yet understand, however, is that other people engage with and relate to the world in much the same way as they themselves do. The first step toward such understanding is the mutual experience provided by the emotional dimension of interpersonal engagement, which enables both interactive partners to 'feel' *within* themselves what they observe in the expressions of the other. From the point of view of the infant, this experience affords her direct access to the emotional directedness of others, both toward herself and toward other objects in the world. There is an obvious problem, with this state of affairs. If the infant experiences the subjective orientations of others within herself, how can she distinguish between her own subjective experiences of the world and that of others? According to Hobson (1993, 2002), the answer lies within the infant's increasingly sophisticated interpersonal skills. By being able to take control of reciprocal social interactions and manipulate the behaviours of others, infants start to direct the subjective orientations of others toward different aspects of their own (the infant's) behaviour. One moment the adult directs his attention to the infant's smile, the next he tickles a moving limb, and after that he places a finger in the infant's opening hand. And when the adult fails to play along, all the infant needs to do is deploy an arsenal of smiles, coos, cries and gaze-patterns in order to re-engage him. What the infant learns through these experiences, is that the subjective orientations of others are 'directable' and that they are 'directable' toward things that the infant herself is engaged with. In other words, the 9 month old infant is beginning to understand that her own orientations toward the world are independent of the orientations of other people and this means that there is something special about times when both direct their orientations to the same objects. This transition in the infant's understanding marks the emergence of joint attentional skills.

In discussing the Mindreading account offered by Baron-Cohen (1995, 2005), we have already seen that the age of 9 months marks an important developmental milestone in relation to ASD. According to Baron-Cohen, this is the time at which the Shared Attention Mechanism (SAM) typically comes online. A developmental failure of SAM is regarded as the onset of the behavioural manifestations of ASD and the evidence for joint attention, mentalising and empathizing impairments are taken as supportive evidence for this account. Hobson (2002) also regards the 9 month developmental milestone as important in relation to ASD but for rather different reasons. For Hobson (2002) the developmental trajectory of ASD does not start with a failure of some mechanism to develop at 9 months. Instead he suggests that the disorder has been developing since birth. He argues that the infant with ASD never experiences the kind of emotional connectedness with others that forms the foundation of understanding other people *as* people. Thus, by 9 months, the child has not had the necessary experiences with others to understand that the subjective orientations of others can be brought in line with the child's own. It is for this reason that individuals with ASD fail to develop the rich joint-attentional skills that characterise typically developing children and not because some computational mechanism fails to come online. Another point on which Hobson (1993, 2002) and Baron-Cohen (1995, 2005) disagree, is how a failure in the development of joint attention around the age of 9 months compromises the subsequent development of reciprocal social behaviour in such a way as to give rise to the clinical manifestations of ASD. As described in the previous section, Baron-Cohen suggests that impairments in joint attention (i.e. SAM) compromise the maturation of ToMM and TESS, which mediate an understanding of behaviour in terms of mental states and empathy. As a result of this developmental knock-on effect, individuals with ASD are limited in their imaginative and symbolic play activities (see Jarrold, 2003 for a review), their understanding of behaviour in terms of mental states and emotions (see Baron-Cohen, 1995, 2001) and their empathic reactions to the distress of others (e.g. Sigman et al., 1992; Yirmiya, et al., 1992). Hobson (1993, 2002) agrees that these manifestations of ASD are linked to the impairments in the development of joint attentional skills, but not in terms of a computational processing chain. Hobson suggests that an infant's failure to share the attentional focus with another individual prevents her from learning about the world *through* the experiences of

others. Recall, that the studies by Sigman and colleagues outlined earlier (e.g. Sigman et al., 1992; Sigman, 1998) showed that children with ASD do not only fail to attend to adults displaying distress but that they also fail to alter their behaviour in relation to the object eliciting this reaction in the adult. In this context Hobson emphasises that infants do not simply observe or attend to what another person is attending to but that they are *relating* to that person's emotional orientation toward the world. Through this relatedness, the child is being moved to different psychological standpoints in relation to objects in the world and, according to Hobson (1993, 2002), this movement in psychological space facilitates the development of imaginative abilities and a deeper understanding of other people's mental lives. In parallel, this joint engagement with others enables a child to learn about the emotional saliency of objects and events in the world. The clinical manifestations of ASD, therefore, are seen as the continuing failure of interpersonal engagement rather than a developmental failure of a cold and calculating cognitive machinery

Summary

I hope that the preceding overview has demonstrated three things. First, that it is well established that individuals with ASD are severely limited in multiple aspects of emotional communication. Second, that explanations of these limitations at the neural level converge on the suggestion that abnormalities of the amygdala are most likely involved. And finally, that explanations at the psychological level continue to differ as to whether ASD is fundamentally a disorder of emotional development (e.g. Hobson, 2002) or of socio-cognitive development (e.g. Baron-Cohen, 2005). Within the scope of the current thesis it has not been possible to outline all the evidence that is relevant to this last debate in detail. A thorough consideration of the similarities and differences between children with ASD, congenitally blind children, and children who are emotionally deprived during infancy (e.g. Romanian orphans), for instance, would have been informative in relation to Hobson's (2002) account, whilst a more focused discussion of the evidence pertaining to the amygdala's involvement in social cognition may have helped to further furnish the arguments of Baron-Cohen (2005). The principal aim, however, was not to provide an in-depth discussion of a debate that has prevailed in the

study of ASD for almost three decades but to convince the reader that this debate continues to be unresolved. In this context the important point to make is that the currently available evidence, even if it stems from various sources, can be accommodated within both Baron-Cohen's (2005) and Hobson's (2002) account.

One of the main difficulties in distinguishing between the validity of the frameworks proposed by Hobson (1993, 2005) and Baron-Cohen (1995, 2005), is the fact that, like all theories, they are open to refinement. Hobson's account, for instance, does not specify what it means to be engaged with another emotionally. The concept is defined only loosely by the behavioural patterns that take place between an infant and her caregiver. As such, the definition of interpersonal engagement can be refined on the basis of new observations regarding interpersonal behavioural patterns. In the case of Baron-Cohen's account, the theoretical malleability arises from the modular structure of the Mindreading System. Between 1995 and 2005, this system has changed from a 3-component computational mechanism to a 5-component one (TED and TESS were added) and as a result the system can now better account for the emotional disturbances that characterise ASD. It is important to note that these observations are not meant as criticisms of the respective theories. Redefining theories in light of new evidence is what scientific progress is all about. Sometimes newly accumulated data require a drastic change in the formulation of theories, such as when Copernicus placed the sun rather than the earth at the centre of our solar system. More often than not, however, theories are not entirely wrong and instead capture something important about the way things really are. They just do not always capture that 'something' in quite the right way, requiring a small adjustment to the definitions and terms of the theory. In psychology, as in every other science, it is this process of theoretical refinement that results in ever more detailed explanations of the phenomenon of interest. The unfortunate state of affairs regarding the debate surrounding the emotional disturbances evident in ASD, is that two competing theories offer equally valid explanations for the same empirical phenomena whilst being equally accommodating of new findings. The question is, how can we resolve this theoretical deadlock?

A closer look at what emotions are

After having discussed the emotional disturbances associated with ASD over dozens of pages, it may seem strange to ask what emotions actually are. Yet, if we want to determine whether abnormalities in emotional development play a central role in the development of ASD, we need to specify what we mean when we say that something is 'emotional'. Neither Hobson (1993, 2002) nor Baron-Cohen (1995, 2005) consider this issue in any great detail, and they do not need to, for their theories are about explaining the *socio*-emotional difficulties associated with ASD and not the emotional difficulties *per se*. Both authors focus their considerations on how emotional behaviours influence reciprocal social development and how development in this domain relates to other areas of cognition. Hobson (1993, 2002), for instance, identifies the mutual experience that emotional episodes provide for an infant and her caregiver as a key factor in social development and he maintains that cognitive capacities mature within this affectively patterned social context. Baron-Cohen (1995, 2005), by contrast, emphasises that emotions need to be understood as mental phenomena in order for one to make sense of the emotional nuances of reciprocal social behaviour, and a pre-condition for this level of understanding is the development of certain neurocognitive mechanisms. What both of these authors overlook, is the fact that emotions are not merely a social phenomenon. Emotions play an important role in our lives regardless of whether we are alone or in the company of others; they influence our attention, modulate our learning, and alter our perceptions of the world. Thus, if there is something severely amiss in terms of how emotional processes develop in ASD, we would expect the consequences to be evident regardless of whether emotions are studied within the context of social behaviour or not. Looking back on the evidence discussed so far, it becomes immediately apparent that we know virtually nothing about how emotional processes operate outside the context of social cognition in ASD and this gap in the literature is what the following experimental chapters will try to fill. Chapter 2 will present the findings from three experiments that explore the influence of emotional factors on learning and memory in ASD. Chapter 3 will consider the influence of emotional factors on perceptual processes, and Chapter 4 will examine fear conditioning. Since four of the five experiments presented in these chapters have either been published, or are currently under review (Gaigg & Bowler, 2007; Gaigg & Bowler, 2008; Gaigg & Bowler,

under review a, Gaigg & Bowler, under review b), they will be presented as stand-alone articles and as such, contain detailed descriptions of the literature most pertinent to them. In order to avoid repetition, therefore, the current chapter will conclude with some general considerations about the nature of emotions and the formulation of the principal hypothesis that motivates the subsequent studies.

Arousal, Cognition and the subjective experience of Feelings

Emotions have been at the centre of fierce debates for more than a century and so it is just as well that, for the current purposes, it suffices to focus on issues that all authors agree on. First, however, it is necessary to start with the rather dull task of commenting on some terminological distinctions since these continue to be the source of many debates. The first point to make, in this context, is that the current thesis uses the terms *emotion* and *affect* in a theoretically neutral sense to describe behaviours, processes and experiences alike. This decision reflects the fact that these terms are used commonly in this manner in day-to-day conversations and so ascribing specific meanings to them would inevitably lead to confusion. Other words, however, are used with more deliberation. The word *'feeling'* and its derivatives, for instance, is reserved to describe the subjective experience people describe when they are asked to report on their current emotional state. It reflects a person's *conscious awareness* of experiencing an emotion. It is worth noting, about the word 'feeling', that it is commonly also used to describe the subjective experience of physical sensations such as temperature (e.g. I feel cold) and homeostatic imbalances (e.g. I feel thirsty, I feel dizzy). There is a thin line between these two uses of the word and it is not a coincidence that descriptions of physical sensations often figure in reports of emotional states (e.g. hot with rage, shaking with fear). What distinguishes the two will become apparent shortly. Another term with a specified meaning is *'arousal'*, which denotes the automatic alterations in bodily states that take place during an emotional episode. Arousal is the product of the sympathetic and parasympathetic branches of the autonomic nervous system (ANS) and as such reflects the state of a multifaceted homeostatic system. An important feature of arousal is that it manifests in various ways such as changes in heart-rate, respiratory function, blood pressure, body

temperature, vasoconstriction, pupil-dilation, muscle tension and gland secretions. The final word worth a brief comment is *'cognition'*, which describes the information processing capacities of our brain (i.e. cognitive neuroscience) and mind (i.e. cognitive psychology). Traditionally, emotions were not considered under the rubric of 'cognition', and during the height of the cognitive revolution the two words almost acquired the status of antonyms (see Damasio, 1994; LeDoux, 1996 for further discussion). Whilst this contrast between emotion and cognition has started to fade in recent years, for the purposes of the current thesis it is useful to keep the two apart. Thus, whenever the word 'cognition' is used here, it refers to inherently non-emotional processes including learning, memory, attention, perception, decision-making, problem-solving and mental imagery. On a final note, it is worth noting that the following discussion is informed by the considerations of several prominent authors in the field of emotions (e.g. Barrett, Mesquita, Ochsner & Gross, 2007; Cannon, 1927; Damasio, 1994, 1999, 2003; Darwin, 1872; Ekman, 1992; James, 1890; Lane, 2006; Lane & Nadel, 2000; Lazarus, 1984; LeDoux, 1996, 2002; Levenson, 1992; Reisenzein, 1983; Russell, 2003; Schachter & Singer, 1962; Zajonc, 1984) as well as by my own thinking on the topic. For this reason, and because the experimental chapters of this thesis will provide a fully referenced introduction to specific aspects of emotional processing, the subsequent summary will contain few references.

The first point of agreement in the study of emotions, is the idea that every emotion is elicited. It should only take a moment's consideration to agree with the fact that emotions do not simply arise out of nowhere (nothing does!). They are the result of some kind of stimulus or event that can either be a real object such as a snake or the sight of a loved one, or a figment of the imagination such as the thought of failing an exam or the memory of a recent argument. As William James (1890) put it, '*One may get angrier in thinking over one's insult than at the moment of receiving it; and we melt more over a mother who is dead than we ever did when she was living*' (p.443). It is not worth dwelling on this issue for too long because the current thesis is not specifically concerned with examining the kinds of objects or events that elicit emotions in individuals with ASD (though some of the findings reported later are relevant to this issue). It is worth noting, however, that we still know very little about this topic. With the exception of a few studies that I will consider in due course, the only stimulus we know not to

elicit typical emotional responses in individuals with ASD is a person – especially one that expresses an emotion. Outside the social domain, however, we know next to nothing about what individuals with ASD consider to be emotional or not.

The point with which the current thesis is most directly concerned is the question of how certain objects or events come to elicit emotions and how these objects come to be considered as 'emotional' as a result. The answer to these questions is fraught with controversy, partly because different authors employ different terminologies in slightly different ways, partly because it depends on the object or event of interest and partly because different types of emotions require different formulations of an answer. Despite many unresolved issues, however, authors have come to several points of consensus. One of these is the notion that emotions are the result of the interaction between arousal and cognition. It is useful to remember that the term 'emotion' in the current thesis is used in a theoretically neutral way and that 'cognition' refers to inherently non-emotional processes. This use of terminology has the advantage that every form of behaviour and cognition can be described as either emotional or non-emotional. Since this is no doubt confusing (and logically dubious), it is worth considering an example. Imagine you are walking down a dark alley when, all of a sudden, you hear footsteps quickening behind you. Your cognitive system immediately homes in on the noise. Your attention is focused, your senses heightened and your memory system lays down a trace of the whole event. Simultaneously, your arousal system starts to prepare you for action by increasing your heart rate, diverting blood-flow to your muscles and increasing adrenalin secretion. Regardless of whether you are an organism capable of self-conscious thought or not, you will quicken your pace in order to avoid potential danger. Now, the quickening of your pace in this scenario, is an *emotional behaviour* because it is a direct consequence to the interaction between the cognitive processing of the sound of quickening footsteps in the dark alley and the arousal elicited by that sound. Equally, the memory you formed during the episode is an *emotional memory*, because it too resulted from the interaction between arousal and cognition (in this case memory). Thus every behaviour and every form of cognition can take on an emotional quality despite the fact that there is nothing inherently emotional about such behaviours or cognitions.

The foregoing arguments directly inform the next point on which most authors agree, which is the idea that feelings are the result of a rather special kind of interaction between cognition and arousal. More specifically, feelings only arise when the interaction between cognition and arousal is *evaluated* or *appraised*[9]. Once again, it is useful to illustrate the point with an example and so I invite you back into the dark alley. In the scenario above, you would most likely have experienced the feeling of fear because, in the context of your arousal response you would have evaluated the quickening footsteps behind you as threatening to your well being. Imagine again, however, that you hear the quickening of footsteps behind you, only this time you know that they belong to your partner, who had stopped to tie his shoelace and is now trying to catch up. In this case your autonomic system is unlikely to respond with alarm and you are unlikely to start feeling afraid. Even if your arousal system is activated with an initial startle response or is in a similar state to that described above at the moment you hear the footsteps quickening – maybe because you ran to catch the last bus before being forced to walk home – you would not necessarily start to feel afraid. The point here is that cognition and arousal are not *sufficient* to elicit a feeling. Feelings only arise when arousal is *evaluated* in relation to the stimulus that gave rise to it. Incidentally, this analysis helps clarify the relationship between the two uses of the word 'feeling' noted earlier. Recall that 'feelings' can either describe one's conscious awareness of experiencing an emotion, or one's awareness of experiencing a physical sensation. Since emotions are, in part, the result of arousal and because many aspects of arousal (e.g. heart rate, breathing rate, etc...) can come under the scrutiny of conscious awareness, it makes sense that emotional feelings should sometimes be associated with descriptions of physical sensations. What distinguishes the two, is that emotional feelings *necessarily* involve an *evaluation* of the physiological sensation in relation to the stimulus that elicited it. Physical feelings on the other hand are primarily conscious perceptions of physical sensations that need not necessarily be *evaluated*.

An important question raised by the foregoing analysis, is what the nature of the evaluative process is that gives rise to feelings. Although this topic remains the matter of

[9]This evaluative process is often described as a cognitive process, so it is easy to see where some of the terminological confusion stems from.

debate, it is widely acknowledged that feelings are intricately linked to the concepts of self awareness and consciousness (e.g. Damasio, 2003; Lane, 2006; LeDoux, 2002). More specifically, ideas converge on the suggestion that the interaction between arousal and cognition shapes interpretations of environmental stimuli and events in terms of their significance to the 'Self'. This view has important implications for developmental theory since it is widely believed that neonatal infants do not yet possess the kind of 'Self' concept that lies at the centre of the evaluative processes necessary for feelings (e.g. Neisser, 1988). Infants do possess a functioning autonomic nervous system, however, and they have a series of functioning cognitive processes at their disposal that allow them to represent the sensory properties of their surroundings. Thus, the building blocks for the emergence of feelings are present very early in life, making it likely that the interactions between arousal and cognition that are necessary for the emergence of feelings also play a role in the development of self-awareness more generally (see Damasio, 2003; LeDoux, 2002). I believe that these arguments hold the key to understanding the developmental trajectory of ASD in its broadest sense and I will return to this argument in more detail in the final chapter. For now let me return to the final point of agreement in the study of emotions.

An immense amount of evidence has accumulated over the past two decades that leaves little doubt that the amygdala plays an important role in mediating the interaction between arousal and cognition that gives rise to emotional behaviours and experiences (e.g. Davis & Whalen, 2001; Lane & Nadel, 2000; LeDoux, 1995, 1996; Phelps, 2006; Zald, 2003). Once again, this evidence will be set out in detail in the following chapters and so I will only note two general points here. First, the amygdala is in an ideal position to moderate the interaction between arousal and cognition. Situated in the anterior portion of the medial temporal lobes (immediately anterior to the hippocampus and adjacent to inferior horn of the lateral ventricle), the amygdala receives sensory information from all modalities, both directly from the thalamus (with the exception of olfactory information which is relayed to the amygdala directly from the olfactory bulbs) and indirectly from cortical areas. Efferent pathways project back to virtually all cortical areas thus allowing the amygdala to modulate sensory representations of information. Efferent pathways also project to many sub-cortical areas such as brain-stem nuclei and the hypothalamus, which are in control of regulating the

homeostatic balance of the autonomic nervous system. In other words, the amygdala sits at the junction between arousal and cognition (see Aggleton, 2000 for a collection of detailed discussions regarding the amygdala). The second point worth a brief comment about the amygdala relates to its involvement in the processes underlying feelings. Here, interactions between the amygdala, the cingulate cortex and frontal areas of the cortex (particularly the orbitofrontal and ventromedial prefrontal areas) are thought to be important (e.g. Damasio, 2003; Lane, 2006). Interestingly, prefrontal areas of the cortex mature much later in life than the amygdala (e.g. Bachevalier & Loveland, 2006; Happaney et al., 2004). Thus, in neurodevelopmental terms, it seems plausible to suggest that young infants are unlikely to experience the kind of feelings an adult would normally associate with emotional experiences. Yet such infants are already equipped with the neural hardware that mediates arousal.

Back to the beginnings

Looking back on the evidence relating to the emotional disturbances that characterise ASD, it becomes apparent that research in this area is restricted, almost exclusively, to the study of the cognitive and behavioural components of emotional experiences within the social domain. This limited range of research makes it impossible to determine whether the emotional processing difficulties arise from abnormalities in social-cognition, cognitive aspects of emotion, arousal responses to emotional stimuli or the interaction between these domains. There is, however, one area of research that I have deliberately ignored until now and that is the study of autonomic arousal in ASD. The decision to delay mention of this evidence is motivated by the fact that it seemed necessary to operationalise the concept of emotions before discussing evidence of arousal in ASD. More importantly, however, this evidence directly informs the principal hypothesis of the current thesis and it seemed natural to conclude the introduction with the formulation of this hypothesis.

It is difficult to summarise the evidence pertaining to arousal responses in ASD in brief, because the studies that have looked at this issue vary widely in scope and methodology and are not always directly concerned with the study of emotion. Table 1.2 includes a brief description of each of the relevant studies to date that have appeared in the

literature since ASD was first officially recognised as a distinct disorder in 1980 (Barry & James, 1988; Ben Shalom, Mostofsky, Hazlett, Goldberg, McLeod & Hoehn-Saric, 2003; Ben Shalom, Mostofsky, Hazlett, Goldberg, Landa, Faran, et al., 2006; Bernier, Dawson, Panagiotides & Webb, 2005; Blair, 1999; Bölte & Poustka, 2003; Corona et al., 1998; Hirstein, Iversen & Ramachandran, 2001; Hubert, Wicker, Monfardini & Deruelle, 2008; James & Barry, 1984; Johnson, Yechiam, Murphy, Queller & Stout, 2006; Kylliäinen & Hietanen 2006; Palkovitz & Wiesenfeld, 1980; Salmond, de Haan, Friston, Gadian & Vargha-Khadem, 2003; Sigman, Dissanayake, Corona & Espinosa, 2003; Stevens & Gruzelier, 1984; van Engeland, 1984; van Engeland, Roelofs, Verbaten & Slangen, 1991; Willemsen-Swinkels, Bakemans-Kranenburg, Buitelaar, van IJzendoorn & van Engeland, 2000). Table 1.2 provides a brief description of the participant groups, sample sizes and matching criteria for each of the studies (CA = chronological age; MA = Mental Age) and sets out a brief description of the stimuli or experimental paradigm employed and what the autonomic and behavioural dependent variables (DVs) were (SCR = Skin Conductance Response; HR = Heart Rate; BP = Blood Pressure; RR = Respiratory Pause; EMG = Electromyogram). Most importantly, however, Table 1.2 summarises the overall outcome of the results. Column 'Mag' indicates whether the *magnitude* of the autonomic responses of participants with ASD were elevated (↑), similar (=) or attenuated (↓) in relation to the comparison groups, or whether subgroups of individuals with ASD needed to be classified in this manner (= ↑). Column 'Diff' indicates whether the autonomic responses of individuals with ASD *differentiated* between different types of stimuli to the same extent (=) or less (↓) than the comparison groups or whether such response differentiation occurred only to some but not all classes of stimuli (= ↓). Column 'Hab' sets out whether participants with ASD *habituated* to the repeated presentation of stimuli to the same extent as the comparison groups (=) or whether they habituated at a slower rate (↓) (habituation is indexed by a decrease in response magnitude as a function of repeated exposure to the same stimulus). And finally, column 'Behav' shows how ASD participants fared on the *behavioural* response measures that were obtained in some studies, with the symbol '≠' indicating group differences and the symbol '=' indicating that groups performed similarly.

Table 1.2

Summary of studies of psychophysiological arousability in ASD

Reference	Participant Groups	*N*	Matching	Stimuli / Paradigm	Autonomic DVs	Behavioural DVs	Results Mag.	Diff.	Hab.	Behav.
Palkovitz & Wiesenfeld (1980)	ASD; Typical	10; 10	CA	tone vs. speech	HR; SCR	NA	↑	=	NA	NA
Stevens & Gruzelier (1984)	ASD; Typical; Dev. Delay	20; 20; 20	CA; MA	tones of different amplitude	SCR	NA	=	=	↓	NA
James & Barry (1984)	ASD; Typical; Dev. Delay	40; 40; 40	CA; MA	tone vs. white squares	HR; SCR; RP	NA	↑	NA	↓	NA
van Engeland (1984)	ASD; Typical; Dev. Delay; Psychiatric	35; 45; 20; 38	CA	tone	SCR	NA	↑	NA	=	NA
Barry & James (1988)	ASD; Typical; Dev. Delay	32; 32; 32	CA; MA	tones of different amplitude vs. images of different complexity	HR; SCR; RP	NA	= ↑	=	↓	NA
van Engeland et al. (1991)	ASD; Typical; Psychiatric	20; 20; 40	CA; MA	images of different complexity vs. Target detection paradigm	SCR	Target detection	↓	NA	=	NA
Corona et al. (1998)	ASD; Dev. Delay	22; 22	CA; MA	simulated distress of adult	HR	NA	↓	NA		NA
Blair (1999)	ASD; Typical; Dev. Delay	20; 20; 20	CA; MA	Distress, Threat & Neutral photographs	SCR	NA	=	= ↓	NA	NA
Willemsen-Swinkels et al (2000)	ASD; Typical; Dev. Delay	32; 19; 19;	CA; MA	separation vs. reunion	HR	Attachment style	=	= ↑	NA	≠
Hirstein et al. (2001)	ASD; Typical	37; 25	Not reported	Eye-contact with mother vs. papercup	SCR	NA	↓	↓	NA	NA
Sigman et al. (2003)	ASD; Dev. Delay	22; 22	CA; MA	video of crying vs. playing infant; interaction with mother; separation vs. reunion	HR	Looks at stimulus & attitude	=	= ↓	NA	≠
Salmond et al. (2003)	ASD; Typical	14; 18	CA	startle response modulation by emotional pictures	EMG	Subjective ratings	=	=	NA	=
Ben Shalom et al. (2003)	ASD; Typical	10; 10	CA	Pleasant, Unpleasant, & Neutral photographs	SCR	Subjective ratings	=	=	NA	≠

Table 1.2 Continued

Reference	Participant Groups	*N*	Matching	Stimuli / Paradigm	Autonomic DVs	Behavioural DVs	Results Mag.	Diff.	Hab.	Behav.
Bernier et al. (2005)	ASD; Typical	14; 14	CA; MA; Anxiety	Fear conditioning paradigm	EMG	NA	=	NA	NA	NA
Kylliäinen & Hietanen (2006)	ASD; Typical	12; 12	CA; MA	direct vs. averted gaze	SCR	Gaze direction detection	=	= ↑	NA	=
Ben Shalom et al. (2006)	ASD; Typical	10; 10	CA	Pleasant, Unpleasant, & Neutral photographs	SCR	Subjective ratings	=	=	NA	≠
Johnson et al. (2006)	ASD; Typical	15; 14	CA; MA	Iowa Gambeling Task	GRS	Card Selections	↓	NA	NA	≠
Bölte et al. (2008)	ASD; Typical	10; 10	CA; MA	Fear, anger, disgust, happiness & sadness eliciting photographs	HR; BP	Subjective ratings	=	=	↓	≠
Hubert et al. (2008)	ASD; Typical	16; 16	CA	Angry vs. Happy Faces; Moving object	SCR	Identification	↓	↓	NA	=

Note: CA (Chronological Age), MA (Mental Age), BP (Blood Pressure), EMG (Electromyogram), HR (Heart Rate), RP(Respiratory Pause), SCR (Skin Conductance Response).

A first glance at the last four columns of Table 1.2 suggests that the observations of the respective studies are rather inconsistent. A closer look, however, reveals some patterns in this inconsistency. More specifically, studies involving simple auditory or visual stimuli tend to show that individuals with ASD respond to such stimuli with heightened physiological responses and reduced habituation but that they nevertheless differentiate between different stimuli to the same extent as comparison groups. By contrast, studies involving more complex and emotionally meaningful stimuli tend to show that individuals with ASD exhibit either typical or reduced response magnitudes but less differentiation between stimuli. Finally, studies that fail to observe group differences in terms of how individuals with and without ASD respond autonomically to emotionally meaningful stimuli, nevertheless indicate that individuals with ASD subjectively experience such stimuli differently from typical individuals (i.e. ratings distinguish groups). Only two studies do not conform to this pattern (Bernier et al., 2005; Salmond et al., 2003) and both of these employed conditioning paradigms that will be discussed in greater detail in Chapter 4.

The evidence summarised in Table 1.2 above suggests two things. First, that stimulus parameters atypically modulate autonomic arousal responses in individuals with ASD and second, that autonomic arousal responses abnormally relate to the cognitive representation of stimulus parameters in individuals with ASD. The latter set of findings, has led Ben-Shalom and colleagues (e.g. Ben Shalom, 2000; Ben Shalom et al., 2003; Ben Shalom et al., 2006) to suggest that ASD is characterised by impairments in feelings but not arousal. Whilst I agree in principle with this suggestion, I believe that it misses the point somewhat, mainly because this idea amounts to a description rather than an explanation of a set of observations. As suggested in the previous section, it is unlikely that neonates have the cognitive capacities necessary to experience feelings because such subjective experiences require relatively mature conceptualisations of the 'Self'. Since such a self concept seems to be acquired throughout early development because of the interaction between arousal and basic forms of cognition, an impairment in feelings in adults, must reflect abnormalities in how arousal and cognition interacted earlier in life. I will return to issues relating to the 'Self' in more detail later. The principal hypothesis I put forward for now, is that the developmental trajectory of individuals with ASD is the result of abnormalities in the interaction between

stimulus induced arousal and cognition and that the neural correlate of this abnormality is a disruption in the connectivity between the amygdala and areas of the brain that mediate aspects of cognition. I will deal with the developmental aspect of this hypothesis in the final chapter of this thesis. The aim of the experimental work presented below is to test one of the main predictions of this hypothesis, namely that cognition and arousal abnormally modulate one another in ASD.

CHAPTER 2: MEMORY FOR EMOTIONALLY SIGNIFICANT STIMULI

Overview

The present chapter will set out three experiments designed to assess the modulatory effect of stimulus induced arousal on memory. Experiment 1 asks whether ASD and typical participants differ in how their memory for emotionally arousing and non-arousing words is affected by the division of attention during a study phase. Experiment 2 employs a free recall paradigm in order to determine whether individuals with ASD, like typical individuals, retain emotionally arousing words better over time than comparable neutral stimuli. And experiment 3 sets out to determine to what extent it is possible to induce false memories of emotionally charged words in individuals with and without ASD. In typically developed individuals, these experimental manipulations help to illustrate that memories of emotionally arousing stimuli not only differ in quantity from those of non-arousing stimuli but that such memories are also retained in qualitatively distinct ways (see Reisberg & Hertel, 2004 for a collection of reviews). Thus, these manipulations provide a sensitive measure of the specific modulatory influence of arousal on memory and of the hypothesis that such modulatory mechanisms may operate abnormally in ASD. Since the influence of arousal on memory is widely known to necessitate the functional integrity of the amygdala (e.g. Hamann, 2001; LaBar, 2007; McGaugh, 2000; Phelps, 2004), the experiments should also provide insight into the possible neural underpinnings of emotional processing abnormalities in ASD.

As mentioned earlier, several experiments of the current thesis (including experiments 2 and 3 of the current chapter) have either been published or are currently under review. Therefore they are presented in the form in which they were submitted for publication, with the exception of minor editorial amendments in order to preserve the format of the thesis (e.g. Abstracts and reference sections have been removed and some terminologies have been standardised). Since it is in the nature of such publications to treat only the most pertinent evidence to the topic under discussion, the respective introductions to the individual experiments provide little background information on general issues such as how the concept

of memory is currently operationalised and what is known about its integrity in ASD. In order to overcome this shortcoming and discuss some general methodological issues, the current chapter will commence with a brief introduction of these more general issues.

Memory in ASD

Memories are the result of learning, which is evident when an organism's behaviour is altered by past experience. Whilst this rather crude description is relatively simple, the underlying processes are complex and multifaceted. First it is important to note that not all memories are alike. Remembering how to ride a bicycle is fundamentally different from knowing what the capital of the UK is, which in turn differs in important ways from recalling one's last visit to the dentist. In addition, some memories only last a brief moment and seem to vanish without a trace, whilst others last a lifetime and cannot be undone even if one tries to forget. The way in which we acquire new memories also varies considerably. Some memories are formed through effortful and conscious attempts to learn (e.g. studying for an exam) whilst others are acquired without much effort or awareness (e.g. recognising your friend's voice). Similarly, the context in which we try to retrieve memories has important consequences for our chances of success in doing so. We might on one occasion struggle to think of the name of a colleague who we met at a recent conference, yet upon our next meeting the same name may materialise on the tip of our tongue without much effort. Differences such as these have led to the suggestion that memory is not a unified phenomenon but rather the result of the operation of separable memory *systems* and *processes* that interact in systematic ways with the properties of what we try to remember and the context in which we try to do so.

As far as memory systems are concerned, one of the first distinctions was proposed by James (1890), who noted that certain memories only last as long as they occupy conscious awareness, whilst others endure even after we have stopped to think about their content. The former is known as *primary, short-term* (STM) or *working memory* (WM), whilst the latter is referred to as *secondary* or *long-term memory* (LTM). As with many of James's

(1890) insights, his considerations on memory turned out to be justified and the study of amnesic patients has provided ample evidence to suggest that the ability to form long-lasting memories is dissociated from the capacity to retain information over a short period of time (Scoville & Milner, 1957; Wickelgren, 1968). Amnesic patients have also stimulated other sub-divisions of memory systems including the differentiation between *procedural* or *implicit* and *declarative* or *explicit* memory (Cohen & Squire, 1980; Warrington & Weiskrantz, 1968). The procedural / implicit memory system allows us to learn new skills, procedures or units of information without the awareness of such learning taking place. Amnesic patients exhibit this ability and have been found to improve in their performance on tasks such as tracking a moving target, reading mirror-reversed words or identifying degraded images that they were shown on previous occasions (e.g. Cohen & Squire, 1980; Corkin, 1968; Graf, Squire & Mandler, 1984; Warrington & Weiskrantz, 1968). The declarative / explicit memory system, on the other hand, endows us with the awareness that we have learned something new and this ability is virtually absent in amnesia (e.g. Cohen & Squire, 1980). Thus, even though such individuals improve their performance on various tasks, they will deny ever having taken part in learning trials. The final commonly cited distinction is one proposed by Tulving (e.g. Tulving, 1985), who suggests that the declarative or explicit memory system is further sub-divided into a *semantic* and an *episodic* one. The former is responsible for the acquisition of factual knowledge about the world such as the boiling point of water or the name of a UK prime minister. Tulving (2002) calls these memories 'timeless facts', because they are associated with a sense of awareness (what he calls 'noetic' awareness) that does not recreate the spatial-temporal context, which places the 'self' at the centre of the remembered experience. This latter kind of awareness, what Tulving (1985, 2002) calls 'autonoetic' awareness, is what makes episodic memories special and it is this self awareness in memory that led Tulving (2002) to liken episodic memories to mental time-travel (see Gardiner, 2001 for a review of evidence pertaining to the distinction between semantic and episodic memory).

In addition to distinguishing among various memory systems, cognitive scientists also differentiate among various memory *processes*, the most important of which are *encoding, consolidation, forgetting* and *retrieval* processes. Crudely put, encoding processes are

responsible for transforming sensory information into memory representations, whilst retrieval processes are involved in recovering these representations for the purposes of guiding behaviour. Consolidation and forgetting, on the other hand, alter the quality or strength of memory representations, with consolidation resulting in longer-lasting and more robust memories whilst forgetting weakens or decays them (see Roediger, Gallo & Geraci, 2002; Wixted, 2004; Wixted, 2005 for relevant reviews). Although the way in which these processes work and how they interact with one another is rather complex, several properties of their operation are relatively well established. For instance, it is widely accepted that encoding processes operate at different levels such that different properties of a stimulus are encoded through separate processing channels (e.g. Craik & Lockhart, 1972; Craik & Tulving, 1975; Roediger, et al., 2002). In itself this is not surprising given that early stages of sensory processing operate to a large degree independently from one another. Interestingly, however, the extent of encoding at different levels can be manipulated by instructing participants to pay particular attention to a certain stimulus property (e.g. meaning of a word, spelling of a word, etc.) and such 'orienting tasks' alter the ease with which participants can later retrieve the stimulus. More specifically, orienting tasks that direct a participant's attention toward conceptual properties of a stimulus (e.g. its meaning or conceptual relations to other stimuli) generally result in better performance on a subsequent memory test than orienting tasks that direct attention toward more perceptually anchored properties of the stimulus such as its graphemic composition (e.g. Bower, Clark, Lesgold & Winzenz, 1968; Craik & Tulving, 1975; Hunt & Seta, 1984). Another well established phenomenon is the observation that the retrieval success of a particular memory depends not only on how it is encoded but also on how suitable the encoded information is for a particular retrieval context (e.g. Morris, Bransford & Franks, 1977; Nairne, 2002; Tulving & Thomson, 1973). For instance, if one learns a list of words by thinking about rhymes of these words, the presentation of such rhymes during a memory test will make it easier to retrieve the studied words than the presentation of semantically related words (e.g. Morris et al., 1977). Similarly, if one learns a set of stimuli in a particular location it is usually easier to retrieve these stimuli in the same location rather than another (e.g. Godden & Baddeley, 1975). Other interactions between memory processes are evidenced by interference effects whereby the encoding of a set of

stimuli hinders the consolidation (or retrieval) of a similar set of stimuli encoded subsequently (e.g. Gershberg & Shimamura, 1995).

The foregoing description, despite by no means complete, paints a rather complex picture of what memory entails. It is important to note, however, that the various distinctions between memory systems and processes outlined above are not mutually exclusive. Nor are they necessarily compatible with one another. They reflect the views of different authors and represent attempts to operationalise the concept of memory in order to account for, and scientifically study, its phenomenology. In relation to ASD, this theoretical heterogeneity is very useful because it provides a rich heuristic for studying how such individuals form lasting impressions of their interactions with their environment, which in turn offers insights into their inner mental experiences (see Bowler & Gaigg, 2008). It is within this context that the study of memory in ASD has enjoyed a long and prosperous history, a history that has recently been celebrated with the publication of an edited book dedicated to this topic alone (Boucher & Bowler, 2008). The purpose of the current discussion is not to provide a summary of the extensive literature in this field but rather to highlight certain aspects of memory functioning in ASD that raise important methodological issues for the experiments presented below. As already mentioned, the principal aim of the following three experiments is to determine whether stimulus-induced arousal modulates memory processes in ASD in the same way as in typical individuals. The methodological challenge that presents itself in this context is two-fold. First, it is important to devise experimental paradigms that assess the specific effects of stimulus induced arousal on memory. In other words, any quantitative or qualitative difference in memory for emotionally arousing compared to non-arousing stimuli must reflect the modulatory influence of arousal on memory and not of other factors. Although this is not an easy task, scientists interested in the influence of arousal on memory have devised rather clever experimental paradigms to achieve this goal and these paradigms serve as the basis for the experiments presented below. The more problematic methodological concern, however, is to ensure that the experimental paradigm assesses how arousal modulates those kinds of memory functions that operate similarly in ASD and typically developed individuals. If not, it is difficult to rule out the possibility that differences (or even similarities) in how arousal

modulates memory in ASD and typically developed individuals are the result of group differences in the operation of generic memory processes or systems.

The main difficulty in satisfying the latter of the two aforementioned criteria, is the fact that it is very difficult to know whether conceptualisations of memory in typically developed individuals are suitable for describing the phenomenology of memory in ASD (see Mottron, Dawson & Soulières, 2008 for a more detailed consideration of this issue). In order to demonstrate this problem more concretely and illustrate its relevance to the subsequent experimental studies, consider the well established finding that typically developed individuals remember semantically and conceptually more coherent information better than information that is less meaningful[10]. Regardless of how such meaning is manipulated or how the relevant information is presented, and regardless of the conditions under which individuals learn or try to retrieve the information, meaning almost always facilitates memory (e.g. see Gardiner, 1976 for a collection of relevant reviews and also Morris et al., 1977). In relation to studying the effects of arousal on memory, this raises the possibility that arousing stimuli are remembered differently from non-arousing ones simply because they differ with respect to their meaning (e.g. Talmi, Luk, MacGarry, & Moscovitch, 2007; Talmi & Moscovitch, 2004). One way to control for this possibility is to try to match arousing and non-arousing stimuli in terms of their meaningfulness, which is not always easy since 'meaningfulness' is a rather vague concept. In ASD, however, the issue becomes even more complicated because meaning, loosely defined, influences memory differently in this population.

Investigations of free recall performance, where individuals are simply asked to reproduce what they can remember from a previously presented set of stimuli, often show that individuals from across the autism spectrum do not benefit from meaning as much as non-autistic comparison groups. For instance, numerous studies have assessed free recall performance for word sequences that are either arranged randomly or according to the syntactic and conceptual rules of language, and found that whereas children without ASD recalled the latter sequences better than the former, children with ASD did not (e.g. Hermelin

[10] The term meaning here is used loosely to describe the meaning of the information *per se* as well as it's meaningfulness within the context of the task (see Morris et al., 1977 for a more detailed discussion of the concept of 'meaning' in the context of memory)

& O'Connor, 1967; Hermelin & O'Connor, 1970; O'Connor & Hermelin, 1967). Similarly, when memory for a set of semantically interrelated words is compared to that for a set of unrelated words, individuals from across the autism spectrum exhibit a much lower recall advantage for the semantically related words than comparison participants (e.g. Bowler, Matthews & Gardiner, 1997; Bowler, Gardiner, Grice & Saavalainen, 2000; Tager-Flusberg, 1991). In addition, individuals with ASD tend to cluster (i.e. recall in sequence) semantically interrelated words less during their recall attempts (e.g. Gaigg, Gardiner & Bowler, 2008; Minshew & Goldstein, 1993) and even when no obvious relations exist among to-be-remembered stimuli individuals with ASD tend to cluster stimuli differently from non-autistic individuals (Bowler, Gaigg & Gardiner, 2008a). If such observations were consistent, one could conclude that meaning does not influence memory in ASD and therefore attempt to design an experimental paradigm that assesses the influence of arousal on memory whilst placing few demands on the processing of meaning. Unfortunately, however, things are not that simple. First, not all studies indicate that free recall performance in ASD is atypically modulated by semantic interrelations amongst stimuli, particularly when pictorial rather than verbal material is used (e.g. López & Leekam, 2003; Ameli, Courchesne, Lincoln, Kaufman & Grillon, 1988). More importantly, studies that employ recognition or cued recall measures of memory generally fail to demonstrate abnormalities in how semantic relations amongst stimuli influence memory in ASD (e.g. Boucher & Warrington, 1979; Bowler, Gaigg & Gardiner, 2008b). Individuals with ASD have also been found to benefit as much as typical participants from orienting tasks that direct attention to semantic aspects of information (Bowler, et al., 1997; Gaigg et al., 2008; Mottron, Morasse & Belleville, 2001; Toichi & Kamio, 2002) and such individuals have been found to be subject to semantically induced memory illusions (Bowler et al., 2000; Beversdorf, Smith, Crucian, Anderson, Keillor, Barrett, et al., 2000), which are false memories of a non-presented word that participants frequently report after having studied a number of words that are highly associated with this illusory item (e.g. bed, pillow, night, dream... for sleep). In short, a vague concept such as 'meaning' seems of little use in trying to specify the memory difficulties experienced by individuals with ASD.

One might wonder why the above analysis poses a problem in relation to studying the effects of arousal on memory in ASD. If there is any doubt as to how meaning influences

memory, then controlling for it experimentally is surely a good option. Of course this is true but recognising that meaning influences memory differently in ASD without actually explaining the patterning of memory functioning in this condition highlights the fact that controlling for meaning alone is not necessarily sufficient to rule out the possibility that generic memory difficulties result in abnormalities in how arousal influences memory in ASD. In order to try and specify the kind of memory processes that pose difficulties for individuals with ASD, it is useful to take a closer look at what the different tasks outlined above require of the participant. Such an analysis has led my colleagues and I (Bowler & Gaigg, 2008; Bowler, Gaigg & Lind, in press; Gaigg, et al., 2008) to suggest that individuals with ASD experience difficulties in processing *relational* information whilst their ability to process *item-specific* information is preserved and possibly even enhanced. Relational processing, refers to the processing of a stimulus in relation to its context, which can include other stimuli, the time of day or the location in which the stimulus is encountered. Item-specific processing, on the other hand describes the processing of information specific to the stimulus such as its physical, semantic or conceptual properties. This distinction has been used to describe several phenomena in the typical memory literature (e.g. Anderson & Bower, 1972; Hunt & McDaniel, 1993) and the patterning of memory functioning in ASD is consistent with the idea that such individuals process information more in an item-specific rather than a relational fashion (see Bowler, et al., in press for a detailed discussion of the relevant evidence). For instance, tasks on which meaning influences memory relatively typically in ASD (i.e. cued recall, recognition, levels of processing & memory illusions) rely more heavily on item-specific rather than relational processing whereas the opposite is true for tasks that pose some difficulty for individuals with ASD. The distinction between item-specific and relational processing also accommodates the fact that individuals with ASD experience difficulties on tasks assessing episodic memory (e.g. Bowler, Gardiner & Gaigg, 2007; Bowler, Gardiner & Grice, 2000; Lind & Bowler, 2008; Toichi, 2008), which require that information is processed *in relation* to temporal and contextual information that define a particular episode. Of course there is a certain danger of circularity in this argument whereby any task that poses difficulties for individuals with ASD may be said to involve relational processing. Nevertheless, direct tests of the hypothesis have so far yielded affirmative results. Thus, when individuals with

ASD are simply asked to learn a list of words that consists of varying numbers of items from different categories, their performance is not only overall worse than that of a comparison group, but they experience disproportionate difficulties recalling words that are not obviously related to one another whereas their recall of words that are more obviously related to one another is less affected (Gaigg et al., 2008). This pattern of results is in line with the suggestion that individuals with ASD experience disproportionate difficulties in relational as compared to item-specific processing (see Hunt & Seta, 1984).

Based on the view presented above, the following three experiments were designed in order to minimize demands on relational processing by either employing recognition test procedures or item-specific encoding instructions. In addition, three very different experimental paradigms were employed in order to ensure that findings would not be confounded by specific task demands. An attempt was also made to control for the possible influence of semantic properties on memory, which not only minimized the chance of confounding between group comparisons but also helped to dissociate the specific influence of arousal on memory. In relation to this last point, the experimental paradigms also employed within-group manipulations that would influence memory for arousing and non-arousing material differentially, thereby lending further support to the notion that arousing stimuli are remembered in qualitatively distinct ways.

Experiment 1: The effect of dividing attention on memory for emotionally arousing and non-arousing words in Autism Spectrum Disorder

Introduction

Although it is now well established that abnormalities in socio-emotional conduct constitute a clinically significant manifestation of ASD, only very few studies have to date assessed the emotional competencies of such individuals outside the broader context of social cognition. This is somewhat surprising given the long standing debate about the

developmental significance of socio-emotional disturbances in this disorder (e.g. Baron-Cohen, 1995; Hobson, 2002; Loveland, 2005) and the related question of what role abnormalities of the amygdala play in the pathology underlying ASD (e.g. Bachevalier & Loveland, 2006; Baron-Cohen et al., 2000; Schultz, 2005). Studying emotional processes outside the context of social cognition could provide valuable insights into both of these issues because it would help clarify to what extent emotional abnormalities are specific to the social domain and because the role of the amygdala in certain non-social emotional processes is well understood.

In the memory literature, one of the most consistent observations is the finding that emotionally significant stimuli and events (as long as they do not elicit severe distress) are better remembered and for longer retained than comparable neutral ones (see Reisberg & Hertel, 2004; Uttl, Ohta & Siegenthaler, 2006 for a collection of reviews). The extensive research of this phenomenon suggests that two properties of emotional information are responsible for making it more memorable. The emotional *valence* of a stimulus and the degree of *arousal* it elicits. Valence refers to whether a stimulus is perceived as positive or negative and this dimension is thought to facilitate memory through relatively generic processes such as promoting deeper and more elaborate levels of processing (e.g. Buchanan, Etzel, Adolphs & Tranel, 2006; Kensinger & Corkin, 2004; Maratos, Allan & Rugg, 2000; Talmi & Moscovitch, 2004). The *arousal* response elicited by certain emotional stimuli refers to changes in autonomic functioning that can either be directly observed (e.g. monitoring heart rate) or inferred from subjective ratings and an extensive body of evidence indicates that this dimension enhances memory through a relatively specialized neural circuitry that centres around the amygdala (e.g. Buchanan & Lovallo, 2001; Buchanan et al., 2006; Hamann, 2001; Kensinger & Corkin, 2004; McGaugh, 2002; Paré, Collins & Pelletier, 2002; Phelps, 2004). Given the widely held belief that abnormalities of the amygdala play an important role in the neuropathology underlying ASD (e.g. Bachevalier & Loveland, 2006), it is easy to see how studies of the influence of arousal on memory could prove fruitful for our understanding of the disorder.

To date, only two studies have investigated memory for emotionally significant stimuli in ASD. In the earliest of these, Beversdorf and colleagues (Beversdorf, Anderson, Manning, Anderson, Dordgren, Felopulos, et al., 1998) asked a group of adults with and without a diagnosis of ASD to try to remember a series of emotionally charged and neutral statements (e.g. 'He talks about death' *vs.* 'He is talking with his roommate') for a subsequent free recall test. The results of this test showed that only the comparison group recalled the emotionally charged statements significantly better than the neutral ones, despite the fact that groups did not differ in terms of their memory for sentences and paragraphs that varied in terms of their syntactic and conceptual coherence or mentalistic content. A more recent study by South and colleagues (South, Ozonoff, Suchy, Kesner, McMahon & Lainhart, 2008) failed to support these observations. These authors presented participants with a list of words containing emotionally charged and neutral items and found that both ASD and typical participants recognised significantly more of the emotional words on a subsequent 'Yes/No' recognition test.

It is important to note that neither of the experimental paradigms used in the studies described above was designed to dissociate the relative effects of valence and arousal on memory. Thus, it is difficult to know to what extent the observations should be interpreted in relation to the kinds of processes that are engaged when emotional stimuli elicit arousal, and in how far the findings may best be described with reference to the more generic memory processes that underlie the influence of valence on memory. Based on current evidence and the particular methodologies employed by Beversdorf et al. (1998) and South et al. (2008), it seems that the discrepant findings can be explained purely in terms of the operation of relatively generic memory processes. More specifically, Beversdorf et al. (1998) employed a free recall paradigm, and it is generally accepted that such test procedures pose more difficulty for individuals with ASD than the kind of recognition test employed by South et al. (2008) (see Bowler & Gaigg, 2008 for a more detailed discussion on free recall and recognition performance in ASD). More importantly, a recent study by Bowler, et al. (2008b) showed that individuals with ASD benefit from semantic relations amongst to-be-remembered words and incidentally encoded context words only on a test of recognition whereas typical participants benefit from such relations on tests of recognition and free recall. This suggests

that certain properties of stimuli that normally enhance memory by promoting elaborated processing only do so in ASD when test procedures offer some retrieval support. Within this Task Support framework (e.g. Bowler, Gardiner & Berthollier, 2004), the difference in findings between Beversdorf et al. (1998) and South et al. (2008) can be accommodated without invoking either the amygdala or any other emotion specific process.

The foregoing analysis shows that it is important to employ experimental paradigms that assess the specific influence of arousal on memory in order to determine whether abnormalities in emotional processes (and associated amygdala functions) generalize to this domain of cognition in ASD. An experiment by Kensinger and Corking (2004) offers such a paradigm. These authors showed that the influence of valence and arousal on memory can be dissociated by manipulating participants' attentional resources whilst they are trying to remember a set of words. In their experiment participants studied words varying in emotional arousal and emotional valence either during a full-attention condition or a divided-attention condition. During the former, participants simply tried to remember the words but during the divided-attention condition participants were also required to monitor a sequence of tones and press a button whenever they heard a change in pitch. During subsequent recognition tests, participants' performance for emotionally arousing words was consistently above 80%, regardless of whether an easy, difficult or no concurrent tone-monitoring task needed to be performed during study. Performance for valenced and neutral words on the other hand declined as the concurrent task during study increased in difficulty. This pattern confirmed that valenced and neutral words are encoded through qualitatively similar processes, whilst arousing words are encoded through qualitatively distinct and relatively automatic processes. That the latter processes involved the amygdala was supported by functional imaging data, which showed that the encoding of valenced words was associated with activity in a prefrontal cortex-hippocampal network whilst the encoding of arousing words was associated with activity in an amygdala-hippocampal network (Kensinger & Corkin, 2004).

The current experiment employs a paradigm similar to the one used by Kensinger and Corkin (2004) in order to test the hypothesis that arousal modulates memory abnormally in ASD. Based on the finding that manipulations of dividing attention moderate recognition

performance similarly in ASD and typical participants (Bowler, et al., 2007), it was predicted that overall recognition levels would be comparable between ASD and typical participants. However, based on the hypothesis that the influence of arousal on memory operates atypically in ASD, it was expected that dividing attention at study would not differentially effect subsequent recognition performance for arousing, valenced and neutral words in this disorder. If these predictions are borne out, it would lend support to the suggestion that emotional processing abnormalities extend to domains outside the broader context of social-cognition in ASD.

Method

Participants

Sixteen ASD (13 male, 3 female) and sixteen typical individuals (12 male, 4 female) were recruited for the current experiment and paid standard University fees for their participation. ASD participants were diagnosed according to conventional criteria and a review of available medical records and/or assessment with the Autism Diagnostic Observation Schedule (ADOS, Lord et al., 1989) confirmed that all met current diagnostic criteria for an Autism Spectrum Disorder (DSM-IV-TR; American Psychiatric Association, 2000). Developmental histories of sufficient detail to show that a diagnosis was made according to the DSM criteria were available for 7 of the 16 ASD participants. For the remaining 9 participants, ADOS scores confirmed that all met relevant cut-off points consistent with an ASD diagnosis (these participants had also provided brief statements of their diagnosis). Typical individuals were recruited from the local community through newspaper advertisements and individually matched to ASD participants to within 7 points of Verbal IQ (Wechsler Adult Intelligence Scale-III^{UK}, The Psychological Corporation, 2000). Groups were also matched in terms of chronological age, Performance IQ and Full-scale IQ (see Table 2.1). Brief interviews with all participants ensured that none had any medical history of psychiatric conditions other than ASD and all participants reported being free of medication. The experimental procedures described below were approved by the University's ethical committee and adhered to the guidelines set out by the British Psychological Society.

Table 2.1

Summary of Age and IQ characteristics for the ASD and Comparison Group

	ASD (n = 16)		Comparison (n = 16)	
Measure	*M*	*SD*	*M*	*SD*
Age (years)	34.2	14.1	32.1	11.2
VIQ[a]	106.2	15.4	105.5	15.1
PIQ[b]	106.7	16.6	104.8	13.2
FIQ[c]	106.8	16.6	105.4	14.8

[a] Verbal IQ (WAIS-R^{UK} or WAIS-III^{UK})

[b] Performance IQ (WAIS-R^{UK} or WAIS-III^{UK})

[c] Full-Scale IQ (WAIS-R^{UK} or WAIS-III^{UK})

Materials and Design

The stimuli for the current experiment included a set of 36 emotionally arousing words (hereafter 'arousing'), 36 emotionally valenced but non-arousing words (hereafter 'valenced'), and 36 categorically related words. The latter were chosen from the Hampton and Gardiner (1983) category norms and included the most typical members of the 'Clothing' category. Typical category members were chosen in order to maximise the semantic cohesiveness of this group of words and thereby rule out the possibility that any memory advantage for arousing over non-arousing words would be the result of the former being semantically highly interrelated. The arousing and valenced words were selected from two sources. 12 words were selected from the Affective Norms for English Words (ANEW, Bradley & Lang, 1999), which provide normative data for ratings of emotional arousal and valence for over 1000 words. Since this data set includes few taboo, offensive or sexually explicit words that one would expect to be amongst the most arousing, however, the majority of emotional words were selected from a pool of 130 words for which normative data were

gathered separately. During this normative study 49 undergraduate students (35 female, 14 male) rated each of 130 words that included a set of profanities, sexually explicit words and taboo words on four dimensions – familiarity, imageability, emotional valence and emotional arousal – using 9 point rating scales. High familiarity scores indicated that the meaning of a word was immediately obvious whilst low scores indicated that some thought was required to bring the meaning of the word to mind. Similarly, high scores on the imageability dimension were used when a word immediately brought a picture to mind whereas low scores were used when some effort was required to picture what a word described. The emotional valence dimension was rated according to whether a word was positive or negative in meaning, with low scores reflecting the former and high scores the latter. Finally, high ratings on the emotional arousal dimension were used to indicate that a word elicited a physical emotional reaction whilst low scores were used when this was not the case. It was made clear to students in this rating task that the emotional valence and emotional arousal dimensions were not necessarily related to one another and that the emotional arousal dimension should reflect the actual physical responses experienced when reading the word rather than the physical responses that might be elicited by the object or event described.

Since the principal aim of the current study was to assess the specific effects of emotional arousal on memory, the arousing and valenced words selected for the current experiment were closely matched in terms of letter length (arousing M = 5.22, SD = 1.67; valenced M = 5.39, SD = 1.25; t = 0.48, df = 70, *ns*) ratings of familiarity (arousing M = 7.58, SD = 0.41; valenced M = 7.76, SD = 0.59; t = 1.48, df = 70, *ns*), imageability (arousing M = 6.21, SD = 0.87; valenced M = 6.00, SD = 1.70; t = .68, df = 70, *ns*) and valence (arousing M = 5.95, SD = 1.38; valenced M = 5.73, SD = 1.93; t = .55, df = 70, *ns*)[11]. For valence, it was also ensured that the number of positive (n = 8) and negative words (n = 16) in the arousing and valenced category was equal. Obviously ratings of arousal differentiated the two types of words (arousing M = 5.46, SD = 0.49; valenced M = 5.07, SD = 0.76; t = 2.56, df = 70, p < .05) although it is important to note that the close matching procedure meant that there was

[11] It should be noted that familiarity ratings and imageability ratings were not available on 9-point scales for the words taken from the ANEW data set. Relevant ratings for these words were therefore estimated on the basis of the norms provided by the University of Western Australia MRC Psycholinguistic Database (Coltheart, 1981).

considerable overlap on this dimension (range for arousing words 4.58 – 6.47; range for valenced words 3.32 – 6.50).

The final set of words was split into 3 sub-sets including 12 arousing, 12 valenced and 12 categorically related words each. Sets A and B always served as the 'Target' words that participants would be required to learn during the full-attention and divided-attention conditions, whilst half of the words of set C (6 or each type) was used as 'Lures' during each of the two recognition tests. By not counterbalancing Targets and Lures it was possible to select Target words that were maximally distinct in terms of arousal whilst remaining closely matched in terms of all other dimensions. During the study phase, words were presented in Bold, 48 point, Times New Roman font in the centre of a Sony Laptop monitor. The rate of presentation was set at 2700 ms per word with no inter-stimulus interval. The order of words within each set was randomised with the constraint that no more than two words of the same type would occur in sequence. During the divided attention condition, a sequence of high-frequency (1575 Hz) and low-frequency (788 Hz) ping tones was continuously played through speakers at a rate of approximately 1 tone per second. The sequence was constructed to produce 13 changes in pitch at random intervals throughout word presentation. Word sets A and B were counterbalanced across the full-attention and divided attention conditions and the order of these conditions was counterbalanced across participants. Finally, during each of the recognition tests all Targets from the studied set and half of the 'Lures' from set C were presented in the same format as during study but remained on the screen until participants gave their responses.

Procedure

Participants were tested individually in a quiet room and fully briefed before the start of the experiment. They were told that they would be asked to try to remember two sets of words and that their memory for each set would be tested through a recognition test in which they had to decide whether they had seen certain words before or not. Participants were further informed that they would be studying one set of words without any distraction but that they would be studying the other set whilst trying to monitor a sequence of sounds for

changes in pitch. Finally, participants were warned that some of the words they would be studying would be rude, offensive and sexually explicit and that they should not take part in the study if they preferred not to be exposed to such materials.

After participants gave their consent, they were familiarised with the divided-attention procedure. First, they were presented with several tone sequences and asked to press the space-bar every time they heard the tone change. After this initial induction procedure, participants were required to monitor another tone sequence for pitch changes whilst reading a series of 16 common words (e.g. house, window, cat) aloud. Since all participants performed near ceiling on these practice trials, no further training was needed. The two experimental tasks followed the practice trials with half of the participants starting with the divided-attention condition whilst the other half commenced with the full-attention condition. A recognition test immediately followed each encoding phase and the two encoding conditions were separated by approximately 1 hour during which participants were either taken for lunch or asked to take part in an unrelated experiment.

Results

Performance on the divided attention task was near ceiling for both groups, with ASD participants responding accurately on 94% of trials and typical comparison individuals on 96% (t = 0.80, df = 30, *ns*). The results from the recognition tests are set out in Table 2.2, which provides average proportions of 'yes' responses to Targets and Lures as a function of participant group (ASD vs. Typical), encoding condition (full vs. divided attention) and word type (arousing vs. valenced vs. category member). Also set out in Table 2.2 are corrected recognition rates (i.e. Target 'yes' minus Lure 'yes' proportions), which provide an estimate of recognition memory that is uncontaminated by participant's response biases (i.e. their tendency to respond 'yes'). A 2 (Group) x 2 (Encoding Condition) x 3 (Word Type) mixed ANOVA of corrected recognition rates revealed main effects for Encoding Condition (F(1,30) = 10.34, p < .01) and Word Type (F(2,29) = 17.22, p < .001) but no other main effects or interactions (all Fs < .65). Thus, although performance during the divided-attention condition (M = .66, SD = .15) was worse than during the full-attention condition (M = .76, SD = .15) and

despite the fact that arousing words (M = .82, SD = .13) were better remembered than the valenced (M = .64, SD = .19; t = 5.51, df = 31, p < .001) and categorically related words (M = .67, SD = .16; t = 5.01, df = 31, p < .001), the current findings failed to replicate Kensinger and Corkin's (2004) observation that memory for arousing words is relatively immune to the effects of dividing attention at study.

Table 2.2

Average recognition proportions for the ASD and Comparison Group as a function of Encoding Condition and Word Type (values in parenthesis reflect standard deviations)

	ASD (n = 16)			Comparison (n = 16)		
	Target	Lure	Corrected	Target	Lure	Corrected
Full attention						
Arousing	0.88 (0.14)	0.03 (0.09)	0.85 (0.15)	0.88 (0.14)	0.01 (0.04)	0.87 (0.14)
Valenced	0.73 (0.20)	0.07 (0.12)	0.66 (0.19)	0.74 (0.21)	0.06 (0.10)	0.68 (0.23)
Category	0.82 (0.18)	0.06 (0.12)	0.76 (0.22)	0.79 (0.21)	0.06 (0.10)	0.72 (0.21)
Divided attention						
Arousing	0.82 (0.17)	0.04 (0.11)	0.78 (0.17)	0.86 (0.19)	0.09 (0.10)	0.77 (0.18)
Valenced	0.71 (0.24)	0.09 (0.17)	0.61 (0.24)	0.69 (0.24)	0.10 (0.15)	0.59 (0.25)
Category	0.71 (0.23)	0.07 (0.12)	0.64 (0.21)	0.72 (0.22)	0.13 (0.16)	0.60 (0.17)

One possible reason for the current failure to replicate the findings by Kensinger and Corkin (2004) in full is the fact that the experimental materials used here were not only matched in terms of valence but also in terms of other stimulus dimensions known to influence memory (i.e. familiarity, imageability and semantic relatedness). In order to determine whether these stringent matching criteria may have contributed to the difference in findings, the data were re-analysed after re-classifying arousing and valenced words

according to ratings of arousal alone. That is, memory for the 24 most arousing words was compared to that for the 24 least arousing words regardless of how these words were matched according to the other stimulus dimensions (in fact high-arousal and low-arousal words only differed in terms of imageability, with the former being rated as more imageable than the latter). According to this re-classification, 10 previously valenced words were now included as high-arousal words. A 2 (Group) x 2 (Encoding Condition) x 3 (Word Type) mixed ANOVA of the resulting corrected recognition rates, however, again only yielded main effects for Word Type ($F(2,29) = 7.44$, $p < .01$) and Encoding Condition ($F(1,30) = 9.50$, $p < .01$) but no other main effects or interaction (Fs < 0.57). Average corrected recognition rates during the full-attention condition were 0.80 ($SD = 0.18$) for high-arousal words and 0.72 ($SD = 0.17$) for low-arousal words whilst during the divided-attention condition the respective values were 0.73 ($SD = 0.17$) and 0.65 ($SD = 0.23$). Recognition rates for categorically related words were not influenced by the re-classification of the emotional words (i.e. see Table 2.2 for values). Somewhat surprisingly, a nearly identical pattern of results also emerged when emotional words were re-classified according to the valence dimension alone (i.e. most negative vs. least negative; the latter were also rated as significantly more familiar). This re-classification resulted in 12 originally valenced and 12 originally arousing words being included in the most and least negative categories each. Again statistical analyses revealed main effects for Word Type ($F(2,20) = 13.56$, $p < .001$) and Encoding Condition ($F(1,30) = 10.55$, $p < .01$) but no other main effects or interactions (Fs < 0.30). Average recognition rates for this re-classification of words were 0.80 ($SD = 0.17$) for the most and 0.73 ($SD = 0.17$) for the least negative words during the full-attention condition and 0.75 ($SD = 0.16$) and 0.63 ($SD = 0.22$) respectively for the divided-attention condition. Thus, regardless of whether emotional words were classified according to arousal ratings or valence ratings, the most extreme words on either of these dimensions were remembered better than all other words. No evidence was uncovered, however, to suggest that arousal modulates memory through qualitatively distinct processes in the current paradigm.

Before turning to a discussion of the current findings, it is important to point out that the failure to replicate Kensinger and Corkin's (2004) observations in full was somewhat surprising because overall recognition levels and the main effects of Word Type and

Encoding Condition in the current study were comparable to those observed by these authors. Recognition rates in the Kensinger and Corkin (2004) study varied between 60% and 88% across the various experimental conditions, which is nearly identical to the values observed here (see Table 2.2). In addition, inspection of individual data ruled out the possibility that a minority of individuals in the current sample performed in an unusual way that may have obscured the expected interaction effect. Most importantly, however, a pilot study of 7 undergraduate students had yielded this interaction between Word Type and Encoding Condition ($F(2,5) = 4.54$, $p = .07$; effect size $r = .69$), making it extremely unlikely that the materials and procedures employed in the present study were not sensitive to the effects observed by Kensinger & Corkin (2004).

Discussion

The purpose of the present experiment was to assess the specific effect of arousal on memory in individuals with ASD, in order to gain insight into the nature of emotional processing difficulties evident in this condition. For this purpose an experimental paradigm used by Kensinger and Corkin (2004) was employed, which, in the original publication, showed that arousing words are not only better remembered than non-arousing ones but that the acquisition of such memories is also relatively unaffected by dividing participant's attention at study. The demonstration of such differences at both the quantitative and qualitative level, together with extensive neuroscientific evidence, lends strong support to the idea that memories of arousing stimuli are mediated by distinct cognitive and neural mechanisms (e.g. Hamann, 2001; McGaugh, 2002).

Although the current experiment confirmed that arousing words are better remembered than non-arousing words, it failed to replicate the finding that the formation of such memories is relatively immune to the effects of dividing attention at study. Moreover, memory was found to be superior for emotional words regardless of whether such words were classified according to their ratings of arousal or their ratings of valence. Thus, the present findings yielded no evidence to suggest that arousal modulates memory through

emotion specific cognitive or neural mechanisms, making it difficult to formulate conclusions regarding the functional integrity of such mechanisms in ASD.

Despite the failure to replicate the findings by Kensinger and Corkin (2004) in full, the current observations nevertheless provide a useful extension to previous research regarding memory for emotionally significant stimuli in ASD. More specifically, the current findings replicate the observations by South et al. (2008) who found that individuals with ASD exhibit a relatively typical recognition memory advantage for emotionally significant compared to neutral words. In addition, the present results show that this memory advantage prevails when emotional and neutral words are closely matched on other (non-emotional) dimensions known to influence memory. In particular, the current findings rule out the possibility that individuals with ASD may remember emotionally significant words better than neutral words simply because the former are semantically more interrelated. Thus, similar to typical individuals, those with a diagnosis of ASD seem to exhibit a relatively specific memory enhancement for emotionally significant information. Whether this memory enhancement reflects the operation of qualitatively similar processes in both groups, and whether these processes are emotion-specific and mediated by the amygdala, however, remains uncertain.

Experiment 2: Free recall and forgetting of emotionally arousing words in Autism Spectrum Disorder

Experiment 1 demonstrated that individuals with ASD exhibit a rather typical recognition advantage for emotionally significant words but failed to confirm that this recognition advantage was the result of the operation of amygdala mediated memory modulation processes. One possibility for the failure of Experiment 1 to replicate the findings by Kensinger and Corkin (2004) in full, is that participants in that experiment did not actually experience arousing words as truly arousing. That is, participants may not have had differential physiological reactions to the arousing compared to non-arousing words. The present study addresses this question by drawing on an experimental paradigm that allows

for the direct assessment of physiological responses to words differing in subjective ratings of arousal. Assessing such physiological responses places certain constraints on the experimental paradigm because they take place over a period of several seconds. Thus, the presentation of stimuli has to be rather slow, which gives participants ample opportunity to process the to-be-remembered stimuli in a variety of ways. In relation to the principal aim of the current experiments, this increases the possibility that differences in performance between ASD and typical participants may arise because the two groups engage in different encoding processes. In order to overcome this difficulty, the following experiment employed an incidental memory paradigm (i.e. participants were not instructed to try to remember the words) in which participants were simply asked to rate each word in terms of how aroused they felt by it. Such rating instructions have the additional advantage of encouraging item-specific rather than relational encoding processes, which, as noted earlier, seem to operate typically in ASD. This experiment has been published in *Neuropsychologia* (Gaigg & Bowler, 2008) and it should be noted that the study by South et al. (2008) appeared in the literature after the present study was accepted for publication. As a result, the study by South et al. (2008) was acknowledged in a note added during the revision process rather than in the introduction of the manuscript.

Introduction

Since the first descriptions of individuals with ASD, it has been noted that marked abnormalities in affective behaviours constitute a prominent feature of their behavioural manifestations. These include the limited sharing of affect (Kasari, et al., 1990; Yirmiya, et al., 1992), restricted and inflexible use of context appropriate emotional expressions (Dawson, et al., 1990; Kasari, et al., 1993; Macdonald, et al., 1989; Sigman, et al., 1992; Yirmiya, et al., 1989) and difficulties in the perception and recognition of emotional expressions in others (Hobson, 1986a,b; Hobson, et al., 1988a,b; Hobson, 1991; Weeks & Hobson, 1987; but see Castelli, 2005 for contrasting findings). Although these atypicalities constitute a diagnostically relevant feature of ASD (American Psychiatric Association, 2000; World Health Organisation, 1992), their causes remain the matter of debate. Some authors maintain that disordered

emotional behaviours may be a secondary manifestation of impairments in other social capacities such as face-processing (Schultz, 2005) or 'Theory of Mind' (ToM) understanding (Baron-Cohen, et al., 2000; Baron-Cohen, et al., 1999), whilst others argue that aberrant emotional behaviours reflect primary impairments in emotional processes (Hobson, 1989; Kanner, 1943; Mundy & Sigman, 1989). In order to gain further insights into this debate, it is important to determine whether emotional processing difficulties in ASD extend to domains outside the broader context of social cognition.

In typically developed individuals, it is well established that emotionally charged events are remembered better than neutral ones (Bradley, Greenwald, Petry & Lang, 1992; Cahill & McGaugh, 1998; Heuer & Reisberg, 1990; Kensinger & Corkin; 2003) but only one study has to date investigated this phenomenon in individuals with ASD. In one of four experiments, Beversdorf and colleagues (Beversdorf, et al., 1998) asked participants to try to remember a series of emotionally charged and neutral statements (e.g. 'He talks about death' *vs.* 'He is talking with his roommate') that were auditorily presented in a blocked fashion. Following each block, participants were asked to free recall as many statements as possible. Results showed that whilst typically developed participants recalled significantly more emotionally charged than neutral statements, those with ASD recalled both types of statements to a similar extent. Since the other 3 experiments of this publication demonstrated that individuals with ASD, like typical participants, exhibited enhanced memory for sentences and paragraphs that were syntactically and conceptually more coherent, the authors concluded that ASD seems to be characterised by a relatively specific memory decrement for emotionally charged stimuli.

Studies such as the one set out above provide valuable insights into the nature of emotional processing abnormalities in ASD by indicating that the emotional significance of environmental stimuli atypically modulates cognitive processes outside the broader context of social cognition. As such the findings by Beversdorf et al. (1998) augment recently accumulating evidence showing that the aberrant emotional processes manifest in ASD do indeed extend to domains outside the broader context of social cognition. Studies of classically conditioned fear responses for example, indicate that individuals with ASD acquire

fear to a previously neutral stimulus similar to typical individuals when this stimulus is consistently and repeatedly paired with a noxious stimulus (Bernier, et al., 2005). However when the association between the neutral stimulus and the noxious stimulus is less consistent, such as in differential fear conditioning paradigms, fear acquisition in ASD is attenuated in comparison to typical individuals (Gaigg & Bowler, 2007). This pattern suggests that whilst relatively basic emotional response mechanisms function relatively typically in ASD, they are not normally modulated by the contingencies that determine the emotional significance of stimuli. Studies assessing the psychophysiological reactivity of individuals with ASD have yielded a similar pattern of observations. Such studies show that although individuals with ASD generally exhibit heightened autonomic responses to emotionally significant stimuli (e.g. Ben-Shalom, et al., 2003; Salmond, et al., 2003), such responses are often abnormally modulated by specific stimulus parameters such as the direction of gaze in facial stimuli (Joseph, Ehrman, McNally & Tager-Flusberg, 2005; Kylliainen & Hietanen, 2006) or the type of emotional content displayed (Blair, 1999; Hillier, Carpenter, Smith, Berntson, & Beversdorf, 2006). In addition, even when autonomic responses are comparable across typical and ASD participants, the accompanying subjectively experienced 'feelings' participants report tend to differentiate these groups (Ben-Shalom, et al., 2003). Based on this literature, we have recently suggested that ASD may be characterised by abnormalities in how psychophysiological and cognitive emotional processes are integrated and how they modulate one another (Gaigg & Bowler, 2007). Memory paradigms provide a useful behavioural tool to test this suggestion because stimulus induced psychophysiological arousal has been shown to modulate memory processes reliably (e.g. Cahill & McGaugh, 1998; Corteen, 1968; Heuer & Reisberg, 1990; Kensinger & Corkin, 2004; Maltzman, Kantor & Langdon, 1966).

In addition to shedding further light on the nature of emotional atypicalities in ASD at the behavioural level, studies of memory for emotionally significant stimuli may also provide important insights into the neuropathological basis underlying them. As Beversdorf et al. (1998) point out, their finding of atypical memory for emotional stimuli is in line with the view that abnormalities of the limbic system, in particular the amygdala, may play an important role in the neuropathology underlying ASD. Although several other lines of inquiry support this

view (e.g. Bachevalier, 2000; Baron-Cohen, et al., 2000; Nacewicz, Dalton, Johnstone et al., 2006), the evidence remains inconclusive and the extent and nature of a proposed amygdala abnormality unspecified (e.g. Amaral, Bauman, Mills & Shumann; 2003; Sweeten, Posey, Shekhar & McDougle, 2002). Based on the behavioural evidence outlined above, it appears that relatively basic amygdala functions such as those necessary for mediating automatic behavioural and autonomic responses to innately emotive stimuli are relatively preserved in ASD. Ashwin, et al. (2006) have recently reached a similar conclusion following their observation that individuals with ASD like typical individuals exhibit a 'pop-out' effect (i.e. faster detection) when searching for an angry face amongst neutral or happy face distracters. What appears to be impaired in ASD is how such basic emotional response processes modulate and are modulated by 'higher' level cognitive and perceptual processes. Together with accumulating evidence suggesting that the neuropathology underlying ASD may be characterised by relatively widespread abnormalities in connectivity between disparate brain areas (e.g. Brock, Brown, Boucher & Rippon, 2002; Just, Cherkassky, Keller, Kana & Minshew, 2007; Rippon, Brock, Brown & Boucher, 2007) this pattern of findings has led several authors to suggest that the amygdala may be functionally disconnected from areas sub-serving other cognitive and perceptual processes (Ashwin, et al., 2006; Gaigg & Bowler, 2007; McAlonan, Cheung, Cheung, Suckling, Lam, Tai, et al., 2005). Memory paradigms provide a useful behavioural tool in this context because it is well established that interactions between the amygdala and hippocampus play a central role in the modulation of memory as a function of the physiological arousal elicited by emotionally significant stimuli (see Cahill & McGaugh, 1998; Hamann, 2001; McGaugh, 2000; Phelps, 2004 for reviews).

In order to test the prediction that memory is atypically modulated by stimulus induced autonomic arousal in ASD, the current study extends the findings by Beversdorf et al., (1998) in two ways. First, by directly measuring participants' Skin Conductance Responses (SCR) and asking them to rate each stimulus on 'arousal', we confirmed whether or not our experimental stimuli did indeed vary on this dimension. In addition to assessing the impact of arousal on memory more directly, this also allowed us to address the possibility that emotional memory deficits in ASD may arise because of abnormalities in how such individuals perceive or physiologically respond to emotionally charged stimuli. As indicated by

the evidence outlined above, this possibility is viable since individuals with ASD do not seem to exhibit typical levels of physiological arousal in response to all emotionally significant stimuli (e.g. Blair 1999) and even when they do, such responses may not alter their perceptions of the stimuli accordingly (e.g. Ben-Shalom, et al., 2003). Second, we assessed participants' memory at three time-points (immediately after encoding and following 1 hour and 24 hour delays) in order to establish forgetting rates for emotionally arousing and non-arousing stimuli. Studies employing such forgetting paradigms consistently show that memories of emotionally arousing stimuli are more resistant to forgetting than non-arousing stimuli (e.g. LaBar & Phelps, 1998; Sharot & Phelps, 2004; Walker & Tarte, 1963). Aided by extensive neuroscientific evidence (e.g., Adolphs, Denburg & Tranel, 2001; Hamann, 2001; Phelps, 2006; Phelps, LaBar, Anderson, O'Connor, Fulbright & Spencer, 1998) this behavioural dissociation of the effects of arousal on memory is widely thought to reflect memory modulation processes that are mediated by the amygdala and should therefore yield further insights into the functional integrity of the amygdala in ASD. Because no study has to date addressed the possibility that the attenuated forgetting rate for emotionally arousing stimuli may in part be the result of the greater semantic relatedness amongst arousing compared to non-arousing stimuli, we included a set of categorically related items in our experimental materials in order to control for this possibility. This experimental control was equally important in terms of addressing the possibility that a memory decrement for emotionally arousing stimuli in ASD may arise simply because such individuals tend to make less use of semantic relations between items to facilitate free recall (e.g. Bowler, et al., 1997; Hermelin & O'Connor, 1967; Smith, Gardiner & Bowler, 2007; Tager-Flusberg, 1991; But see López & Leekam, 2003 for contrary evidence).

Method

Participants

Eighteen participants with a diagnosis of ASD (15 male; 3 female) and 18 typically developed adults (14 male; 4 female) participated in the current experiment. All participants in the clinical group had been diagnosed according to conventional criteria and a review of

available medical records or assessment with the Autism Diagnostic Observation Schedule (ADOS; Lord, et al., 1989) confirmed that all met DSM-IV (American Psychiatric Association, 2000) criteria for an autism spectrum disorder. Brief interviews ensured that none of the participants suffered from any mental or neurological disorder other than ASD and all participants were free of medication. ASD and typical participants were individually matched to within 7 points of verbal IQ as measured by the Wechsler Adult Intelligence Scale (WAIS-III^{UK}; The Psychological Corporation, 2000) and groups did not differ on performance IQ, full-scale IQ or age (see Table 2.3).

Table 2.3

Summary of Age and IQ characteristics for the ASD and Comparison Group

	ASD (n = 18)		Comparison (n = 18)	
Measure	*M*	*SD*	*M*	*SD*
Age (years)	33.0	13.1	30.4	10.0
VIQ[a]	107.8	15.5	107.4	14.7
PIQ[b]	107.8	16.8	105.1	12.5
FIQ[c]	108.6	16.8	104.6	12.4

[a] Verbal IQ (WAIS-R^{UK} or WAIS-III^{UK})
[b] Performance IQ (WAIS-R^{UK} or WAIS-III^{UK})
[c] Full-Scale IQ (WAIS-R^{UK} or WAIS-III^{UK})

Materials & Design

The experimental materials consisted of 16 emotionally arousing, 16 semantically related neutral and 16 semantically unrelated neutral words. The arousing words included profanities, sexually explicit and taboo words and were between 3 and 10 letters in length. Written frequency norms (Kucera & Francis, 1967) were available for 8 of these items and

ranged from 1 per million (e.g. Puke) to 84 per million (e.g. Sex) (M = 15.25, SD = 28.08). The semantically related words consisted of 16 items of fruit taken from the Battig and Montague (1969) category norms and were matched to the arousing words on letter length (range 4-10) and written frequency (M = 10.88, SD = 17.47). Similarly, the 16 unrelated neutral words were selected to match the arousing and semantically related words in terms of letter length (range 3-10) and written frequency (M = 16.31, SD = 9.24) and none of the these words was semantically or conceptually related to any other words in this study.

Words were presented in pseudorandom order in 46 point Arial font in the centre of a 15" laptop monitor with the constraint that no more than two words of the same type (i.e. arousing, related neutral, unrelated neutral) would occur in succession. An additional 4 filler words, 2 at the beginning and 2 at the end of the list, were included to counteract primacy and recency effects on memory. Words were presented for 5 seconds each and were immediately followed by on-screen instructions asking participants to rate the word on a 4 point 'arousal' scale (1 = not at all arousing; 4 = very arousing). Once the participants had indicated their rating by typing the appropriate number on the keyboard, a fixation cross appeared in the centre of the screen and remained there until the experimenter triggered the presentation of the next item via a wireless mouse. Throughout the experiment participants SCRs were recorded via two surface electrodes attached to the medial phalanges of the first and third digit of the participants' non-dominant hand and a new stimulus word was presented only when there was no sign of galvanic activity for at least 2 seconds. SCR data acquisition was controlled by PowerLab hardware (ADInstruments, 2004), which sampled electrodermal activity at 1 kHz and Chart 5 software (ADInstruments, 2004) was used for the storing and assessment of the data.

Procedure

Participants were tested individually in a sound attenuated laboratory and upon arrival, were informed that the current experiment was concerned with assessing individuals' emotional responses to neutral and emotionally charged words. More specifically, participants were told that the purpose of the experiment was to determine the degree to which their

physiological responses to different words would relate to their subjective experience of arousal. We explained that for this purpose we would measure their SCR and ask them to rate each word in terms of how much they thought they physiologically reacted to these stimuli. It was clarified that their rating should reflect the degree to which they actually '*felt*' an emotional response to each word rather than base their ratings on the meaning of the words. No mention was made of any impending memory tests.

Following the instructions, the SCR electrodes were attached and participants were asked to find a comfortable position in front of the screen and relax for a few minutes. Once SCR had reached baseline activity, the presentation of stimuli commenced. Immediately after the experiment and removal of all equipment, participants were given a surprise free recall test in which they were asked to write down as many words from the experiment as possible in any order. Following approximately 1 hour which was filled by a lunch break or by non-verbal tasks, free recall was again requested. For logistical reasons it was unfortunately not possible to ask participants to return again the next day for the final free recall test. Instead, they were asked to take home a sealed envelope which they should not open until the following day. The envelope contained instructions to once again write down as many words as possible from the experiment of the previous day and to return responses via the envelope provided.

Results

For the analyses of participants' SCR, the largest deflection of galvanic activity during the 5 second stimulus presentation was calculated and square root transformed in order to normalise the distribution of the data. Average responses to the arousing, related neutral and unrelated neutral words are set out in Table 2.4, which also presents the participants' average arousal ratings for these stimuli. A 2 (Group) x 3 (Word Type) ANOVA of SCR confirmed that the emotive words elicited higher levels of physiological arousal than the other classes of stimuli ($F(2,33) = 17.54$, $p < .001$) and the absence of a main effect of Group ($F(1,34) = 0.78$, ns) and interaction ($F(2,33) = 2.36$, ns) suggests that both groups exhibited similar levels of autonomic activity across the different stimuli. An analysis of participants'

ratings equally revealed a significant main effect of Word Type ($F(2,33) = 66.21$, $p < .001$) but no main effect of Group ($F(1,34) = 0.75$, ns) or interaction ($F(2,33) = 0.75$, ns). Together, these results suggest that the manipulation of arousal was successful and that both groups similarly perceived and autonomically experienced the emotional significance of arousing words. Interestingly, an assessment of correlations between autonomic activity and subjective ratings of arousal indicated that only for the comparison group this association was statistically reliable. These correlations were computed for each participant and whilst the average correlation coefficient ($M = 0.29$; $SD = 0.20$) across typical participants was significantly above chance ($t = 6.11$, $df = 17$, $p < .001$) the average across individuals with ASD ($M = 0.10$; $SD = 0.23$) was only marginally significant ($t = 1.85$, $df = 17$, $p = .08$)[12].

Table 2.4

Average SCR and arousal ratings for ASD and Comparison Group as a function of Word Type

	ASD (n = 18)		Comparison (n = 18)	
Word Type	*M*	*SD*	*M*	*SD*
SCR (√μS)				
Arousing	0.720	0.347	0.880	0.880
Related Neutral	0.546	0.340	0.503	0.503
Unrelated Neutral	0.574	0.378	0.559	0.559
Arousal Rating				
Arousing	2.49	0.76	2.49	2.49
Related Neutral	1.64	0.64	1.49	1.49
Unrelated Neutral	1.51	0.42	1.27	1.27

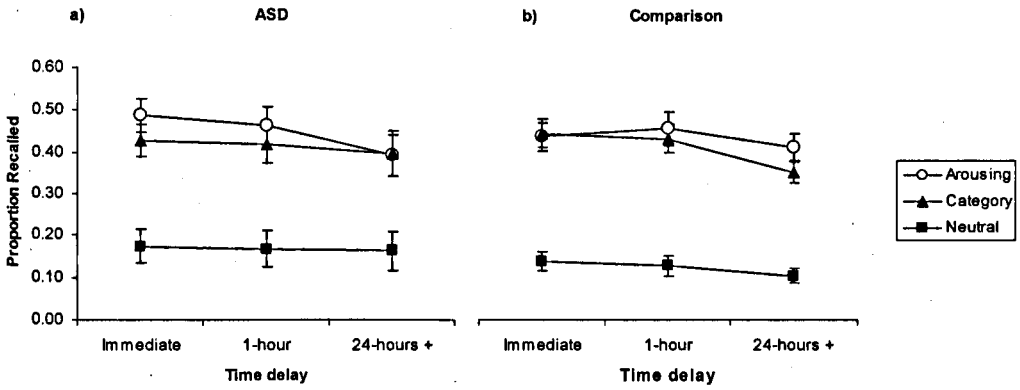
[12] The difference between groups was also statistically reliable ($t = 2.62$, $df = 34$, $p < .05$)

Prior to assessing the free recall data we determined whether participants in the two groups completed the final free-recall test within similar timeframes. All but two typical and one ASD participant returned their final free recall responses and since the exclusion of these participants did not significantly alter the results reported below, their final free recall score was substituted with the respective group averages. Inspection of postmarks indicated that typical participants posted their responses on average 2 days after the experiment (SD = 1.26; Range = 1-5) and ASD participants 3 days after the experiment (SD = 2.27; Range = 1-9). Since this difference was statistically not reliable (t = 1.29, df = 31, ns) and response delay did not correlate with free recall for either group (r < 0.25, ns) it is unlikely that the results reported below are confounded by group differences in study-test delays. For simplicity we will refer to the last free recall test as a 24 hour+ delay test to indicate that for all participants at least 1 day had elapsed before they returned their responses.

Figures 2.1a and 2.1b illustrate the average proportions of arousing, related neutral and unrelated neutral words recalled by the ASD and Comparison group as a function of time of recall. A 2 (Group) x 3 (Word Type) x 3 (Time) mixed ANOVA of these data revealed main effects for Word Type (F(2,33) = 155.84, p < .001) and Time (F(2,33) = 8.78, p < .01, Greenhouse-Geisser corrected). As the figures suggest, the main effect of Word Type was due to the arousing (M = 0.44; SD = 0.15) and semantically related words (M = 0.41; SD = 0.16) being recalled significantly more frequently than the neutral words (M = 0.15; SD = 0.14) whilst the main effect of Time was mainly due to a significant drop in recall between the 1 hour (M = 0.34; SD = 0.14) and 24 hour+ (M = 0.30; SD = 0.15) delay (t = 3.36, df = 35, p < .01). Although no other main effects or interactions were statistically significant, the three-way interaction was (F(4,31) = 2.28, p = .08). Post-hoc analysis indicated that the Comparison group exhibited the expected pattern of results, forgetting the unrelated neutral (t = 2.46, df = 17, p < .05) and related neutral (t = 3.66, df = 17, p < .005) words whilst their recall of arousing words (t = 0.98, df = 17, ns) did not decrease significantly over time. For the ASD group on the other hand, only the forgetting rate of arousing words over the 24 hour+ period (t = 2.57, df = 17, p < .05) was significant.

Figure 2.1

Average recall performance for the ASD and Comparison Group as a function of Word Type and Time of Recall (Error Bars represent Standard Errors)

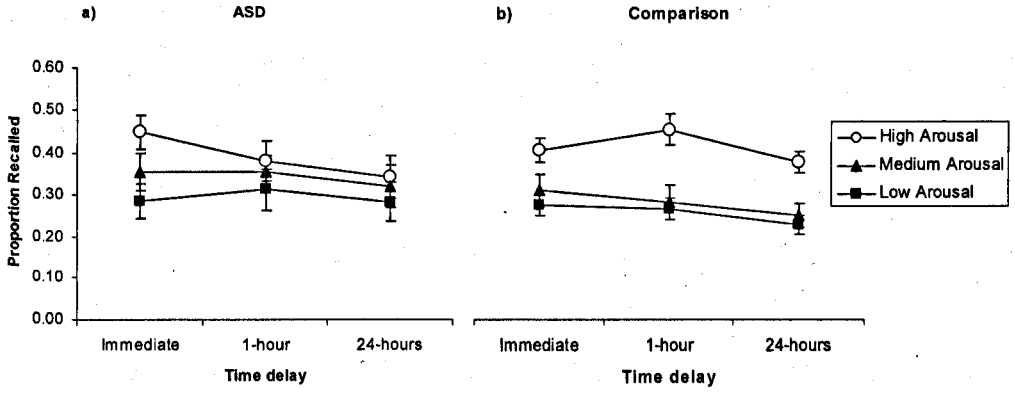


The results above indicate that although individuals with ASD remember emotionally charged and semantically related items better than neutral unrelated items, they forget these words more quickly than typical participants. In order to establish to what extent these results are attributable to abnormalities in how physiological arousal modulates memory, we re-analysed recall performance as a function of actual levels of physiological arousal rather than as a function of the conceptual classification of words. For this purpose, words were re-classified as either 'High Arousal' (16 words eliciting highest SCRs), 'Medium Arousal' (16 words eliciting moderate SCRs) or 'Low Arousal' (16 words eliciting the lowest SCRs) on the basis of each participant's SCRs. Figure 2.2a and 2.2b illustrate the relevant recall data for this classification of words and again show a markedly atypical pattern of forgetting in the clinical group. A 2 (Group) x 3 (Level of Arousal) x 3 (Time) ANOVA of these data revealed main effects for Level of Arousal ($F(2,33) = 18.75$, $p < .001$) and Time ($F(2,33) = 5.81$, $p < .01$) and a significant three-way interaction between the factors ($F(4,31) = 4.38$, $p < .01$). The main effect of Level of Arousal represents the fact that the high arousal words ($M = 0.40$; $SD = 0.15$) were recalled significantly better than the medium arousal ($M = 0.31$; $SD = 0.17$) and low arousal words ($M = 0.28$; $SD = 0.15$), whilst the main effect of Time was again mainly due to a significant drop in recall performance between the 1 hour ($M = 0.34$; $SD = 0.14$) and 24 hour+ ($M = 0.30$; $SD = 0.15$) delay period. As Figure 2.2 suggests, the three-way interaction

was due to the ASD participants exhibiting a recall advantage for highly arousing words only on the immediate test of memory ($F(2,16) = 8.78$, $p < .01$) but not following a 1hour ($F(2,16) = 1.35$, ns) or 24 hour+ delay ($F(2,16) = 1.82$, ns). Typical participants on the other hand exhibited a highly reliable recall advantage for high arousal words across all time delays (Immediate: $F(2,33) = 9.27$, $p < .01$; 1 hour: $F(2,33) = 16.16$, $p < .001$; 24 hour+ $F(2,33) = 18.68$, $p < .001$).

Figure 2.2

Average recall performance for the ASD and Comparison Group as a function of Level of Arousal and Time of Recall (Error Bars represent Standard Errors)



Discussion

Previously, Beversdorf and colleagues (Beversdorf et al., 1998) have observed a relatively specific memory decrement for emotionally charged stimuli in individuals with ASD and concluded that such a decrement may be the result of abnormalities of the amygdala. Current views regarding the role of the amygdala in memory postulate that this limbic structure modulates hippocampally based memory consolidation processes when stimuli elicit physiological arousal (e.g. Phelps, 2006). As a result of this modulation, memory for arousing over non-arousing stimuli is quantitatively enhanced and also more resistant to forgetting over time. Particularly the attenuated forgetting rate of emotionally arousing items is thought to

reflect amygdala mediated memory modulation processes, whilst the enhancement of memory over short periods of time can in part be accounted for by the semantic interrelatedness of emotionally charged stimuli (Talami, et al., 2007; Talmi & Moscovitch, 2004). The current study was aimed at trying to provide further insights into the functional integrity of amygdala mediated memory modulation processes in ASD by assessing participants' SCR and subjective experience of arousal in response to a series of emotionally charged and neutral words and subsequently assessing memory for these stimuli at 3 points in time.

Our results may be summarised as follows. Individuals with ASD, like typical individuals, exhibited significantly increased SCR to emotionally charged compared to neutral words. Similarly, both groups rated the emotive words as more arousing than the non-emotive ones. Interestingly, the correlation between SCRs and subjective ratings was significantly higher for the comparison than the clinical group for whom this relationship was only marginally above chance. In terms of the participants' recall performance, both groups exhibited enhanced recall for emotive and semantically related compared to semantically unrelated neutral words. However, whilst the results from the comparison group replicated previous findings of reduced rates of forgetting of arousing words (e.g. LaBar & Phelps, 1998), the ASD group exhibited the opposite pattern by forgetting emotionally arousing but not non-arousing words. Further analysis of the recall data as a function of each participant's SCR confirmed this group difference, again demonstrating significantly attenuated forgetting rates of arousing words in the typical but not the ASD group. This analysis furthermore showed that whilst typical participants maintained a highly reliable recall advantage for physiologically arousing items over time, this recall advantage was only present on a test of immediate memory in ASD participants. Already following 1 hour, the clinical group's recall no longer varied as a function of whether the words had elicited high, medium or low levels of arousal during encoding, strongly suggesting atypicalities in how physiological arousal modulates consolidation processes in this group.

The degree to which neurological processes can be inferred from behavioural data depends on the extent to which the observed behaviour is associated with specific neural

processes. In this respect our observations from the comparison group yield important new evidence regarding the specificity with which physiological arousal modulates memory. Although it is well established that emotionally charged stimuli are better remembered than neutral stimuli, there remains some debate regarding the extent to which this memory enhancement may be an artefact of the semantic interrelatedness of emotionally arousing stimuli (e.g. Buchanan, et al., 2006; Maratos, et al., 2000; Talmi & Moscovitch, 2004). Our current findings contribute to this debate by demonstrating that incidentally encoded emotionally significant words, despite being better remembered than unrelated neutral words, are not remembered better than semantically related ones on an immediate test of free recall. Importantly, however, our assessment of forgetting rates showed that only emotive words were resistant to forgetting whilst neutral words were forgotten at a similar rate regardless of whether they were semantically interrelated or not. This suggests that although quantitatively memory for emotional words may be similar to that of semantically related non-emotional words over short periods of time, such stimuli are consolidated in a qualitatively different manner. This qualitative difference is further highlighted by the fact that when memory is considered in relation to actual levels of physiological arousal rather than a conceptual classification of stimuli, arousing stimuli are not only resistant to forgetting but remembered better than non-arousing stimuli even on immediate tests of memory. Together with the extensive neuroscientific evidence implicating the amygdala in the modulation of memory due to arousal (e.g. Cahill & McGaugh, 1998; Hamann, 2001; Phelps, 2004), our results suggest that, in particular, forgetting rates of emotionally arousing stimuli provide a relatively reliable behavioural marker of amygdala mediated memory modulation processes.

If one accepts our interpretation above, our results from the ASD group provide important new insights into the functional integrity of the amygdala in this condition. Our observation of relatively typical autonomic responses to emotionally charged words in ASD suggests that the basic process by which the amygdala modulates autonomic arousal in response to verbal stimuli is functionally intact. The markedly abnormal forgetting rate of emotionally arousing stimuli, on the other hand, suggests that the amygdala abnormally modulates hippocampally based consolidation processes as a function of arousal in ASD. As such our observations provide further behavioural evidence for our suggestion that the

amygdala may not be grossly impaired in ASD but that instead it abnormally modulates the functional activity in other areas of the brain because of poor connectivity to those areas (Ashwin, et al., 2006; Gaigg & Bowler, 2007; McAlonan, et al., 2005). One may criticise these conclusions on the basis of our observation that individuals with ASD did exhibit enhanced memory for arousing words on a test of immediate free recall. As we argued above, however, the quantitative memory enhancement for arousing stimuli over short periods of time can be accounted for on the basis of such stimuli being semantically interrelated. In the current study individuals with ASD were able to draw on such semantic relationships to facilitate free recall as indicated by their superior recall of semantically related compared to semantically unrelated neutral words. Thus, although individuals with ASD exhibited a quantitative memory enhancement for arousing stimuli, these memories were not consolidated in the qualitatively distinct way that characterises emotionally charged memories in typical participants. In addition, our assessment of correlations between subjective ratings of arousal and the magnitude of SCRs replicates an earlier study in showing that this correlation is significant for typical individuals only (see Hillier, et al., 2006 for a similar observation). Again, this may be interpreted as indicating that cognitive representations of the emotional significance of environmental stimuli, which are thought to be mediated by cortical areas such as the cingulate cortex and frontal areas (e.g. Kensinger & Schacter, 2006; Lane, Reiman, Axelrod, Yun, Holmes & Schwartz, 1998; Maddock, Garrett & Buonocore, 2003), are inadequately modulated by physiological responses to these stimuli.

Regardless of whether one accepts or rejects our suggestion that abnormalities of amygdala connectivity may be causally related to our observations, our results provide interesting insights into memory processes in ASD. First, our results confirm the observations by Beversdorf et al. (1998) in demonstrating that emotionally significant stimuli are atypically remembered by individuals with ASD. Although our observation of a relatively preserved memory enhancement for emotionally charged words on an immediate test of memory may be viewed as inconsistent with the observations by Beversdorf et al. (1998), as we have noted above, the atypical forgetting rate of these stimuli strongly suggests that qualitatively these memories are abnormal in individuals with ASD. Our observations thus provide further evidence that emotional processing atypicalities in ASD extend to domains outside the

broader context of social cognition, favouring the view that such abnormalities constitute a relatively basic feature of the ASD phenotype (e.g. Hobson, 1989) rather than a secondary manifestation of atypical social capacities (e.g. Baron-Cohen, et al., 2000; Schultz, 2005). Second, our findings also support recently accumulating evidence, which suggests that under some circumstances individuals with ASD make similar use of semantic relationships amongst items to facilitate free recall as do typical individuals (Gaigg et al., 2008; López & Leekam, 2003). The current findings confirm our recent findings which showed that when semantically related words are presented in a mixed list, recall performance in individuals with and without ASD is nearly identical when individuals encode words through an item-specific encoding task in which individuals are asked to rate each word on pleasantness (Gaigg et al., 2008). Thus our current findings provide further support for our suggestion that item-specific memory processes function in a relatively intact manner in ASD.

In summary, we have shown that individuals with ASD exhibit atypical forgetting rates of emotionally arousing words, which on the basis of current neuroscientific evidence provides further support for the suggestion of abnormal amygdala connectivity in this condition (Ashwin, et al., 2006; Gaigg & Bowler, 2007; McAlonan, et al., 2005). Furthermore, the current findings replicate our recent observations of intact free recall performance following item-specific encoding (Gaigg et al., 2008), providing further support for the suggestion that item-specific memory processes function typically in this disorder. Finally, the present findings add to the recently accumulating evidence, which suggests that emotional processing atypicalities extend to domains outside the broader context of social cognition, making it increasingly unlikely that the emotional difficulties characterising autistic social behaviour are the sole result of abnormal socio-cognitive development. Instead it seems more plausible that emotional processing atypicalities form a primary feature of the ASD phenotype.

Note

During the process of revising this manuscript for re-submission, we came across the recent publication by South, et al. (2008), who reported the results of four behavioural

experiments that are relevant to amygdala function. In one of these experiments the authors asked individuals with ASD and typical individuals to study a list of words containing emotionally charged and neutral words for a subsequent recognition memory task. The results showed that both groups of participants exhibited a memory enhancement for emotionally charged over neutral words. These observations are not inconsistent with our observations since we also noted that individuals with ASD recalled emotionally charged words better than neutral (unrelated) words on an immediate test of memory. What our results add to this observation is the suggestion that in ASD such emotional memories are not consolidated in the qualitatively distinct manner that characterises consolidation in typical individuals, thus implicating functional abnormalities in how the amygdala modulates such consolidation processes. It is worth noting that the other three experiments of the South et al. (2008) publication also revealed no indication of behavioural abnormalities leading these authors to suggest that amygdala dysfunction may be specific to social situations in ASD. Our current and previous findings (Gaigg & Bowler, 2007) suggest otherwise and further research is clearly needed in order to elucidate precisely what amygdala functions are and are not compromised in ASD.

Experiment 3: Illusory memories of emotionally charged words in Autism Spectrum Disorder: Further evidence for atypical emotion processing outside the social domain

Experiment 2 above provides strong evidence for the suggestion that stimulus induced arousal atypically modulates memory in individuals with ASD. Although it remains possible that over relatively short periods of time such modulatory processes operate relatively normally in this condition, it seems clear from the foregoing observations, that individuals with ASD do not retain emotionally significant stimuli in distinct ways over time. If this is the case, one would expect such individuals to accumulate atypical representations of emotional stimuli (at least words) over the course of their lives. The present experiment puts this hypothesis to the test through the use of a memory illusion paradigm. This experiment

has been accepted for publication by the *Journal of Autism and Developmental Disorders* (Gaigg & Bowler, in press a).

Introduction

Research over the past four decades has firmly established that individuals from across the autism spectrum are severely compromised in various aspects of affectively patterned communication (e.g., Corona, et al., 1998; Hobson, et al., 1988a, Joseph & Tager-Flusberg, 1997; Kasari, et al., 1990; Loveland, 2003; Moore, et al., 1997; Sigman, et al., 1992). Although these atypicalities are widely accepted to constitute a diagnostically sensitive manifestation of Autism Spectrum Disorder (ASD) (Lord, et al., 1989; Lord, et al., 1994; Magyar & Pandolfi, 2007; Robins, et al., 2001), their causes remain the matter of debate. Some authors maintain that the emotional disturbances associated with ASD are the result of abnormalities in the development of socio-cognitive capacities such as face processing or 'Theory of Mind' understanding (e.g., Baron-Cohen, 1995; Baron-Cohen, et al., 2000; Frith, 2003; Leslie & Frith, 1990; Schultz, 2005). Others, by contrast, suggest that abnormal affective development lies at the root of the interpersonal abnormalities characterising the disorder (e.g., Hobson, 2002; Loveland, 2005, Mundy & Sigman, 1989). The major difficulty in resolving this debate rests with the fact that most of the relevant evidence for it stems from studies assessing how individuals with ASD understand and react to the emotional signals of others. As such, the evidence can be accommodated by both theoretical accounts. In order to advance our understanding of the role of affective disturbances in ASD, it is therefore important to broaden the focus of research and assess how the emotional significance of stimuli impacts on processes that are not primarily of a social nature. Studies of memory are ideally suited for this purpose because the influence of emotional factors in this domain are well established and understood (see Reisberg & Hertel, 2004 and Uttl, et al., 2006 for a collection of extensive reviews).

One of the most reliable findings in the typical memory literature is that emotionally significant information is better remembered than neutral information (Bradley, et al.,1992; Cahill & McGaugh, 1998; Heuer & Reisberg, 1990; Kensinger & Corkin; 2003). To date only

four studies have investigated this phenomenon in individuals with ASD. The first, was conducted by Beversdorf and colleagues (Beversdof, et al., 1998) who presented a group of 10 ASD and 13 typically developed adults with an audiotape of 10 emotionally salient statements (e.g., Carl shot his gun at someone) and 10 emotionally neutral statements (e.g., Mike is talking on the phone). After each block of statements participants were asked to write down as many of them as possible. Results indicated that only typically developed adults, and not those with a diagnosis of ASD, recalled significantly more of the emotionally salient statements. In direct contrast to this finding, South and colleagues (South, et al., 2008) reported no differences in memory for emotional material between a group of 36 adults with a diagnosis of ASD and 38 typically developed adults. In their experiment, participants were asked to study a random sequence of emotionally salient and neutral words for a subsequent recognition memory test. This time both groups of participants were found to exhibit superior memory for the emotionally salient material. Whilst the findings from these two studies are clearly at odds, a study from our own laboratory (Gaigg & Bowler, 2008) may help to resolve this inconsistency. Similar to South et al. (2008) we also presented participants with a random sequence of emotionally charged and neutral words and also found no group differences in memory for the emotional material between ASD and typically developed adults on an immediate test of memory. When assessing participants' memory again after 1 hour and once more after 1 day, however, the memory advantage for emotional words increased for typical participants whilst it diminished for those with a diagnosis of ASD. Importantly, we had had included a set of semantically related but neutral words (i.e. names of fruit) in our materials, allowing us to determine to what extent the memory superiority for emotional words may be accounted for in terms of the semantic similarity between them. In this respect our results showed that neither group of participants exhibited enhanced memory for emotionally charged over semantically related neutral words on the immediate free recall test. This emotion specific enhancement of memory only emerged on the 1 hour and 1 day delay tests and it only emerged for typical but not ASD participants. Together, this pattern of findings suggests that individuals with ASD may exhibit a quantitative enhancement in memory for emotionally charged as compared to neutral information over short periods of time because of semantic aspects of the stimuli rather than their emotional quality *per se*. In the Beversdorf et

al. (1998) study, the blocked presentation and use of statements rather than single words, may not have rendered semantic aspects of the emotional material salient enough to facilitate such a 'semantic modulation' of memory in ASD, whereas in the South et al. (2008) study such semantic influences on memory may explain why these authors failed to observe differences between ASD and typical participants. The evidence from the verbal domain, therefore, suggests that the emotional quality of verbal information does not impact on memory in ASD in the same qualitatively distinct fashion as in typical individuals and this conclusion has recently received additional support from the non-verbal domain. More specifically, Deruelle, Hubert, Santos and Wicker (2008) found that, unlike typical individuals who exhibit a memory advantage for negative as compared to positive and neutral pictures, participants with ASD demonstrated no such memory advantage (if anything they tended to remember neutral images more than positive and negative ones).

Considering the available evidence, one might expect that memories of emotional material in individuals with ASD are qualitatively no different (or only minimally so) from their memories of non-emotional information. Over a lifetime, this would mean that individuals with ASD would accumulate representations of emotional information that are qualitatively indistinct from representations of non-emotional information. One way to test this contention is through a memory illusion paradigm developed by Pesta, Murphy and Sanders (2001). In their experiment, participants were presented with a list of words comprising blocks of orthographically associated words (e.g., Book, Nook, Cook, Look,...Cape, Tape, Shape, Nape,...) that participants were instructed to try to remember for a subsequent recognition memory test. Unbeknownst to the participants, this recognition test included a number of words that were orthographically very similar to the words that had been on the studied list, but which participants had not actually seen. Half of these so-called Target Lures were neutral (e.g., Hook) and half of them were emotionally charged (e.g., 'Rape'). Pesta et al. (2001) found that typical individuals were extremely unlikely to falsely identify the emotionally charged Target Lures as having been on the original study list whilst their false recognition rates of neutral Target Lures was far above chance. In other words, whilst the orthographic similarity between the studied words and the Target Lures gave rise to illusory memories of

certain neutral words, the distinctive nature of the emotional words attenuated such illusory memories.

In the current experiment we replicated one of Pesta et al's. (2001) experiments in order to test the hypothesis that individuals with ASD would be as likely to succumb to illusory memories of emotionally charged as neutral words. In addition, we were interested in whether individuals with ASD would generally be as susceptible to the illusory memory phenomenon as typically developed individuals. To date, most studies investigating this phenomenon in ASD (Beversdorf, et al., 2000; Bowler, et al., 2000; Hillier, Campbell, Keillor, Phillips & Beversdorf, 2007) have employed a paradigm developed by Deese (1959) and Roediger and McDermott (1995), in which participants study groups of semantically rather than orthographically associated words (e.g., bed, dream, pillow,...) before memory is tested for some of these words together with semantically associated Target Lures (e.g. sleep). Overall the evidence suggests that individuals with ASD are susceptible to such semantically induced memory illusions, even if they may sometimes be less susceptible to them than typical comparison individuals (Beversdorf et al., 2000). Interestingly, Hillier et al. (2007) found that individuals with ASD were less susceptible to illusory memories of abstract visual patterns that were induced by asking participants to study sets of similar visual patterns, despite the fact that groups did not differ on a standard semantic memory illusion paradigm. This pattern of results is informative in relation to the 'Weak Central Coherence' (WCC) (e.g. Shah & Frith, 1993) and 'Enhanced Perceptual Functioning' (EPF) accounts of ASD (Mottron & Burack, 2001); Mottron et al., 2006). The former suggests that individuals with ASD are impaired in processing globally constituted and conceptual meaning, which seems at odds with the finding that individuals with ASD are subject to semantically induced memory illusions. The EPF account, on the other hand, predicts superior perceptual processing in ASD and this account seems to be in line with the patterning of findings in the memory illusion literature. To date, no study has assessed memory illusions in ASD in the verbal domain that are induced by structural (i.e. orthography) rather than semantic associations between words. Given the evidence so far, one might expect individuals with ASD to be less susceptible to such illusions.

Method

Participants

Twenty two individuals with a diagnosis of ASD (18 male, 4 female) and 22 typically developed individuals (17 male, 5 female) participated in the present experiment. Individuals with ASD were diagnosed by experienced clinicians and a review of available records and/or assessment with the Autism Diagnostic Observation Schedule (ADOS; Lord, et al., 1989) confirmed that all met DSM-IV (American Psychiatric Association, 2000) criteria for Autism Spectrum Disorder. ADOS scores were only used as an exclusion criteria when participants provided a statement of their diagnosis that did not include information about their developmental history. The 10 individuals included in this study who were unable to provide such details all met relevant cut-offs for an ASD on the ADOS assessment. Two individuals whose ADOS scores fell below the cut-off for an ASD were included in the present study because their medical records clearly suggested that such a diagnosis was appropriate (exclusion of these participants did not alter the results presented below significantly). Typical participants were recruited from the local community through newspaper advertisements, and brief interviews ensured that no participants in either group suffered any psychiatric or neurological disorder (other than ASD) or were taking any psychotropic medication. Typical and ASD participants were individually matched to within 7 points of verbal IQ (WAIS-III^{UK}; The Psychological Corporation, 2000) and, as Table 2.5 indicates, groups did not differ significantly in terms of performance IQ (t = 1.41, df = 42, *ns*), full-scale IQ (t = 1.29, df = 42, *ns*) or age (t = 0.53, df = 42, *ns*). The experimental procedures outlined below adhere to the ethical guidelines set out by the British Psychological Society and were approved by the University's Senate Ethical Committee.

Table 2.5

Summary of Age and IQ characteristics for the ASD and Comparison Group

	ASD (n = 22)		Comparison (n = 22)	
Measure	*M*	*SD*	*M*	*SD*
Age (years)	33.5	12.3	35.2	9.6
VIQ[a]	101.1	12.6	103.1	11.5
PIQ[b]	96.7	13.3	102.5	14.1
FIQ[c]	98.7	12.6	103.7	13.1

[a] Verbal IQ (WAIS-R^{UK} or WAIS-III^{UK})

[b] Performance IQ (WAIS-R^{UK} or WAIS-III^{UK})

[c] Full-Scale IQ (WAIS-R^{UK} or WAIS-III^{UK})

Materials & Design

The experimental materials used in the current study were taken directly from Pesta et al's. (2001) second experiment and included 12 groups of 10 orthographically related words, 12 Target Lures, and 3 so-called 'distinctiveness attenuators'. Of the 12 orthographic word groups, six were designated as Set A and six as Set B. Within each of these sets, three groups of words included the orthographic neighbours of three neutral Target Lures (*Hook, Shave* and *Peach* for Set A and *Rink, Park* and *Digit* for Set B), whilst the other three groups included the orthographic neighbours of three emotionally charged Target Lures (*Rape, Bitch* and *Whore* for Set A and *Slut, Hell* and *Penis* for Set B). As outlined by Pesta et al. (2001), the emotional and neutral Target Lures were matched on word frequency (Kučera & Francis, 1967) and letter length, whilst the lists comprising the orthographic neighbours of each type of Target Lure were matched as closely as possible in terms of the number of strict orthographic neighbours of their respective Target Lure. The three 'distinctiveness attenuators' (*Fuck, Piss* & *Asshole*) were presented during the study phase of the experiment and served to satisfy the participants' expectations of seeing emotionally charged words during the experiment (for

ethical reasons participants were fully informed about the nature of the experimental materials).

During the study phase of the experiment, participants were presented with a sequence of 67 words, including the sixty words from the orthographic word groups of either Set A or Set B, the three 'distinctiveness attenuators', and four buffer words (2 at the beginning and 2 at the end of the list). The buffer words were included to counter primacy and recency effects. Words were presented visually on a lap-top monitor at a rate of one word every 3 seconds. Each word appeared in bold, Times New Roman font (size 60) in the centre of the screen, remained there for 2 seconds and was followed by a 1 second blank screen before the onset of the next word. Following Pesta and colleagues, the order of presentation followed a pseudorandom blocked design, whereby sets of 5 words from a given orthographic word group were presented in sequence, whilst the order of these blocks was counterbalanced across participants. The order of words within each block was randomised. The three 'distinctiveness attenuators' were inserted in serial positions 13, 34 and 50. During the recognition test participants were presented with a random selection of 18 words that they had studied (3 from each orthographic word group), 18 words from the set that they had not studied (3 from each orthographic word group) and all 12 Target Lures. The order of presentation was random and the format of presentation was similar to that during the study phase with the exception that the words remained on the screen until participants gave their response.

Procedure

Participants were tested individually in a sound attenuated laboratory. Prior to the experiment, participants were briefed about the nature of the experiment. They were told that they would be required to try to remember a list of words for a subsequent memory test and that some of the words in the experiment would be 'sexually charged, vulgar or offensive'. The illusory memory phenomenon was not mentioned at this stage. Once participants had given their consent to take part in the experiment, they were presented with the to-be-remembered list of words. Immediately after the last word, participants were given

instructions about the impending recognition memory test. The instructions specified that participants would again see a list of words and that this list would include some of the words that they had just seen and some new words. Participants were instructed to indicate whether or not they had seen a particular word before and the experimenter noted these responses on an answer sheet. After the end of the recognition test procedure, participants were fully debriefed about the nature of the experiment and paid according to standard University fees.

Results

Prior to analysing the illusory recognition rates of Target Lures, we assessed participants' true and false recognition rates of the orthographic word group items. Following Pesta and colleagues (2001), we computed participants' 'Yes' responses to these items as a function of whether these were part of the studied or unstudied set and according to whether they comprised the orthographic neighbours of the emotional or neutral Target Lures. A 2 (Group) x 2 (Studied vs. Unstudied) x 2 (Orthographic neighbour of emotional vs. neutral Target Lure) mixed ANOVA of these data, which are set out in Table 2.6, revealed a main effect of Studied/Unstudied ($F(1,42) = 328.84$, $p < .001$; effect size $r = 0.94$) but no other main effects or interactions ($F < 2.20$; effect size $r < 0.22$). The main effect of Studied/Unstudied simply confirms that true recognition rates exceeded false recognition rates. The important result, however, is that both groups performed similarly well in terms of their overall recognition rates and across words comprising the orthographic neighbours of the emotional and neutral Target Lures. Thus, any differences in illusory Target Lure recognition between groups or across emotional and neutral Target Lures are unlikely to be due to differences in terms of how well participants remembered the orthographic neighbours of these Target Lures.

Table 2.6

Proportion of 'Yes' responses to Studied and Unstudied words and the Target Lures related to them as a function of Emotionality and Group (values in parenthesis reflect the SD)

	ASD (n = 22)		Typical (n = 22)	
Word Type	Studied	Unstudied	Studied	Unstudied
Orthographic word group				
Related to neutral Target Lure	.63 (.27)	.09 (.14)	.66 (.19)	.06 (.08)
Related to emotional Target Lure	.59 (.28)	.04 (.08)	.69 (.26)	.03 (.05)
Target Lures				
Neutral	.39 (.32)	.11 (.19)	.30 (.31)	.12 (.19)
Emotional	.32 (.36)	.09 (.18)	.18 (.20)	.00 (.00)

Note: In relation to the Target Lures, the column labels 'Studied' and 'Unstudied' refer to the distinction between Target Lures that were either orthographically related to the Studied or Unstudied word groups.

Table 2.6 also presents the illusory Target Lure recognition rates. A 2 (Group) x 2 (Orthographic neighbour of Studied vs. Unstudied list items) by 2 (Emotional vs. Neutral) mixed ANOVA of these data revealed main effects for the Studied/Unstudied ($F(1,42) = 50.75$, $p < .001$; effect size $r = 0.74$) and Emotional/Neutral ($F(1,42) = 4.78$, $p < .05$; effect size $r = 0.32$) factors, confirming that Target Lures are more likely to be misremembered when they are orthographically similar rather than dissimilar to the studied words, and that emotionally charged Target Lures are less likely to be misremembered than neutral Target Lures. A closer inspection of Table 2.6, also indicates the expected lack of an emotional modulation of illusory memories in the ASD group and planned within-group comparisons confirmed this impression. For the typical group, main effects were observed for both, the Studied/Unstudied ($F(1,21) = 23.08$; $p < .001$; effect size $r = 0.72$) and Emotional/Neutral ($F(1,21) = 9.27$; $p < .01$; effect size $r = 0.55$) factors, whilst for the ASD group only the main effect of Studied/Unstudied was significant ($F(1,21) = 27.97$; $p < .001$; effect size $r = 0.75$)

whilst the Emotional/Neutral factor had virtually no effect in this group ($F(1,21) = 0.49$; *ns*; effect size $r = 0.15$).

Discussion

The principal motivation for the current experiment was the observation that ASD individuals seem to retain emotionally significant information in a qualitatively rather indistinct fashion over time (Gaigg & Bowler, 2008). On the basis of this observation, we suggested that individuals with ASD would be unlikely to have accumulated distinct representations of emotionally salient information throughout their lives, leading us to predict that they should not exhibit an emotional modulation of the illusory memory phenomenon. Before we discuss our results in relation to this hypothesis, we will briefly address the more general question of whether individuals with ASD are as susceptible to orthographically induced memory illusions as typically developed individuals.

As outlined in the introduction, previous studies of memory illusions in ASD seem to suggest that such individuals may be less susceptible than typical individuals to illusory memories that are induced through perceptually anchored associations between to-be-remembered words whilst their susceptibility to conceptually induced memory illusions is more in line with that of typical individuals (Beversdorf et al., 2000; Bowler et al., 2000, Hillier et al., 2007). On the basis of the EPF model (Mottron & Burack; Mottron et al., 2006) this pattern may be explained in terms of the superior perceptual processing abilities of individuals with ASD, which may enhance their ability to discriminate amongst very similar patterns of perceptual information. In a way, one might consider the present findings to pose a challenge to such an interpretation because we observed no differences between ASD and typical participants in terms of their susceptibility to orthographically induced memory illusions. We hasten to add, however, that orthographic associations between words are not perceptually anchored in the same way as the similarities between the abstract visual patterns used in the Hillier et al. (2007) study. In this context it would be of interest to devise an experiment in which memory illusions of verbal stimuli are induced through similarities amongst words in terms of font style or font colour, which would more closely resemble the

associations of the abstract visual patterns used by Hillier et al. (2007). It is clear that further studies will be needed in order to clarify what kinds of associations amongst stimuli determine the degree to which individuals with ASD are susceptible to illusory memories. All we can conclude for the moment is that studies of this phenomenon may be fruitful in informing theoretical frameworks such as the WCC (e.g., Shah & Frith, 1993) and EPF accounts (Mottron & Burack, 2001; Mottron, Dawson, Soulières, Hubert & Burack, 2006), which attempt to specify the perceptual and conceptual processing abilities of individuals with ASD.

In relation to our principal hypothesis, our findings support our prediction that individuals with ASD would be as likely to experience illusory memories of emotionally charged as neutral words. More specifically, whilst our findings from typical participants replicated the observations of Pesta et al. (2001), that the experience of illusory memories is attenuated for emotionally charged compared to neutral words, we observed no such attenuation for participants with ASD[13]. In the context of our recent observation that individuals with ASD do not retain emotionally significant words in a qualitatively distinct fashion over time (Gaigg & Bowler, 2008), we interpret the current findings as follows. Throughout development, individuals with ASD accumulate representations of emotionally significant information that are rather indistinct from their representations of neutral information. More specifically, we argue that autonomic responses during emotionally charged situations in ASD are atypically integrated with the subjectively experienced perception of the situation, resulting in an alteration in how relevant information is consolidated into long-term memory. In support of this view, the atypical pattern of memory for emotional material in ASD tends to be associated with abnormalities in how stimulus induced arousal influences the subjective perception of stimuli. In our previous memory study (Gaigg & Bowler, 2008), for instance, we found that the subjective ratings of emotionality of words in ASD did not correlate with participants' autonomic responses to the words, whereas for typical participants this correlation was significant. Several other studies have reported similar findings in relation to picture stimuli rather than words (Ben Shalom, et al., 2003;

[13] One may criticise our conclusions in this respect on the basis that we observed no Group x Emotion interaction in our analysis. It is important to note, however, that even if this interaction were significant, it would be irrelevant in relation to our predictions, since the main effect of Emotion may nevertheless be significant within each group (i.e. the effect could simply be larger in the Typical group), which would suggest that both groups represented emotional words to some extent as qualitatively distinct. Our planned within-group analyses, therefore, are the most appropriate statistical analyses in relation to our predictions.

Gaigg & Bowler, 2008; Hillier, et al., 2006) and as the recent study by Deruelle et al. (2008) suggests, individuals with ASD remember emotionally significant pictures no better than emotionally neutral ones (unlike typical participants).

Of course, there are other ways to interpret our observations. On the basis of the EPF framework (e.g., Mottron et al., 2006) for instance, one could argue that the enhanced perceptual abilities of individuals with ASD interfered with the processing of the emotional quality of words, thus rendering them relatively indistinct and equally susceptible to the illusory memory phenomenon as neutral words. Such an explanation, however, would have to presume that the emotional quality of a stimulus is processed at a higher-order conceptual level than whatever perceptual process interfered with it. We do not think that such an assumption is tenable since the emotional quality of a stimulus tends to be processed relatively rapidly and automatically at a pre-conceptual level (LeDoux, 1996). Even if it were the case, however, that an atypically well functioning perceptual system interfered with the processing of the emotional quality of information, it would not contradict our interpretation of the current findings. On the contrary, such interference effects might explain why autonomic emotional responses are abnormally integrated into perceptual and cognitive processes in ASD. In other words, perceptual interference may explain why individuals with ASD do not accumulate truly *emotional* representations of environmental stimuli.

Another way of interpreting the current observations would be to suggest that individuals with ASD did not exhibit an emotional modulation of illusory memories because of more general abnormalities in language development. In fact, since language is acquired in the broader context of social cognition, one might argue that the current findings are the result of atypical reciprocal social development, an argument that could lead back to the theoretical debate that motivated the present experiment in the first place. In this context, however, it is important to consider the present findings not in isolation but in relation to other findings in the field. First, invoking atypical language development as an explanation for the current observations would not specify why individuals with ASD should remember emotionally significant words better than neutral ones over short periods of time (Gaigg & Bowler, 2008; South et al., 2008) whilst at the same time not retaining such words in a

qualitatively distinct manner over time (Gaigg & Bowler, 2008). Second, it is unclear how language development could compromise memory for emotional pictures (Deurelle et al., 2008) and how it could alter the extent to which subjective ratings of such pictures are related to arousal responses elicited by them (e.g. Ben Shalom, et al., 2003; Hillier et al., 2006). Third, a language account would be stretched to explain the finding that individuals with ASD acquire classically conditioned fear responses atypically when stimulus contingencies are unpredictable (Gaigg & Bowler, 2007) but not when they are predictable (Bernier et al., 2005) and such an account would also not clarify why decision making behaviours in ASD should be atypically related to the level of arousal participants exhibit in response to their decision making choices (Johnson et al., 2006). Because of this line of evidence, we feel that an explanation of the emotional processing difficulties in ASD in terms of a purely social phenomenon seems no longer tenable. In our opinion this facet of the ASD phenotype is more appropriately explained with reference to emotional processes that operate outside as well as within the social domain and in this respect we argue that a core atypicality in ASD lies in how physiological aspects of emotional experiences influence perception and cognition.

Regardless of whether one accepts or rejects our current interpretation, the observation itself remains the same and suggests that individuals with ASD do not only process socially relevant emotional signals atypically but that they are also relatively insensitive to the emotional significance of stimuli that are not directly social in nature. As such, our findings contribute to a growing literature, which favours the view that emotional disturbances constitute a relatively central feature of the ASD phenotype (e.g. Hobson, 2002).

Summary

The principal aim of the experiments set out in this chapter was to test the hypothesis that stimulus induced arousal modulates memory atypically in individuals with ASD. This hypothesis was motivated by evidence suggesting that abnormalities of the amygdala, which is known to play a vital role in modulating memory as a function of arousal, are involved in the neuropathology underlying ASD. Experiment 1 showed that individuals with ASD, like typical

individuals, exhibit a quantitative memory enhancement for emotionally significant words on a test of recognition. However, that study failed to replicate the finding by Kensinger & Corkin (2004) that memory for arousing stimuli is relatively immune to the effects of dividing attention at study. Since the involvement of the amygdala in memory can only be inferred when arousal specific effects on memory are observed, Experiment 1 therefore did not permit any conclusions regarding the functional integrity of amygdala mediated memory modulation processes in ASD. Experiment 2 provided relatively strong evidence for arousal specific effects on memory and so provided a much more sensitive measure of amygdala functioning. This study demonstrated that, at least in typical individuals, memories for arousing words are much more resistant to forgetting than those of non-arousing words even when the latter are semantically highly interrelated. ASD participants did not exhibit this differential rate of forgetting despite the fact that both groups rated the arousing words as more physiologically stimulating, exhibited similar levels of actual physiological arousal and even demonstrated similar levels of enhanced recall of arousing words on an immediate test of free recall. This specific pattern of abnormality is difficult to account for in terms of generic memory processes especially since the finding was replicated when memory was assessed in relation to the actual levels of physiological arousal elicited by the words. At the behavioural level, therefore, this finding provides strong evidence for the hypothesis that stimulus induced arousal modulates memory abnormally in ASD and at the neural level this pattern of observations is consistent with the suggestion that abnormalities of amygdala connectivity (especially amygdalo-hippocampal connections) may be functionally compromised in this condition. The third, and final, experiment took a somewhat different approach and employed a memory illusion paradigm in order to determine whether individuals with ASD, like typical individuals, would be less likely to experience illusory memories of emotionally charged words. The rationale for this experiment was that, in order to experience illusory memories of emotionally charged words less frequently, one must perceive them as distinct, which necessitates that one has acquired distinct representations of emotional over non-emotional words on previous occasions. Since Experiment 2 showed that individuals with ASD retain emotionally arousing words no differently from non-arousing words, it was hypothesised that such individuals would not have acquired very distinct representations of emotional words throughout their

lives. Thus, individuals with ASD were predicted to experience illusory memories of emotionally charged words to the same extent as of neutral words, which was precisely what Experiment 3 demonstrated.

In summary, the experiments of this chapter lend support to the principal hypothesis of the current thesis, namely that ASD is characterised by abnormalities in the interaction between stimulus induced arousal and cognition. On the basis of neuroscientific evidence, this evidence is suggestive of abnormalities in the functional connectivity between the amygdala and the hippocampus. The evidence, so far, however, is restricted to the domain of memory, and even more specifically to the memory of visually presented words. Although this restriction allowed for the control of variables that might explain group differences in performance without reference to emotion-specific processes, it leaves open the possibility that the observed abnormalities are relatively specific do the domain of memory, or perhaps even specific to verbal stimuli. The following two chapters address this issue by first determining whether abnormalities in the influence of arousal on cognition extend to the domain of perception when visually presented words are used as stimuli (Chapter 3) and second by employing a fear conditioning paradigm that assesses the influence of arousal on learning at a purely non-verbal level (Chapter 4).

CHAPTER 3: PERCEPTION OF EMOTIONALLY SIGNIFICANT WORDS

Overview

The concept of perception, like that of memory, is very broad and it would lead too far afield to provide a detailed overview of this domain of cognition. Such an overview would also be a digression from the principal focus of the following experiment, which simply asks whether individuals with ASD, like typical individuals, are more accurate at detecting emotionally arousing compared to non-arousing words when these are embedded within a very rapid sequence of distracter words. The experiment that follows has been accepted for publication as a brief report by the *Journal of Autism and Developmental Disorders* (Gaigg & Bowler, in press b) and as such includes only a brief introduction and discussion. Before I present the submitted manuscript, it is useful to elaborate on the rationale for the study somewhat.

The previous set of experiments indicated that individuals with ASD may not accumulate distinct representations of arousing words throughout their lives because arousal responses induced by the perception of such words abnormally modulate the formation of long lasting memories. Although every attempt was made to control for the possibility that generic memory difficulties might result in the observed pattern of results, it is nevertheless possible that such memory difficulties contributed to the observations reported earlier. As noted at the outset of Chapter 2, it is difficult to know whether our operational definitions of memory processes and systems apply to the way in which memory functions in individuals with ASD. As a consequence, it is possible that the observations of the experiments reported above, rather than reflecting abnormalities in how arousal modulates memory in ASD, are the consequence of differences in how memory operates in this disorder. However, if it is indeed the case that individuals with ASD accumulate relatively indistinct representations of emotional words throughout their lives, one would expect to observe evidence of this in other domains of cognition. For instance, one would expect that perceptual processes in ASD are rather unaffected by the emotional significance of words. This hypothesis is put to the test in

the present chapter, which sets out an experiment that employed an attentional paradigm in order to determine whether individuals with ASD, like typical individuals, are more accurate at identifying emotionally significant compared to neutral words embedded in a rapid sequence of distracters. As in the previous experiments, the current design attempted to control experimentally for the possibility that individuals with ASD may exhibit abnormal performance patterns as a result of generic perceptual and/or attentional difficulties rather than emotion-specific processing difficulties.

Experiment 4: Attenuated emotional suppression of the Attentional Blink in Autism Spectrum Disorder: Another non-social abnormality?

Introduction

Traditionally, investigations of the emotional competences of individuals with ASD have focused on the study of emotional behaviours within the context of social cognition. Behavioural abnormalities are well established in this domain (e.g. Hobson, 2002) and although the developmental significance of these remain the matter of debate (see Baron-Cohen, 1995; Frith, 2003; Hobson, 2002; Leslie & Frith, 1990; Loveland, 2005 for relevant discussions), neuroscientific investigations converge on the idea that abnormalities of the amygdala are most likely responsible (e.g. Bachevalier, 2000; Baron-Cohen, et al., 2000; Schultz, 2005). The amygdala, however, is not only involved in the mediation of socially relevant emotional behaviours and processes. It plays an important role in our emotional lives regardless of whether we are alone or in the company of others – modulating learning and memory (e.g. LeDoux, 1994; Hamann, 2001; McGaugh, 2000; Phelps, 2004), altering decisions (e.g. Bechara, Damasio & Damasio, 2003; Bechara, Tranel, Damasio & Damasio, 1996) and influencing perceptions of the world (e.g. Anderson & Phelps, 2001). In recent years, several researchers have started to use the extensive literature on amygdala function heuristically to study the integrity of emotional processes in ASD outside the context of social cognition.

Studies of fear conditioning have shown that individuals with ASD only learn the association between a noxious stimulus and a neutral one when the contingencies between the two are relatively predictable (Bernier, et al., 2005) but not when they are more variable (Gaigg & Bowler, 2007). Individuals with ASD have also been found to retain emotionally significant information no differently from non-emotional information (Beversdorf, et al., 1998; Gaigg & Bowler, 2008; Gaigg & Bowler, under review a; but see South, et al., 2008) and decision-making processes in such individuals seem to be atypically influenced by the motivational significance of the decision-making choices (Johnson, et al., 2006). In short, accumulating evidence suggests that emotional processing abnormalities in ASD extend to domains outside the broader context of social cognition.

The current experiment was designed to extend the aforementioned literature to the domain of attention where the amygdala is also known to play a modulatory role as a function of the hedonic value of environmental stimuli (e.g. Anderson & Phelps, 2001; Armony & Dolan, 2001). To date most studies relevant to this domain in ASD (see Schultz, 2005 for a relevant review), have involved assessments of attention to socially relevant emotional signals, with some studies noting abnormalities (e.g. Corona, et al., 1998; Kasari, et al., 1993) whilst others do not (Ashwin, et al., 2006). Only two studies have attempted to extend this literature to the non-social domain, and here too the findings are inconsistent. South et al. (2008) found that typical and ASD participants exhibit similarly enhanced detection of fear-relevant (e.g. snake) vs. fear-irrelevant (e.g. flower) stimuli in a visual search task, which supports the finding by Ashwin et al. (2006) that individuals with ASD demonstrate a typical anger-superiority effect in visual search paradigms employing socially relevant stimuli (i.e. faster detection of angry vs. non-angry facial expressions). A recent study exploring a phenomenon known as the 'Attentional Blink' (AB), on the other hand, showed that the emotional significance of words did not capture the attention of individuals with ASD to the same extent as for typical participants (Corden, Chilvers & Skuse, 2008), which is in line with our observation that individuals with ASD do not seem to retain physiologically arousing words in qualitatively distinct ways over time (Gaigg & Bowler, 2008). The AB (Raymond, Shapiro & Arnell, 1992) describes a period of reduced awareness, elicited during tasks where participants are required to identify two target stimuli embedded among distracters in rapid

serial visual presentation (RSVP). Correct identification of the first target (T1) markedly attenuates identification of a second target (T2) occurring between 180-500 ms following T1. When T2 is emotionally charged, however, the AB phenomenon is attenuated (Keil & Ihssen, 2004), and this attenuation is thought to be mediated by a neural system involving the amygdala (Anderson & Phelps, 2001).

The experiment we report here can be thought of as a replication of Corden et al. (2008), even though the two studies were conceived independently of one another (Corden et al's., 2008 findings only came to our attention after having prepared this manuscript). In fact, the two studies differ in important ways methodologically, thus strengthening the findings of each. Our rationale for the experiment is based on our previous finding that individuals with ASD do not seem to retain physiologically arousing words in qualitatively distinct ways over time (Gaigg & Bowler, 2008; Gaigg & Bowler, under review). On the basis of this finding we hypothesised that such individuals would not accumulate distinct representations of emotional words in long-term memory, making it unlikely that such words would capture their attention in an AB paradigm.

Method

Participants

Twenty five individuals with a diagnosis of ASD (20 male, 5 female) and 25 typically developed individuals (20 male, 5 female) participated in the current study. Individuals with ASD were diagnosed by experienced clinicians and a review of available medical records and/or assessment with the Autism Diagnostic Observation Schedule (ADOS; Lord, et al., 1989) confirmed that all met DSM-IV (American Psychiatric Association, 2000) criteria for Autism Spectrum Disorder. Typical participants were recruited from the local community, and individually matched to within 7 points of verbal IQ (WAIS-III^{UK}; The Psychological Corporation, 2000) to ASD participants. Groups were also matched in terms of performance IQ, full-scale IQ and age. The relevant descriptive statistics for these group characteristics are set out in Table 3.1. The experimental procedures outlined below adhere to the ethical

guidelines set out by the British Psychological Society and were approved by the University's ethical committee.

Table 3.1

Summary of Age and IQ characteristics for the ASD and Comparison Group

	ASD (*n* = 25)		Comparison (*n* = 25)	
Measure	*M*	*SD*	*M*	*SD*
Age (years)	38.4	13.6	36.2	11.8
VIQ[a]	106.9	14.4	106.3	13.8
PIQ[b]	104.8	17.0	104.5	16.6
FIQ[c]	105.2	15.5	105.8	15.1

[a] Verbal IQ (WAIS-R^{UK} or WAIS-III^{UK})

[b] Performance IQ (WAIS-R^{UK} or WAIS-III^{UK})

[c] Full-Scale IQ (WAIS-R^{UK} or WAIS-III^{UK})

Materials & Design

The stimuli included a pool of 590 distracter words and 120 target words. Distracter words were 7 letters long and had a minimum written frequency (Kucera & Francis, 1967) of 10 per million (average 60.2 per million). In order to ensure adequate masking during the RSVP stream, target words were only 3-5 letters long. Sixty of these were designated T1 and included only emotionally neutral words. The remaining sixty were designated T2 and included 20 emotionally neutral words, 20 emotionally charged words (profanities, taboos, etc...) and 20 male first names. The latter were included to control for the possibility that semantic distinctiveness, rather than the emotional quality of words, facilitates T2 detection. In this context it is also worth noting that in previous studies ASD participants and typical participants did not differ in terms of either their galvanic skin responses to emotionally charged words or their subjective rating of arousal of such words (Corden et al., 2008; Gaigg & Bowler, 2008). T1 words and T2 words were closely matched on letter length as were the

three sub-categories of T2 words. Neutral and emotional T2s were furthermore equated on ratings of familiarity, which we obtained in a separate normative study in which 49 undergraduate students (35 female, 14 male) rated a set of 130 emotionally charged and neutral words on a 9-point scale (1 = not at all familiar; 9 = very familiar). The mean ratings for the neutral and emotional words included in the current experiment were 8.08 (*SD* = .50) and 7.84 (*SD* = 0.32) respectively. In order for the male first name T2s to be maximally distinct, we chose the ones most common in the UK.

Stimulus presentation was controlled by E-Prime software (Psychology Software Tools, 1996-2002), which presented words in bold, 26-point, Arial font in the centre of a Sony Laptop 15" monitor at a rate of 10 Hz (50 ms word durations + 50 ms blank intervals). Distracter words were always presented in blue ink, target words were always presented in red ink and the background colour of the screen was grey. Each trial started with a 1 second central fixation cross followed by the RSVP stream. Each of these streams consisted of 26 distracter words and 2 red targets. The first target (T1) occurred randomly between serial positions 5 and 20 whilst the second target (T2) occurred either one, three, or five distracter words after T1 (hereafter Lag 2, Lag 4 and Lag 6 trials). The resulting SOAs (Stimulus Onset Asynchrony) between T1 and T2 were therefore 200 ms for Lag 2 trials, 400 ms for Lag 4 trials and 600 ms for Lag 6 trials. Thus, Lag 2 and Lag 4 trials presented T2s within the AB period whilst on Lag 6 trials T2s occurred after this critical period.

The experiment consisted of a total of 180 trials including 60 trials for each of the three Lag conditions. Within Lag conditions, 20 trials each included either a neutral, emotionally charged or male first name T2. Target words were repeated three times during the experiment such that each of the T1 and T2 words appeared once in each of the Lag conditions. Distracter words were repeated 7-8 times during the experiment. The order of presentation of the various trial types was random without constraints.

Procedure

Participants were tested individually in a sound attenuated laboratory and informed that they would be shown 180 very rapid word sequences that consisted mainly of blue words but also two red words that they should try and identify (written responses were requested). For ethical reasons, participants were forewarned about the sexually explicit and offensive nature of some of the words included in the study. To avoid a bias in favour of detecting emotionally charged words, participants were also told that they would see male first names. They were not told that the first red word was always neutral. Following the instructions, participants were asked for their consent and given a series of practice trials constructed from a separate pool of words (all neutral). Once they were confident that they had understood what was required of them, the experimenter started the experimental trials and left the room.

Results

Prior to analysing T2 detection rates, we computed the proportion of correctly reported T1 words for each of the 9 experimental conditions (3 T2 word types x 3 lag conditions). As expected, detection rates for these targets were very high (M = .97, SD = .04). A 2 (Group) x 3 (T2 word type) x 3 (Lag) mixed ANOVA of T1 reports revealed a main effect of Lag ($F(2,47) = 3.68$, $p < .05$), with detection rates on Lag 6 trials (M = .97, SD = .04) being significantly higher than on Lag 2 trials (M = .96, SD = .04; $t = 2.69$, $df = 49$, $p < .05$). Detection rates on Lag 4 trials fell in between (M = .97, SD = .04). No other main effects or interactions were significant (Fs < 1.10).

For the analysis of T2 detection rates, only trials on which T1 was correctly identified were taken into account since only these trials reliably index the AB phenomenon (e.g. Keil & Ihssen, 2004). Figures 3.1a and 3.2b set out the proportions of correctly identified T2s as a function of the experimental manipulations and participant group. A 2 (Group) x 3 (T2 word type) x 3 (Lag) mixed ANOVA of these data revealed the expected main effects of T2 word type ($F(2,47) = 14.09$, $p < .001$, Greenhouse-Geisser corrected) and Lag ($F(2,47) = 82.12$, $p < .001$, Greenhouse-Geisser corrected), and a T2 word type x Lag interaction ($F(4,45) =$

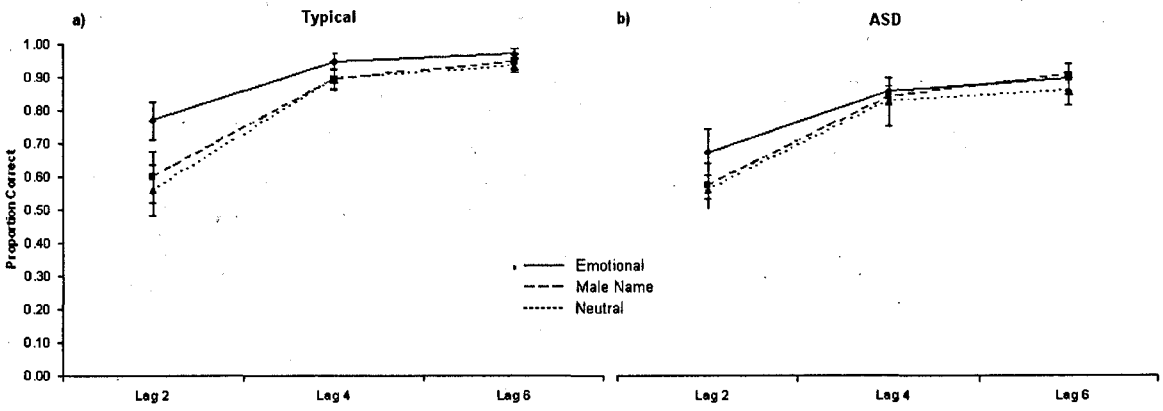
13.48, $p < .001$, Greenhouse-Geisser corrected). The main effect of T2 word type confirms that detection rates of emotionally charged words ($M = .85$, $SD = .17$) were significantly higher than detection rates of male first names ($M = .79$, $SD = .17$; $t = 3.55$, $df = 49$, $p < .01$) and neutral words ($M = .77$, $SD = .18$; $t = 4.11$, $df = 49$, $p < .001$). In addition, the detection of male first names was superior to that of neutral T2s ($t = 2.34$, $df = 49$, $p < .01$). The main effect of Lag, replicates the AB phenomenon with detection rates during Lag 2 trials ($M = .62$, $SD = .28$) being significantly lower than during Lag 4 trials ($M = .88$, $SD = .14$; $t = 9.50$, $df = 49$, $p < .001$), which in turn were lower than during Lag 6 trials ($M = .92$, $SD = .10$; $t = 2.34$, $df = 49$, $p < .001$). As indicated by Figures 3.1a and 3.1b, the interaction between T2 word type and Lag was partially due to the near ceiling performance during Lag 4 and Lag 6 trials, which compressed detection rates across word types in comparison to Lag 2 trials. There is, however, more to this interaction than is first apparent. More specifically, whilst male first names and neutral T2s were detected with similar frequency during Lag 2 (Male names $M = .59$, $SD = .31$; Neutral $M = .56$, $SD = .32$; $t = 1.29$, $df = 49$, *ns*) and Lag 4 trials (Male names $M = .87$, $SD = .15$; Neutral $M = .86$, $SD = .17$; $t = 0.45$, $df = 49$, *ns*), during Lag 6 trials male first names were detected more frequently than neutral T2s (Male names $M = .93$; $SD = .11$; Neutral $M = .90$; $SD = .12$; $t = 2.91$, $df = 49$, $p < .01$). Thus, semantic distinctiveness seemed to facilitate T2 detection only after, but not during, the critical AB time-window.

The data set out in Figures 3.1a and 3.1b suggests that the ASD group was less susceptible to the emotional modulation of the AB than the typical comparison group. Although the foregoing analysis yielded no interactions or main effects involving the group factor, as predicted, within group analyses showed that the effect of T2 word type on target detection was only significant in the typical comparison group ($F(2,23) = 21.69$, $p < .001$, Greenhouse-Geisser corrected) but not the ASD group ($F(2,23) = 2.61$, *ns*, Greenhouse-Geisser corrected). Calculations of effect sizes indicated that the effect of T2 word type was more than twice the size in typical ($r = .61$) compared to ASD ($r = .28$) participants[14]. To put these effect sizes into perspective, the sizes of the main effects of Lag within each group were .79 for the typical and .75 for the ASD group.

[14] These effect sizes are calculated on the basis of the Greenhouse Geisser adjusted degrees of freedom.

Figure 3.1

Proportion of correctly reported 2nd Target Words (T2s) as a function of Word Type, Lag and Participant Group (Error Bars represent Standard Errors)



In addition to the quantitative differences revealed by the within-group analyses, we also noted that the magnitude of the emotional modulation of the AB (i.e. difference between emotional T2 detection and male first name T2 detection[15]) in the ASD group was significantly correlated with VIQ (Lag 2: $r = .53$, $p < .01$; Lag 4: $r = .37$, $p = .07$), which was not the case for the comparison group (Lag 2: $r = -.24$, *ns*; Lag 4: $r = -.19$, *ns*). Fisher's z transformations showed that the differences in these correlations between groups were significant for Lag 2 ($p < .01$) and marginally significant for Lag 4 ($p = .052$). Figures 3.2a and 3.2b depict the relevant scatter plots for these correlations (for illustrative purposes average difference scores across Lag 2 & Lag 4 trials are presented) and show that the association in the ASD group was not merely an artefact of individual variability (Fisher's transformations showed that correlations calculated on the basis of averages across Lags 2 & 4 (ASD $r = .502$, $p < .05$; Typical $r = -.252$, *ns*) significantly differed between groups ($p < .01$)).

[15] It should be noted that difference scores between emotionally charged and neutral T2s yield the same pattern of results, which is not surprising given that semantic distinctiveness does not seem to facilitate T2 detection during the AB time-window. We present difference scores based on male first name T2s here as these more conservatively estimate the impact of emotion on the AB.

Figure 3.1

Proportion of correctly reported 2nd Target Words (T2s) as a function of Word Type, Lag and Participant Group (Error Bars represent Standard Errors)

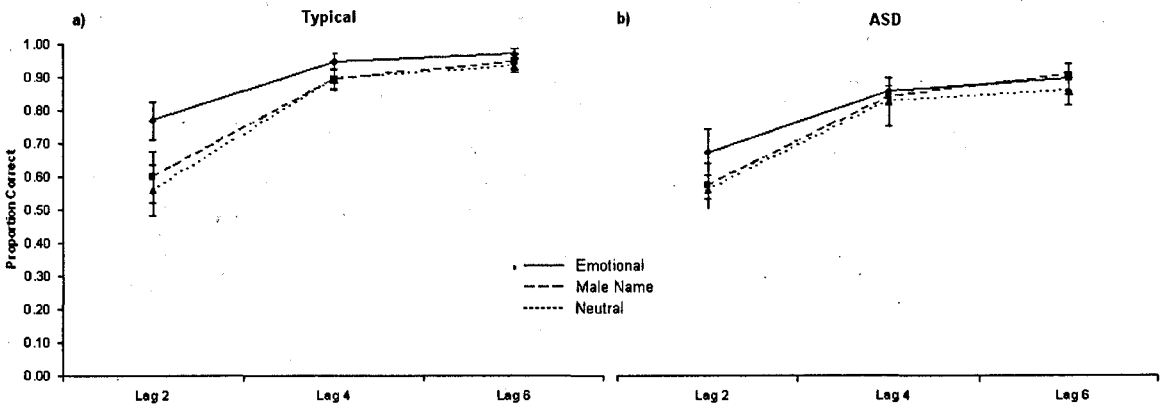


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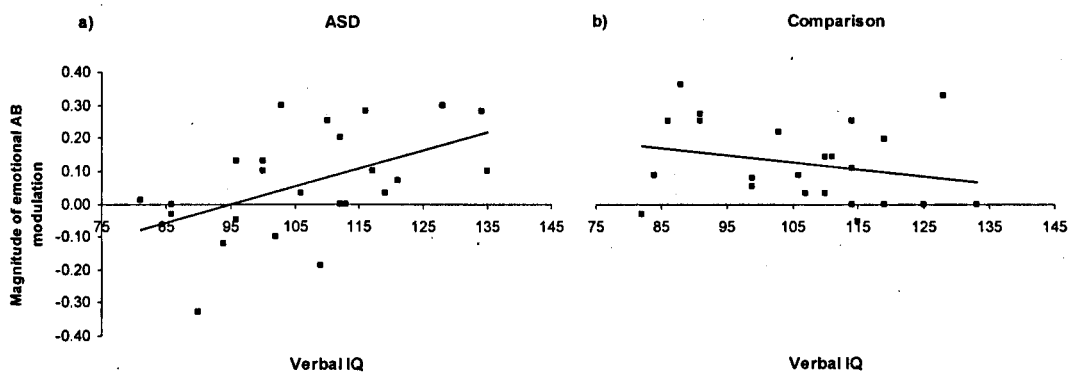
Figure 3.2

Scatter plots depicting the association between the magnitude of the emotional modulation of the Attentional Blink and VIQ for the ASD and Comparison Group



Discussion

The current experiment adopted the Attentional Blink paradigm in order to determine whether a group of individuals with ASD, like typically developed individuals, would exhibit enhanced attention to emotionally significant words. Based on our previous observation that individuals with ASD do not retain emotionally charged words in a qualitatively distinct manner over time (Gaigg & Bowler, 2008; Gaigg & Bowler, under review a), we predicted that the magnitude of the AB would not be modulated by the emotional significance of words in this group. Although our analyses revealed no between-group differences that would support this prediction, within-group analyses clearly indicated that the effect of emotion on T2 detection was reduced (and actually not statistically reliable) in the ASD group. In addition, the emotional modulation of the AB in the ASD group was unusually associated with participants' verbal IQ. Together, this pattern of results supports the recent observations by Corden et al. (2008) in showing that the emotional significance of words does not capture the attention of individuals with ASD as readily as that of typical individuals. Importantly, the present and Corden et al. (2008) studies differed with respect to the use of control measures to rule out the possibility that general processing demands, rather than emotion-specific demands, were responsible for the atypical pattern of performance by individuals with ASD.

Corden et al. (2008) manipulated perceptual properties of T2s (i.e. brightness) for this purpose whereas we manipulated the semantic properties of T2 words (i.e. by including male first names). In both cases, these manipulations affected performance in ASD and typical participants similarly and in both cases performance of typical participants was affected much more by the emotional properties of T2 words. Since it is difficult to know how best to equate emotional and non-emotional words on distinctiveness, the two studies together present a strong case for the specificity with which emotion *per se* modulates early perceptual processes in the AB paradigm. In turn, this strengthens the conclusion that the atypical pattern of performance observed in individuals with ASD represents abnormalities in emotion specific processes.

The present findings did not only strengthen the observations by Corden et al. (2008) but also revealed an interesting and unusual association between VIQ and the magnitude of the emotional modulation of the AB in the ASD group. Such associations between VIQ and task performance parallel findings from socio-emotional tasks, such as those assessing the ability to identify emotion from facial or bodily expressions (e.g. Ozonoff, et al., 1990). This may suggest that difficulties in emotional processes within and outside the social domain in ASD are the result of a common developmental pathway. Corden et al. (2008) came to a similar conclusion after noting that the attenuated emotional modulation of attention in the AB paradigm was associated with poorer performance on a facial fear recognition task. It will be important for future research to clarify how emotional processing difficulties within and outside the social domain relate to one another over the course of development, because such clarification would contribute valuable information to the long-standing debate as to whether ASD is fundamentally a disorder of socio-cognitive or emotional development (e.g. Baron-Cohen, 1995; Hobson, 2002). It will also be important to extend the work on non-social emotional processing to lower functioning individuals from the autism spectrum in order to establish whether findings such as the current ones are representative of the broader phenotype of the disorder.

At the neural level of analysis, the present findings invite the inference that abnormalities in amygdala functioning may be responsible for the atypical pattern of

performance by individuals with ASD. Although several lines of evidence from both the typical and ASD literature (e.g. Anderson & Phelps, 2001; De Martino, Kalish, Rees & Dolan, 2008; Schultz, 2005) would support this suggestion, it is important to remember that the amygdala operates within complex neural systems. In the context of the AB paradigm, for instance, interactions between the amygdala, cingulate cortex and frontal cortical areas seem to be important (De Martino et al., 2008) and the abnormality in ASD may lie anywhere in this system (or perhaps even outside it). As Corden et al., (2008) point out, functional imaging studies will be an important next step in this context and our prediction for such studies would be that the functional connectivity between the amygdala and relevant cortical areas of the brain would be compromised in ASD (see Gaigg & Bowler, 2007; Gaigg & Bowler, 2008 for further discussion). Regardless of the nature of the neural correlate, however, a hypothesised amygdala involvement seems to serve a useful heuristic purpose for furthering our understanding of the cognitive characterisation of emotional processing difficulties in ASD. In this respect the current findings add to a growing literature that demonstrates atypicalities in this domain outside the broader context of social cognition.

Summary

The memory experiments presented in Chapter 2 suggested that individuals with ASD retain emotionally significant words no differently from comparable neutral words and that, as a consequence, they do not seem to accumulate distinct representations of such words in long-term memory. The findings of Experiment 4 lend support to this suggestion by showing that emotive words do not capture the attention of individuals with ASD any more than neutral words. This indicates that the abnormalities observed in Chapter 2 extend outside the memory domain, making it unlikely that generic memory difficulties in the ASD group were responsible for the pattern of the earlier observations. Instead, this condition seems to be characterised by rather specific abnormalities in how the emotional significance of words impacts on the cognitive processing of those words. The present experiment also supports the idea that abnormalities in amygdala function may be responsible for the abnormal way in which emotionally significant words are processed in ASD. Patients with

amygdala lesions have been shown not to exhibit enhanced detection rates for emotionally charged words in the kind of AB paradigm used here (Anderson & Phelps, 2001) and although arousal was not directly measured in the present experiment, the observations confirmed that the enhanced perception of emotionally significant words during the AB time-window was not simply the result of such words being semantically distinct. Together with the independent observations by Corden et al. (2008), which only came to light during the reviewing process of the manuscript presented above, these observations support the notion that the emotional suppression of the AB is mediated by the amygdala and so the atypical pattern of performance seen in the ASD group is suggestive of abnormalities in this limbic structure.

The foregoing claims are compromised by the fact that all experiments reported so far were concerned with the study of how the emotional significance of individual words influences cognition. Since abnormalities in language development constitute a clinically defining manifestation of ASD, one may argue that the findings so far simply reflect abnormalities in linguistic processing in this condition rather than more specific atypicalities in how stimulus induced arousal modulates cognition. Several aspects of the experimental designs employed so far make this unlikely. First, many ASD individuals who participated in the above experiments met diagnostic criteria for Asperger's syndrome, which is not thought to be associated with abnormalities in language development (Lord et al., 1989). Second, participant groups were closely matched (on a person by person basis) on the basis of their performance on a standardised verbal ability test. Third, the performance of ASD participants differed from that of typical participants only with respect to emotionally significant words. Performance for non-emotional words was very similar between groups regardless of whether or not these stimuli were semantically distinct and / or interrelated. In other words, individuals with ASD seemed to process semantic aspects of words similarly to typical participants. Most importantly, however, individuals with ASD did not fail to process the emotional significance of words altogether. Like typical individuals they rated such words as emotionally significant and like typical participants they exhibited arousal responses to such words. Individuals with ASD even remembered emotionally significant words better than non-emotional words over short periods of time. The differences between ASD and typical participants, therefore, were

very specific. Individuals with ASD were unable to retain emotionally distinct representations of emotional words over time and they were relatively poor at providing subjective ratings of words that reflected the individual's actual physiological responses to them. It is difficult to imagine what kind of language difficulty could give rise to this particular pattern of observations. A more plausible account would invoke abnormalities in the kind of processes that are normally associated with amygdala functioning.

Even if it is unlikely that generic language difficulties were responsible for the pattern of observations so far, the hypothesis that ASD is characterised by abnormalities in amygdala functioning that disrupt the acquisition of distinctly emotional representations of stimuli, needs to be validated for non-verbal stimuli. The next chapter will provide a very rigorous test of this hypothesis by drawing on a fear-conditioning paradigm, which is known to necessitate the functional integrity of the amygdala.

CHAPTER 4: LEARNING ABOUT THE EMOTIONAL SIGNIFICANCE OF NON-VERBAL STIMULI IN ASD

The following experiment has been published in *Neuropsychologia* (Gaigg & Bowler, 2007) and includes sufficient background information not to warrant a more general introduction.

Experiment 5: Differential fear conditioning in Autism Spectrum Disorder: Implications for an amygdala theory of autism

Introduction

In the current paper we draw on the fear conditioning literature in order to provide further insights into emotional processes in ASD. Fear conditioning is a form of Pavlovian conditioning through which individuals learn the hedonic values of previously neutral stimuli via a process of association. In a typical fear conditioning study participants are presented with a simple visual or auditory stimulus alongside a painful or noxious stimulus such as a startling noise or mild electric shock (the unconditioned stimulus; UCS). Naturally, individuals will respond to such noxious stimuli with species-typical defence behaviours (the unconditioned response; UCR) such as increased autonomic activity, which in humans can readily be measured by monitoring skin conductance responses (SCR; Frederikson, Annas, Georgiades, Hursti & Tersman, 1993). After a few pairings of the neutral stimulus and the UCS, participants will start to exhibit such fear responses to the neutral stimulus alone (the stimulus has become a conditioned stimulus; CS), indicating that they have learned the association between the noxious and neutral stimuli.

To date, only one investigation has examined fear conditioning in ASD. Bernier and colleagues (Bernier, et al., 2005) employed a potentiated startle paradigm in order to assess

simple fear conditioning in a group of adolescents and adults with ASD. The authors aversively conditioned participants to a red square by pairing its presentation with an aversive puff of air to the throat. Following several pairings of the red square and the puff of air, the authors assessed participants' eye-blink startle response to either a loud noise presented alone or accompanied by the red square. The results showed that as in typical participants, eye-blink startle responses in the ASD group were enhanced during the trials including the red square indicating that both groups had learned the aversive nature of the conditioned stimulus to similar extents.

There are several reasons why such studies of fear conditioning are of value to our understanding of ASD. First, as Bernier and colleagues point out, such studies contribute to our understanding of the neuropathology underlying this spectrum of disorders. Extensive animal (see LeDoux, 1994, 1995, 1998, 2000 for detailed reviews) and human (Bechara, Tranel, Damasio, Adolphs, Rockland & Damasio, 1995; Büchel, Morris, Dolan & Friston, 1998; Büchel, Dolan, Armony & Friston, 1999; Cheng, Knight, Stein & Smith, 2003; Knight, Smith, Cheng & Stein, 2004; LaBar, LeDoux, Spencer & Phelps, 1995; LaBar, Gatenby, Gore, LeDoux & Phelps, 1998; Morris, Friston & Dolan, 1997, Morris, Friston & Dolan, 1998; Morris, Őhman & Dolan, 1997; Phelps, et al., 1998) research has demonstrated that the associative learning in fear conditioning paradigms is mediated by the amygdala, a limbic structure which has attracted increasing attention in relation to ASD in recent years. Although several lines of research have implicated the amygdala in the pathology underlying this disorder (e.g. Bachevallier, 1994; Bachevallier, 2000; Baron-Cohen et al., 1999; Baron-Cohen, et al., 2000; Fotheringham, 1991; Howard, Cowell, Boucher, Broks, Mayes, Farrant, et al., 2000; Sweeten, et al., 2002), the evidence is somewhat inconsistent and the extent and nature of the proposed amygdala pathology remain unclear (see Sweeten et al., 2002; Amaral, et al., 2003; Palmen, van Engeland, Hof & Schmitz, 2004 for recent reviews). Fear conditioning paradigms are valuable in this respect because different forms of conditioned fear behaviour have been shown to rely on different amygdala nuclei or pathways. The acquisition of fear in simple conditioning paradigms such as the one employed by Bernier and colleagues (2005), for example, is thought to be mediated primarily by a sub-cortical amygdala system involving direct sensory afferent projections from thalamic nuclei and

efferent connections to various brainstem and hypothalamic nuclei that mediate the behavioural and physiological fear responses (LeDoux, 1998; LeDoux, 2000). As Bernier and colleagues (2005) point out, their findings suggest that at least this sub-cortical system appears to be functionally relatively intact in ASD. Important for our current investigation are findings which suggest that fear acquisition in more complex differential fear conditioning paradigms, in which participants acquire fear to only one of several different stimuli (e.g. different colours), have been shown to rely on cortical modulation of the sub-cortical amygdala system (Jarrell, Gentile, Romanski, McCabe & Schneiderman 1987; Morris et al., 1997). This cortical modulation is thought to be important for the regulation of fear responses according to the specific conditioning contingencies (i.e. responding to the conditioned stimulus but not to the non-conditioned stimuli). Since several lines of evidence indicate that ASD may be characterised by poor connectivity between disparate brain regions (Belmonte, Allen, Beckel-Mitchener, Boulanger, Carper & Webb, 2004; Ben Shalom, 2000; Brock, et al., 2002; Castelli, Frith, Happe & Frith, 2002; Just, Cherkassky, Keller & Minshew, 2004; Just, et al., 2007; McAlonan, et al., 2005; Rippon, et al., 2007), an investigation of differential fear conditioning in ASD may provide valuable behavioural insights into the functional integrity of cortico-amygdala connectivity in this population.

In addition to providing further insights into the functional integrity of amygdala systems, studies of fear conditioning may also inform debates about the developmental role of emotional atypicalities in the clinical presentation of ASD. To date most investigations relevant to this debate have focused on how individuals with ASD perceive and express emotions within the broader context of social behaviour. Although the evidence in this area is relatively consistent in illustrating that ASD is characterised by difficulties in the recognition (Hobson, 1986a,b; Hobson, et al., 1988a,b; Hobson, 1991; Weeks & Hobson, 1987) and context appropriate expression of emotions (Dawson, et al., 1990; Kasari, et al., 1990; Kasari, et al., 1993; Sigman, et al., 1992; Yirmiya, et al., 1989; Yirmiya, et al., 1992), these findings can be accommodated within competing explanatory frameworks. In line with Kanner's (1943) original conclusion, some authors have argued that emotional atypicalities constitute a primary and possibly innate feature of the autistic phenotype. Hobson (1989) for example suggests that individuals with ASD are characterised by difficulties in understanding the

hedonic value of their sensory-motor environment which results in an abnormal developmental progression of interpersonal relatedness (See Mundy & Sigman, 1989 for a similar suggestion). Others, however, argue that the emotional difficulties evident in ASD are secondary to impairments in more general socio-cognitive processes. Schultz (2005), for example, argues that primary face processing atypicalities are responsible for the aberrant development of socio-emotional behaviours, whilst Baron-Cohen and colleagues (Baron-Cohen et al., 1999, Baron-Cohen et al., 2000) have suggested that difficulties in Theory of Mind (ToM) understanding give rise to the abnormal social and emotional behaviours characterising the autism spectrum. These latter accounts are supported by evidence which suggests that individuals across the autism spectrum experience difficulties in processing faces (e.g. Gross, 2005; Joseph and Tanaka, 2003; Partland, Dawson, Webb, Panagiotides & Carver, 2004; Spezio, et al., 2007) and understanding mental states such as beliefs and desires of others (e.g. Baron-Cohen, et al., 1985; Happe, 1995; but see Bowler, Briskman, Gurvidi & Fornells-Ambrojo, 2005). Fear conditioning paradigms may provide important new insights into this issue because they assess a relatively basic and automatic emotional process that does not necessitate intact socio-cognitive processes. In addition, because fear conditioning paradigms assess the processes by which individuals learn the hedonic value of sensory stimuli, such paradigms constitute a relatively direct test of Hobson's (1989) suggestion that ASD may be characterised by atypicalities in understanding the hedonic value of their sensory-motor environment.

Finally, studies of fear conditioning in ASD will add to a small but growing number of studies that have investigated emotional processes in this population at the psychophysiological rather than the behavioural level. Since it is widely accepted that psychophysiological responses form an integral part of emotional experiences and behaviours (Cannon, 1929; James, 1884), the investigation of such responses in ASD is vital to understanding the nature of emotional atypicalities in this population. The limited evidence in this area to date suggests that like typical individuals, individuals with ASD exhibit changes in autonomic activity, such as increases in skin conductance responses (SCR) or changes in heart rate, when presented with emotionally salient pictures (Ben Shalom et al., 2003; Blair, 1999; Hillier, et al., 2006; Salmond, et al., 2003), aversive auditory stimuli (Bernier, et al.,

2005; Salmond et al., 2003) or emotive words (Gaigg & Bowler, 2008). However, these physiological responses seem to be atypically modulated by specific stimulus properties in ASD. Blair (1999) for example found that although children with ASD exhibited typically increased SCRs to distress cues (e.g. crying face) compared to neutral images, their responses to threatening images (e.g. gun) were less consistently elevated than in the comparison group (see Hillier et al., 2006 for similar findings). Similarly, SCRs to faces in ASD have been found to be abnormally modulated by the direction of gaze (Joseph, et al., 2005; Kylliainen & Hietanen, 2006). As Ben Shalom (2000) suggests, this pattern of results would be in line with the suggestion that ASD is characterised by atypicalities in the connectivity between cortical areas responsible for the cognitive appraisal of emotional stimuli and the amygdala which mediates our physiological reactions to such stimuli. Such a view is also supported by the finding that unlike in typical individuals, SCRs do not seem to correlate with subjective ratings of emotionality in ASD (Gaigg & Bowler, 2008; Hillier et al., 2006).

As this brief overview of the literature illustrates, there are several reasons why the study of fear conditioning is important for our understanding of ASD. In the current study we draw on a differential fear conditioning paradigm employed by Bechara and colleagues (Bechara et al., 1995) in order to test the hypothesis that individuals with ASD would exhibit a pattern of atypicality consistent with the suggestion that the amygdala is abnormally modulated by cortical areas. Thus, on the basis of the evidence suggesting that the sub-cortical amygdala system is functionally intact in ASD (e.g. Bernier et al., 2005) we hypothesised that a group of ASD participants would exhibit typical patterns of physiological responses to aversive stimuli and that their autonomic activity would exhibit evidence of learning the hedonic value of a previously neutral stimulus. However, based on the suggestion that the amygdala may not be modulated normally by cortical areas, we expected that participants with ASD would not exhibit a typical pattern of acquiring fear discriminatively to conditioned and non-conditioned stimuli. If our predictions are borne out this pattern of results would lend support to Hobson's (1989) suggestion that ASD may be characterised by difficulties in understanding (in this case learning about) the hedonic value of their sensory-motor environment.

Method

Participants

Fifteen individuals with ASD (12 male, 3 female) and sixteen typical individuals (13 male, 3 female) participated in this experiment. One female ASD and two male comparison participants were excluded from all analyses as they failed to exhibit detectable changes in skin conductance in response to the UCS (80dB - 100dB foghorn sound). Participants in the final sample (N = 14 per group) were matched on chronological age (ASD mean = 29.7 yrs., SD = 10.2; Comparison mean = 30.4 yrs., SD = 12.2) and WAIS-III^{UK} (The Psychological Corporation, 2002) full scale IQ (ASD mean = 111, SD = 17.3; Comparison mean = 109, SD = 12.7). Individuals with ASD had all received their diagnosis according to conventional criteria (DSM-IV-TR, American Psychiatric Association, 2000; ICD-10, World Health Organisation, 1992) by experienced clinicians and none suffered any co-morbid anxiety disorders. The comparison group was recruited locally through newspaper advertisements. All individuals were free of medication and none of the participants exhibited discrepancies between verbal and non-verbal IQ of more than 15 points (i.e. 1.5 SDs), which could indicate neuropathology non-specific to ASD. The experimental procedures outlined below adhered to the ethical guidelines set out by the British Psychological Society and were approved by the University's Senate Ethical Committee. All participants were fully briefed before the experiment and all provided informed consent to participate in the study.

Materials & Design

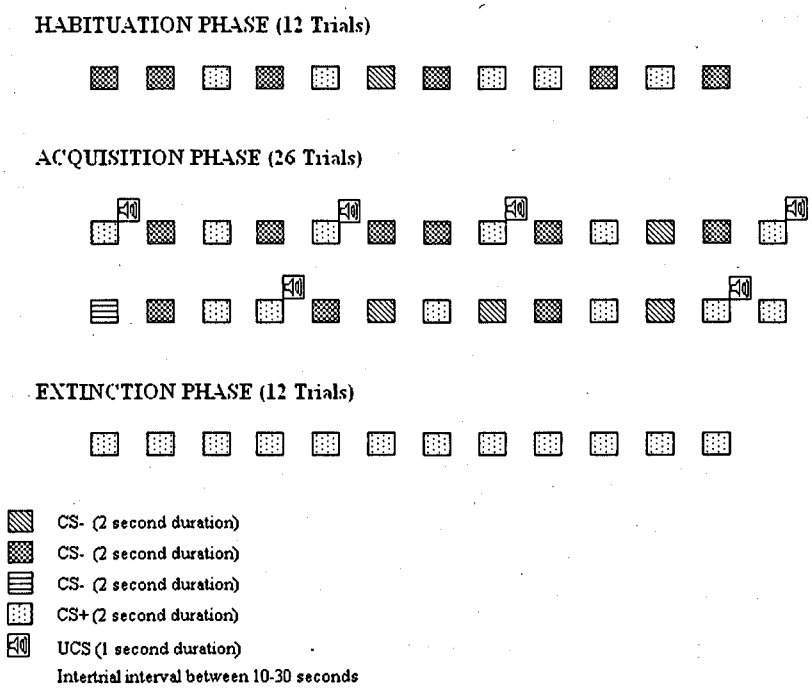
The conditioning protocol was based on that employed by Bechara et al. (1995) and consisted of 12 habituation trials, 26 acquisition trials and 12 extinction trials. Four coloured slides (yellow, green, blue and red) presented for 2 seconds each on a Sony laptop 15" monitor served as stimuli. One of these colours was designated CS+ and was to be followed by the UCS (1sec foghorn between 85 and 100 dB) during the acquisition phase of the protocol. The remaining colours were designated CS- and were never paired with the UCS. The choice of colour for CS+ was counterbalanced across participants. During habituation

and acquisition, colours were presented at different frequencies in a fixed pseudorandom order (see Bechara et al., 1995 for details). The colour used as the CS+ stimulus occurred 5 times during the habituation phase and 12 times during the acquisition phase and appeared on the same trials for all participants. The remaining trials consisted of the CS- colours. The rate of presentation was one colour approximately every 10-30 seconds, dependent on the participants' skin conductance responses (a new stimulus was presented only when there was no sign of galvanic activity for at least 2 seconds). During the acquisition phase CS+ was reinforced according to a variable ratio schedule. Thus, six of the 12 occurrences of CS+ were immediately followed by the UCS ($CS+_{paired}$ trials), whereas the other 6 presentations of CS+ were not ($CS+_{unpaired}$ trials). During extinction, CS+ was presented repeatedly without any further presentations of the UCS. The illustration in Figure 4.1 summarizes this protocol.

Throughout the experiment, SCR was recorded via two surface electrodes attached to the medial phalanges of the first and third digits of the non-dominant hand (assessed by asking participants). Data were recorded using PowerLab hardware (ADInstruments, 2004), which sampled electrodermal activity at 1 kHz. Chart 5 software (ADInstruments, 2004) was used for the recording and assessment of the data. SCRs were computed according to standard criteria with the largest deflection during an 8 second window following the onset of a stimulus serving as a measure of autonomic response to that stimulus. All SCRs were square-root transformed prior to statistical analyses in order to normalise the distribution of the data.

Figure 4.1

Experimental procedure for the differential fear conditioning paradigm



Procedure

Participants were tested individually in a semi-soundproof air-conditioned room. They were warned that the experiment would involve hearing some startling noises, which were demonstrated at an initially low volume through speakers. All participants were then allowed to choose a volume level that they would find startling but in no way painful. Subsequent to the attachment of the electrodes, participants were asked to relax and find a comfortable seating position approximately 50 cm in front of the screen. They were asked to try and pay attention to the colours on the screen and to move as little as possible throughout the task in order to avoid movement artefacts. The experiment commenced following a few minutes during which SCRs were allowed to reach baseline activity. The experimenter was present throughout the whole of the procedure to control stimulus presentation and monitor SCRs.

Seating arrangements were such that the participant was seated at approximately 1.5 m from the experimenter with no equipment apart from the attached electrodes and the presentation laptop in line of sight.

Following Bechara and colleagues (1995) participants' declarative memory for the experimental contingencies was probed around 5 minutes after the experimental procedure by asking them; 1) How many colours did you see? 2) What colours were they? 3) How many colours were followed by the loud noise? 4) What colour(s) was/were it/they? Correct responses to questions 1, 2 and 3 received a score of 0.5 whereas a correct response to question 4 received a score of 2.5, reflecting the fact that question 4 asks for the most important aspect of the experimental contingencies.

Results

Groups did not differ significantly in terms of the UCS intensities they chose (ASD group: M = 94dB, SD = 6; Comparison group: M = 97dB, SD = 3). Similarly, a 2 (group) x 6 (trial) mixed ANOVA of SCRs elicited during the 6 $CS+_{paired}$ trials of acquisition revealed no main effects or interactions (All Fs < 1). Thus, the UCS was similarly effective for both groups in eliciting startle responses and neither group seemed to habituate to the UCS during the acquisition phase. An analysis of participants' declarative knowledge revealed 100% accuracy for the comparison group but 5 of the ASD participants made errors in response to at least 1 of the questions. Thus the ASD group performed significantly worse in terms of noticing or remembering the experimental parameters (U = 63, z = 2.41, p < .05). As we will illustrate below, an assessment of individual data suggested that failing to remember the experimental contingencies was not directly related to autonomic fear acquisition, making it unlikely that the results described below are confounded by this group difference.

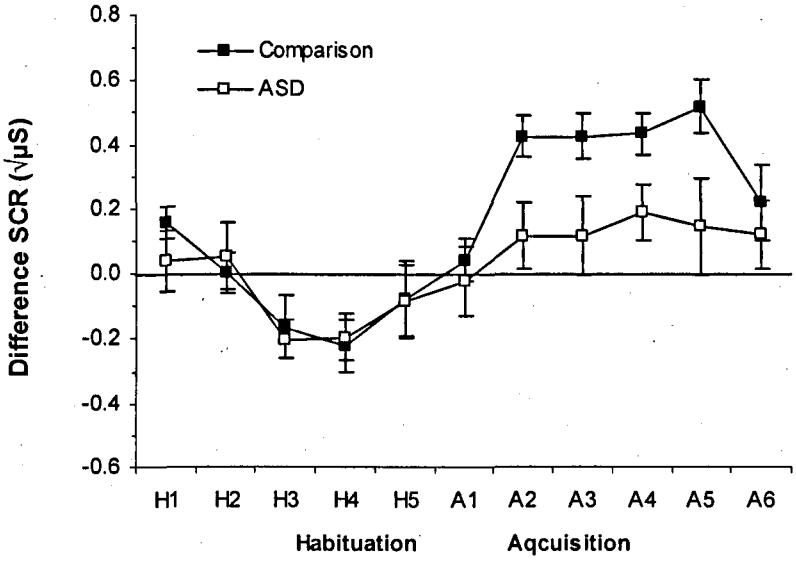
In order to assess the conditioning data we adopted a similar method to that of LaBar and colleagues (1995). Thus for our first analysis we computed difference scores by subtracting the average response elicited during CS- trials from SCRs elicited by each of the $CS+_{unpaired}$ trials. The resulting difference scores thus indicate to what extent SCRs during

$CS+_{unpaired}$ trials exceeded the average response elicited by CS- presentations. Figure 4.2 illustrates these difference scores for the relevant five habituation and six acquisition trials as a function of group. For the analysis of these data the first acquisition difference score was omitted since in differential conditioning paradigms the association between CS+ and the UCS only becomes fully apparent after the second pairing between these stimuli. A 2 (Group) x 2 (Phase) x 5 (Trial) mixed ANOVA revealed main effects for Phase (Habituation: M = -.07√μS, SD = .11; Acquisition: M = .27√μS, SD = .15; $F(1,26) = 50.11$, $p < .001$) and group (ASD group: M = .03√μS, SD = .13; Comparison group: M = .17√μS, SD = .13; $F(1,26) = 8.00$, $p < .01$), which were further characterised by a Phase x Group interaction ($F(1,26) = 6.90$, $p < .05$). Post-hoc comparisons showed that this interaction was due to the fact that SCR difference scores were similar for both groups during habituation (ASD group: M = -.08√μS, SD = .15; Comparison group: M = -.06√μS, SD = .15), whilst the ASD group exhibited significantly attenuated difference scores relative to the comparison group during the acquisition phase (ASD group: M = .14√μS, SD = .21; Comparison group: M = .40√μS, SD = .21; $F(1,26) = 11.30$, $p < .01$). Importantly, however, separate analyses of the groups, revealed main effects of experimental Phase for both the ASD ($F(1,13) = 13.27$, $p < .01$) and the comparison group ($F(1,13) = 37.59$, $p < .001$). Thus, although participants with ASD exhibited attenuated fear acquisition in comparison to typical participants, the data confirm our prediction that individuals with ASD would show evidence of acquiring autonomic fear responses to a previously neutral stimulus.

The attenuation of difference scores in the ASD group during acquisition could have several sources. First, it is possible that ASD participants compared to typical participants exhibited either attenuated SCR responses to $CS+_{unpaired}$ stimuli or excessive responses to CS- stimuli during the acquisition phase of the protocol. Second, it is possible that only a subgroup of ASD participants exhibited abnormalities to the effect of significantly reducing the groups' average in relation to the comparison group.

Figure 4.2

Skin conductance difference scores ($CS+_{unpaired}$ – average CS-) during habituation and acquisition for the ASD and Comparison Group (Error Bars show Standard Errors)

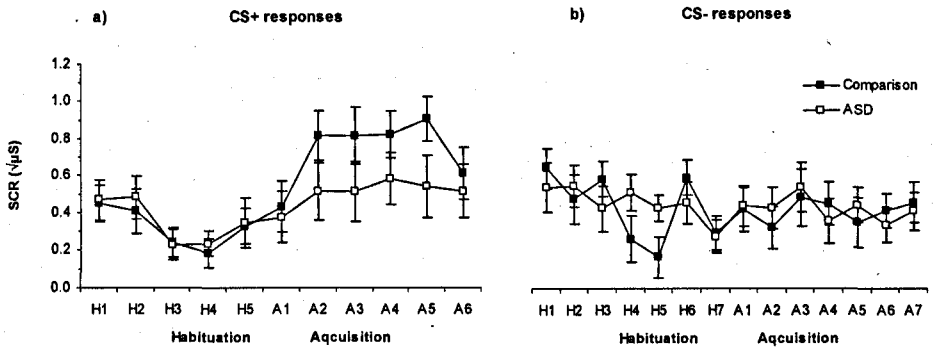


In order to assess these possibilities we carried out two further analyses. First we assessed SCRs separately for $CS+_{unpaired}$ and CS- stimuli as a function of group and experimental phase. Figure 4.3a and 4.3b illustrate these data and suggest that the attenuated differential fear acquisition in the ASD group was largely the result of reduced SCRs to $CS+_{unpaired}$ stimuli during the acquisition phase of the experiment. A 2 (group) x 2 (phase) x 5 (trial) mixed ANOVA of these data (again the first $CS+_{unpaired}$ trial of acquisition was excluded) revealed a main effect of Phase ($F(1,26) = 16.40$, $p < .001$) confirming that $CS+_{unpaired}$ responses during acquisition ($M = .66\sqrt{\mu}S$, $SD = .31$) were higher than during habituation ($M = .35\sqrt{\mu}S$, $SD = .17$). Furthermore, the data were characterised by a significant interaction between Phase and Trial ($F(4,23) = 4.92$, $p < .01$) and a marginal interaction between Phase and Group ($F(1,26) = 2.94$, $p = .098$). Post-hoc analyses revealed that the interaction between Phase and Trial was due to a decrease in responses over trials during habituation ($F(4,23) = 3.86$, $p < .05$) but not during the acquisition phase. The marginal interaction between phase and group was due to the fact that for the ASD group the effect of

experimental phase was only marginally significant (Habituation: M = .35√μS, SD = .24; Acquisition: M = .53√μS, SD = .44; $F(1,13) = 3.36$, p = .090) whereas for the comparison group this effect was highly reliable (Habituation: M = .35√μS, SD = .24; Acquisition: M = .79√μS, SD = .44; $F(1,13) = 14.00$, $p < .01$). As Figure 3b suggests, responses to CS- stimuli were not characterised by any main effects or interactions[16].

Figure 4.3

Skin conductance responses to CS+ (a) and CS- (b) presentations during habituation and acquisition for the ASD and Comparison Group (Error Bars indicate Standard Errors)



The analysis above suggests that the attenuated difference scores in the ASD group are mostly attributable to an attenuation of SCRs to $CS+_{unpaired}$ stimuli during the acquisition phase. However, the magnitude of the standard errors illustrated in Figure 4.3a, together with the marginally significant effect of experimental phase of these data in the ASD group, would also be consistent with the possibility that abnormalities in fear acquisition were present in only a subgroup of ASD participants. In order to explore this possibility further, we carried out a second analysis and computed indices of fear acquisition and discrimination for each participant.

For the computation of these indices we considered the standard error of the mean habituation trials (hereafter SE_h) to reflect the error of measurement of SCRs of each individual, since these responses reflect galvanic activity during a relatively relaxed period

[16] Note: For the analysis of these data, responses during the 14 CS- trials during acquisition were averaged across blocks of two consecutive trials.

and are therefore uncontaminated by the aversive stimulation that took place during acquisition[17]. For the index of acquisition, we subtracted the average SCRs to $CS+_{unpaired}$ trials during habituation from those to $CS+_{unpaired}$ trials during acquisition and divided this difference by SE_h. The resulting score thus represents the change in SCR to $CS+_{unpaired}$ trials between habituation and acquisition in units of the standard error of measurement, which has the advantage of removing inter-individual differences in baseline SCR variance from the data. For the index of discrimination, we subtracted the average SCRs to CS- trials during acquisition from the average SCRs to $CS+_{unpaired}$ trials during acquisition, again dividing the result by SE_h. This index thus provides the magnitude by which responses to $CS+_{unpaired}$ trials during acquisition exceeded responses to CS- trials during acquisition. The data for these indices, together with the values for SE_h are set out in Table 4.1. Based on the t-criterion one can consider an individual to have acquired fear if the acquisition index falls above 2. According to this criterion, 7 ASD and 9 typical participants exhibited reliable fear acquisition to the CS+ stimulus, with 1 additional ASD participant falling just short of the criterion. Closer inspection of these data furthermore suggest that with the exception of two typical participants who exhibited extremely high acquisition indices, the distribution of acquisition scores is relatively similar for the two groups. In fact, neither the group averages ($Z = 1.06$, $p = .291$), nor the proportions of participants reaching the criterion value of 2 ($X^2 = .58$, $df = 1$, $p = .352$) are statistically significant. Thus, on the basis of the acquisition indices, and in line with the findings by Bernier and colleagues (2005), we obtained no reliable evidence to suggest that ASD participants differed from typical participants in terms of generally acquiring autonomic fear responses to a previously neutral stimulus.

[17] Note: In fact there was no significant difference in the standard error of measurement between the habituation and acquisition trials, indicating that the presentation of aversive stimuli did not seem to increase the error variance in measurements of SCR activity.

Table 4.1

Indices of acquisition and discrimination for each participant in the ASD and Comparison Group. For clarity the data within each group have been arranged in descending magnitude of the acquisition index.

	ASD Group			Comparison Group		
Participant	SE_h	Acquisition index	Discrimination index	SE_h	Acquisition index	Discrimination index
1[c]	0.055	7.462[a]	5.150[b]	0.025	43.965[a]	31.686[b]
2	0.165	6.889[a]	1.779	0.126	11.468[a]	4.656[b]
3	0.062	5.761[a]	3.777[b]	0.082	7.288[a]	7.341[b]
4	0.070	4.745[a]	-0.619	0.073	5.559[a]	3.764[b]
5[c]	0.043	3.506[a]	2.550[b]	0.083	4.443[a]	4.893[b]
6	0.105	2.581[a]	1.558	0.083	4.097[a]	3.861[b]
7	0.112	2.416[a]	3.060[b]	0.125	3.071[a]	3.350[b]
8	0.044	1.970	0.991	0.174	3.029[a]	2.404[b]
9[c]	0.096	1.019	0.633	0.132	2.140[a]	2.012[b]
10	0.083	-0.644	0.288	0.089	1.080	-0.085
11	0.103	-1.891	1.217	0.119	1.022	3.170[b]
12[c]	0.035	-2.160	-1.756	0.100	0.237	2.608[b]
13	0.098	-2.923	0.011	0.059	-0.279	1.821
14[c]	0.042	-7.822	-1.218	0.103	-2.313	-0.114
Median	**0.076**	**2.193**	**1.104**	**0.095**	**3.050**	**3.260**

[a] Acquisition index scores that indicate reliable fear acquisition

[b] Discrimination index scores that indicate reliable differential fear responses

[c] ASD participants who did not recall all aspects of the experimental contingencies

In contrast to this relatively typical level of fear acquisition, the data in table 4.1 indicate that ASD participants compared to typical participants did not seem to acquire fear responses discriminately to CS+ and CS- stimuli during the acquisition phase. Compared to 11 of the typical participants who exhibited discrimination scores of at least 2, only 4 participants in the ASD group reached this criterion. This difference is statistically reliable both at the group level ($Z = 2.39$, $p < .05$) and in terms of the difference in the proportion of participants within each group who reached the criterion value of 2 ($X^2 = 7.04$, $df = 1$, $p < .05$). It is important to note that, since our indices of acquisition and discrimination are normalized against the standard error of the 12 habituation trials, it is possible that our analyses of these indices are confounded by group differences in baseline variability of SCRs (i.e. SE_h). As the data set out in Table 1 indicate, however, SE_h was similar for the two groups ($t = 1.35$; $df = 26$; *ns*)[18].

Several other aspects of these data merit further comment. First, it may seem paradoxical that two typical participants, who did not reach the criterion value of 2 for the acquisition index, reached this criterion for the index of discrimination. Closer inspection of the data for these individuals revealed that they had not fully habituated during the 12 habituation trials. During the acquisition phase, however these individuals seemed to learn that the CS- colours were 'safe' as indicated by a decrease in SCRs during these trials over the course of acquisition. In contrast CS+ trials continued to elicit relatively high responses. Thus although SCRs for these individuals during CS+ trials did not increase during acquisition, the fact that their responses to CS- trials decreased indicates that they successfully learned the differential significance of CS- and CS+ stimuli. Although relatively few studies report intersubject variability of fear acquisition in paradigms such as the one used here, our observation that 78% of our comparison participants reliably acquired fear closely resembles the 80% reported by Phelps, Delgado, Nearing and LeDoux (2004). A related issue concerns our observation that 3 ASD and 1 typical participant exhibited reliable acquisition scores in the wrong direction. On the basis of these data one may question the

[18] Note: It is also worth noting that although the indices of acquisition and discrimination are highly correlated, even when the typical individual with indices greater than 30 is excluded (ASD group: $r(12) = .723$, $p < .01$; Comparison group: $r(11) = .762$, $p < .01$), an analysis of covariance on the discrimination index with the acquisition index as the covariate still reveals a main effect of group ($F(1,24) = 5.02$; $p < .05$). This furthermore confirms a relatively specific impairment in discriminate fear acquisition in our ASD group.

validity of the acquisition index as a measure of fear acquisition. However, these negative acquisition indices simply reflect that rather than acquiring fear to the CS+ stimulus, the individuals continued to habituate to this stimulus throughout the acquisition phase. In other words, these individuals simply failed to acquire fear rather than acquiring fear in the wrong direction, which would be indicated by a reliable negative discrimination index (i.e. < -2) that was not observe for any individual. Finally it is worth pointing out that the ASD individuals who did not receive maximum scores on the questions probing their declarative knowledge about the experimental contingencies (highlighted in bold font in Table 4.1) are not clearly identifiable in terms of their conditioning responses. Thus it seems unlikely that the attenuated level of differential autonomic conditioning in the ASD group is due to some of these individuals failing to correctly recall the experimental contingencies.

Discussion

In the current experiment we examined differentially conditioned autonomic fear responses in a sample of participants with a diagnosis of ASD and matched typical comparison participants in order to gain further insights into emotional processing difficulties and the functional integrity of the amygdala in ASD. On the basis of the relevant literature we hypothesised 1) that ASD participants would exhibit typical levels of autonomic responses to aversive stimulation 2) that they would exhibit evidence of acquiring autonomic fear responses to a previously neutral stimulus and 3) that they would show an attenuated level of discriminate fear responses to conditioned and non-conditioned stimuli.

Our results were largely in line with our predictions. Groups were equivalent in terms of their baseline galvanic activity and their autonomic responses to an unconditioned aversive noise. An analysis of differential fear responses revealed that although individuals with ASD exhibited attenuated fear responses in comparison to typical participants, they did show a residual level of differentially acquired fear. In an attempt to identify the source of this atypical pattern of differential fear acquisition we carried out additional analyses, which indicated that although individuals with ASD were not impaired in acquiring fear *per se,* their learned fear responses to the conditioned stimulus did not differ reliably from autonomic responses to non-

conditioned stimuli. It is important to note that this pattern of findings was not due to individuals with ASD acquiring fear to both the conditioned and non-conditioned stimuli but rather the result of significantly attenuated fear responses to the former.

At first glance, our observation of impaired differential fear acquisition in ASD may seem at odds with the intact acquisition of potentiated startle responses reported by Bernier et al., (2005). Particularly on the basis of our separate analyses of SCRs to CS+ and CS- stimuli, one may argue that in the current study ASD is characterised by general impairments in acquiring fear. Although the current data do not allow us to refute this possibility conclusively, there are several reasons why we argue that individuals with ASD are characterised by impairments in fear discrimination rather than fear acquisition. First, our group analyses revealed a residual level of fear acquisition in ASD, which together with the findings by Bernier et al. (2005) suggest that a gross impairment in the processes necessary for fear acquisition are not likely to be present in this disorder. Second, an assessment of individual data indicated that an equivalent number of participants in both groups reliably acquired fear to the conditioned stimulus. Most importantly, however, we feel that the attenuated level of fear acquisition to CS+ in our ASD group needs to be interpreted within the context of the differential fear conditioning paradigm employed here. Such paradigms differ from simple conditioning paradigms such as the one employed by Bernier and colleagues (Bernier et al., 2005) in that the presentation of the conditioned stimulus is mixed with other neutral stimuli (i.e. CS-). In addition, unlike Bernier and colleagues (2005) who employed a 100% reinforcement schedule we utilized a 50% reinforcement schedule such that the CS+ stimulus was paired with the UCS on only half of its occurrences during the acquisition phase. Both of these factors make the CS+ stimulus a less reliable cue for the UCS and although additional studies will be needed to clarify what aspects of the experimental contingencies contribute to the atypical pattern of fear acquisition in ASD, we argue that the additional complexity of differential conditioning paradigms underlies the impairment in fear acquisition in ASD.

As we have noted in our introduction, the additional complexity of differential fear conditioning paradigms requires cortical modulation of the sub-cortical amygdala system that

mediates fear responses (Jarrell, et al., 1987; Morris et al., 1997). Our results thus lend support to the notion that atypical amygdala function may play a central role in the neuropathology characterising the disorder (e.g. Bachevallier, 1994; Bachevallier, 2000; Baron-Cohen et al., 1999; Baron-Cohen et al., 2000; Fotheringham, 1991; Howard, et al., 2000; Sweeten, et al., 2002). However, rather than a basic amygdala abnormality, our findings suggest that atypical amygdala function may arise from poor connectivity between this structure and functionally associated cortical areas. As noted in our introduction, this conclusion is in line with increasing evidence suggesting that ASD may be characterised by an underconnectivity of disparate brain regions (e.g. Belmonte et al., 2004; Brock et al., 2002; Rippon et al., 2007). The only direct evidence for this suggestion to date stems from functional imaging studies involving tasks assessing sentence comprehension (Just et al., 2004), executive function (Just et al., 2007), working memory (Koshino, Carpenter, Minshew, Cherkassky, Keller & Just, 2005) and mental state attribution (Castelli et al., 2002), all of which concluded that intra-cortical connectivity is atypical in ASD. Although Ben Shalom (2000) has suggested that poor connectivity between the amygdala and cortical areas may constitute a source for the emotional processing atypicalities in ASD, to the best of our knowledge, our study constitutes the first behavioural evidence to directly support this view. In this context it was unfortunate that we were unable to assess the extinction phase of our protocol. Since fear extinction has also been shown to rely on interactions between the cortex (particularly the medial prefrontal cortex) and the amygdala (Morgan, Romanski & LeDoux, 1993; Morgan, Schulkin & LeDoux, 2003; Phelps, et al., 2004; Quirk, Russo, Barron & Lebron, 2000), we would predict atypical extinction learning in ASD. However, since the majority of ASD participants exhibited marked abnormalities in acquiring differential fear responses in the current paradigm, it would be impossible to interpret results from the extinction phase meaningfully. More specifically, any atypicality of fear extinction could either reflect atypical extinction processes or be a side-effect of the atypical pattern of fear acquisition. Equally, equivalent fear extinction between groups would not necessarily indicate typical extinction processes in ASD since the possibility remains that atypicalities would arise if both groups acquired fear to similar extents. Future studies may be able to assess fear extinction more closely by employing simple conditioning paradigms, which as the study by

Bernier and colleagues has demonstrated, lead to relatively typical patterns of fear acquisition in this population (Bernier et al., 2005).

Regardless of the neurological basis of the attenuated differential fear acquisition in ASD, our finding that such atypicalities exist at least behaviourally bears some important implications for our conceptualisation regarding the role of atypical emotional processes in the clinical manifestations of ASD. Since the acquisition of fear is amongst the most basic mechanisms by which an individual, and indeed any organism, learns about the emotional significance of sensory stimuli, our findings provide strong support for Hobson's (1989) suggestion that individuals with ASD are characterised by difficulties in understanding the hedonic value of their sensory-motor environment. Although future research will be needed in order to determine when such abnormalities emerge in ASD, it seems likely that this aspect of emotional development would play an important role in the aberrant development of affective behaviours in this condition. Thus, whilst difficulties in processing faces and theory of mind understanding may further contribute to the socio-emotional atypicalities manifest in ASD (e.g. Baron-Cohen et al., 2000; Schultz, 2005), it seems no longer plausible to suggest that atypicalities in these relatively complex socio-cognitive capacities are solely responsible for the clinically defining feature of this condition.

Our findings also bear some important practical implications for the design and implementation of behavioural intervention programmes. Several programmes that are currently in use, especially those based on the findings by Lovaas (1987), draw on operant conditioning principles to aid children in their learning. Similar to classical conditioning, operant conditioning mediates learning by means of association, although in this case the relevant associations are between an individuals own behaviour and an emotionally significant consequence (i.e. 'reward' or 'punishment') rather than between a neutral and an emotional stimulus. Another parallel between classical and operant conditioning, is that both types of learning are mediated primarily by the amygdala (see Aggleton, 2000 for detailed reviews). Given our findings, the question thus arises whether operant conditioning provides an effective way of mediating learning in ASD. Based on our findings we fear that the answer to this question might be no. In fact, one of the most common problems therapists working on

such programmes report, is that children with ASD often struggle to either generalize their learned responses to relevant problems outside of the immediate context, or discriminate their responses adequately to different but related stimuli (e.g. learning the names of different animals or the letters of the alphabet). Although such reports are merely anecdotal, they underline the importance for further investigations in this area.

Summary

Acquiring fear responses to a previously neutral stimulus through a process of association is one of the most basic mechanisms by which an organism learns about the emotional significance of environmental stimuli. The finding that this learning mechanism is compromised in ASD, together with the observations of the previous two chapters, lends strong support to the suggestion that abnormalities in emotional processes extend to domains outside the social sphere. The evidence is also consistent with the idea that abnormalities of amygdala function play an important role in the neuropathology underlying ASD. The amygdala plays a necessary role in the associative learning process that is responsible for fear acquisition and the pattern of abnormality observed in ASD in the current and a previous study (Bernier et al., 2005), suggests that a functionally preserved sub-cortical amygdala system may be abnormally modulated by cortical areas in this condition. But how do these abnormalities relate to the socio-emotional disturbances of the disorder that were outlined at the beginning of this thesis? And how does the knowledge of such abnormalities influence our conceptualisation of the developmental trajectory of ASD? These and other questions will be addressed in the final chapter of this thesis, which will attempt to integrate the present findings with those of past research in order to formulate a neurodevelopmental framework for ASD.

CHAPTER 5: GENERAL DISCUSSION

The starting point for this thesis was the observation that there is currently insufficient evidence to distinguish between competing developmental accounts of the emotional abnormalities characterising autistic social behaviour. Although the relevant evidence consistently shows that individuals from across the autism spectrum are compromised in multiple aspects of reciprocal emotional communication (see Table 1.1) and despite a growing consensus that abnormalities in amygdala functioning are most likely involved in the neural underpinnings of this facet of the disorder (e.g. Bachevalier & Loveland, 2006; Baron-Cohen et al. 1999; Schultz, 2005), scientists continue to debate whether abnormalities in emotional development constitute a primary or secondary feature of the ASD phenotype. According to authors such as Baron-Cohen (e.g. Baron-Cohen, 1995; 2005, see also Frith, 2001; Frith, 2003), the emotional disturbances characterising ASD are a developmental sequel of earlier emerging abnormalities in a 'Mindreading' system that is responsible for facilitating an understanding of other people's behaviours in terms of non-observable mental states such as desires, beliefs but also emotions. According to this view, a child needs to be able to understand behaviour in terms of mental states before she can make sense of the emotional nuances of reciprocal social behaviour and since a substantial amount of evidence indicates that individuals with ASD exhibit difficulties in such mental state understanding, the emotional disturbances associated with the disorder are viewed as a consequence rather than a cause of the developmental trajectory of the disorder. Hobson (1993, 2002) objects to this account and instead argues that much of an infant's cognitive and socio-emotional development is anchored in affectively patterned interpersonal contact early in life. According to this view, early emotional interchanges provide a mutual experience for an infant and her caretaker that allow the infant to come to understand that other people have similar propensities to orient psychologically toward the world as she herself does. In relation to ASD, therefore, Hobson (2002) suggests that abnormalities in affectively patterned interpersonal contact are the cause rather than the consequence of the difficulties individuals with ASD experience in terms of understanding the particular characteristics of the mind.

In reviewing the evidence relevant to this debate, it became apparent that the competing theories can accommodate much of the existing literature with equal plausibility. I suggested that we can only get around this theoretical impasse by closely examining the concept of 'emotions'. A consideration of this topic revealed that emotional processes and behaviours are not only relevant within the context of social interchanges but also in relation to our non-social lives. Such processes shape the way in which we remember objects or events, for instance, and they alter our perceptions of the world regardless of whether or not other people are present. By considering how the concept of emotion is currently operationalised, I showed that a distinction between physiological and cognitive response mechanisms is crucial in order to understand the diverse phenomenology of emotional behaviours and experiences. I also pointed out that an extensive body of neuroscientific evidence implicates the amygdala as a mediator of physiological aspects of emotions. Following these observations I returned to the field of ASD and noted that relatively few studies have to date attempted to assess the physiological reactivity to environmental stimuli of individuals with this disorder. Despite the paucity of research in this domain, I argued that the patterning of observations in the relevant studies to date indicates that physiological response mechanisms do not always seem to operate typically in ASD. More specifically, I suggested that physiological responses in this disorder do not seem to be typically modulated by different stimulus parameters and that such responses do not always appear to modulate behavioural responses normally. This pattern led me to formulate my principal hypothesis, namely that ASD may be characterised by abnormalities in the interaction between physiological and cognitive aspects of emotional experiences. Based on relevant neuroscientific evidence, I also suggested that the neural substrate of this abnormality may be a functional impairment in the connectivity between a relatively basic amygdala system that is responsible for modulating autonomic activity, and brain areas that are involved in various aspects of cognition. In the following section I will briefly evaluate these ideas in the context of the five experiments presented in Chapters 2-4.

Critical evaluation of the experimental findings

The cognitive level: ASD and the interaction between arousal and cognition

Chapter 2 presented three experiments designed to determine whether memory processes in ASD are typically modulated by the degree to which to-be-remembered words elicit physiological arousal. Experiment 1 drew on a paradigm developed by Kensinger and Corkin (2004) who showed that in typical individuals the influence of arousal on memory can be dissociated from other factors known to influence memory by asking participants to study a set of words during either a full attention or a divided attention condition. Dividing participants' attention at study usually interferes with encoding processes and so results in a diminution of performance on a subsequent memory task. Kensinger and Corkin (2004), however, showed that recognition memory for arousing words is relatively immune to the effects of dividing attention, indicating that arousal modulates memory through relatively automatic processes that are qualitatively different from the more generic memory processes that normally underlie the encoding of information. Unfortunately the replication of Kensinger and Corkin's (2004) paradigm in Experiment 1 failed to confirm this arousal specific effect on memory and so provided a poor test for the hypothesis that arousal modulates memory atypically in ASD. Nevertheless, this experiment replicated an earlier study by South et al. (2008) in demonstrating that individuals with ASD, like typical individuals, exhibit enhanced recognition memory for emotionally significant compared to neutral words. In a sense this finding may be regarded as evidence against the hypothesis that arousal abnormally modulates memory in ASD since the failure to demonstrate arousal-specific effects of dividing attention on memory, does not necessarily indicate that arousal did not play a role in modulating performance on the recognition task. Thus, arousal may have influenced memory in ASD and typical participants similarly. Since arousal was not objectively measured in Experiment 1 it was impossible to address this alternative and so it remained possible that, at least over short periods of time, memory processes in ASD might be typically modulated by arousal.

Experiment 2 turned out to be a much more sensitive test of the specific effects of arousal on memory. In this experiment participants incidentally encoded emotionally charged and neutral words by rating these on a 4-point arousal scale. Skin conductance responses (SCR) were also collected during this encoding phase in order to ascertain that arousing words actually elicited higher levels of physiological arousal than non-arousing words. Following the encoding stage, free recall was assessed at three points in time – immediately after the words had been rated, again after 1 hour and once more after at least 1 day. For typical participants the results of these recall tests replicated and extended previous work (e.g. LaBar & Phelps, 1998; Sharot & Phelps, 2004) in showing that memory for arousing words decreased significantly less over time than memory for non-arousing words, regardless of whether the latter were semantically interrelated or not. For the ASD group, by contrast, memory for arousing words was not immune to the effects of forgetting. This group difference could not be accounted for by differences in the degree to which ASD and typical participants experienced the emotionally charged words as arousing since both groups provided higher ratings of arousal for the emotionally charged compared to the neutral words and both groups also exhibited typically enhanced SCR to these words. Most importantly, the arousal specific attenuation of forgetting in the typical group and the lack thereof in the ASD group was observed regardless of whether memory was assessed as a function of a conceptual classification of words or as a function of each participant's SCR. Thus, whilst arousal modulated memory consolidation processes in qualitatively distinct ways in typical participants, this did not appear to be the case for individuals with ASD, suggesting that the processes responsible for this arousal specific modulation of memory are dysfunctional in this group.

Although Experiment 2 lent support to the idea that arousal abnormally modulates memory atypically in ASD, it also suggested that not all aspects of memory may be affected by this abnormality. More specifically, arousal was found to facilitate memory on the immediate recall test to a similar extent in both groups of participants, a finding that emerged particularly in an analysis of memory based on participants' actual physiological responses. The difference between the groups, therefore, only emerged when free recall was assessed after a delay. Thus, whilst the findings provided further evidence for the possibility that over

short periods of time arousal might modulate memory relatively typically in ASD, the observations also showed that the consolidation of these memories into Long-Term storage may be compromised.

On the basis of the findings from Experiment 2, I suggested that individuals with ASD may never acquire the same kind of qualitatively distinct representations of emotional words as typical participants. Experiments 3 and 4 provided some support for this suggestion. In Experiment 3 participants were required to study a list of words that included orthographic neighbours of several emotionally charged and neutral words (i.e. Target Lures) that were subsequently included in a recognition memory test. Typical participants falsely identified the neutral Target Lures as having been on the study list relatively frequently but they were very unlikely to experience such illusory memories of the emotionally charged Target Lures. Individuals with ASD, on the other hand, falsely endorsed the neutral and emotionally charged Target Lures as having been studied with approximately equal frequency. In other words, the distinctive nature of emotional words did not prevent individuals with ASD from falsely recognising them. In Experiment 4, participants were required to identify two target words that were embedded amongst distracter words in rapid serial visual presentation (RSVP). Identification of the second target (T2) in such paradigms is very difficult because the rapid presentation of words strains attentional resources. When T2s are emotionally charged, however, typical participants identify them more readily than when they are neutral and the performance of the comparison group in Experiment 4 confirmed this phenomenon. In addition, Experiment 4 showed that the attenuation of the Attentional Blink is unlikely the result of the semantic properties of emotionally charged words alone since male first name T2s were not identified more easily than non-distinct neutral T2s. Unlike typical participants, individuals with ASD in this experiment did not exhibit enhanced identification of emotionally charged compared to neutral or male first name T2s. Again, therefore, the special characteristics of emotional words did not seem to impact on how individuals with ASD responded to them.

Experiments 1 – 4 suggested that the learning mechanisms responsible for the acquisition of hedonically distinct representations of words may be operating abnormally in

ASD, leading such individuals to process emotionally charged words as if they were neutral. Since these experiments were restricted to the verbal domain, however, it remained possible that the findings merely reflected more generic difficulties with the processing of language. This possibility gains further weight through the finding in Experiment 4 where the emotional attenuation of the Attentional Blink in the ASD group was correlated with participants' verbal IQ. As noted in the introduction (pp. 23-24), such associations have also been noted in the domain of emotion perception (e.g. Ozonoff et al., 1990). Thus, despite the fact that groups of participants were matched on verbal ability in the present experiments, the possibility remains that individuals with ASD performed atypically because language development does not follow a typical trajectory in this population. In fact, since language is acquired within the broader context of reciprocal social interactions, one may even argue that an inability to process the emotional quality of language is a consequence of early emerging abnormalities in socio-cognitive processes such as mentalising. Obviously this alternative would bring us right back to the debate that stimulated the experimental work of this thesis in the first place and so it was important to turn to a rather different experimental paradigm in Experiment 5 – fear conditioning.

Fear conditioning represents one of the most basic learning mechanisms by which organisms acquire hedonic representations of environmental stimuli, and learning in such paradigms is mediated by a process of association that relies on neither language nor on any form of social cognition. Participants in Experiment 5 were presented with a random sequence of colours on a computer monitor whilst their skin conductance responses were recorded. At a certain point one of these colours was occasionally paired with an aversive startling noise. Aversive stimuli such as these, typically trigger innately specified defence reactions, which in humans include a rather marked increase in SCRs. Both typical and ASD participants in Experiment 5 exhibited such defence reactions, suggesting that the noise was similarly aversive for both groups. The critical outcome measure in fear conditioning paradigms, however, is whether participants start to exhibit increases in SCR to the colour that is predictive of the noise even when this noise is not presented. In this respect ASD participants were characterised by marked abnormalities. More specifically, although like typical individuals they exhibited increased SCRs to the critical colour after it had been paired

with the aversive noise, these acquired fear responses were not sufficiently marked to differentiate the critical colour from other colours that had never been paired with the aversive noise. In other words, the autonomic responses of individuals with ASD did not distinguish between the hedonic properties of the different colours, suggesting that the associative mechanisms by which a previously neutral stimulus acquires emotional meaning operate differently in ASD. In addition, a significant proportion of individuals with ASD failed to retain an awareness of the contingency between the critical colour and the loud noise. That is, after the experiment, several ASD participants experienced difficulties recalling which of the colours had been paired with the startling noise. This last finding provides a parallel between the memory experiments of Chapter 2 by showing that arousal atypically facilitates the formation of hedonically distinct memory representations in this group. All in all, the findings from Experiment 5 make it seem unlikely that the observations in Experiments 1 – 4 simply reflected language difficulties in ASD.

Experiment 5 not only made it unlikely that Experiments 1 – 4 simply reflected atypicalities in language processing in ASD, but it also helped to clarify an apparent paradox raised by the findings in Experiment 2. If it is true that individuals with ASD are somehow compromised in learning about the emotional significance of environmental stimuli and as a result accumulate atypical representations of these stimuli in long-term memory, why would they exhibit typical autonomic responses to these stimuli and perceive them as emotionally significant? The answer, I believe, lies in the pattern of abnormality observed in Experiment 5. The results of this experiment, together with the observations of an earlier investigation of fear conditioning in ASD (Bernier et al., 2005), indicated that the learning mechanisms that mediate the acquisition of hedonic representations of environmental stimuli are not entirely absent in individuals with this disorder. Similar to typical individuals, those with ASD seem to acquire fear responses to some extent but this fear acquisition is inadequately modulated by specific stimulus contingencies. This makes it possible for individuals with ASD to acquire hedonic representations of environmental stimuli, whilst forming atypical representations of, and action tendencies toward, them. In this context, it is important to remember that whilst the magnitude of SCR to words in Experiment 2 correlated with the subjective ratings of arousal of these words for typical participants, this was not the case for the ASD group. Thus, despite

quantitative similarities in these measures between the two groups of participants, qualitative differences remained.

In summary, the experimental work presented in this thesis lends some support to the hypothesis that cognitive processes in ASD are atypically modulated by stimulus-induced arousal. I hasten to add, however, that this conclusion is much too vague to be of much use, not least because the concept of cognition can refer to all sorts of processes of the mind and brain. Moreover, Experiments 1 and 2 suggested that memory processes that operate over relatively short time-spans may be modulated relatively typically by arousal in ASD and two recent studies indicate that certain classes of usually arousing stimuli, such as angry facial expressions (Ashwin et al., 2006) and snakes (South et al., 2008), may capture the attention of individuals with ASD relatively typically, which contrasts the finding of abnormalities in the emotional modulation of the Attentional Blink in ASD presented in Experiment 4. Thus not all aspects of cognition seem to be abnormally modulated by arousal in ASD and not all classes of arousing stimuli seem to be atypically processed by such individuals. Although a great deal more research is needed in this area, the evidence seems consistent enough to formulate hypotheses that might prove useful in guiding future work in this domain. More specifically:

1) The learning mechanisms responsible for the acquisition of stable (over time) emotionally significant representations of stimuli are functionally compromised in ASD.

2) As a consequence such individuals atypically respond to and act upon at least those kinds of stimuli that acquire emotional significance through a process of learning.

3) Objects and events that are emotionally meaningful because of innate predispositions may be processed typically in ASD at least in so far as that they engage evolutionarily predisposed response mechanisms that are designed to ensure an organism's survival (e.g. basic approach and avoidance behaviours).

In seeking verification (or falsification) for these claims, one of the priorities for future research should be a thorough assessment of what kinds of stimuli elicit typical physiological

arousal responses in ASD and what stimuli individuals with ASD experience as subjectively emotional. My prediction would be that such work would highlight a distinction between stimuli that are inherently emotional and those that acquire emotional meaning through a process of learning. What precisely this distinction might look like is difficult to specify but one possibility is that inherently emotional stimuli would elicit a more coherent set of physiological and subjective responses than stimuli that acquire emotional meaning through learning. In any event, it seems unlikely that a classification of stimuli in terms of their social relevance is sufficient to characterise the emotional processing difficulties in individuals with ASD.

Before I consider a possible neural framework for the above hypothesis, one more caveat of the present thesis needs to be addressed and that is the fact that all of the experiments presented above involved assessments of individuals with relatively high cognitive and intellectual abilities. This leaves open the possibility that the present findings may not be representative of the autism spectrum as a whole. Although it will certainly be important to try and extend the work of the present thesis to individuals with ASD who are characterised by global cognitive developmental delay (and non-verbal paradigms such as fear-conditioning tasks would be suitable for such an endeavour), I do not consider it very likely that such individuals would exhibit no abnormalities where individuals with typical levels of cognitive functioning do. If we adopt a spectrum view of autism, as I do, we must assume a certain degree of homogeneity in the causal mechanisms that give rise to the disorder regardless of the heterogeneity with which it ultimately manifests. In this context I will make the rather strong claim that much of the developmental trajectory of ASD is caused by abnormalities in the learning processes responsible for the acquisition of hedonically salient representations of environmental stimuli. Before I defend this claim I will briefly address the question of whether it is sustainable on the basis of what is known about the neural underpinnings of emotion.

The neural level: ASD and the functional connectivity of the amygdala

There is absolutely no doubt that the functional integrity of the amygdala is necessary for an organism to effectively deal with and respond to emotionally significant events and

objects in its environment (e.g. LeDoux, 1996; LeDoux, 2000). If one observes abnormalities in this domain, therefore, there is a very good chance that the functional integrity of the amygdala has been compromised, either because of abnormalities within this structure or its afferent and efferent projections. Given the evidence reviewed in Chapter 1 and the findings from the experiments presented in Chapters 2 – 4, it is not surprising that a consensus is emerging that abnormalities in amygdala functioning are likely to play a role in the neural basis of ASD (e.g. Bachevalier & Loveland, 2006; Baron-Cohen et al., 2000; Schultz, 2005; Sweeten et al., 2002). Schultz (2005), for instance, suggests that early emerging abnormalities in amygdala functioning in ASD disrupt an infant's natural tendency to attend to socially relevant information, thereby depriving other brain areas involved in socio-cognitive functioning (e.g. Fusiform Gyrus) of sensory input that would normally ensure their functional specialisation. Baron-Cohen and colleagues (e.g. Baron-Cohen, 1995; Baron-Cohen et al., 1999; Baron-Cohen et al., 2000) offer a similar account. According to these authors the amygdala is involved in directing an organisms attention to the eye-region of faces, and this attentional modulation is argued to play an important role in the maturation of a more widespread neurocognitive mechanism (including areas such as the Dorsolateral Prefrontal Cortex and Superior Temporal Sulcus) that is responsible for mentalizing (see also Frith, 2001; Frith & Frith, 2006). A somewhat different approach is taken by Bachevalier and Loveland (2006), who focus not only on the amygdala's involvement in socio-perceptual functioning but also on its role in modulating reciprocal social behaviour. Similar to Hobson (2002), these authors point out that ASD is not only characterised by difficulties in the perception of socio-emotional signals but also by difficulties in the regulation of behaviour in accordance with these signals. Their argument, therefore, is that the socio-emotional disturbances of ASD result from abnormalities in an orbitofrontal – amygdala circuit since the orbitofrontal cortex has been implicated in the self-regulation of behaviour (see Bachevalier & Loveland, 2006 for a review).

If one considers the diversity of theoretical starting points for the various neural accounts outlined above (see the relevant sections in Chapter 1 for further details), it is truly remarkable that all come to virtually the same conclusion, namely that it is not necessarily an abnormality in amygdala functioning *per se* that underlies the socio-emotional disturbances of

ASD but rather the abnormal functioning of a wider neural system that supports socio-emotional functioning and of which the amygdala forms a key component. This account is consistent with the broader consensus that the neuropathology of ASD is not likely to be localised to any particular structure or neural system but rather involves widespread atypicalities in neural connectivity that are the result of developmental abnormalities in brain maturation (e.g. Akshoomoff, Pierce & Courchesne, 2002; Belmonte, et al., 2004; Belmonte, Cook, Anderson, Rubenstein, Greenough, Beckel-Mitchener, et al., 2004; Brock, et al., 2002; Just, et al., 2004; et al., 2007; Koshino, et al., 2005; Rippon, et al., 2007). There is, however, one major problem with these accounts, and that is the implication that the amygdala's dysfunction in ASD is restricted to its operation within a social brain network. In fact, one may even question the more fundamental assumption of the existence of a social brain, but this is an issue for another time.

The findings presented in this thesis suggest that functional aspects of the amygdala in ASD may not only be compromised in relation to socio-emotional behaviours but also in relation to basic processes involved in mediating the acquisition of hedonically distinct representations of stimuli. Of course, this suggestion is speculative at present since none of the experiments presented in this thesis has assessed amygdala functioning directly. Nevertheless, the experimental paradigms employed here, particularly Experiments 2 and 5, are thought to be sensitive to amygdala functioning (e.g. see Cahill & McGaugh, 1998; Hamann, 2001; Phelps, 2004; LeDoux, 2000 for relevant reviews). For instance, it is well established that the influence of arousal on memory is mediated by interactions between the amygdala and the hippocampus (e.g. Cahill & McGaugh, 1998; Dolcos, LaBar & Cabeza, 2004; Kensinger & Corkin, 2004; McGaugh, 2002; Phelps, 2004). Although these interactions involve complex neural and hormonal processes (e.g. Abercombie, Kalin, Thurow, Rosenkranz & Davidson, 2003; Buchanan & Lovallo, 2001) that operate in areas extending beyond these limbic structures (e.g. Maddock, 1999; Maddock, et al., 2003; Medford, Phillips, Brierley, Brammer, Bullmore & David, 2005) few would suggest that amygdalo-hippocampal interactions involved in emotional memory formation constitute part of the operation of a social-brain circuit (although emotional memory formation certainly plays a role in the context of social cognition). The behavioural abnormalities observed in Experiment 2, therefore,

suggest that the dysfunction of the amygdala in ASD extends outside the realms of a social brain network. Further support for this suggestion stems from the fear conditioning experiment presented in Chapter 4. Differential fear acquisition is thought to rely on cortical modulation of a subcortical amygdala system (e.g. Jarrel et al., 1987; LeDoux, 2002; Morris et al., 1997) and so the observation of abnormalities in this domain in ASD is suggestive of a disruption in cortico-amygdala interactions. Again, it is difficult to see how the processes involved in fear conditioning would fall under the realms of a social brain and so the amygdala dysfunction in ASD again seems to extend outside this neural construct. It is important to note that the neural underpinnings of the behavioural abnormalities noted in Experiments 2 and 5 may not necessarily lie within the neural structures most directly implicated (i.e. amygdalo-hippocampal system; cortico-amygdala system). It is, for example, possible that incoming neural signals to these structures carry atypical information that does not permit normal processing within them. Similarly, it could be the case that these circuits operate typically in principle, but that their outputs are somehow compromised. Whatever the precise nature of the abnormality may be, one thing seems almost certain; A conceptualisation of amygdala dysfunction in ASD within the framework of a social-brain network will not suffice to capture the full extent of emotional processing abnormalities that characterises the disorder.

If the idea of a social-brain is inadequate to capture the neural underpinnings of the emotional difficulties characterising ASD, what kind of neural framework might be better suited? To answer this question, it is necessary to appreciate the complexity of the amygdala's connectivity with other areas of the brain. Although it is merely the size of an almond, the amygdala comprises more than a dozen interconnected sub-regions that receive and transmit neural signals to virtually all cortical and subcortical regions of the brain (see Aggleton, 2000 for relevant reviews). In order to understand in what respects this complex system is functionally compromised in ASD it is not very useful to start by tackling the cognitions and behaviours that mark the pinnacle of this system's evolutionary history. A more fruitful strategy would be to start by uncovering phylogenetically older properties of the system on which the more complex functions rest. In this respect, the approach taken by neuroscientists interested in the neural basis of emotions could serve as a useful heuristic model. Rather than trying to uncover the neural correlates of emotions in general, many

scientists in this field have focused their attention on much more fundamental process such as those involved in fear conditioning (e.g. LeDoux, 1995, 1996, 1998, 2002, Phelps & LeDoux 2005 for reviews). Through meticulous research of this emotional learning process, it has been shown that the amygdala controls the autonomic and humoral responses characteristic of defence behaviours through efferent pathways to brainstem and hypothalamic nuclei. The sensory information necessary for triggering these responses is delivered directly from the thalamus and indirectly from primary sensory and association cortices. The direct thalamic pathways allow the organism to respond rapidly to potential threat on the basis of crude sensory input whilst the slower cortical pathways can modulate these responses according to a more detailed analysis of the stimulus or event that first triggered them. Cortical areas, however, not only send information to the amygdala, they also receive information from this structure, both directly and indirectly through structures such as the hippocampus. This reciprocity means that the amygdala can modulate sensory representations in cortical areas in order, for instance, to heighten the organism's sensory awareness to potentially threatening stimuli or store relevant information about them. All of this we have learned from the study of a process that outdates the phylogenetic origins of humans by millennia and the knowledge gained to date stands on firm scientific ground. So what does any of this have to do with ASD?

In the previous section I suggested that the learning mechanisms responsible for the acquisition of emotionally significant representations of stimuli are functionally compromised in ASD. I also proposed that the consequence of this abnormality is that individuals with ASD atypically respond to and act upon all those stimuli that acquire their emotional significance through a process of learning. Stimuli that are inherently emotional, on the other hand, should trigger relatively typical automatic response mechanisms. If we take what is known about the amygdala's involvement in fear conditioning as a model for how this structure deals with emotionally significant stimuli in general, we could predict that subcortical amygdala pathways involved in rapidly preparing an organism to deal with an emotionally significant stimulus or event are principally spared in ASD. By contrast, the reciprocal connections between this system and areas responsible for the consolidation and storage of memories of these stimuli may be disrupted. Although at the moment these possibilities remain

speculative, they constitute testable hypotheses that can as easily be falsified as verified. Together with the fact that these speculations rest on an extensive body of neuroscientific evidence, I believe the exploration of these possibilities to be more valuable for our understanding of the neural basis of ASD than a continuation of an over-focus on the rather vague and speculative concept of a social brain.

Implications for developmental theory

Learning is a process that starts the minute we are born and lasts for as long as we are alive. Throughout this time it shapes the way in which we interact with the world and how we learn things about it. If this process is altered, a vicious cycle can ensue that might change the course of development of the organism. The experimental work of this thesis suggests that adults with ASD are characterised by abnormalities in the mechanisms responsible for the acquisition of stable emotionally salient representations of the sensory environment. I have already presented the argument that this disturbance in emotional learning may alter the way in which such individuals come to interact with and represent the emotional significance of their surroundings. I have also proposed that these disturbances may be rooted in dysfunctional interactions between a relatively basic amygdala system and areas of the brain responsible for the consolidation and storage of memories (i.e. hippocampus and relevant cortical areas). The issue I will consider in this final part of my discussion, is what the implications for developmental theories of ASD would be, if these arguments turn out to be justified.

To start with, it is important to note that none of the developmental theories of ASD discussed so far offer suitable explanations for why individuals with this disorder should be compromised in the acquisition of emotionally distinct representations of sensory stimuli. Authors such as Baron-Cohen and Frith (e.g. Baron-Cohen, 1995, 2005; Frith, 2001, 2003), for instance, argue that the core deficit in ASD is an abnormality in the development of a neurocognitive mechanism responsible for mentalizing. Unless, one considers the processes involved in emotional learning to constitute part of this mentalizing system or assumes that

such learning processes can be altered as a consequence of abnormalities within it, it is difficult to imagine how an abnormal mentalizing system could give rise to abnormalities in processes that are as basic as those involved in fear conditioning. It is equally difficult to imagine how the acquisition of emotionally salient representations of environmental stimuli could be compromised by a disturbance in behavioural self regulation (i.e. Bachevalier & Loveland, 2006) or how a developmental alteration of affectively patterned interpersonal relations (e.g. Hobson, 2002) could compromise the processes involved in emotional learning. One might wonder why it should be problematic for current theories if they can not accommodate findings such as those presented in this thesis. After all, the principal focus of the theories discussed thus far is to try to explain the clinically defining reciprocal socio-emotional impairments characterising ASD and not the characteristics of the disorder that fall outside this domain. As noted in the previous section, however, there is a growing consensus amongst theorists that the neural underpinnings of the reciprocal socio-emotional disturbances characterising ASD involve abnormalities in the functioning of the amygdala (Bachevalier & Loveland, 2006; Baron-Cohen, et al., 2000; Schultz, 2005; Hobson, 1989) and since this structure is known to be involved in the mediation of emotional learning processes, the social and non-social emotional processing difficulties in ASD seem to arise from one and the same abnormally functioning neural system. Given this common neural denominator, it is unlikely that the developmental trajectory of socio-emotional functioning in ASD is independent from the development of non-social emotional processes. Thus developmental theory must either specify reasons for why social and non-social emotional processes should be treated separately or explain how abnormalities in one of these domains relate to the atypical functioning in the other. To date, no theory seems able to meet this challenge.

Since it seems unlikely that abnormalities in emotional learning processes constitute the developmental sequel of socio-emotional dysfunction in ASD, what about the reverse? Could it be that abnormalities in emotional learning mechanisms disrupt the development of socio-emotional functioning to such a degree as to give rise to the developmental trajectory of ASD? I believe the answer to be yes. Hobson (2002) has already offered a convincing account of how early emerging abnormalities in emotional development can result in severe perturbations of reciprocal social development. Hobson, like all other authors concerned with

trying to explain the socio-emotional difficulties characterising ASD, however, has framed his account exclusively in terms of emotional processes that are anchored in reciprocal social interactions. More specifically, he argues that the developmental trajectory of ASD is the result of abnormalities in the innate predisposition to affectively relate to other people. Similar to other developmental theories of ASD, this account has difficulty explaining why individuals with ASD should be compromised in acquiring emotional representations of environmental stimuli through non-social learning processes. Unlike alternative theories, however, the suggestions by Hobson (2002) may be brought in line with the evidence presented in the present thesis. More specifically, one could suggest that ASD is the result of a disturbance not only in the predispositions to affectively relate to other people but in the processes necessary to relate to, and learn about, the hedonic properties of environmental stimuli and events in general. In order to demonstrate what such a reformulation of Hobson's theory might add to our understanding of the developmental trajectory of ASD, the remainder of this discussion will consider what role emotional learning processes might play during early infant development. As I hope to demonstrate, such a consideration reveals that a single disturbance early in life may not only account for the socio-emotional disturbances of ASD but also for the non-social characteristics of the disorder's phenotype.

The role of emotional learning in the development of self-awareness and ASD.

The arguments I will formulate in this final section are principally anchored in neurobiological accounts of the origins of self-awareness and consciousness (particularly Damasio, 1994, 1999, 2003 and LeDoux, 2002), which suggest that the processes involved in emotional learning play an important role in the development of an *evaluative awareness* of our surroundings (this terminology will be explained shortly). These accounts are generally compatible with psychologically oriented theories of consciousness and self-awareness (e.g. Neisser, 1988; Tronick, 2005; Zelazo, 1996) and I will suggest that they provide a neurologically plausible foundation for the notion that an understanding of other people grows out of richly emotional interactions between an infant and her environment (e.g. Hobson, 2002; Trevarthen, 2005; Tronick, 2005). Although the following discussion will digress

somewhat from the principal focus of the experimental work presented in this thesis, I hope to demonstrate that a broader consideration of the role of emotional learning in infant development may hold the key to understanding the ontogenetic origin of autistic spectrum disorders.

The starting point for my argument is the assumption that infants are born with innately specified propensities to respond to certain properties of the world with changes in autonomic activity. The properties of the world in question are those that signal survival relevant information such as the presence of potential danger, the opportunity to obtain nutrients, or the presence of a conspecific, and the changes in autonomic activity triggered by such information serve to ready the infant for appropriate action. Infants are also endowed with a number of perceptual and cognitive capacities that permit the formation of at least primitive sensory representations of the environment. When I use the term *representation*, I am not referring to a psychological or mental abstraction of what the sensory organs perceive (or rather what they transduce), but rather a pattern of neural activity that specifies the physical properties of the world in a format that permits an infant to respond appropriately. Importantly, the infants' perceptual systems not only represent properties of the external environment in this manner, but they also represent the homeostatic and kinaesthetic state of the infant's own body. Damasio (1994, 2003) makes the important point that the ability to represent the body proper and the environment in parallel provides a means for an organism to delineate one (i.e. the body) from the other (i.e. the environment), because representations of the environment vary on average more than those of the body proper on the one hand, whilst on the other hand, changes in the representations of the body and the environment correlate (e.g. small changes in the orientation of the neck muscles can cause drastic changes in the visual representations of the environment). Soon after birth (and maybe even beforehand), therefore, human infants most probably posses the capacity to represent the 'Self' in the form of a perceptually delineated entity, or what Neisser (1988) might call the 'Ecological Self' (see also Zelazo's, 1996 concept of minimal consciousness).

Simply representing one's own body as delineated from the environment is far from sufficient to give rise to the kind of self-aware thought that characterises human cognition.

The key to understanding how this level of self-awareness comes about is to appreciate that the representation of one's own body in relation to the environment is not a static but a continuously dynamic process. This dynamic allows an infant to discover patterns in the relations between the physical properties of the environment as perceived and the body's own state as kinaesthetically and homeostatically represented. Importantly, neural assemblies in the brain are functionally and structurally altered as a result of experience, thus continuously altering the way in which the brain neurally represents the environment. Through this neural plasticity the infant starts to perceive properties in objects that are linked to the infants own propensities to act toward it. The infant learns that some things are harder and heavier than others because they resist different amounts of muscle tension when acted upon and the infant learns that objects vary in size and shape because they require different orientations of the limbs in order to be explored, reached or manipulated. Actively engaging in these actions is not needed in order to perceive these affordances in objects, they have become anchored in the perceptual representations of the objects themselves because of the experience driven changes in the relevant perceptual neural systems. The infant, therefore, is starting to entertain what I will call '*affordance representations*' of the environment. Affordances, however, are not the only property of objects that infants start to directly perceive as a result of experience. The infant also learns that certain objects and affordances of objects are more or less desirable than others because they influence the body's homeostatic state in a way that triggers the kind of neural responses that are inherently appetitive or aversive (see LeDoux, 2002 for further discussion). I will call the resulting representations '*evaluative representations*' and it is important to note that part of what is *evaluated* is the affordance of objects for action.

There is an important difference between the ways in which an infant accumulates affordance and evaluative representations of the environment. Affordance representations arise when an infant's perceptual representations of the stimulus world is altered because of a child's inter*action* with the object. Affordances, in other words, are anchored in the infant's own behaviour. More specifically, the motor commands that originate within the child bring about a temporally correlated change in the perceptual representations of the object in motion and the kinaesthetic representation of the infant's own body and this temporal correlation

brings about a change in the perceptual representations of the object that partially reflects the body's relation to it. Evaluative representations, by contrast, are not as directly related to action because they arise from a temporal linkage between the sensory representation of the object and the homeostatic representation of the body. Again this temporal correlation alters the sensory representations of the object in a way that reflects the body's own homeostatic state but in this instance the infant's actions (and motor programs) are not responsible for these changes. The hypothesis I put forward in relation to ASD, is that both the social and non-social characteristics of the disorder are the result of abnormalities in the temporal linkage between sensory representations of the stimulus world and homeostatic representations of the body that is necessary for an accumulation of evaluative representations of the world. Before I consider this hypothesis in more detail, however, it is useful to briefly consider how an infant becomes consciously aware of her evaluations of the world.

According to Damasio (1994, 2003) and LeDoux (2002), what makes human consciousness special is the fact that the evolution of the cerebrum, especially the prefrontal cortex, has resulted in a remarkable working-memory capacity that allows an individual to represent presently constructed affordance and evaluative representations of the environment in parallel with previously constructed representations of this kind. As this working-memory capacity matures during early infancy (see Happaney et al., 2004), a child starts to represent multiple versions of her own kinaesthetic and homeostatic relation to the stimulus world simultaneously. This creates a situation that is not dissimilar to that which gives rise to a delineated representation of the body as separate from the environment (i.e. the Ecological Self). In other words, across multiple affordance and evaluative representations, the child's kinaesthetic and homeostatic relation to objects is less variable than the perceptual properties of the objects themselves. Thus something becomes delineated, and this particular something is what Neisser (1988) might call the 'Extended Self': A relatively abstract but nevertheless neurally anchored representation of an infant's temporally stable relation to her stimulus environment.

Having formulated an account of what role the homeostatic state of the body (i.e. arousal) might play during early infant development, I will now return to the hypothesis I formulated above, namely that an infant will develop ASD if the processes involved in the accumulation of evaluative representations of the environment are disturbed. One of the non-social characteristics of the disorder that such an abnormality may be able to account for, is the presence of stereotyped and repetitive forms of behaviour (SRB). A series of recent studies by Noyce and Messer (2008) suggests that certain SRBs in ASD seem to be preceded by changes in autonomic arousal whilst other forms of SRB seem to serve an arousal modulating function. Individuals with ASD, therefore, seem to seek behavioural control over the changes in the homeostatic state of their body. This is precisely what one might expect if during infancy sensory representations of the environment were atypically integrated with changes in the homeostatic state of the body whilst action centred affordances of objects in the world were accumulated relatively typically. The infant with ASD, in other words, may inappropriately associate changes in the body's level of arousal with an object-oriented (or self-oriented) behaviour rather than with the appropriate perceptual representation of the object. There is relatively little research to date on how young infants, who are later diagnosed with ASD, interact with objects. My prediction in this context, however, would be that the physiological responses to objects in infants with a later diagnosis of ASD would be atypically modulated by the perceptual properties of the stimulus whilst physiological responses during physical interactions with the objects would be relatively typical.

The second non-social characteristic of ASD that would be explained by the present account is the heterogeneity of the autistic phenotype. As I noted in Chapter 1, part of this heterogeneity is most likely due to an underlying variability in the genetic, neurological and biochemical factors associated with the disorder. A heterogeneous symptom manifestation, however, would also be expected if the infant with ASD accumulates atypical evaluative representations of the stimulus environment. Damasio (1994) suggests that the homeostatic state of the body in relation to the object or event that triggers this state, serves as a 'somatic marker' that provides an organism with a principled way for organising experiences. Typical infants all share this principled way of learning about the significance of objects and so they

develop a shared understanding of the world and the objects within it. If in ASD this learning mechanism is disrupted because the infant's homeostatic representations of the body are temporally not adequately correlated with the appropriate sensory representations of the environment, every infant with ASD would come to organise experiences somewhat differently. They would still share a conceptualisation of the world in terms of the affordances for action of the objects within it but every individual would evaluate the objects and their associated affordances in a relatively idiosyncratic way. One child might find the sensory feedback from the touch of a sticker aversive, whilst another might seek such stimulation, and where one individual may develop a fascination for performing calendrical calculations, another may wish to use a calendar in order to keep a log of the weather conditions during the morning and afternoon of every day of the year.

A third non-social characteristic of the ASD phenotype that may be explained by assuming that the disorder is the result of atypicalities in the accumulation of evaluative representations early in life, is the observation that such individuals exhibit a somewhat atypical perception of themselves. Recall that the 'Extended Self' (Neisser, 1988) emerges out of the ability of the infant to represent multiple evaluative representations of the world in parallel. In ASD, each of these evaluative representations would be somewhat compromised because the necessary association between the object or event in question and the homeostatic state of the body would be disrupted. The ASD infant, therefore, would construct a qualitatively different representation of the self in time, a representation that is anchored more in actions and behavioural affordances than in the relation between the environment and the homeostatic state of the body. Evidence of episodic memory difficulties in ASD are consistent with this notion (e.g. see Bowler & Gaigg, 2008 & Lind & Bowler, 2008 for reviews) as is the finding that individuals with ASD describe themselves with a less varied emotional vocabulary than comparison individuals (e.g. Lee & Hobson, 1998) and that they report difficulties in introspecting on their own emotions (e.g. Silani, Bird, Brindley, Singer, Frith & Frith, 2008). Individuals with ASD have also been found to retrieve fewer specific autobiographical memories than typical individuals when cued with emotionally salient words (Goddard, Howlin, Dritschel & Patel, 2007), and dream reports of individuals with ASD are relatively void of emotional content (Daoust, Lusignan, Braun, Mottron & Godbout, 2008).

So far, I have concentrated exclusively on the non-social characteristics of the ASD phenotype and so in this last section I will briefly consider how an abnormality in the accumulation of evaluative representations of the world may also give rise to the reciprocal social atypicalities characterising ASD. I will keep this argument brief because I believe that Hobson (2002) has already offered a useful account of how an infant's atypical engagement with other people may give rise to the various reciprocal social impairments that characterise ASD (e.g. engagement in joint attention, mentalizing, acquisition of language). Rather than repeating these arguments here, I will focus on what might underlie an infant's atypical engagement with other individuals to begin with. To answer this question, it is important to appreciate that other people are the most complex 'object' in terms of the influence they exert on a developing infant's homeostatic state. Typical infants soon discover that there is something rather special about the way in which perceptual representations of another person relate to changes in the body's homeostatic state. Affordance representations of most objects are relatively static, directly anchored in the child's own behaviour, and to a certain degree independent of the evaluative representations of the objects in question. Representations of other people, however, are much more dynamic, their affordances sometimes change in anticipation of the infant's behaviours and most importantly, these changes in affordances often facilitate the maintenance of a desirable homeostatic state in the infant's body (i.e. changes in evaluative representations are associated with changes in affordance representations). One moment the other alleviates an undesirable homeostatic state by offering an object, and the next he facilitates a desirable one by maintaining a rather pleasant face-to-face interaction. Initially, the infant may not always directly associate the other person with the resulting desirable homeostatic state. The infant's perceptual system may be more concerned with the representation of the offered object than with the representation of the person that offered it. Nevertheless, the other is continuously (or almost continuously) present, and he repeatedly alters the environment in a way that facilitates a positive homeostatic state. As the typical infant starts to represent multiple evaluations of the world in parallel, she discovers patterns in the relationship between the actions of the other and the influences these actions have on the body's homeostatic state. The infant, in other words, is soon privy to information that allows her to delineate the other person as a rather

special kind of object – a kind of proto-person. I call this early form of representing other people a 'proto-person' because the infant still has much to learn about many of the non-observable characteristics of other people such as the fact that their behaviours are guided by their own representations of the world. As Hobson (2002) notes, such understanding comes through repeated interactions with others, which provide the infant with the necessary information to start to directly perceive more obscure properties of others, much in the same way as she learned to perceive her own relation to objects in the objects themselves. Whatever way an infant comes to ultimately understand other people, the important point to stress is that such understanding necessitates that the infant can makes sense of the complex dynamic properties of other people. A typical infant's representational systems achieve this because every change in the sensory representation of the other person is temporally linked to the appropriate change in the body's homeostatic state. For the developing infant with ASD, by contrast, other people are about as confusing as objects can get. Being compromised in relating representations of bodily changes in arousal with the appropriate sensory representations of the environment, the infant is confronted with a percept that continuously and unpredictably alters its influences on the body. Encounters with others may often turn out to be generally desirable (because other people are very skilled in interacting with infants), but the infant has difficulty predicting the changes in the affordances that the other person initiates (see also Loveland, 1991; Loveland, 2001) because these too are often linked to the infant's homeostatic state rather than her behaviour (as mentioned above, these changes in affordances primarily serve to facilitate the infant's homeostatic regulation). The infant with ASD, therefore, is starting to experience difficulties in regulating his behaviour in accordance with the other person (e.g. Bachevalier & Loveland, 2006), which results in the accumulation of atypical affordance representations in addition to the already atypical evaluative representations that are accumulating. Ultimately, the infant relates to other people in fundamentally different ways (e.g. Hobson, 2002), setting the course for the atypical developmental trajectory that characterises autistic social behaviour.

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