Free Recall and Forgetting of Emotionally Arousing Words in Autism Spectrum Disorder

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Abstract

Since the earliest descriptions of individuals with autism spectrum disorders (ASD) abnormalities in affective behaviours have been considered a prominent feature in their clinical manifestations. What remains unclear, however, is whether these altered emotional behaviours are a mere facet of abnormalities in socio-cognitive processes or whether they constitute a primary feature of the condition. A number of studies now indicate that emotional processing atypicalities in ASD extend to domains outside the broader context of social cognition leading us to suggest that the disorder may be characterised by basic abnormalities in how psychophysiological and cognitive emotional responses modulate one another (Gaigg & Bowler, 2007). In the current study we show that although individuals with ASD, like typical individuals, exhibit a free recall advantage for emotionally arousing and semantically related neutral as compared to unrelated neutral words, they do not show reduced forgetting rates for arousing stimuli as do typical individuals. These observations provide further support for the view that psychophysiological emotional responses do not modulate cognitive processes normally in ASD and further implicate abnormalities of amygdala connectivity (in particular with the hippocampus) in the neuropathology underlying this disorder.

Key Words: Amygdala; Connectivity; Memory; Psychophysiological modulation of Cognition.
Introduction

Autism spectrum disorders (ASD) are clinically defined by abnormalities in the domains of communication and reciprocal socio-emotional behaviour and the presence of narrow, stereotyped and repetitive patterns of interests (Wing, 1993). Although current diagnostic criteria (American Psychiatric Association, 2000; World Health Organisation, 1992) distinguish between Asperger’s disorder and Autistic Disorder on the basis of language development, which presents no clinically significant delay in the former, it is generally accepted that both conditions form part of the same underlying syndrome. The current paper adopts such a view and will make use of the term autism spectrum disorder to cover all conditions unless a distinction is necessary.

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, Sigman, Mundy & Yirmiya, 1990; Yirmiya, Sigman, Kasari & Mundy, 1992), a restricted and inflexible use of context appropriate emotional expressions (Dawson, Hill, Spencer, Galpert & Watson, 1990; Kasari, Sigman, Baumgartner & Stipek, 1993; McDonald, Rutter, Howlin et al., 1989; Sigman, Kasari, Kwon & Yirmiya 1992; Yirmiya, Kasari, Sigman & Mundy, 1989) and difficulties in the perception and recognition of emotional expressions in others (Hobson, 1986a,b; Hobson, Ouston & Lee, 1988a,b; Hobson, 1991; Weeks & Hobson, 1987; but see Castelli, 2005 for contrasting findings). Although these atypicalities are regarded as diagnostically relevant (American Psychiatric Association, 2000; World Health Organisation, 1992), their causes remain the matter of debate. Some authors maintain that disordered emotional behaviours constitute a secondary manifestation of impairments in basic social capacities such as face-processing (Schultz, 2005) or ‘Theory of Mind’ (ToM) understanding (Baron-Cohen, Ring, Bullmore, et al., 2000; Baron-Cohen, Ring, Wheelwright, et al., 1999), whilst others argue that aberrant emotional behaviours reflect primary impairments in emotional development (Hobson, 1989; Kanner, 1943; Mundy & Sigman, 1989). In part the difficulty in resolving this debate lies in the fact that most relevant research to date has centred around assessing socially relevant affective behaviours. Since the findings from such investigations can be accommodated within the competing theoretical frameworks, an
important question that remains largely unanswered is whether emotional processing abnormalities extend to domains outside the broader context of social cognition in ASD.

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, Anderson, Manning 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 are not restricted to social contexts. Studies of classically conditioned fear responses for example, indicate that individuals with ASD acquire fear to a previously neutral stimulus similarly to typical individuals when this stimulus is consistently and repeatedly paired with a noxious stimulus (Bernier, Dawson, Panagiotides & Webb, 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 basic emotional response mechanisms may function relatively
typically in ASD, they may not be normally modulated by the contingencies that determine the emotional significance of stimuli. Studies assessing the psychophysiological reactivity of individuals with ASD lend further support to this view. Such studies show that although individuals with ASD generally exhibit heightened autonomic responses to emotionally significant stimuli (e.g. Ben-Shalom, Mostofsky, Hazlett et al., 2003; Salmond, Haan, Friston, Gadian & Vargha-Khadem, 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) and the impact of such autonomic arousal on decision making processes have also been found to be atypical (Johnson, Yechiam, Murphy, Queller & Stout, 2006). 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 processing 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, Ring, Bullmore, 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, one may argue 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, Wheelwright & Baron-Cohen (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 functionally atypical in ASD is how such basic emotional responses 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, 2006; Rippon, Brock, Brown & Boucher, 2007) this pattern of findings has led several authors to suggest that the amygdala may be functionally under-connected with areas sub-serving other cognitive and perceptual processes (Ashwin, Wheelwright & Baron-Cohen, 2006; Gaigg & Bowler, 2007; McAlonan, Cheung, Cheung et al., 2005). Once again, memory paradigms provide a useful behavioural tool in this context because it is well established that interactions between the amygdala and the 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 the experimental stimuli did indeed vary on this dimension for this population. 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 tenable 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, following 1 hour and again after at least 24 hours) in order to establish forgetting rates for emotionally arousing and non-arousing stimuli. Studies employing such 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 et al., 1998) this attenuated forgetting rate is widely thought to reflect amygdala mediated modulation of hippocampally based memory consolidation processes. Therefore such a paradigm should 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 as 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 concerns 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, Matthews & Gardiner, 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 ASD 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, Rutter, Goode, et al., 1989) confirmed that all met DSM-IV (American Psychiatric Association, 2000) criteria for 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<sub>UK</sub>; The Psychological Corporation, 2000) and groups did not differ on performance IQ, full-scale IQ or age (see Table 1). 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.

**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 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 free recall. 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 experiment they would take part in 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 ratings 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 (i.e. no noticeable activity for at least 20 seconds), 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 and without time restrictions. Following approximately 1 hour which was filled with lunch or 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 were asked not to 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. We also computed the curve integrations (i.e. area under the curve) of SCR during this 5 second window but since the relevant analyses yielded equivalent results for both measures we only present the peak response data here\(^1\). Average responses to the arousing, related neutral and unrelated neutral words are set out in Table 2, 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 \((F(1,34) = 0.78, \text{ ns})\) and interaction \((F(2,33) = 2.36, \text{ ns})\) suggests that both groups exhibited similar levels of autonomic activity across the different stimuli\(^2\). 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 \((F(1,34) = 0.75, \text{ ns})\) or interaction \((F(2,33) = 0.75, \text{ ns})\). 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 \((t = 1.85, df = 17, p = .08)\). The difference between groups was also statistically reliable \((t = 2.62, df = 34, p < .05)\).

\(^1\) Details on the curve integration data are available upon request from the first author.

\(^2\) Although inspection of Table 2 suggests that arousing words elicited somewhat higher SCR in typical than ASD participants, this difference was not statistically reliable \((t = 0.99, df = 34, \text{ ns})\).
Prior to assessing the free recall data in detail we determined whether participants in the two groups completed the final free recall test within similar timeframes and whether they may have differed in the number of intrusions they committed (i.e. recalling words not originally presented). Regarding the final free recall test, all but two typical and one ASD participant returned their final free recall responses. 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. In fact, entering the delay (in days) between the second and last free recall test as a covariate in the relevant analyses did not alter the results reported below significantly. 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. With respect to the number of intrusions, these were generally relatively low, with typical individuals producing on average 0.50 (SD = 0.79) and ASD participants 1.27 (SD = 1.60) new words across all three recall trials. Again groups did not differ significantly in this respect (t = 1.85, df = 34, ns) and co-varying for the number of intrusions in the relevant analyses below did again not alter the results significantly.

Figures 1a and 1b illustrate the average proportions of arousing, related neutral and unrelated neutral words recalled by the ASD and typical 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 1hour (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 marginally so (F(4,31) = 2.28, p = .08). Post-hoc analyses indicated that the typical 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 decrease in recall of arousing words over the 24 hour+ period (t = 2.57, df = 17, p < .05) was significant.

**INSERT FIGURES 1A AND 1B**

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 findings 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 Arousing’ (16 words eliciting the lowest SCRs) on the basis of each participant’s SCRs. This re-classification of words resulted in no significant group differences in terms of the average magnitude of SCR elicited by the words in each electrophysiological category. For the ASD participants the average SCRs (measured in √μS) elicited by the High, Medium and Low arousal words respectively were 0.937 (SD = 0.482), 0.437 (SD = 0.315) and 0.129 (SD = 0.175) whilst for the typical group the corresponding values were 1.032 (SD = 0.613), 0.498 (SD = 0.403) and 0.131 (SD = 0.149). Similarly there was no between group difference in how words from the a priori conceptual groupings fell into the electrophysiologically defined categories. For the ASD group 48% of words conceptually classified as arousing were reclassified on SCR criteria as ‘High Arousing’, 32% as ‘Medium Arousing’ and 19% as ‘Low Arousing’. The corresponding percentages for the typical group were 56%, 29% and 15%. The categorised neutral and unrelated neutral words each comprised roughly 25% of the
'High Arousing', 35% of the 'Medium Arousing' and 40% of the 'Low Arousing' categories for both groups.

Figures 2a and 2b illustrate the recall data for the electrophysiological reclassification of words and again show a markedly atypical pattern of forgetting in the ASD 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 Figures 2a and 2b suggest, 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+ $F(2,33) = 18.68, p < .001$).

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 hippocampal 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. In particular 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, Luk, McGarry & Moscovitch, 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 for which we assessed memory 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 as 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 typical than the ASD 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 as 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 ASD 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. We believe the results from the second analysis to provide particularly strong support for this suggestion. First, because this analysis addresses the issue more directly by assessing memory as a function of actual physiological arousal. Second, because this analysis reveals marked group differences already
following a delay of one hour, which overcomes any concerns about the poor experimental control over the 24 hour period. Third, because the re-arrangement of the data according to the actual level of physiological arousal equated the groups more closely in terms of the magnitude of arousal they exhibited in response to the most stimulating words.

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 typical 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, Etzel, Adolphs & Tranel, 2006; Maratos, Allan & Rugg, 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 forgetting rates of emotionally arousing stimuli provide a relatively reliable behavioural marker of amygdala mediated memory modulation processes.
If one accepts our reasoning 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 this group.

As such our observations provide further behavioural evidence for the 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). Additional support for this conclusion stems from our assessment of the correlations between subjective ratings of arousal and the magnitude of SCRs, which replicated an earlier study in showing that only for typical but not ASD individuals these measures are significantly correlated (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. Lane, Reiman, Axelrod, et al., 1998; Maddock, Garrett & Buonocore, 2003; Kensinger & Schacter, 2006), are inadequately modulated by physiological responses to these stimuli (i.e. by the amygdala).

In relation to the neural interpretation of our data presented above, two aspects of our observations merit further comment. The first concerns the fact that individuals with ASD did exhibit enhanced memory for arousing words on a test of immediate free recall. This finding may indicate that at least over short time delays the amygdala contributes normally to the arousal induced enhancement of memory in ASD. On the basis of our current observations we can not rule out this possibility but it is worth reiterating that 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. That individuals with ASD were able to draw on such semantic relationships to facilitate free recall in the current study is illustrated by their superior recall of semantically related as compared to semantically unrelated neutral words. Thus the observation of enhanced memory for emotional material on an immediate test of free recall in ASD is not necessarily
inconsistent with the suggestion that the amygdala may abnormally modulate memory consolidation processes in this condition. To address this issue more closely, however, future studies need to establish whether the memories of arousing stimuli over the short term are qualitatively different from memories of non-arousing stimuli in this group, which would implicate the amygdala (see Kensinger & Corkin 2003; Kensinger & Corkin, 2004 for a divided attention paradigm that may be suitable here). The second aspect of our data worth further comment centres around the finding that individuals with ASD tended to forget non-arousing stimuli to a lesser extent than typical individuals. This observation may give rise to the suggestion that rather than amygdala mediated memory consolidation processes being impaired in ASD, the consolidation of non-arousing stimuli may be enhanced and in turn interfere with arousal induced consolidation. We would refute this alternative on the grounds that interference theories of memory rest on the assumption that the stimuli that interfere with one another do so because of competition for the same processing resources. As we have argued throughout this article, arousal induced consolidation operates in a qualitatively distinct manner that involves the modulation of hippocampal consolidation processes by the amygdala (see LeDoux, 2002 for further details). If it were indeed the case that the increased rate of forgetting of arousing material in ASD is due to interference from the consolidation of non-arousing stimuli, one would have to conclude that in this group both arousing and non-arousing stimuli were consolidated via the same processes. Such an interpretation would take us back to the conclusion that the amygdala functions abnormally in terms of modulating hippocampally based consolidation processes for arousing stimuli. Ultimately it will require further behavioural and more direct neuroscientific investigations to settle issues such as these. For now we simply hope to have generated a testable hypothesis – that the amygdala atypically modulates hippocampally mediated memory consolidation processes in ASD – that will stimulate future research.

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

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3 It is furthermore worth noting that an analysis of correlations between the rate of forgetting of arousing and non-arousing stimuli did not yield any negative relationships that would indicate that neutral words were remembered at the expense of arousing words in ASD.
demonstrating that emotionally significant stimuli are atypically remembered by individuals with ASD. As such our findings add to a growing body of evidence suggesting 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). 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 (Bowler, Gaigg & Gardiner, in press; Gaigg, Bowler & Gardiner, in press; López & Leekam, 2003). More specifically, the current findings confirm our recent observation that when semantically related words are presented in a mixed list, recall performance in individuals with and without ASD is nearly identical when words are encoded through an item-specific processing task in which individuals are asked to rate each word on pleasantness (Gaigg, Bowler & Gardiner, in press). 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 that the amygdala is functionally under-connected with other brain structures (Ashwin, et al., 2006; Gaigg & Bowler, 2007; McAlonan, et al., 2005). More specifically, our current findings lead us to suggest that in ASD the amygdala atypically modulates hippocampal consolidation processes. Furthermore, the current findings replicate our recent observations of intact free recall performance following item-specific encoding (Gaigg et al., in press), 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 processing atypicalities remain the result of atypical social capacities.

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.
difficulties characterising autistic social behaviour are the sole result of abnormal socio-cognitive development.

Note

During the process of revising this manuscript for re-submission, we came across the recent publication by South, Ozonoff, Suchy and colleagues (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.
References:


Emotional Memory in Autism Spectrum Disorder


Tables & Figures:

Table 1

*Summary of Age and IQ characteristics of the ASD and Typical Group*

<table>
<thead>
<tr>
<th>Measure</th>
<th>ASD (n = 20)</th>
<th>Typical (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Age (years)</td>
<td>32.8</td>
<td>12.4</td>
</tr>
<tr>
<td>VIQ(^a)</td>
<td>105.2</td>
<td>14.7</td>
</tr>
<tr>
<td>PIQ(^b)</td>
<td>106.4</td>
<td>17.5</td>
</tr>
<tr>
<td>FIQ(^c)</td>
<td>106.3</td>
<td>17.2</td>
</tr>
</tbody>
</table>

\(^a\) 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\(^UK\))
Table 2

Average SCR and arousal ratings for ASD and Typical participants as a function of Word Type

<table>
<thead>
<tr>
<th>Word Type</th>
<th>ASD (n = 18)</th>
<th>Typical (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>SCR (√μS)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arousing</td>
<td>0.720</td>
<td>0.347</td>
</tr>
<tr>
<td>Related Neutral</td>
<td>0.546</td>
<td>0.340</td>
</tr>
<tr>
<td>Unrelated Neutral</td>
<td>0.574</td>
<td>0.378</td>
</tr>
<tr>
<td>Arousal Rating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arousing</td>
<td>2.49</td>
<td>0.76</td>
</tr>
<tr>
<td>Related Neutral</td>
<td>1.64</td>
<td>0.64</td>
</tr>
<tr>
<td>Unrelated Neutral</td>
<td>1.51</td>
<td>0.42</td>
</tr>
</tbody>
</table>
Figure 1
Average recall performance for ASD (panel A) and Typical (panel B) participants as a function of Word Type and Time of Recall (Error Bars represent Standard Errors)

Figure 2
Average recall performance for ASD (panel A) and Typical (panel B) participants as a function of Level of Arousal and Time of Recall (Error Bars represent Standard Errors)