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The effect of glucose administration on the recollection and familiarity components of recognition memory

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Abstract

Previous research has demonstrated that glucose administration facilitates long-term memory performance. The aim of the present research was to evaluate the effect of glucose administration on different components of long-term recognition memory. Fifty-six healthy young individuals received (a) a drink containing 25 g of glucose or (b) an inert placebo drink. Recollection and familiarity components of recognition memory were measured using the 'remember-know' paradigm. The results revealed that glucose administration led to significantly increased proportion of recognition responses based on recollection, but had no effect on the proportion of recognition responses made through participants' detection of stimulus familiarity. Consequently, the data suggest that glucose administration appears to facilitate recognition memory that is accompanied by recollection of contextual details and episodic richness. The findings also suggest that memory tasks that result in high levels of hippocampal activity may be more likely to be enhanced by glucose administration than tasks that are less reliant on medial temporal lobe structures.

Keywords: Glucose; Aspartame; Recognition memory; Recollection; Familiarity; Healthy young adults

1. Introduction

The role of glucose in the modulation of cognitive processes in healthy young and aged animals and humans has been clearly demonstrated (see Messier, 2004 for a recent review). Previous studies have utilized a variety of procedures and paradigms, and benefits in cognitive performance have been found to occur in a range of cognitive tasks, including central processing speed and reaction times (Benton et al., 1994), working memory (Martin and Benton, 1999; Kennedy and Scholey, 2000; Sünram-Lea et al., 2001, 2002b) and attention (Messier et al., 1997). However, in general it appears that glucose administration and/or impairments in glucoregulatory mechanisms have a pronounced effect on declarative long-term memory performance associated with hippocampal function (e.g. Craft et al., 1994; Messier and Gagnon, 1996; Korol and Gold, 1998; Foster et al., 1998; Messier, 2004; Sünram-Lea et al., 2001, 2002a,b, 2004; Riby, 2004; Riby et al., 2006; Meikle et al., 2004, 2005) and smaller and/or less reliable effects on other aspects of cognitive functioning.

More specifically, robust glucose facilitation has been observed on memory tasks entailing intentional or conscious recollection of previous experiences, i.e. those tasks tapping explicit or declarative memory. For example, glucose administration has been shown to significantly improve delayed paragraph recall performance, but not procedural memory (Craft et al., 1994). Glucose ingestion significantly improved memory performance on explicit word recall tasks (Foster et al., 1998; Messier et al., 1999; Sünram-Lea et al., 2001; Sünram-Lea et al., 2002a,b; Meikle et al., 2004) and paired associate learning (Riby, 2004; Riby et al., 2006), whereas no facilitation of implicit memory performance was observed (Manning et al., 1997). Therefore, in healthy young people glucose seems to facilitate most reliably verbal long-term memory for complex associations. In addition, there is evidence that the glucose memory facilitation effect seems to be mediated by enhanced *retention* of information in the long-term memory store: the glucose memory facilitation effect observed in young people is

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typically maintained after controlling for both (a) participants' differential baseline blood glucose levels and (b) individual levels of immediate memory performance, and (c) retrograde glucose administration also significantly enhances memory performance (Foster et al., 1998; Sünram-Lea et al., 2001; Sünram-Lea et al., 2002a,b).

A recent influential theoretical position postulates that the 'extended hippocampal system' is primarily involved in the mediation of recall memory (that is spontaneous reproduction of material) rather than being involved in recognition memory (Aggleton and Brown, 1999). According to this framework, if the hippocampus is preferentially affected by glucose administration, significant facilitation effects on explicit memory performance should be noted on memory when this is tested by recall, but not by recognition. However, improved performance on verbal and facial recognition memory tasks under elevated blood glucose levels has previously been observed in some studies (Foster et al., 1998; Sünram-Lea et al., 2002a,b; Metzger, 2000; Metzger and Flint, 2003). This point notwithstanding, in our own laboratories facilitatory effects of glucose on recognition memory have proven to be more variable – compared to the more reliable glucose-mediated enhancement of long-term recall performance (Foster et al., 1998; Sünram-Lea et al., 2002a,b). This finding may be due to the application of simple 'yes'/'no' recognition paradigms in previous research. It has been suggested that the hippocampal-diencephalic system is not critical for efficient recognition, as recognition is considered to be composed of at least two independent processes, only one of which appears to be hippocampally dependent (Aggleton and Brown, 1999). More specifically, the proposition here is that item recognition occurs either through (i) recollection of the stimulus (a process which is hippocampally dependent) or (ii) detection of stimulus familiarity (which does not require the hippocampus) – or through some combination of these two processes. It has been further suggested that the familiarity process is mediated by the perirhinal cortex in the temporal lobes (Aggleton and Brown, 1999). These observations indicate that further research is merited in order to draw definitive conclusions about the possible fractionation of glucose enhancement effects on different long-term memory processes.

Recollection and familiarity are subjective memory experiences that refer to how we recognise a previously experienced event and/or a previously encountered individual (see Yonelinas, 2002, for a review). According to Tulving (1985), these subjective memory experiences can be based on the psychological experience of either 'remembering' or 'knowing'; by this framework, 'remembering' refers to an experience of recognition that is accompanied by recollection of contextual details, whereas 'knowing' lacks this episodic richness and is based on feelings of familiarity alone. Based on Tulving's theory, the 'Remember'-'Know' paradigm has been used to measure the different subjective experiences that can accompany recognition (for a review see Gardiner and Richardson-Klavehn, 2000). This paradigm is used widely in recognition memory testing: participants are shown a set of studied and unstudied items, and are required to decide whether each item was presented in the study phase (i.e. it should be judged to be 'old') or not (i.e. it should be judged 'new'). Following an 'old' decision, participants are then further required to make a 'remember', 'know' or 'guess' decision (the 'guess' response category is included so that the 'know' response category is not erroneously inflated by guesses; Gardiner et al., 1996).

Although there has been debate over the degree to which 'remember' and 'know' responses are process pure (Donaldson, 1996), there is ample evidence that 'remember' and 'know' responses can be dissociated experimentally (see Gardiner and Richardson-Klavehn, 2000). There is also evidence from psychopharmacological studies that 'remember' and 'know' responses can be dissociated by substances such as lorazepam (Curran et al., 1993) and alcohol (Curran and Hildebrandt, 1999). In addition, studies have shown that 'remember' and 'know' responses can be dissociated in terms of brain activity, both temporally and spatially (Henson et al., 1999; Eldridge et al., 2000; Mangels et al., 2001; Rugg et al., 1998; Düzel et al., 1997). Of particular relevance to the present study is previous evidence suggesting that 'remember' but not 'know' responses require hippocampal involvement (Henson et al., 1999; Eldridge et al., 2000). These findings demonstrate that recognition processes can be dissociated. Additionally, these findings buttress the notion that the 'remember-know' procedure will allow a more precise investigation of the effects of glucose administration on long-term recognition memory, thereby clarifying the somewhat inconsistent results observed when testing recognition memory in our previous studies.

The present experiment attempted to evaluate further the relationship between (i) glucose availability and (ii) different components of verbal long-term recognition memory. Our provisional working model specifies that the effects of glucose upon memory functioning may be mediated via the hippocampus. It has been suggested that the hippocampal-diencephalic system is vital for item recognition occurring through recollection of the stimuli, whereas item recognition mediated through detection of stimulus familiarity is independent of the 'extended hippocampal system' (Aggleton and Brown, 1999). Therefore, if glucose facilitation of long-term memory performance is indeed mediated predominantly via the hippocampus, we anticipated in this study that recognition based on recollection (as measured by 'remember' responses) will be improved by glucose administration, whereas familiarity-based recognition (as measured by 'know' responses) will be unaffected by glucose.

2. Materials and methods

2.1. Participants

Ffifty-six healthy young individuals with no history of neurological or psychiatric illness, or diabetes took part in this study. The age range was 18–25 years (mean age 20 years), with a mean BMI of 22.85 kg/m^2. Participants were recruited via an opportunity sample from the Lancaster University. Participants received £5 for taking part in the experiment. The study was approved by the Department of Psychology Ethics Committee at Lancaster University, and was

conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from each participant prior to participation in the study.

2.2. Recognition memory test

A set of 80 words was selected from Gilhooly and Logie (1980) and divided into 2 lists of 40. The two lists were matched for length (in letters), frequency, concreteness, imageability, and age-of-acquisition, though none of these variables were manipulated. One of the lists was presented to participants at study and later used as target items in the recognition test, while the other list of words provided the distractor items for the recognition test. Half the participants in each condition studied list 1 at the time of encoding, and half studied list 2. Study items within each set were presented in a different random order for each participant. The dependent measures were the numbers of 'remember' (R) and 'know' (K) responses produced in the recognition test.

2.3. Treatment and design

A double-blind, placebo controlled, between-participant-design was employed in order to investigate the effect of drink administration on 'remember' and 'know' responses. Participants received either 25 g of glucose or 5 tablets of aspartame (i.e. sweetness matched to the glucose treatment) dissolved in 300 ml of water. Glucose was used in a dose of 25 g, as previous studies in this and other laboratories have shown this dose to be effective for memory enhancement (Riby, 2004). All drinks were flavoured with two teaspoons of lemon juice in order to improve palatability and participants' compliance. The trial material was prepared in the laboratory and refrigerated prior to testing.

2.4. Procedure

Each participant attended one test session that lasted approximately 30 min. Participants were informed that they should not eat or drink anything (except water) for a period of 2 h before testing. This time interval was chosen as previous research demonstrated that a 2 h period of fasting is sufficient to demonstrate the glucose memory facilitation effect (Sünram-Lea et al., 2001). Testing was carried out between 09:00 and 11:00 h. All participants were informed that they would undergo cognitive testing relating to human memory performance, and that they were required to drink a non-harmful, non-intoxicating liquid. Participants were asked to give information about their age, weight, and height, and whether they were taking any medication.

At the beginning of each session (i.e. before drink consumption), baseline glucose levels were measured. Participants were assured that they were permitted to withdraw from the study without prejudice if they were not willing to have small samples of blood taken. Blood glucose readings were obtained using the ExacTech blood glucose monitoring equipment (supplied by MediSense Britain Ltd., 16/17 The Courtyard, Gorsey Lane, Coleshill, Birmingham B46 1JA), following the recommended procedure. Participants then received one of the two treatments (glucose or aspartame). These treatments were randomly allocated to participants as they entered the laboratory. Drink administration followed a double-blind procedure, i.e. the nature of the drink was not divulged to either the participant or the experimenter. After a 10 min delay, participants' blood glucose levels were measured again. This was directly followed by study phase, in which participants were presented with a pre-recorded set of to-be-remembered items (40 words) at a rate of one word every 2.5 s. Participants were instructed to listen to the list silently in preparation for a later (unspecified) memory test. Following the list study phase, all participants gave a second blood glucose sample, and then completed some multiplication problems for 10 min (as a distractor phase). They were then given the recognition test, which consisted of the 40 target items plus the 40 distractor items, presented on both sides of a single sheet of paper. Each side of the paper contained 2 columns of 20 words each. The letters R, K and G appeared to the right of each word. Participants were given the following instructions:

"These pages contain a set of words, some of which appeared in the list you heard earlier. The letters 'R', 'K', and 'G' are printed to the right of each item. Your task is to identify those words that appeared in the earlier phase of the experiment. If you believe a word did not appear in the earlier phase, leave it and go on to the next one. If you believe a word did appear in the earlier task then underline it. In addition, each time you underline a word please circle either R, K, or G. These stand for 'remember', 'know', and 'guess', and refer to the nature of your conscious experience as you recognize the item. A 'remember' response is one in which you can consciously recollect the appearance of that word in the first part of the experiment. You may recall details of the event, such as any thoughts, feelings, or memories you experienced when you heard the word, an association you formed with another word, or some aspect of the word's physical appearance. A 'know' response is one in which you recognize the word because it feels familiar from the first part of the experiment, but you cannot recall any details of its occurrence. You recognize it purely on the basis of familiarity. There may be other words that you neither recollect nor recognize on the basis of familiarity, but which you cannot definitely reject. You have the option of making a 'guess' response to these items if you wish. Please think carefully about each word, and make a 'remember', 'know', or 'guess' decision for each one you recognize". The recognition test was participant-paced, and took approximately 5 min to complete. A final blood glucose reading was obtained immediately after the recognition phase (approximately 30 min after consumption of the earlier drink).

2.5. Statistical analyses

Blood glucose values were examined using a two-way analysis of variance (ANOVA) with repeated measures on one factor. The between-subjects factor was drink (aspartame versus glucose). The within-subject factor was time (i.e. at what point blood glucose was measured: T0 = baseline blood glucose levels, T10 = 10 min post ingestion, T30 = 30 min post ingestion). Where significant statistical effects were identified by ANOVA, Tukey honest significant difference (HSD) testing was subsequently conducted. Overall correct recognition hits, and correct 'remember', 'know' and 'guess' responses were then each submitted to separate one way ANOVAs to investigate the effect of drink (glucose versus placebo). A further series of correlation analyses was carried out in order to test (i) whether there was a relationship between blood glucose levels at 10 and 30 min and recognition memory performance and (ii) whether individual differences in the glycaemic response to a glucose drink had any effects on recognition memory.

3. Results

3.1. Glycaemic response

A two-way ANOVA (drink, 2 levels; time, 3 levels) produced significant main effects of drink [$F(1,53) = 26.26$; $p < 0.0001$], and time [$F(2,106) = 51.45$; $p < 0.0001$] and a significant drink × time interaction [$F(2,106) = 36.73$, $p < 0.0001$]. Post hoc testing indicated that although baseline BGLs (T0) did not differ across groups ($p = 0.99$; n.s.), as anticipated those receiving the glucose drink had significantly higher BGLs 10 min (T10) and 30 min (T30) post consumption compared to the aspartame group ($p < 0.01$ and $p < 0.001$; respectively). (See Fig. 1).

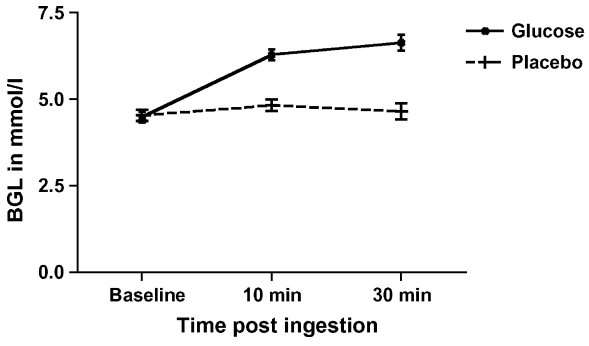

Fig. 1. Blood glucose levels (mmol/l) as a function of drink condition and time.

Table 1
Mean proportion of studied words correctly recognized (Hits) and non-studied words falsely recognized (FA = false alarms) as a function of drink condition and subjective experience

Drink	Glucose		Placebo	
	Hits	FA	Hits	FA
Overall	0.68 (0.15)	0.37 (0.23)	0.60 (0.13)	0.36 (0.20)
Remember (R)	0.38 (0.15)	0.06 (0.09)	0.28 (0.15)	0.04 (0.04)
Know (K)	0.16 (0.10)	0.12 (0.11)	0.18 (0.09)	0.10 (0.09)
Guess (G)	0.14 (0.10)	0.19 (0.14)	0.14 (0.11)	0.22 (0.15)

Note: Standard deviations in parentheses.

3.2. Overall recognition

Overall hits were submitted to a one-way ANOVA. There was a significant effect of drink on overall recognition hits [$F(1, 54) = 4.38$, $p = 0.04$]. Following glucose administration participants correctly recognized significantly more items (0.68 ± 0.15) compared to placebo (0.60 ± 0.13).

3.3. Subjective experience

Correct 'remember', 'know' and 'guess' responses were then each submitted to separate one-way ANOVAs. The analysis of 'remember' responses revealed a main effect of drink [$F(1,54) = 6.10$; $p = 0.017$], with participants receiving the glucose drink displaying significantly more correct remember responses than participants receiving placebo. No effects of drink were found on correct 'know' [$F(1,54) = 0.58$; $p = 0.45$; n.s.] or 'guess' responses [$F(1,54) = 0.01$; $p = 0.97$; n.s.]. (see Table 1 for all treatment means) (Fig. 2).

3.4. Relationship between blood glucose level and memory

Analysis of the Pearson's product moment correlation coefficient (two-tailed) across drink conditions showed no significant correlation between blood glucose levels following drink administration (T10 and T30) and number of overall recognition hits. Further analysis of response type revealed no significant correlation between blood glucose levels at either time point and the number of correct 'remember', 'know' or 'guess' responses. In addition, no significant correlations were observed between blood glucose levels and the number of false alarm responses (see Table 2 for correlation matrix).

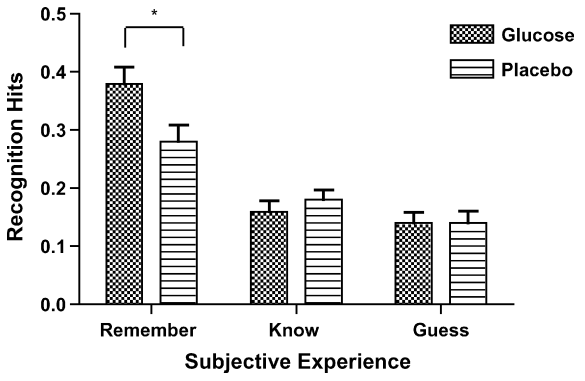

Fig. 2. Mean proportions of studied words correctly recognized (Hits) as a function of drink condition and subjective experience. $^{*}p < 0.05$.

Table 2
Correlation matrix for blood glucose levels and glucoregulatory indices and recognition memory

	Hits				FA			
	Total	R	K	G	Total	R	K	G
Fasting BGL[a]	−0.05	−0.10	0.01	0.07	0.00	−0.04	−0.09	0.08
BGL T10[a]	0.12	0.13	−0.11	0.06	0.14	0.22	0.06	0.06
BGLT30[a]	0.21	0.09	−0.07	0.20	0.10	0.14	−0.02	0.08
Evoked[b]	−0.01	−0.16	−0.06	0.27	0.00	0.09	−0.14	0.05
AUC[c]	−0.08	−0.16	−0.02	0.15	0.18	0.22	0.01	0.13

[a] Irrespective of drink.
[b] Calculation for evoked glycaemic response following glucose ingestion: BGL T30-T10.
[c] Calculation for Area under the curve (AUC) following glucose ingestion: [((BGL10 − BGL0)/ 2) × (10 − 0)] + [{((BGL10 − BGL0) + (BGL30 − BGL0))/2} × (30–10)].

3.5. Glucoregulation indices and recognition memory

Previous research has suggested that individual differences in the ability to regulate blood glucose levels following a glucose drink influences the degree to which glucose administration affects memory performance (e.g. Awad et al., 2002). As the effect of glucose administration on subjective recognition experience may also be moderated by glucoregulatory control, we decided to examine the relationship between the ability to regulate glucose levels and recognition memory performance following administration of a glucose drink. In order to assess the degree of glycaemic control we used two glucoregulation indices: (i) recovery from evoked glucose levels (defined in this study as the difference between evoked glucose levels 30 min post glucose administration and fasting glucose levels; previously used for example by Awad et al., 2002; Donohoe and Benton, 2000; Manning et al., 1990; Messier et al., 1997, 1999), and (ii) area under the curve of evoked glucose levels (see, for example, Awad et al., 2002). A high evoked glycaemic response is generally thought to be the result of poor insulin secretion and/or increased tissue sensitivity to insulin. Both indices (i) and (ii) above have previously been shown to predict susceptibility to glucose induced memory enhancement (Awad et al., 2002). Although there was a significant positive correlation between these two glucoregulation indices ($r = 0.69$; $p < 0.05$), neither predicted memory performance in terms of overall recognition hits or subjective recognition experience (i.e. correct 'remember' and 'know' responses). No significant correlations between glucoregulatory indices and the number of false alarms were observed (see Table 2 for correlation matrix).

4. Discussion

The aim of the present study was to investigate the effects of glucose administration on the recollection and familiarity components of recognition memory. With regard to the

glycaemic response, as expected blood glucose levels were significantly higher after glucose consumption compared to placebo (aspartame-sweetened drink). However, no significant relationship between blood glucose levels and memory performance was observed. It has been suggested that glucose improves memory in humans that have poor glucose regulation but that effects are less likely to be observed in humans with good glucose regulations (Awad, 2002). However, in the current study individuals' blood glucose regulation did not affect the degree to which they were susceptible to the effects of glucose administration on recognition memory. It is important to note that Awad et al. (2002) assessed glucose tolerance following administration of 75 g glucose load, whereas in the current study only 25 g was administered. This may account for the failure to observe a relationship between glucoregulation and memory performance since the glucose load used in the current study (25 g) might not have been sufficient to expose differences in glucose regulation and tolerance in healthy young adults.

Regarding recognition memory performance, participants recognized significantly more words following glucose administration compared to placebo. Further analysis of the subjective recognition experience revealed that glucose consumption did not affect all aspects of recognition memory equally. Glucose administration led to significantly increased recollection ('remember' responses), but had no effect on stimulus familiarity ('know' responses). In addition, no drink specific effects were observed on 'guess' responses. Consequently, glucose administration appears to increase recognition memory that is accompanied by recollection of contextual details and episodic richness.

The present results may help explain why some previous studies have failed to identify an effect of glucose administration on recognition memory. Assessing the effects of glucose administration from a mere quantitative perspective (i.e. 'old'/'new' distinction) might not demonstrate effect of glucose administration on recognition memory, since glucose administration appears to have differential effects on the different processes underlying recognition memory. Hence, the failure (in some previous studies) to observe a glucose effect on overall recognition memory using an 'old'/'new' decision might be due to the fact that this approach does not allow examination of selective effects on specific elements (i.e. different subjective experiences) of 'remembering'.

These findings add to the growing body of evidence that 'remember' and 'know' responses in recognition memory can be dissociated empirically (see Gardiner and Richardson-Klavehn, 2000, for a review). The enhancement of 'remember' but not 'know' responses by glucose is also consistent with previous findings that remembering is selectively influenced by the administration of other psychopharmacological interventions such as the administration of lorazepam (Curran et al., 1993) and alcohol (Curran and Hildebrandt, 1999). These substances are known to influence conscious processes.

The present findings indicate that the behavioural consequence of glucose administration encompasses the facilitation of more richly episodic representations that include details of the encoding context. Rajaram (1996) has suggested that 'remember' responses are increased by encoding operations that enhance the distinctiveness of studied items. So, for example, these responses are selectively enhanced by deep levels of processing and generation (Gardiner, 1988), elaborative rehearsal (Gardiner et al., 1994), and the presentation of more distinctive stimuli such as pictures relative to words (Dewhurst and Conway, 1994). The selective effect of glucose administration on 'remember' responses observed in this study is consistent with this account, and suggests that glucose enhances the distinctiveness of encoding.

Numerous neuroimaging studies have provided evidence for activation of the hippocampal system during encoding of faces, words, scenes or objects (e.g. Bernard et al., 2004). More specifically, fMRI studies have shown that the amount of hippocampal activity at the time of encoding predicts how well that item is subsequently remembered (Brewer et al., 1998; Wagner et al., 1989; Kirchhoff et al., 2000). This is known as the 'subsequent memory effect' or the 'difference due to memory' (Dm) effect (Paller et al., 1987). By this effect, subsequently remembered items are associated with greater brain activation at the time of encoding. With respect to the current study, it could be argued that glucose administration leads to a stronger memory trace, i.e. glucose administration results in the rich encoding of stimuli that are more likely to be recognised on the basis of episodic 'remembering', in comparison to a placebo treatment. However, further studies that explicitly compare the effects of glucose administration at different stages of the memory process (encoding, consolidation or retrieval) on recollection components of recognition memory are needed before any firm conclusions can be drawn.

Although the exact mechanisms enabling increased peripheral and/or central glucose availability to influence memory processes are not known, microdialysis measurements of rat brain glucose have shown that hippocampal extra cellular fluid (ECF) glucose levels fall during maze testing (a memory task), suggesting that metabolic demands associated with cognitive performance in the hippocampus are limited by glucose supply. The fall in ECF glucose can be prevented by (intraperitoneal) administration of glucose, correlating with enhanced memory performance (McNay et al., 2000). In addition, it has recently been argued that the depletion of extracellular glucose after cognitive challenge could be caused by learning induced up-regulation of hippocampal glucose tranporter 1 (GLUT 1; Choeiri et al., 2005). Moreover, there is substantial evidence indicating that glucose may enhance memory by augmenting cholinergic functions in the hippocampus (Durkin et al., 1992; Messier and Gagnon, 1996; Messier et al., 1990; Ragozzino et al., 1996, 1998; Kopf and Baratti, 1996). Further, it has been argued that elevated insulin in response to hyperglycaemia (rather than hyperglycaemia *per se*) may boost glucose utilisation in the hippocampus, and thereby result in improved memory performance (Craft et al., 1996, 1999; Benedict et al., 2004; Watson et al., 2006; Benedict et al., 2007). Indeed, at the molecular level, insulin and/or insulin receptors seem to contribute in the regulation of learning and memory via the activation of specific signalling pathways, one of which is

shown to be associated with the formation of long-term memory (for a more detailed account see Zhao and Alkon, 2001).

In addition, glucose facilitation of memory performance might be mediated by amygdala-hippocampal interaction. Data suggest that emotional arousal activates the amygdale, and that such activation results in the modulation of memory storage occurring in other brain regions (McGaugh et al., 1996). More specifically, engagement of the amygdala (especially basolateral nucleus) by emotional stimuli is thought to up-regulate gate responses in the hippocampus, resulting in memory enhancement (McGaugh et al., 1996; Cahill et al., 1996; Kilpatrick and Cahill, 2003). Although it has been argued that the amygdala is only involved in memory processes for emotional material, intra-amygdala glucose injections have been shown to attenuate morphine-induced performance impairments on a 'neutral' spatial memory task (McNay and Gold, 1998). Presentation of emotionally arousing material not only increases subsequent memory performance, but also raises plasma glucose levels (Blake et al., 2001; Parent et al., 1999; Scholey et al., 2006). Glucose administration might 'mimic' exposure to emotionally arousing material and lead to amygdalar involvement in remembering neutral material. Further investigations are needed to explore the extent to which glucose administration modulates interactions of the amygdala-hippocampal complex.

In summary, the present study demonstrated that glucose administration has differential effects on 'recollection' and 'familiarity' components of recognition memory. Glucose administration significantly increased recognition based on recollection ('remember' responses), whereas familiarity-based recognition ('know' responses) was unaffected by glucose treatment. Consequently, glucose administration appears to increase recognition memory that is (a) accompanied by recollection of contextual details and episodic richness, and (b) hippocampally dependant. In addition, these findings suggest that memory tasks that result in high levels of hippocampal activity may be more likely to be enhanced by glucose administration than tasks that are less reliant on medial temporal lobe structures.

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