Impaired word and face recognition in older adults with type 2 diabetes

Nicola Jones[a], Leigh M. Riby[a] & Michael A. Smith[a,b,*]

[a] Department of Psychology, Faculty of Health and Life Sciences, Northumbria University, Newcastle upon Tyne, United Kingdom

[b] Faculty of Medicine, Dentistry and Health Sciences, University of Western Australia, Perth, Australia

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*Corresponding author: Dr Michael Smith

Department of Psychology

Faculty of Health and Life Sciences

Northumbria University

Northumberland Building

Newcastle upon Tyne NE1 8ST

United Kingdom

Tel: +44 (0) 191 243 7169

Fax: +44 (0) 191 227 4515

michael4.smith@northumbria.ac.uk

Abstract

Background: Older adults with type 2 diabetes mellitus (DM2) exhibit accelerated decline in some domains of cognition, including verbal episodic memory. Few studies have investigated the influence of DM2 status in older adults on recognition memory for more complex stimuli, such as faces. *Aims:* In the present study, we sought to compare recognition memory performance for words, objects and faces under conditions of relatively low and high cognitive load. *Methods:* Healthy older adults with good glucoregulatory control (n = 13) and older adults with DM2 (n = 24) were administered recognition memory tasks in which stimuli (faces, objects and words) were presented under conditions of either i) low (stimulus presented without a background pattern), or ii) high (stimulus presented against a background pattern) cognitive load. *Results:* In a subsequent recognition phase, the DM2 group recognised fewer faces than healthy controls. Further, the DM2 group exhibited word recognition deficits in the low cognitive load condition. *Conclusions:* The recognition memory impairment observed in the patients with DM2 has clear implications for day-to-day functioning. While these deficits were not amplified under conditions of increased cognitive load, the present study emphasises that recognition memory impairment for both words and more complex stimuli, such as faces, are a feature of DM2 in older adults.

Keywords: Type 2 Diabetes Mellitus; Memory; Aging; Cognition

Introduction

Glucose regulation is a process which is required to maintain homeostasis. Glucoregulatory problems are typically characterised by either i) a high baseline blood glucose in the fasted state, or ii) a prolonged elevation in blood glucose following the ingestion of carbohydrates (1). Problems with glucoregulation in older adults[1] have been shown to adversely impact upon cognitive processing above and beyond normal aging (2, 3). Adults with type 2 diabetes mellitus (DM2) suffer with chronic glucoregulatory problems and exhibit deficits in a number of cognitive domains. In particular, DM2 older adults have demonstrated impaired executive functioning and information processing speed (4-7), although the most frequently reported deficits are seen in episodic memory (2, 8) .

Research investigating cognitive functioning in individuals with glucoregulatory problems has concentrated on verbal memory, as it has been demonstrated that this cognitive domain is most reliably affected in individuals with poor glucoregulatory efficiency (for a review see 9). On this basis, relatively few studies have investigated glucoregulatory influences on nonverbal memory, such as object or face recognition and results in this domainhave been mixed (2). For example, Messier and colleagues (10) investigated the impact of glucoregulation on a variety of cognitive processes including tests of verbal episodic memory and visual memory in healthy young adults. They found only verbal episodic memory to be significantly compromised in participants with poor glucoregulation compared to those with good glucoregulation. However, Vanhanen and colleagues (11) found that when investigating the same processes in healthy older adults, participants with poor glucoregulation performed poorly on both verbal and visual aspects of episodic memory. There are indications from research in older adults with DM2 that impaired glucoregulation

[1] The definition of 'older adult' is somewhat arbitrary and inconsistent in this research area. For the purpose of the present study, we use the World Health Organisation (WHO) definition of 60 years

affects non-verbal episodic memory. A study by Zaslavsky et al (12) compared the performance of DM2 patients with and without autonomic neuropathy, as well as healthy controls, on a range of verbal and visual memory tasks, including a face recognition task. The authors found that there were no differences between the three groups on verbal memory but DM2 patients with autonomic neuropathy exhibited relatively poorer performance on the visual memory tasks. Further, Fontbonne and colleagues (13) found that face recognition performance declined to a greater extent over a four year period in DM2 older adults, relative to individuals with impaired fasting glucose (IFG) and healthy controls. This suggests that the decline in episodic memory performance in older adults with DM2 may not be restricted to the verbal domain. Evidence from the literature suggests that structural and functional changes within the fronto-parietal cortices, the medial temporal lobes and hippocampus are of particular interest when investigating memory decline in DM2, as these structures are associated with episodic memory function (14-16). Research comparing DM2 participants and healthy controls suggests that DM2 patients have increased hippocampal atrophy (17, 18) and that this is related to the cognitive deficits seen in DM2, such as verbal declarative memory impairments (19). Damage to the hippocampus in amnesic patients and those with Alzheimer's disease (AD; a condition which shares common pathological features with DM2; 20) also reveals impairments in face recognition (21, 22). The reduction in hippocampal volume coupled with reportedly reduced connectivity to surrounding brain regions (23) may provide a mechanistic underpinning for the types of cognitive impairment seen in DM2. It is therefore critically important to further investigate diabetes-related cognitive decline on non-verbal aspects of episodic memory, particularly given the potential impact of impaired face recognition on quality of life.

In addition to a relative lack of evidence pertaining to the influence of DM2 status on non-verbal episodic memory, few studies have investigated the influence of cognitive load on

memory performance in this group. Limited findings from studies in healthy older adults has suggested that the performance of individuals with relatively poor glucoregulation is lower on tasks requiring greater cognitive load, such as switching attention (24). In a recent set of studies, source monitoring ability in verbal episodic memory (a more demanding aspect of recognition memory of being able to recall specific details of the context of an item) was found to be compromised in those with impaired glucose tolerance (IGT), and those with DM2 (25). The authors speculated that this may be due adverse effects of poor glucoregulation on the hippocampus, a brain region associated with source monitoring that is typically compromised in those with DM2. On this basis it is reasonable to infer that diabetes status may impact upon episodic memory performance to a greater extent under conditions of increased cognitive load. Cognitive load has been demonstrated previously to impact upon processes essential to daily living; for example, postural control has been found to be compromised under dual-task conditions in healthy older adults (26), and in older adults with DM2 (7). It is therefore of interest to investigate the extent to which increases in cognitive load impact upon memory performance in DM2 older adults, relative to healthy age-matched controls.

The aim of this study was to investigate the effects of DM2 status and cognitive load manipulation on recognition memory performance in older adults. As previous research has predominantly focussed on the verbal domain, this study sought to investigate whether memory for stimuli in other visual domains (i.e. object and face recognition) is impaired in DM2. Previous studies have administered cognitive tests which have not enabled a thorough investigation of the influence of cognitive load on performance in DM2 across different stimulus types (7, 24, 25). This study aimed to use a novel approach by focussing on recognition memory for words, objects and faces using a cognitive load manipulation paradigm in order to mimic the variability in cognitive load typically experienced in everyday

life. Considering previous reports of verbal memory deficits in individuals with compromised glucoregulatory efficiency (9), it was predicted that DM2 older adults would exhibit deficits in word recognition. In addition, we hypothesised that impaired recognition memory for objects and faces would also be observed here. Further, given that performance is impaired in individuals with compromised glucoregulatory efficiency under conditions of increased cognitive load (24), we sought to investigate whether cognitive load moderates recognition memory deficits in DM2. It was hypothesised that recognition memory performance across all stimulus types would be poorer under conditions of increased cognitive load, and that recognition memory deficits would be particularly pronounced in the high cognitive load condition for the DM2 group.

Method

Design

A quasi-experimental mixed design was used, with one between-subjects and one within-subjects factor. The between-subjects factor was group (2 levels: diabetes; control). The within-subjects factor was cognitive load (2 levels: low; high).

Participants

A total of 24 older adults with DM2 took part (7 females, mean age = 71.38 years, SD = 5.77). The DM2 participant group were selected on the basis of a self-reported clinical diagnosis of diabetes. DM2 participants were asked how they controlled their diabetes: ten participants controlled their DM2 using medication (no participants were insulin dependent) alone; eight participants controlled their DM2 through diet and lifestyle; and six participants controlled their DM2 through a combination of diet/lifestyle and medication. How

participants managed their DM2 did not impact on performance ($p > .05$ across all tasks) , so DM2 participant data was considered as a whole group. The mean duration of disease was 10.81 (SD = 8.18) years, the range was 2-30 years.

Their data were compared to 13 healthy older adults who were screened for diabetes via a two-hour oral glucose tolerance test (OGTT; 7 females, mean age = 71.92 years, SD= 5.48), and were found not to meet the diagnostic criteria for diabetes as shown in Figure 1 (fasting blood glucose ⩾ 7.0 mmol/L or 2-hour blood glucose ⩾ 11.1 mmol/L; 27). All participants were white Caucasian. Further participant details are shown in Table 1. Participants were eligible to participate if they were aged over 60 years; were proficient in English; were not suffering from any serious medical (other than type 2 diabetes if part of the DM2 group) or psychiatric condition e.g. coronary disease, asthma, depression, anxiety related disorders; had a caffeine intake that did not exceed ten cups of tea/coffee per day; did not have a diagnosis of dementia or a memory disorder (e.g. mild cognitive impairment); did not have a visual impairment that could not be corrected by glasses or contact lenses; and they did not have any known blood disorders or infections. Participants were recruited through local older adult groups and newsletters, as well as through the North East Age Research participant pool. DM2 participants were also recruited through the Newcastle upon Tyne Diabetes UK support group. Control participants were reimbursed £20 and DM2 participants were reimbursed £10 as compensation for their time and out-of-pocket expenses (control participants were required to attend an additional session to enable the OGTT to be performed in order to screen for diabetes, hence a greater reimbursement value). Written informed consent was obtained from all participants prior to testing. Ethical approval was obtained from the School of Life Sciences Ethics committee at Northumbria University.

INSERT TABLE 1 AND FIGURE 1 ABOUT HERE

Materials

OGTT and blood glucose monitoring equipment. Control participants were required to attend an OGTT session, approximately one week prior to the main testing session. The purpose of this session was to ensure that the control participants did not meet the diagnostic criteria for diabetes. Participants fasted overnight before completing an OGTT the next morning. Baseline blood glucose levels were measured by taking capillary blood drawn from the fingertip using Accu-Chek Safe-T-Pro Plus lancets. The blood was applied to Optium Plus glucose reagent strips (Abbott Diabetes Care Ltd, Oxford, UK) for blood glucose quantification using an Optium Xceed glucometer (Abbott Diabetes Care Ltd, Oxford, UK). Subsequently, participants were allowed 10 minutes to drink a solution of 75g glucose, 200 ml water and 30 ml of no-added sugar orange squash. Blood glucose levels were then measured 30 minutes, 60 minutes, 90 minutes and 120 minutes post-ingestion. Following blood glucose measurements, participants were provided with a £5 breakfast voucher.

Recognition memory stimuli. All tasks were presented using E-Prime software (E-Prime 2.0, Psychology Software Tools) on a Windows desktop PC. For the verbal recognition tasks, 120 monosyllabic words were taken from the online MRC Psycholinguistic database. The words were 4-6 letters in length, with a high familiarity rating (between 400-700). They were presented in the centre of the screen in Courier New font, font size 40. For the object recognition tasks, 120 colour images of object stimuli were taken from the Amsterdam Library of Object images (28). They were presented in the centre of screen and were standardised to a size of 384 by 288 pixels. For the face recognition tasks, 120 colour images of white Caucasian faces (60 male, 60 female of varying age) were taken from the Max Planck Institute for Human Development FACES database (29). Images were presented in

the centre of the computer screen against a white background in a standard size of 251 by 314 pixels.

Recognition memory tasks. For each of the three stimulus types (faces, objects and words), two tasks were created: one with low cognitive load and one with high cognitive load. Figure 2a shows the experiment format. Stimuli were split into 30 'targets' and 30 'foils' for each task. All recognition tasks followed the same format; each task had an encoding phase and a recognition phase. Each encoding phase comprised 30 trials which were presented in a random order. Each trial began with a fixation cross for 500 ms followed by the stimulus for 3000 ms. A two-minute retention interval followed each encoding phase during which the participants were asked to sit quietly. The recognition phase had 60 trials comprising target stimuli interleaved randomly between foil stimuli. Each trial began with a fixation cross for 500 ms followed by the stimulus. The next trial began once participants had made their response; either responding 'Yes' (they had seen the stimulus previously) or 'No' (they had not seen the stimulus previously). Participants indicated their responses by pressing 1 or 3 on the computer keyboard.

In the low cognitive load condition participants were shown the target stimuli against a white background and asked during recognition to indicate whether they had previously seen the stimulus. In the high cognitive load condition, the target stimuli were presented against one of two patterned backgrounds (Figure 2b). Participants were asked to remember both the stimulus and the pattern against which it was presented and at recognition were asked to indicate whether or not they had previously seen the stimulus and the pattern against which it had been presented. Four versions of each task were created to counterbalance the presentation of target and foil stimuli, and also key press responses. Participants were required to make a key press response of 'yes' if they remembered seeing the item during the study phase or to depress a response key marked 'no' if they did not remember seeing the

item previously. Whether the yes response was made with the left or right hand and the condition to which items were assigned (i.e. old versus new) was counterbalanced between participants. The order of presentation of the different types of stimuli was also counterbalanced across participants. Verbal targets and foils were matched for word length; face stimuli were matched in terms of numbers of male and female faces and age; object stimuli were matched according to stimulus size on the screen. Each test period lasted approximately 5 minutes in total, with an overall testing time of approximately 30-35 minutes.

INSERT FIGURE 2 ABOUT HERE

Procedure

All testing sessions took place between 8:30 and 10:30am. Control participants were asked to attend the laboratory having consumed their normal breakfast. DM2 participants were asked to attend the laboratory having fasted overnight (at least 12 hours), so that a fasting blood glucose measure could be obtained. The DM2 participants were provided with a standard breakfast of 40 g cornflakes and 200 ml semi skimmed milk following measurement of fasting blood glucose. Participants were given 10 minutes to consume their breakfast and any medication they required. For the DM2 group, there was a 15 minute interval between end of breakfast consumption and administration of the memory tasks.

Both groups of participants completed the National Adult Reading test (NART) as a measure of premorbid intelligence, and their height, weight and blood pressure were recorded using an inflatable brachial cuff (Omron M3 IntelliSense, Omron Healthcare UK Limited, Milton Keynes, United Kingdom). DM2 participants were asked to report the date of their diagnosis and illness duration. Following this, participants were administered the recognition

memory tasks (the order of words, faces and objects tasks were counterbalanced). After completing the tasks, participants were debriefed and given the opportunity to ask any questions about the study.

Results

Measurements of accuracy and reaction time were collected. Accuracy was measured by subtracting the total number of false alarms from the total number of hits (d-prime). Hits, false alarms, accuracy and reaction time data for each stimulus type were analysed using 2 (cognitive load: low, high) x 2 (group: DM2, control) ANOVAs. All analyses were Bonferroni-corrected to reduce type 1 errors. Figure 3 contains the descriptive statistics for hits, false alarms, accuracy across stimulus types of each cognitive load condition for control and DM2 participants.

Hits

There was a main effect of cognitive load for all stimulus types ($p \leq .01$). For objects, there was a significant cognitive load x group interaction [$F (1, 35) = 6.25$, $p = .02$, $\eta_p^2 = .15$]. Post-hoc tests revealed no significant differences in numbers of hit responses between control and DM2 participants in either low or high cognitive load conditions [equal variances assumed - low load: $t (35) = 1.73$, $p = .09$; equal variances not assumed - high load: $t (18.65) = -1.31$, $p = .21$]. Both groups made significantly more hit responses when cognitive load was low [control: $t (12) = -4.27$, $p = .001$; DM2: $t (23) = -3.56$, $p = .002$]. All other main and interaction effects were nonsignificant.

False Alarms

For objects, there was a main effect of cognitive load [F (1, 35) = 4.94, p = .03, η_p^2 = .12]. All other main and interaction effects were nonsignificant.

Accuracy

There was a main effect of cognitive load for all stimulus types (p < .001). For faces, there was a significant main effect of group in that control participants recognised more faces (mean = 15.08, SD = 5.75) than participants with DM2 (mean = 10.73, SD = 5.33) [F (1, 35) = 5.30, p = .03, η_p^2 = .13]. No significant interaction was observed. For words, a significant cognitive load x group interaction was observed [F (1, 35) = 5.91, p = .02, η_p^2 = .14]. Post-hoc tests revealed that there was no significant difference in accuracy between DM2 and control participants in the high cognitive load condition [t (35) = .41, p = .69]. However, in the low cognitive load condition control participants recognised significantly more words than DM2 participants [t (35) = 2.79, p = .008]. Both groups recognised significantly more words in the low cognitive load condition than in the high cognitive load condition [control: t (12) = -4.77, p < .001; DM2: t (23) = -4.35, p < .001]. All other main and interaction effects were nonsignificant.

Given that NART scores significantly differed between the DM2 and control groups, it was of interest to ascertain whether diabetes status continued to predict overall face recognition accuracy (hits minus false alarms summed across the low and high load conditionss) when NART scores were controlled for statistically in a regression analysis. Doing so revealed that diabetes status was a significant predictor of face recognition accuracy (β = -0.36, R^2 = .28, p = .03), but this relationship became nonsignificant when NART scores, which were a significant predictor of face recognition accuracy (β = 0.46, R^2 = .33, p = .004) were added to the model.

INSERT FIGURE 3 ABOUT HERE

Reaction time

Descriptive statistics for each response (hits, false alarms, correctly identified foils, misses) for each stimulus type at each level of cognitive load are displayed in Figure 4.

Faces. There was a main effect of cognitive load for hits, false alarms and correctly identified foils ($p \leq .004$). There were no other significant main or interaction effects for faces.

Objects. There was a main effect of cognitive load for hits, misses and correctly identified foils ($p \leq .02$). There were no other significant main or interaction effects for objects.

Words. There was a main effect of cognitive load for hits, false alarms, misses and correctly identified foils ($p \leq .01$). For false alarms, a significant interaction was found between cognitive load and group [$F (1, 33) = 9.12$, $p = .005$, $\eta_p^2 = .22$]. Post-hoc tests revealed that when cognitive load was high, control participants made false alarm responses significantly faster than DM2 participants [$t (35) = -2.37$, $p = .02$]. No significant difference was found between the groups when cognitive load was low [$t (33) = .40$, $p = .69$]. There was no significant difference in reaction time between the two load conditions for control participants [$t (11) = 1.08$, $p = .30$]. However, DM2 participants made significantly faster false alarm responses in the low cognitive load condition compared to the high load condition [$t (22) = 5.79$, $p < .001$]. There were no further significant main or interaction effects for words.

INSERT FIGURE 4 ABOUT HERE

Discussion

This study investigated the influence of diabetes status and cognitive load on recognition memory performance for faces, words and objects. The results revealed that DM2 older adults exhibited face recognition deficits irrespective of cognitive load. In addition, they indicated that each stimulus type was more accurately recognised when cognitive load was low. A further salient finding to emerge from the present study was that word recognition deficits were observed in the DM2 group, relative to healthy controls, but only when cognitive load was low. Taken together, these findings provide evidence that, as expected, recognition memory for both words and faces is impaired in older adults with DM2. However, contrary to our hypotheses, the memory deficits observed in the DM2 group were not amplified under conditions of greater cognitive load. In fact, in the case of word recognition, the opposite pattern of findings was observed, in that memory impairment emerged only when cognitive load was low. No between-group differences on object recognition were detected, and there were no overall between-group differences with respect to reaction time across all response types detected.

It is important to acknowledge that premorbid intelligence (NART scores) differed between the DM2 and control groups. On this basis, we conducted a secondary regression analysis to ascertain whether diabetes status uniquely predicted face recognition performance, or whether any such effects were due to shared variance with NART scores. It was clear from the secondary analysis conducted that NART scores accounted for substantial variance in the relationship between diabetes status and face recognition accuracy, to the extent that diabetes status was no longer a significant predictor of face recognition accuracy once NART scores were controlled for statistically. On the basis of this finding, it may be concluded that it is in fact premorbid intelligence, rather than diabetes status per se which is accounting for the observed differences between the DM2 and control groups on face recognition accuracy.

However, despite the well supported validity of the NART as a measure of premorbid intelligence, it has been suggested that NART performance can decline post-onset of some conditions, including Alzheimer's Disease which is a known comorbidity of DM2 (30). Additionally, lower premorbid intelligence has been frequently reported in DM2 individuals (7, 31), thus lower intelligence is an integral part of this condition which may in fact account for some of the cognitive deficits observed. Indeed, low intelligence is considered a risk factor for DM2 and a range of other chronic conditions, possibly in part because such individuals may be more inclined to engage in adverse health behaviours that increase the risk of disease (32). Therefore, relatively lower intelligence could be considered an important mechanism underlying the cognitive deficits observed both in the present study and in other studies which have observed DM2-related cognitive deficits, and the between-group difference in NART scores should not detract from the important observation that face recognition is impaired in DM2.

The present study findings support previous research in this area which has reported episodic memory deficits in older adults with compromised glucoregulatory efficiency (2). Our observation that word recognition was impaired in DM2 older adults was unsurprising given that verbal memory deficits have been more widely reported in individuals with glucoregulatory problems (9). However, our finding that face recognition was also impaired in DM2 patients was relatively more novel, given that few previous investigations of face recognition have been conducted in DM2 individuals. Our findings support the limited previous studies which also report impairments in face recognition in DM2 (12, 13). There are a number of methodological differences between previous research assessing face recognition and the current study. Fontbonne and colleagues (13) used a test of face recognition to assess spatial attention, so the results of the present study whilst not directly comparable still reflect detriments in DM2 cognition. Comparatively, Zaslavsky et al.'s (12)

study used famous faces in their face recognition task and it was only their DM2 participants with autonomic neuropathy that displayed impairments in visual memory, leading the authors to suggest the deficits were a result of central autonomic dysfunction. Presentation of novel exemplars of items (such as unfamiliar faces, as used in the present study) and familiar exemplars (such as famous faces) have been found to activate the anterior part of the hippocampus, whilst posterior activation is only detected in response to presentation of familiar exemplars (33). This suggests that the anterior part of the hippocampus is important for initial encoding, whilst the posterior part of the hippocampus may be related to successful retrieval of semantic knowledge. In light of this, it may be that in those with DM2, there is disruption in connectivity between the fusiform face area (an area of the brain which is considered to be involved in face-specific processing (34)) and the anterior part of the hippocampus, leading to impairments in face encoding. This is supported by previous research that reports reduced connectivity between the hippocampus and surrounding regions in DM2 (23). As face recognition is such an important life skill, any accelerated decline in this domain may affect quality of life for older adults with DM2, particularly with respect to social interactions and the recognition of trusted individuals. Although the psychosocial implications of impairments in face recognition have not been investigated in DM2, in patients with AD, deficits seen in face processing may result in patients showing poor judgement in social situations as a consequence of an inability to judge external cues from those around them (35). The deficits in face recognition observed here may be a result of problems with specific memory processes, such as the encoding, storage or retrieval of memories. Future studies should seek to investigate both i) the precise neurocognitive deficits which may mediate the behavioural impairment observed in the present study, and ii) the functional living implications of face recognition deficits in DM2. These questions are

particularly salient given the limited previous work which has investigated face recognition deficits in DM2 (2).

As mentioned above, an interaction effect was observed for word recognition, in that word recognition was impaired in DM2 older adults only when cognitive load was low. This implies that high cognitive load had an equalising effect on verbal memory, thereby 'neutralising' any effect of diabetes status. This finding is inconsistent with previous observations that cognitive performance is particularly compromised in individuals with relatively poorer glucoregulatory efficiency under conditions of increased cognitive load (24). However, previous studies comparing cognitive performance in younger and older adults at varying levels of cognitive load suggest that younger adults tend to outperform older adults when the cognitive load is low, but such differences are not typically detected in much higher cognitive load conditions (36, 37). This may be because the cognitive capacity of even those individuals at their cognitive peak is exceeded when task demands reach very high levels. In the present study, it appears that control participants performed very well on the 'easier' (low cognitive load) condition, possibly due to a greater cognitive capacity. On the other hand, the individuals with DM2 appeared to lack the cognitive capacity to perform as well as the controls, even on the less difficult task.

It was predicted that DM2 participants would have significantly slower reaction times than controls across all tasks, as previous research has suggested that older adults with DM2 have impairments in processing speed (4). Despite this, the results of this study did not reveal any differences in reaction time between healthy controls and DM2 participants for hit responses. As these tasks were not specifically designed to test reaction time, they may have been insensitive to any reaction time differences. However, the means are in the expected direction (i.e. the more difficult tasks produce slower reaction times overall) thus

demonstrating ‘proof of paradigm’ of our task by establishing that an increased cognitive load impacted on task performance.

The study findings must be considered in context of the limitations. Firstly, the sample size was small. However, the findings on the word and face recognition tasks demonstrate a clear pattern of deficits in the DM2 group, relative to the healthy controls, therefore an increase in sample size may result in an increase in power to detect clearer reaction time differences between the two participant groups across all the recognition tasks. The gap between control participants’ breakfast consumption and computer testing was also not controlled so they may therefore have been at a different stage in the glycaemic response cycle compared to DM2 participants, particularly as their consumption of breakfast would have been earlier than that of the DM2 participants under controlled conditions. Participants’ blood glucose levels were also not measured immediately prior to testing. However, despite potential differences in time gap between breakfast and testing, control participants still outperformed DM2 participants across tasks. Equally, all participants were not screened for dementia, so it is possible that there were more participants in the DM2 group who potentially had early stages of dementia. However, the differences in recognition behaviour for words between the healthy and DM2 participants observed are consistent with the previous literature (9). A further limitation of this study was that diabetes diagnosis and duration was based entirely on self-report. While we were able to confirm objectively (via OGTT) that our control group did not meet the diagnostic criteria for diabetes, we were not able to confirm the diagnosis of diabetes in our DM2 group. An alternative measurement of glucoregulation would have been to consider levels of glycosylated haemoglobin (HbA1c levels), to obtain an indication of glucoregulatory functioning up to three months prior to testing. However, if anything, these limitations would likely reduce the prospect of observing significant between-group differences in the present study, so it is unlikely that any of the

significant between-group differences reported are false positives. Additionally, diabetes can be asymptomatic for several years prior to formal diagnosis (38), thus accurate information with respect to diabetes duration is difficult to ascertain even when clinical records are available. Furthermore, we did not measure subjectively whether the high cognitive load versions of each task were considered to be more demanding by the participants. Despite this, as mentioned above, the consistency in the pattern of reaction times (i.e. slower reaction times for each high cognitive load condition) gives some indication of proof of paradigm, as previous research has shown that more difficult tasks result in slower reaction times in older adults (39). Further, anecdotes from participants indicated that they found the high cognitive load conditions more difficult than the low cognitive load forms of the tasks. Finally, there were between-group differences with respect to premorbid intelligence (as discussed above), blood pressure and BMI which may at least partially explain the between-group differences observed. However, these factors are often found to differentiate older adults with DM2 from healthy age-matched controls (7), and comorbidities such as hypertension and obesity are typical features of DM2 which characterise this condition.

Conclusions

The results of this study failed to find support for the notion that recognition memory may be particularly compromised under conditions of increased cognitive load in older adults with DM2. However, the present study findings indicate that participants with DM2 exhibit clear deficits in both word and face recognition. As face recognition plays an integral role in our everyday lives, it is important to investigate and understand the neurocognitive mechanisms which underlie this deficit, and the potential implications for everyday functional living. Future research should consider the neurocognitive mechanisms involved, and the neuroanatomical processes that mediate recognition memory which may be adversely

affected by diabetes complications. By considering these issues further, it is possible that interventions can be put in place in the early stages post-diagnosis to aid those with DM2. Overall, the present study findings suggest that cognitive load has little bearing on recognition memory deficits in DM2. However, a particularly salient finding reported here is that the frequently reported DM2 related deficits in verbal memory can be extended to face recognition.

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Table 1. Background and demographic characteristics of the DM2 and control groups (means with standard deviations in parentheses).

	Control	**DM2**
Age	71.92 (5.48)	71.38 (5.77)
BMI	23.93 (2.87)	29.23 (4.10)*
NART	40.15 (4.51)	35.79 (7.73)*
Resting Systolic Blood Pressure	125.04 (14.61)	140.90 (20.54)*
Resting Diastolic Blood Pressure	70.74 (13.07)	60.93 (9.08)*
Baseline fasting glucose level (mmol/l)	5.06 (0.35)	6.35 (1.95)*

*indicates $p<.05$

Figure captions

Figure 1. OGTT data of control participants (0 represents baseline fasting levels; standard errors shown in error bars)

Figure 2. The task format used in the present study (example 'object' condition trials are displayed) (a). Examples of the two 'background patterns' used in the task (b).

Figure 3. Mean number of hits, false alarms (FAs) and accuracy (hits – false alarms) (and standard errors), given by each participant group in the low and high cognitive load conditions for each stimulus type on the recognition memory task. Maximum score is 30. *indicates control group significantly different from DM2 group ($p < .05$). †indicates low cognitive load significantly different from high cognitive load ($p < .05$)

Figure 4. Mean reaction time in milliseconds (and standard errors) of hit responses (Hits), false alarm responses (FA), correctly identified foils (CI) and missed target responses (Misses), given by each participant group in the low and high cognitive load conditions for each stimulus type on the recognition memory task. †indicates low cognitive load significantly different from high cognitive load ($p < .05$). $*_a$ indicates control group significantly different from DM2 group ($p < .05$). $*_b$ indicates DM2 group significantly faster in low cognitive load condition compared to high cognitive load condition ($p < .05$)

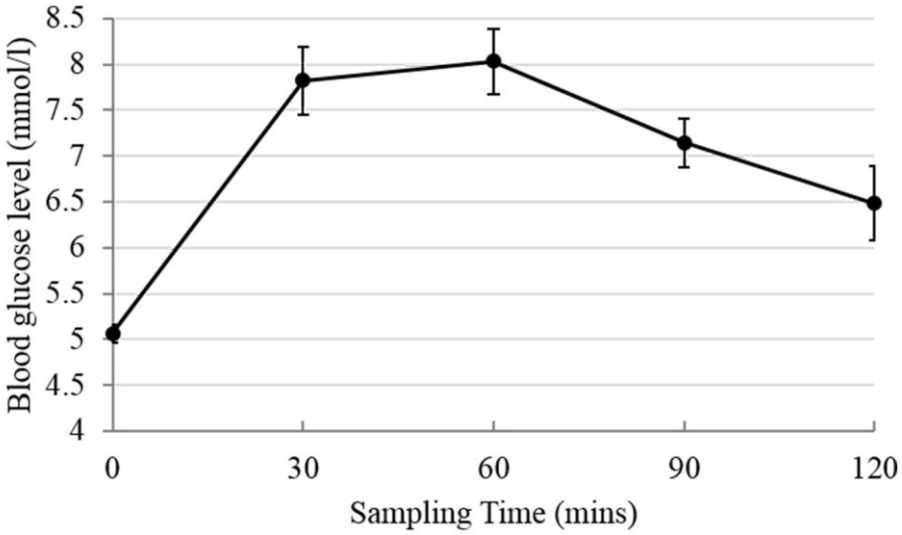
Blood glucose level (mmol/l)
8.5
8
7.5
7
6.5
6
5.5
5
4.5
4
0
30
60
90
120
Sampling Time (mins)

Figure 1

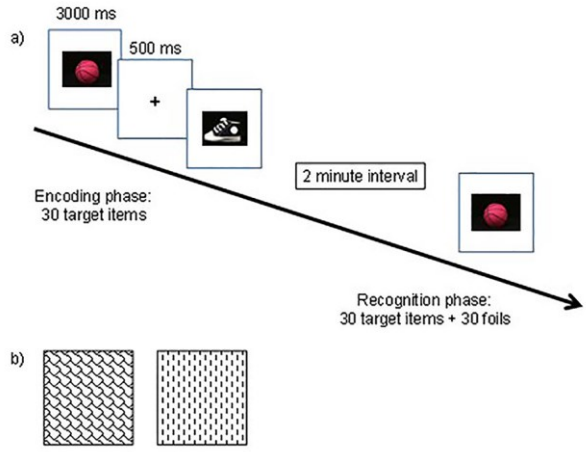
3000 ms
a)
500 ms
+
2 minute interval
Encoding phase:
30 target items
Recognition phase:
30 target items + 30 foils
b)

Figure 2

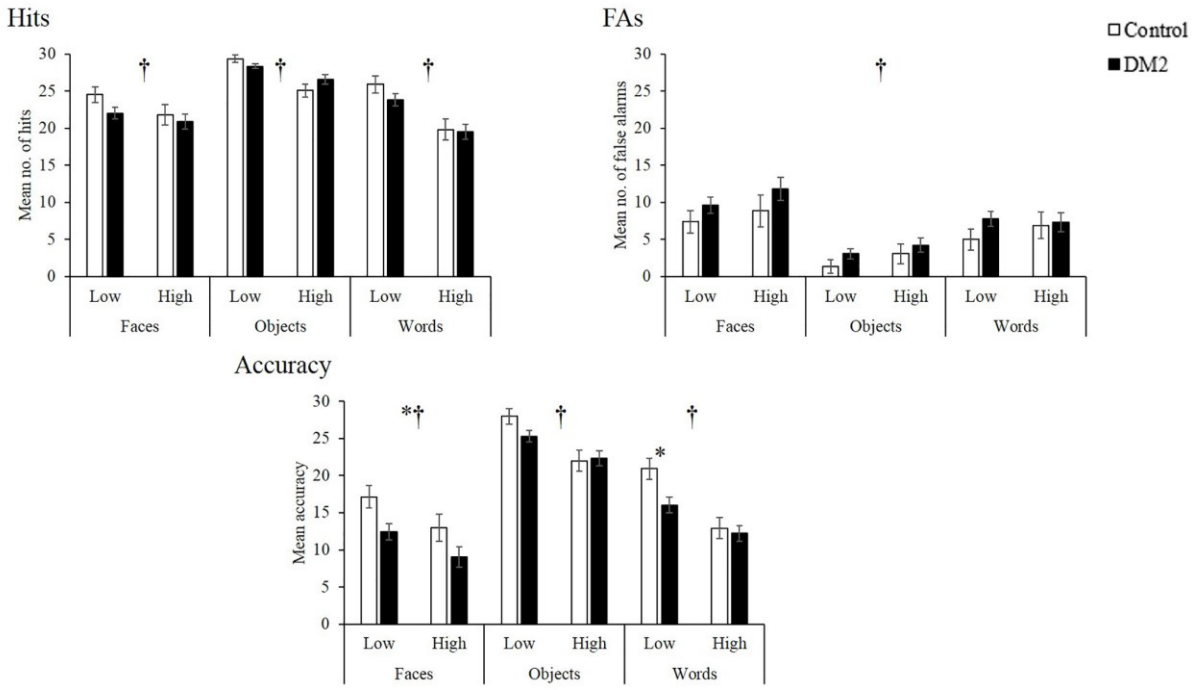
Hits
Mean no. of hits
30
25
20
15
10
5
0
†
†
†
Low
High
Low
High
Low
High
Faces
Objects
Words
FAs
Mean no. of false alarms
30
25
20
15
10
5
0
†
Low
High
Low
High
Low
High
Faces
Objects
Words
Control
DM2
Accuracy
Mean accuracy
30
25
20
15
10
5
0
*†
†
†
*
Low
High
Low
High
Low
High
Faces
Objects
Words

Figure 3

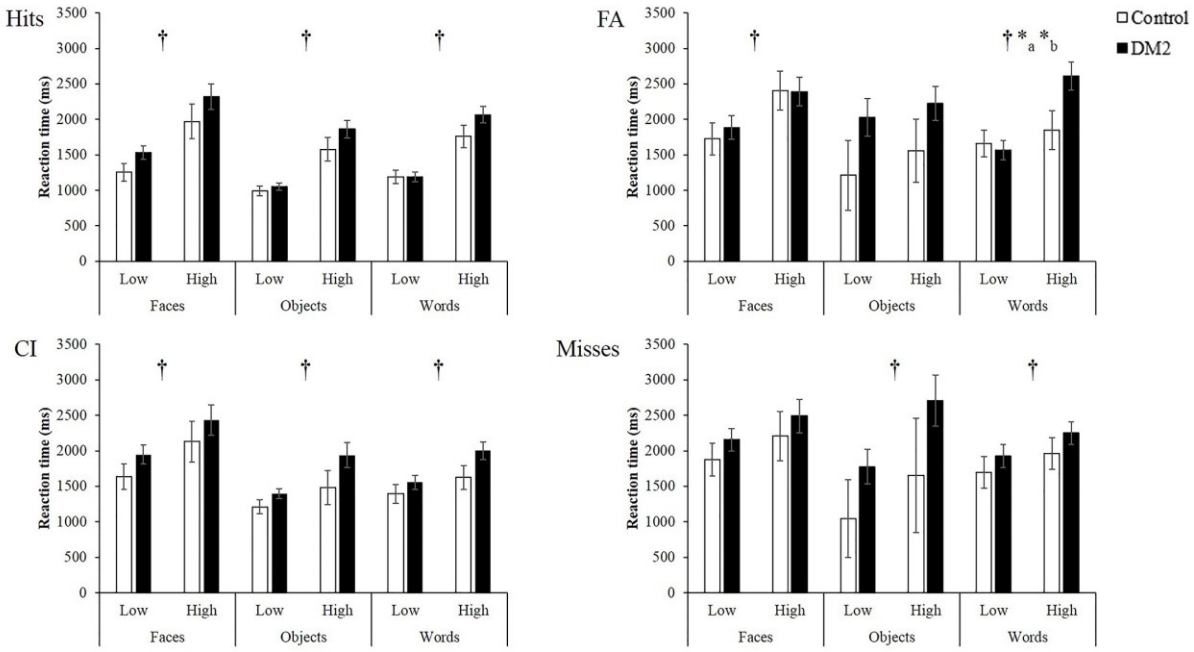
Hits
FA
CI
Misses
Control
DM2
Reaction time (ms)
0
500
1000
1500
2000
2500
3000
3500
Low
High
Faces
Objects
Words
†
*a
*b

Figure 4