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The role of nondeclarative memory in the skill for language: Evidence from syntactic priming in patients with amnesia

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ABSTRACT

Syntactic priming, the phenomenon in which participants adopt the linguistic behaviour of their partner, is widely used in psycholinguistics to investigate syntactic operations. Although the phenomenon of syntactic priming is well documented, the memory system that supports the retention of this syntactic information long enough to influence future utterances, is not as widely investigated. We aim to shed light on this issue by assessing patients with Korsakoff's amnesia on an active-passive syntactic priming task and compare their performance to controls matched in age, education, and premorbid intelligence. Patients with Korsakoff's syndrome display deficits in all subdomains of declarative memory, yet their nondeclarative memory remains intact, making them an ideal patient group to determine which memory system supports syntactic priming. In line with the hypothesis that syntactic priming relies on nondeclarative memory, the patient group shows strong priming tendencies (12.6% passive structure repetition). Our healthy control group did not show a priming tendency, presumably due to cognitive interference between declarative and nondeclarative memory. We discuss the results in relation to amnesia, aging, and compensatory mechanisms.

1. Introduction

The human language system is often characterized by a tripartite architecture (Jackendoff, 2002) that enables us to map sound onto meaning (in listening) or meaning onto sound (in speaking). Next to sound and meaning, there is syntax, which enables the well-formed grouping of words into longer utterances. At a very general level, for all three information types (sound, syntax, meaning), one can make a distinction between two crucial components. The one relates to the common assumption that the basic building blocks of linguistic knowledge get encoded and consolidated in the course of language acquisition. This is what we refer to as the *Memory* component of the human language system, and is more usually called the mental lexicon in the field of psycholinguistics. Crucially, however, language processing is more than the retrieval of lexical knowledge and goes beyond the simple concatenation of retrieved lexical items. The expressive power of human language derives from the possibility to combine elements from memory in often novel ways. This creative aspect led Wilhelm Von Humboldt (1829) to characterize language as a system which *"makes infinite use of finite means"*. This process of deriving new and complex meaning from the lexical building blocks is referred to by some as *Unification* (Hagoort, 2005, 2013, 2016). This process supports the on-line assembly of lexical building blocks into larger structures, with contributions from context and general world knowledge. It instantiates what in linguistic theories is often called the compositionality of language. Although the mental lexicon is part of semantic memory, and hence a component of declarative memory (Hagoort, 2005; Ullman, 2001), it is less clear which memory structure supports the on-line assembly of utterances that are not prestored in the mental lexicon. It has been argued (Ullman, 2001) that the on-line composition (speaking) or decomposition (listening/reading) of sound, morphological, and syntactic structures is subserved by procedural memory (Gupta and Cohen, 2002). Here we investigate a group of patients with severe amnesia that might provide relevant information on the contribution of procedural memory to human language skills, more in particular to the Unification component of the language system.

A core process in language production and comprehension is the production and comprehension of the syntactic relations between the

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lexical items in an utterance; i.e., the processing of the relationships between words in a sentence. The same words can be combined, but in different syntactic roles (e.g., subject, object), to produce different meanings (*the man kisses the woman/the woman kisses the man*) or different words fulfilling the same syntactic roles can be combined to produce the same meaning (*the man kisses the woman/the woman is kissed by the man*). Without a functioning syntactic processing system, the ability to understand language as well as to produce it is severely impaired. As language production and comprehension are so tightly linked, interlocutors are prone to repeat the syntactic structure in which their partner formulates her utterance. Indeed, corpus studies have shown that interlocutors adapt their syntactic language behaviour to match that of their partner (Giles and Powesland, 1975).

In 1982, Levelt and Kelter were the first to experimentally reproduce this repetition of syntax; they showed that the question "*On which instrument does Paul play?*" was more commonly answered (89%) with "*On the piano*" as opposed to "*The piano*" by the 36 participants they tested. The language adaptation behaviour, referred to in this article as syntactic *priming,* but also known as *accommodation* or *alignment,* has been used in a wide-range of applications. Syntactic priming studies have shown that abstract linguistic structures have a basis in psychological reality (Bock, 1986), how these are acquired during language development (Kidd, 2012), and which role syntactic priming plays in social cueing (Balcetis and Dale, 2005). However, the memory system that is needed to retain this linguistic information long enough to be used in producing utterances has not been seriously investigated.

Most studies that have examined the retention of linguistic information over time did not distinguish between different memory types. However, studies that investigated the effect of intervening irrelevant linguistic information or just time itself (Branigan et al., 2000), using either spoken (Bock and Griffin, 2000) or written modalities (Bernolet et al., 2016; Hartsuiker et al., 2008), did not observe a significant decrease in priming ability. Although the primed structures may remain active over some intervening trials, the length of the decay (sometimes even a week; Kaschak et al., 2011b) does not rule out that the participant may have consciously learnt the relevant linguistic structures. This points towards the involvement of declarative memory, the memory that underlies the acquisition, representation, and use of knowledge about facts and events.

At the same time, other studies have suggested that priming might be a form of statistical learning, a subcomponent of nondeclarative memory: participants automatically and unconsciously pick up on the frequency of event occurrences, which could explain why they produce these events with increasing probability over the length of the experimental session (Bock and Griffin, 2000; Jaeger and Snider, 2013; Kaschak et al., 2011a). Indeed a detailed computational model has been developed which supports these claims (Chang, 2002; Chang et al., 2006, 2000). One critical note is that empirial demonstrations of priming studies suffer from the problem of possible declarative contamination (Light, 1991). That is, in healthy participants it is difficult to rule out the possibility that priming effects may be mediated by declarative memory processes as well.

The most direct method to ensure that there is no influence of the declarative memory system is to measure participants that have amnesia. Until now, only one study has used this approach: Ferreira et al. (2008) had patients with declarative memory deficits complete a syntactic priming task (a task that focuses only on grammatical adaptation in language behaviour) and compared their performance to age- and IQ-matched controls. Their results showed that patients' ability to repeat syntactic structures did not differ significantly from the control group, even though their declarative memory performance was significantly worse compared to the controls. This led the authors to conclude that syntactic priming does not require declarative memory. However, this is only a single study, which examined only four patients with a mixed aetiology. The mixed aetiology could potentially be a confound, as both the declarative and nondeclarative memory systems have extensive neural networks, and thus lesions in different areas may not effect the four patients to the same extent.

The declarative memory system is mainly based in the diencephalon and the medial temporal lobe (MTL) structures. These include the hippocampus proper, the entorhinal cortex, the perirhinal cortex and the parahippocampal cortex (Squire and Dede, 2015; Squire and Knowlton, 2000; Suzuki and Eichenbaum, 2000). The hippocampus projects to the midline diencephalic nuclei, including the mammillary bodies and portions of the thalamus (Kopelman, 2014), although there is increasing evidence that the involvement of the hippocampus is not limited to the declarative memory system (Hannula and Greene, 2012; Schapiro et al., 2016). This diencephalic-MTL circuitry is involved in several memory related functions, including encoding, consolidation, and retrieval of new memories (Eichenbaum and Cohen, 2001; Squire and Knowlton, 2000), although memories eventually become mostly independent of the medial temporal lobe structures and dependent upon neocortical regions, particularly the temporal lobes (Hodges and Patterson, 1997; Squire et al., 2001). For language, memory for items stored in the mental lexicon has usually been related to inferior, middle, and superior temporal lobe regions (Hagoort, 2013, 2014; Hagoort and Indefrey, 2014; Ullman, 2001).

The nondeclarative (procedural) memory system is composed of an extensive neural network with the root in the frontal-striatal circuits and branching out to include portions of the parietal cortex, superior temporal cortex, and the cerebellum (De Renzi, 1989; Schacter and Tulving, 1994; Squire and Dede, 2015). The input to the basal ganglia (including the striatum) depend upon the type of information involved; for example motor learning might be projected from the supplementary motor area (SMA) and pre-supplementary motor area (Middleton and Strick, 2000), whereas syntax-related combinatorial operations (i.e., syntactic unification) could be projected from areas such as Broca's region (Conway and Christiansen, 2001). The information is then processed in the basal ganglia and projected back to prefrontal cortex, closing the loop. As the network is so extensive, it is imperative to ensure that whatever the cause of the patient's declarative memory deficit, their nondeclarative memory is not affected.

Korsakoff's syndrome is a neurological disorder caused by a chronic deficiency of thiamine (vitamin B_1) due to severe malnutrition usually associated with chronic alcoholism. Patients display profound amnesia due to bilateral lesions to the thalamus and mammillary bodies (Pitel et al., 2014) which, as mentioned above, are structures relevant for the encoding and consolidation of new memories via the declarative memory system. Patients therefore display deficits in all subdomains of declarative memory, but nondeclarative memory remains intact (Cermak et al., 1991; Oudman et al., 2011), making them an ideal patient group to include in this study.

In this study we aim to shed light on which memory system underlies syntactic priming. To control for any influence of the declarative memory system, we will be comparing the performance of amnesia patients with age-, education-, and premorbid intelligence-matched controls in a syntactic priming task. Overall, if syntactic priming is supported by nondeclarative memory, the amnesia patients should show robust priming effects.

2. Materials and methods

2.1. Participants

2.1.1. Patients with amnesia

Eighteen patients with Korsakoff's syndrome (13 men) were recruited from the Centre of Excellence for Korsakoff and Alcohol-Related Cognitive Disorders of Vincent van Gogh Institute of Psychiatry in Venray, The Netherlands. For all patients, the current intelligence level of each participant had to be in concordance with the estimation of premorbid functioning based on occupational and educational history, to exclude possible alcohol-related dementia (Oslin et al., 1998). All

patients fulfilled the DSM-5 criteria for Alcohol-Induced Major Neurocognitive Disorder, Amnestic Confabulatory Type (DSM-5 American Psychiatric Association, 2013), and the criteria for Korsakoff's syndrome described by Kopelman (2002). The diagnosis was supported by extensive neuropsychological testing. All patients were in the chronic, amnestic stage of the syndrome. None of the patients were in the confusional Wernicke psychosis at the moment of testing. No brain abnormalities were found that are at odds with the diagnosis of Korsakoff's syndrome (i.e., stroke, tumour, etc.). Patients had an extensive history of alcoholism and nutritional depletion, notably thiamine deficiency, verified through medical charts or family reports.

All testing occurred after the patients had been abstinent from alcohol for at least six weeks. The study was approved by the Vincent van Gogh Institutional Review Board (*Commissie Wetenschappelijk Onderzoek Participatie* U14.012). All the patients were informed about the study by the clinical staff and asked whether they were willing to participate; if so, written informed consent was obtained.

2.1.2. Controls

Eighteen healthy participants (8 men) were recruited from the Max Planck Institute for Psycholinguistics database and tested at the institute. These participants were matched with the patients in age, education level, and verbal IQ (see Table 1). No control participants reported any neurological deficits or psychiatric disorders and none had been treated for addiction. At the time of testing, none of the patients were taking any psychoactive medication. The study was approved by the ethics commission of the Faculty of Social Sciences at Radboud University, Nijmegen (Ethics Approval # ECG2013-1308-120).

2.2. Materials

All participants completed a syntactic priming experiment and were also tested on their declarative and nondeclarative memory ability. For the Korsakoff's patients, the implicit and explicit memory test scores were obtained as part of the routine neuropsychological assessment. The healthy controls completed the syntactic priming task, implicit memory task, and explicit memory task (in that order) in one session of approximately 90 min. Syntactic priming data for all participants (patients and controls) were collected by the same experimenter (E.H).

2.2.1. Implicit memory test

For healthy controls, it is impossible to measure pure nondeclarative memory without possible declarative memory contamination, as outlined previously. Therefore, this test was mainly executed to ensure that the *patients* still had nondeclarative learning ability.

To test nondeclarative memory using an implicit memory test, we used the Fragmented Pictures Test (Kessels et al., 2011). Participants are shown a set of 7 line drawings, in a sequence of 8 pictures of decreasing degradation. Each picture in the sequence was presented for 3 s. The participant is instructed to name the picture, to answer only if s/he is quite sure and not to guess. For each line drawing sequence, the sequence number is recorded at which the participant correctly identified the picture. There are 3 consecutive runs of this task and a fourth run after a delay of 10 min. The participant's performance reflects their average sequence number out of the 8 pictures, for each trial type (3 learning trials and one delay trial).

Table 1
Descriptive statistics of the two participant groups.

	Controls	Amnesia patients	*p* value
Age (mean(SD))	62.0 (6.73)	62.2 (8.0)	.919
Education level (mode(range))	5 (2)	4 (6)	.077
NART-IQ (mean(SD))	99.3 (20.78)	95.50 (20.1)	.451

Education level was measured using seven categories in accordance with the Dutch educational system (1 = less than primary school; 7 = academic degree; Verhage, 1964); premorbid intelligence (IQ) was measured using the Dutch version of the National Adult Reading Test (NART; Schmand et al., 1992). There were no significant differences between groups (Mann-Whitney *U* test, $p > .077$)

2.2.2. Explicit memory test

To test declarative memory, we used the Rivermead Behavioural Memory Test - Third Edition (RBMT-3; Wester et al., 2013; Wilson et al., 2008). This extensive test battery consists of a range of everyday memory types (face recognition, picture recognition, story recall, prospective memory route recall, etc.). The participant's overall performance (Global Memory Index; GMI) is a summary of their scores at each subtest, corrected for age.

2.2.3. Syntactic priming test

To test syntactic priming ability, we presented 80 prime-target picture pairs.

2.2.3.1. Stimulus pictures. The pictures used in this task have been described elsewhere (Segaert et al., 2011). The stimulus pictures depicted 40 transitive events such as *kissing, helping,* or *strangling* with a depiction of the agent and patient of this action. Each event was depicted with two pairs of adults and two pairs of children. One male and one female actor were shown in each picture, and each event was depicted with each of the two actors serving as the agent. To prevent the forming of strategies, the position of the agent (left or right) was randomized. Studies have suggested that lexical repetition (a boost in priming magnitude seen when verbs or nouns are repeated in consecutively presented stimuli) is based on declarative memory (Hartsuiker et al., 2008; Kidd, 2012). Thus, to ensure that the control group did not have an advantage over the patient group, no verb or actor type (adult/children) was consecutively repeated. Studies have shown that priming still occurs without this lexical repetition (Bernolet et al., 2016; Branigan et al., 1999; Hartsuiker et al., 2008).

Each transitive picture had three versions: one grayscale version and two colour-coded versions with a green and a red actor (which elicited sentences with either an active or passive transitive). Fillers elicited intransitive sentences, depicting events such as *running, singing, bowing* with one actor (in greyscale or green).

2.2.3.2. Experimental design. Participants were instructed to describe pictures with one sentence, naming the green actor before the red actor if the actors are depicted in colour. This allowed us to manipulate whether the prime sentence produced had an active or a passive syntactic structure. Fig. 1 depicts the order of events for the syntactic priming task. If the actors were not depicted in colour, the participants could describe the photo however they wished, producing voluntarily either an active or passive sentence. To ensure the patients did not forget the instructions, they were written at the top of the screen for each picture. Additionally, the verb that the picture is depicting was written at the bottom of the screen.

Each trial consisted of a prime (a coloured picture) followed by a target (a greyscale picture). There were 20 passive prime trials (a passive picture followed by a transitive greyscale target), 20 active prime trials (an active picture followed by a transitive greyscale target), and 20 baseline trials (an intransitive picture followed by a transitive greyscale target), all randomized in one experimental session. This resulted in 80 transitive pictures and 20 intransitive pictures. The baseline trials allowed us to measure the frequency of producing active and passive transitives on subsequent targets without any immediate prior influence. All pictures were presented until the participant responded. Filler trials were also included (20% of all trials, consisting of an intransitive prime followed by an intransitive target). This brings the total up to 60 intransitive pictures and 100 transitive pictures.

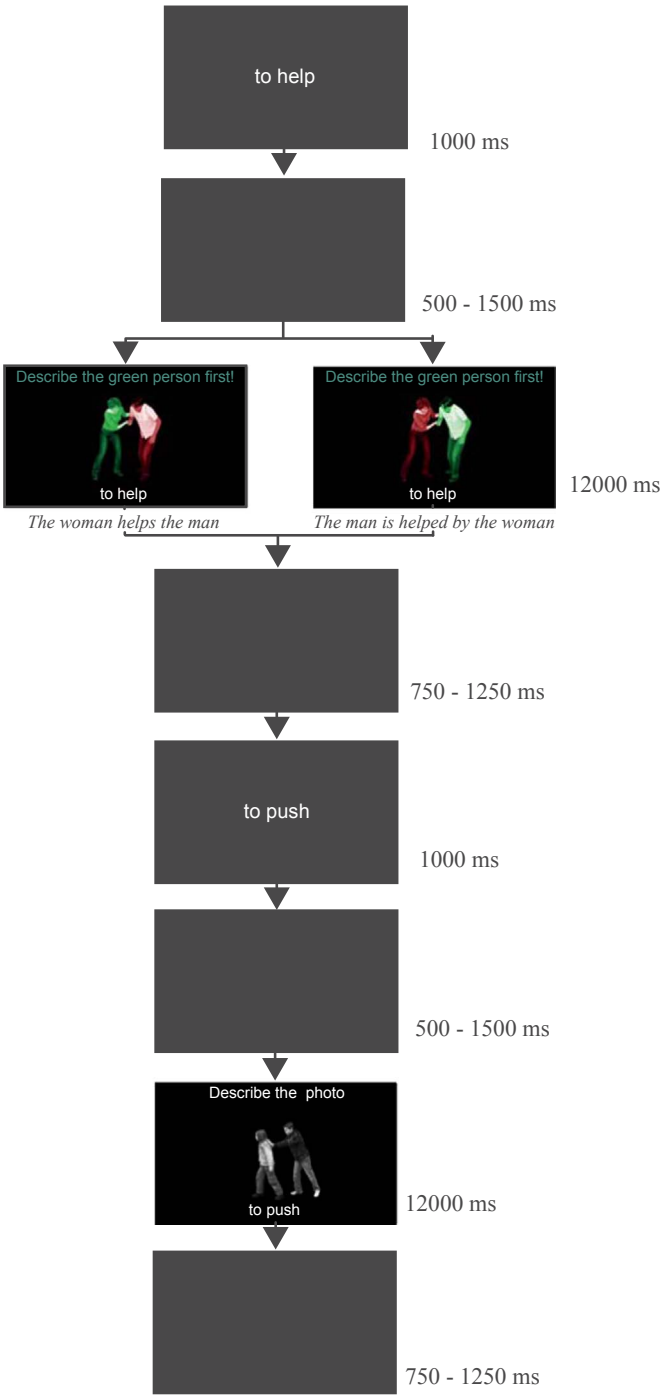

Fig. 1. Order of events for the syntactic priming task. Pictures are presented until a response is produced.

2.3. Coding and analysis

Responses during the syntactic priming task were manually coded by the experimenter as either active (0) or passive (1). An independent rater blind to the purpose of the experiment verified that the coding was performed correctly. Trials in which the descriptions did not match one of the coded structures were discarded. Target responses were included in the analysis only if 1) both actors and the verb were named (a sentence naming only one of the actors does not qualify as a transitive sentence) and 2) the structures used were active or passive. In total 127 trials (9.34%) in the patient group were discarded; 144 trials (8.38%) in the control group were discarded. One patient had over 30% unusable trials and was discarded entirely from the data set; that patient's age-, education-, and IQ-matched control was also discarded to maintain an equal number in each group.

The responses were analysed using a mixed-effects logit model, using the glmer function of the lme4 package (version 1.1.−4; Bates et al., 2012) in R (R Core Development Team, 2011). Target responses were coded as 0 for actives and 1 for passives in factor *Prime*. We used a maximal random-effects structure (Barr et al., 2013; Jaeger, 2009): the repeated-measures nature of the data was modelled by including a per-participant and per-item random adjustment to the fixed intercept ("random intercept"). We began with a full model and then performed a step-wise "best-path" reduction procedure, removing interactions be-

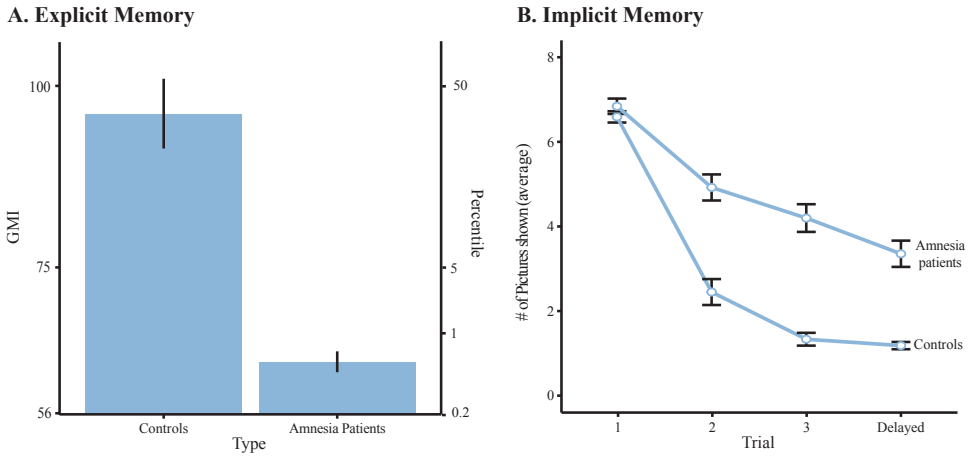

Fig. 2. Results of Memory Tests. Only 14 (out of 17) amnesic patients agreed to complete these tests. A. Explicit Memory: Controls showed significantly higher explicit memory performance compared to the amnesia group on the RBMT-3 ($p < .001$). B. Implicit Memory: Amnesia patients showed a significant learning trend on the Fragmented Pictures Test, indicating that their nondeclarative memory ability is still intact. Error bars represent standard errors of the mean.

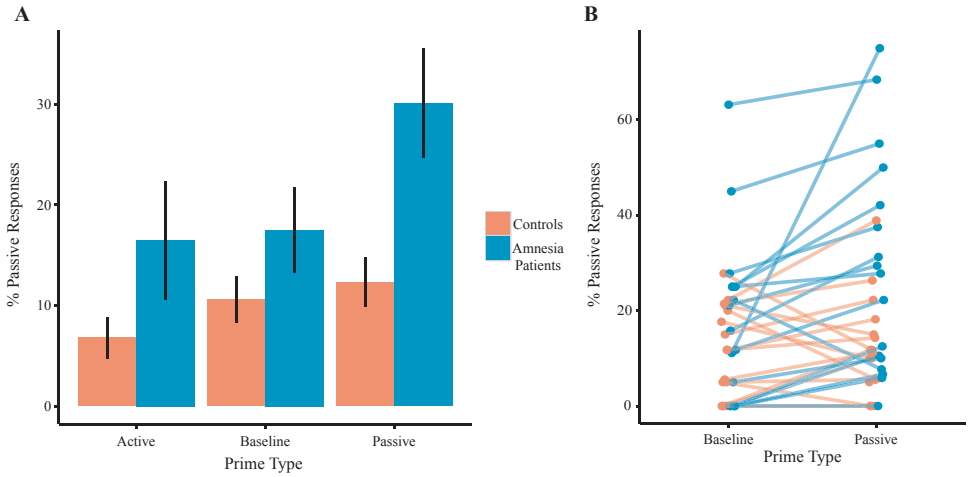

Fig. 3. Percentage of passive responses per prime per group. Following a passive prime, the production of a passive sentence increases with 12.6% for the amnesia group and 1.7% for the control group compared to the baseline condition. In line with previous research, there were no priming effects for actives. Panel A shows the average of both groups (error bars represent standard error), whereas panel B plots the individual performances for the baseline and passive prime trials.

Table 2
Summary of the best mixed logit model for passive vs. active response choices.

Results for the Control Group					
Predictor	coefficient	*SE*	*Wald Z*	*p*	
Intercept (baseline)	−2.84	.35	−8.07	< .001	***
Active Prime	−.59	.55	−1.07	> .250	
Passive Prime	.41	.42	.97	> .250	
Cum. Passive Prop	8.17	1.27	6.41	< .001	***
Results for the Amnesia Group					
Predictor	coefficient	*SE*	*Wald Z*	*p*	
Intercept (baseline)	−2.20	.26	−8.30	< .001	***
Active Prime	−.23	.42	−.55	> .250	
Passive Prime	.90	.34	2.64	.008	**
Cum. Passive Prop	8.35	.78	10.68	< .001	***

Note: N = 927, log-likelihood = −246.9.

Note: N = 909, log-likelihood = −324.0.

fore main effects, to locate the simplest model that did not differ significantly from the full model in terms of variance explained. Factorial predictors were dummy coded (all means compared to a reference group) and all numeric predictors were centred. We included a factor *Cumulative Passive Proportion* to reflect any learning trend exhibited by the participants. This factor was calculated as the proportion of passives out of the total transitive responses produced on the target trials before the current target trial.

We used intransitives as the reference group for *Prime*. Collinearity between factors was low (VIF < 1.37).

3. Results

Fig. 2 shows the results for the explicit and implicit memory tests. Controls showed a significantly higher explicit memory result ($M = 96.18$, $SD = 19.80$) compared to the amnesia group ($M = 62$, $SD = 5.38$, Mann-Whitney $U = 0$, $p < .001$), who performed in the impaired range (in the 20th percentile).

In the amnesia group, a significant learning curve was present for the Fragmented Picture Task performance[1] (Friedman $\chi^2(3) = 39.686$; $p < .001$), with an increase in performance on trial 3 compared to trial 1 (Friedman $\chi^2(3) = -3.298$; $p < .001$), and an increase in performance between trial 3 and the delayed trial (Friedman $\chi^2(3) = -3.236$; $p < .001$) indicating that the patients retained information between the trials, even with a 10-min delay. As the amnesia patients performed

[1] Note that only 14 out of 17 amnesia patients gave consent to conduct the implicit memory task.

within the impaired range on their explicit memory test, presumably their performance in the implicit memory task relies mostly on the nondeclarative memory system.

The controls performed significantly better than the patients ($F(1192) = 174.57$, $p < .001$) on the implicit memory task as they were also able to use their declarative memory to enhance their performance.

Fig. 3 summarizes the relative proportion of passive target responses after each prime structure. The fixed effects of the model fit for these data are summarized in Table 2.

The negative estimate for the intercept indicates that in the baseline condition active responses were more frequent than passive responses. For both groups there was no increase in active responses following active primes, compared to baseline ($p = .283$), indicating there was no increase in the number of actives produced after an active prime.

Significantly more passives were produced compared to baseline ($p = .008$) by the amnesia patients, indicating that despite their declarative memory deficits, they were still able to retain syntactic information. This is also reflected in their *Cumulative Passive Proportion*, which was calculated as the proportion of passives out of the total transitive responses produced on the target trials before the current target trial. Any passives produced during prime trials are not included in this calculation. A positive and significant *Cumulative Passive Proportion* therefore suggests that the proportion of passives previously produced positively influences the probability of producing a passive on the current target trial. In other words, there is a cumulative effect of syntactic priming (i.e., the more passives produced, the stronger the effect), supporting a statistical learning effect of priming and also reflects any delayed priming influence, as opposed to the baseline condition which reflects immediate prior influence. As the patients have an impaired declarative memory system, this ability is most likely supported by their nondeclarative memory ability.

Somewhat unexpectedly, the control group did not show a significant priming effect. They demonstrated a significant learning trend, as reflected in *Cumulative Passive Proportion*, suggesting they produced more passives throughout the length of the experimental sessions, but not enough to produce higher than a 1.7% priming effect.

We included *Education Level* as a factor in the full model, as the difference between the groups was nearly significant ($p = .077$). Including this factor did not make the fit of the model significantly better ($p > .290$), and therefore this was not included in the best model reported above.

4. Discussion

Our results support the theory that syntactic priming is based on nondeclarative memory. We examined 17 patients with amnesia due to Korsakoff's syndrome in a syntactic priming experiment. Memory tests supported the claim that these patients did have a severely impaired declarative memory system, yet a functional nondeclarative one. Fully in line with predictions, the Korsakoff's patients showed a strong passive priming tendency, providing unequivocal support that syntactic priming is a nondeclarative memory process. Somewhat unexpectedly, however, our healthy control group did not show a significant priming effect.

Our results are at odds with an early study also investigating which memory process supports syntactic priming. Ferreira and colleagues (2008) tested four amnesia patients with a mixed aetiliogy with four age-, education-, and IQ-matched healthy controls. They found a significant priming effect not only for their amnesia group, but also for their control group. Although the ages of their participants are younger than ours (*M:* 50.875 vs. 62.09 years), a discussion we will address below, another major difference between our study and that of Ferreira are the syntactic structures used (dative vs. transitive). Research has suggested that priming effects for transitives are generally weaker and more fragile than priming effects for datives (Bock and Griffin, 2000) even though the characteristics of the priming effects are comparable (Bernolet et al., 2016). This may be one explanation as to why our results differ in terms of the control group, although other potential explanations are addressed below. Overall, however, the discrepancy in the results between our study and that of Ferreira and colleagues again illustrates the importance of replication in the psychological sciences.

In this study we used a production-production design, in which the participant's description of the colour coded pictures would act as his or her own prime when describing the grey pictures. However, other methods exist to test syntactic priming ability. These designs include listening to the prime being described by either a recording or a confederate (comprehension design; Bock and Griffin, 2000; Menenti et al., 2011), or having the participant read the prime sentence and then write out the target sentence (Branigan et al., 1999; Hartsuiker et al., 2008), or any combination of the above. As all of these have shown robust syntactic priming effects, it suggests that the underlying mechanism should be independent of the modality used, and therefore we are confident that our results are applicable to other modalities of priming as well and are not unique to the production-production methodology.

Of course, if the underlying mechanism is independent of the modality used, then investigating which brain regions are involved in all modality types should help elucidate the core of syntactic operations, and thereby which memory type(s) supports it. A neuroimaging study by Segaert et al. (2012) did just that: they compared the brain areas involved when syntactic priming was measured in a sentence production task with measurements in a sentence comprehension task. They demonstrated that in both cases adaptation effects were found in the left inferior frontal gyrus (IFG), left middle temporal area (MTG), and bilateral supplementary motor area (SMA). These three areas are known to be involved in language processing, in particular in the unification of language information (IFG; Hagoort, 2003, 2005; Snijders et al., 2009), in the process of sequencing syllable structures (SMA; Segaert et al., 2012), and in the retrieval of lexical-syntactic information from memory (MTG; Snijders et al., 2009), respectively. This latter process is thought to refer to the retrieval of syntactic frame information (Vosse and Kempen, 2000). From these previous studies however, no clear conclusions could be drawn about which memory system underlies syntactic priming.

In the current study we extended these findings by using a lesion model. By examining patients with a deficit in a specific cognitive system, in this case declarative memory, we can determine whether that system is involved in the behaviour of interest. In our study we show that amnesia patients with declarative memory deficits are still able to show robust syntactic priming ability, further supporting the claim that syntactic priming is supported by nondeclarative memory. Our results pertain to syntactic processing. This result does not suggest that all language processes require nondeclarative memory. For instance, patients with primary progressive aphasia have a strong deficit in single-word comprehension (Mesulam et al., 2015), due to cortical atrophy of the left anterior temporal lobe. This area is part of the declarative knowledge base for lexical items. In contrast, patients with Parkinson's disease are impaired in producing correct inflections when these are regular and hence rule-based (Ullman, 2001; Ullman et al., 1997). The degeneration of the basal ganglia in these patients affects the nondeclarative memory system. These patients show a deficit in procedural aspects of word formation, but not in retrieval of lexical information from memory. The Korsakoff's amnesia patients in our study show, on the other hand, a preservation of implicit knowledge relevant for syntactic encoding, that is, the formation of grammatically well-formed sentences, while at the same time suffering from serious impairments in declarative memory. In all, this is a strong indication that language processing recruits multiple memory systems. Within the language domain, syntactic processing is a way to solve the problem of serial order in speaking (Lashley, 1951); that is, to put the lexical items retrieved from declarative memory (the mental lexicon) in a specific

word order. In general terms, nondeclarative memory is known to be involved in sequencing and timing (Dehaene et al., 2015; Nemeth et al., 2011). This might explain why nondeclarative memory is centrally involved in syntactic skills.

In remarkable contrast to the patients and to the younger healthy participants (Segaert et al., 2011, 2016), the age- and education-matched controls failed to show a syntactic priming effect.[2]The finding that an older control sample exhibits less priming compared to the cognitively impaired patients is not a new observation: in a study testing patients with Broca's aphasia, the patient group showed stronger syntactic priming while the healthy age-matched controls showed no significant priming effect for transitives or datives (Hartsuiker and Kolk, 1998b).

The one consistent element between the Hartsuiker and Kolk (1998) and our study that sets them apart from other syntactic priming studies is the age of the participants. So far, most syntactic priming studies are limited to using the undergraduate population: students around 20 years of age. As patients with general amnesia, Korsakoff's syndrome, or Broca's aphasia are on average older and also may have an average or below-average education, most patient studies use non-academic older healthy controls. Therefore, the somewhat unexpected lack of a priming effect seen in the control group could be due to age. As syntactic priming has been observed in children as young as 3 years of age (Branigan and Messenger, 2016; Messenger et al., 2011), the accumulating evidence that older, healthy participants do not show a priming effect (Hartsuiker and Kolk, 1998a; Sung, 2015, 2016) clearly indicates that an adaptation to syntactic priming models to include these lifespan differences is necessary. An age-related decline in syntactic priming, as observed in this study, will help to further elucidate the memory system related to syntactic processing. For example, a recent studies have suggested that statistical nondeclarative learning (modelled to support syntactic processing; Chang et al., 2006) is prone to age-related decline (Neger et al., 2014), whereas perceptual nondeclarative memory is not (Fleischman et al., 2004). However, as our current study was not designed to investigate these age effects, we will also discuss two other possible explanations for the lack of a syntactic priming effect in the control group compared to the patient group.

A first possible explanation is the competitive nature of declarative and nondeclarative memory systems (Krupa, 2009; Rieckmann and Bäckman, 2009). It has been found that these two memory systems are not strictly independent, but also interact with each other (Poldrack and Packard, 2003). This has been highlighted by recent studies showing that the hippocampus is not exclusively involved in the declarative memory system, as previously assumed, but is involved in aspects of nondeclarative memory as well, such as statistical (e.g., Hannula and Greene, 2012; Schapiro et al., 2016) and relational/conceptual learning (e.g., Chun and Phelps, 1999). Additionally, in the case of impairments in one system, the other might play a compensatory role (Ullman and Pullman, 2015). Indeed, animal studies have shown that the lesioning of one of the memory systems can result in an enhanced task performance relative to brain intact animals (Poldrack and Packard, 2003). This results in the intriguing possibility that in the healthy, aging population the nondeclarative memory contribution suffers from interference of the declarative memory system. Studies have suggested that certain aspects of priming such as lexical overlap (which we controlled for in this study) or the use of strategies may be supported by declarative memory (Bernolet et al., 2016; Bock and Griffin, 2000; Chang et al., 2006; Ferreira and Bock, 2006) and hence recruitment of these systems provides an opportunity for competition. The difference between the advantageous effect of using both memory systems as seen in the implicit memory task and the debilitating effect seen in the syntactic priming task could be purely a matter of task difficulty. Dysfunction of the declarative memory system, such as in the patient group, would prevent the use of explicit strategies and hence removes the competition/interference between the two memory systems, which in our case surfaces as a syntactic priming effect. Therefore, a combination of increased competition between memory systems for the healthy controls and an enhanced performance for the patients results in the large difference in priming magnitude that we observed in our study.

An alternative explanation is based on the evidence from neuroimaging research, animal work, and patient studies that nondeclarative memory depends on a subcortical-cortical network with particularly strong involvement of the striatum (Knopman and Nissen, 1994; Packard, 2009; Willingham and Preuss, 1995) and the hippocampus (Schapiro et al., 2016). As one ages, the putamen and caudate shrink by 5–10% (Raz et al., 2003) and dopamine in the striatum decreases as well (up to 10% per decade; Bäckman et al., 2006). The volume of the hippocampus, shown to play an important role in statistical learning tasks (Schapiro et al., 2014) also decreases with age (Rieckmann and Bäckman, 2009). As the striatum is central in maintaining implicit information, the ability to maintain implicit information may also degrade. As mentioned earlier, studies that have looked at the effect of aging on syntactic priming have suggested that, indeed, as we age our ability to prime decreases (Sung, 2015, 2016). Secondly, as we age the speed with which information is processed decreases (Howard et al., 1986; Salthouse, 1996). Consequently, the chance that the information has decayed before it is retrieved is increased. In terms of syntactic priming, this could mean that the information is not retained long enough to be incorporated in future utterances. Indeed, one study has shown an increase in priming after administering dopamine (via administration of levodopa) to healthy participants (Angwin et al., 2004).

The interesting question, however, is why do the amnesia patients in these studies not show a decrease in priming effect? One explanation may be that Korsakoff's patients have an increased 5-HT (a serotonin precursor) in the striatum (Langlais et al., 1987), which facilitates dopamine production (Navailles and De Deurwaerdère, 2011; Zhou et al., 2005). As the Angwin et al. (2004) study suggests, this increase in dopamine production may offer the Korsakoff's patients better priming ability relative to their age-matched healthy peers.

In all, our results show unequivocally that syntactic priming is supported by nondeclarative memory. Language processing, therefore, seems to rely not only on neocortically consolidated declarative memory, but also engages nondeclarative memory structures, such as frontostriatal circuits, to engage in combinatorial processing, at least at the level of syntactic operations. To what degree reduced nondeclarative memory contributions can be compensated by support from declarative memory remains to be seen.

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[2] To verify the absence of a syntactic priming effect in the controls, we ran an independent group of 54 subjects (M_{Age} =67.54 years) with the exact same paradigm. We replicated earlier findings: there was no priming effect for passives (p= > .250) or actives (p= > .250).

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