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Original Article

Nightmare sufferers show atypical emotional semantic associations and prolonged REM sleep-dependent emotional priming

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ABSTRACT

Study Objectives: The objective of this study was to investigate whether nightmare (NM) sufferers exhibit an abnormal network of emotional semantic associations as measured by a recently developed, rapid eye movement (REM) sleep-sensitive, associational breadth (AB) task.
Design: NM sufferers were compared to healthy controls (CTL) for their performance on an emotional AB task containing positive and negative cue words both before and after a nap with REM sleep. AB was assessed in both a priming condition, where cue words were explicitly memorized before sleep, and a non-priming condition, where cue words were not memorized. Performance was assessed again 1 week later.
Setting: The study was conducted in a sleep laboratory with polysomnographic recording at the Hôpital du Sacré-Coeur de Montréal
Participants: Twenty-eight participants between the ages of 18 and 35 years ($M_{age} = 23.3 \pm 3.4$) were included in the study.
Measurements and Results: The NM group scored higher than the CTL group on both positive and negative AB, with group differences persisting at the 1-week retest. However, the two groups did not differ as expected in the AB priming effect following REM sleep. Both groups showed decreased REM sleep-related AB priming for negative cue words and increased AB priming for positive cue words. However, the NM group maintained these effects 1 week later, whereas the CTL group did not.
Conclusions: NM sufferers may access broader than normal emotional semantic networks in the wake state, a difference that may lead to this group being perceived as more creative. The fact that the AB priming effect is maintained at the 1-week retest for NM sufferers suggests that the presence of frequent NMs may alter REM sleep-dependent emotional processes over time.
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1. Introduction

1.1. Nightmares, sleep, and emotional cognition

Nightmares (NMs) are powerful unpleasant dreams associated with feelings of threat, anxiety, fear, or other negative emotions that are clearly recalled upon awakening and that arise primarily during late-night rapid eye movement (REM) sleep [1]. Individuals suffering from frequent idiopathic NMs may have disturbed sleep patterns, both during the NM experience itself and potentially in its absence [2–6]. For example, during an ongoing NM, participants may experience increased heart rate (HR), eye movements, and shortened breath [3,7]; REM periods not marked by NMs have also shown altered structure, including increased REM latency, or increased high alpha spectral power (10–14.5 Hz) [8,9]. Such disturbances may be disruptive to emotional regulation mechanisms provided by sleep.

REM sleep in particular is theorized to regulate emotion via a reduction in amygdala activation, while simultaneously improving the cognitive processing of emotion through increased functional medial prefrontal cortex (mPFC) connectivity [10]. Accordingly, sleep loss leads to increased emotional reactivity as measured both physiologically and behaviorally, as well as to decreased

Abbreviations: NM, Nightmare; AB, Associational Breadth; RT, Reaction Time.

The research was conducted at the Dream & Nightmare Laboratory, Center for Advanced Research in Sleep Medicine, Hôpital du Sacré-Coeur de Montréal.

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outward expression and experience of emotion by sleep-deprived individuals [11,12]. This pattern mirrors clinical findings that patients with frequent NMs suffer from both hyperarousal and alexithymia, a deficit in cognitive labeling of emotion [13]. Further, NM sufferers are prone to affective disorders, including anxiety and depression, implicating a relationship between NMs and dysfunctional emotion processing [14]. NM sufferers also show increased cognitive perseveration in a word fluency task and perform poorly in an emotional Stroop task [4,15]. Thus, frequent NMs may contribute to deficits in both the affective and cognitive domains, although more research is warranted to assess emotional cognition specifically.

1.2. REM sleep associative integration of emotional memory

REM-dependent emotional memory consolidation in the NM population has been largely unexplored. One generally supported theory is that REM sleep functions to adaptively integrate emotional experiences into the vast autobiographical network. The unique neurophysiological state of REM sleep allows the activation of emotional memory traces within an environment of increased associative cortical connections, thus promoting emotional memory integration [16–20]. Substantial research supports the role of REM sleep in emotional memory consolidation, such as in the consolidation of fear and safety memories or of the negative component of complex pictures [2,18]. Further, behavioral experiments support an associative function for REM sleep, showing increased associative capacity immediately upon awakening from REM sleep and improved performance on associative tasks that had been primed before a REM sleep nap [21–23]. Both of these findings support claims that REM sleep enables enhanced access to associative semantic content. Notwithstanding such findings – and theoretical speculations on an emotional integration function of REM sleep – the interaction between emotional memory and associative access has not been studied in depth.

Our recent study of healthy college students specifically assessed the effects of REM sleep on the associative integration of emotional semantic stimuli, as measured by an associational breadth (AB) task [24]. Following a morning nap, participants were asked to provide word associations to emotional cue words they had memorized before sleeping. Participants who had REM sleep during their nap gave less common word associations (scored relative to word associate norms) [25] than did participants who had only non-rapid eye movement (NREM) sleep during their nap or who stayed awake. This finding suggests that the emotional words studied before sleeping were consolidated within a broad semantic network exclusively during REM sleep. Further, this priming effect was particularly strong for positive, compared to negative, cue words. The positively valenced cue words were considered to stimulate an increased spread of activation in REM sleep, similar to an effect observed in waking-state priming studies. Specifically, induction of a positive mood facilitates spread of activation [26], improves success with Remote Associates Test solutions [27–29], and increases the uniqueness and diversity of word associations [30]. Further, positive stimuli promote faster speed of semantic access [31]. Thus, positive cue words in our study may have led to increased breadth and speed of spreading activation in REM sleep.

These findings support the validity of the emotional AB task as being sensitive to both REM sleep and the emotional valence of experimental stimuli. Thus, it is an appropriate task for assessing REM sleep-related emotional processes among frequent NM sufferers. However, the available literature points to two distinct hypotheses about whether NM sufferers are expected to show a *restricted* or an *expanded* breadth of emotional semantic associations.

1.3. Restricted AB in the NM sufferer

The most important symptom of the NM sufferer, the NM experience itself, is composed of rather repetitive and perseverative content, suggesting that NM psychopathology is characterized by restricted emotional semantic access. The NM experience has vivid sensory imagery and intense emotional expression. The NM often depicts an unrelenting threat, whether being pursued by an aggressor, an out-of-control car, or an imminent tidal wave. The NM's main theme and imagery seem to grow more potent and imposing over time, with an increase in emotional arousal, and a resistance to the associative fluidity that normally permeates dreams [32]. This characterization of NMs as being associationally restricted is in line with findings in the literature on waking cognition, which shows that positive emotion increases associative access while negative emotion restricts and slows it [33,34]. Thus, the NM may reflect a temporary failure of REM processes to integrate a dysphoric emotional memory. Given this possibility, we would expect the presence of NMs to correlate with reduced AB and longer associational reaction times (RTs) on the AB task and a blunting of REM sleep-dependent emotional priming.

1.4. Expanded AB in the NM sufferer

A second possible explanation of NM pathology is that frequent NM sufferers are characterized by a broader than normal access to emotional semantic networks. NM sufferers report higher than average recall of positively toned dreams, e.g., non-NM dreams and other "intensified" dreams [35,36] such as lucid dreams that contain self-reflective awareness and subtle kinesthetic imagery, and archetypal dreams that contain blissful emotion and, often, spiritual encounters. Even in the waking state, NM participants report more bizarreness in their daydreams – bizarreness has been likened to broad semantic access, as in the AB task [22,24]. NM sufferers are also characterized by "thin boundaries," a personality construct that includes creativity and artistic expression, both of which seem to draw more flexibly and frequently upon unusual associations [32]. Together, such findings support the expectation that NM sufferers may demonstrate an expansion of associational processing relative to control subjects.

In sum, while research supports the role of REM sleep in the associative integration of emotional memory, any potential effect of frequent NMs on this process is unknown. On the one hand, the NM experience seems to reflect associative restriction imposed by intense negative emotion; on the other hand, NM sufferers often report positive and bizarre dreams, and they are frequently characterized as being creative, artistic individuals. Assessment of their performance on an emotional AB task both before and after REM sleep will provide further insight into waking and REM sleep-dependent processes.

1.5. Objectives and hypotheses

To determine whether the presence of persistent NMs is associated with either restriction or broadening of access to emotional semantic networks, we used a nap protocol and a recently developed task for assessing semantic AB in response to negative and positive emotional words [24]. Our objective was to assess baseline and REM sleep-dependent changes in emotional word associations among frequent NM sufferers and controls (CTL). We also assessed whether group differences were maintained at a 1-week follow-up.

Hypotheses: Current evidence is scarce, although it tends to suggest that NM sufferers will show restricted associative access, particularly in response to negative cue words, on an emotional AB task both without and with REM-sleep dependent priming: 1) lower

AB scores, particularly for negative cue words; 2) slower RTs for associational responses; 3) lower scores on the REM sleep-dependent Priming Effect, particularly for negative cue words; and 4) slower RTs for Priming Effect responses.

All effects are expected to be maintained in NM sufferers after a 1-week delay.

2. Materials and methods

2.1. Participants

Twenty-eight participants (20 female) between the ages of 18 and 35 years ($M_{age} = 23.3 \pm 3.43$) were recruited for a nap study through advertisements and posters. CTL participants reported recalling <1 NM per month for the past 5 years, whereas NM participants reported recalling at least two NMs per week for the past 6 months. Potential participants underwent a telephone screening questionnaire. Exclusion criteria included self-reported sleep disorders other than NM disorder; neurological, psychological, or other chronic illnesses; addictions; use of certain medications; or other conditions that interfere with sleep. Participants completed an informed consent form that was approved by the ethics committee of the Hôpital du Sacré-Coeur de Montréal.

2.2. Procedures

Participants arrived at 8 am, filled out informed consent forms, and completed a series of questionnaires that took approximately 30 min (not reported here).

At 9 am, participants completed an AB task with two negative and two positive cue words, and studied an eight-item word list (see Fig. 1). They also completed a waking daydream procedure (daydream results are not reported here).

At 10 am, a sleep technician attached an electrode montage for polysomnography (PSG) and performed biocalibration. Subsequently, the participants were given a 2-h opportunity to nap. A technician trained in sleep stage scoring monitored each nap and awakened participants 10 min into REM sleep, provided a minimum of 50 min of total sleep time (TST) had elapsed.

Upon awakening, participants were asked to recall their sleep-related cognitions ("Please recall what was going through your mind prior to the *beep*") and to complete a 14-item questionnaire about the content of that experience (not reported here). The electrode montage was then removed.

Approximately 1 h post awakening, participants again completed an AB task, which now contained cue words familiar to them from the previous memorization task (Primed), along with novel cue words (NonPrimed), which were used to calculate a relative Priming Effect (Primed minus NonPrimed AB scores). Participants were then free to leave.

Participants returned to the laboratory one week later to complete the AB task again with novel cue words for comparison with their initial AB performance; they also completed a second Primed AB task for comparison with performance on the first Primed AB task.

2.3. Polysomnography

Participants slept in bedrooms with continuous audiovisual surveillance and a two-way intercom. They were recorded with an electrode montage of six standard 10–20 electroencephalographic (EEG) channels (F3, F4, C3, C4, O1, and O2) referenced to A1, four electrooculographic (EOG) (vertical and horizontal channels), four electromyographic (EMG) channels (chin and corrugator), and three electrocardiographic (EKG) channels. Biosignals were recorded using Grass M12 and Grass M15 Neurodata Acquisition Systems (–6-dB filters with cutoffs at 0.30 and 100 Hz) and archived under the control of Harmonie 5.4 software (Natus Medical Inc., Montreal, QC, Canada). The PSG tracings were scored according to current American Academy of Sleep Medicine (AASM) standards [37] by an experienced PSG technician, and standard sleep variables (REM min, %REM, NREM min, %NREM, and TST) were calculated by in-house software.

2.4. AB task (emotional version)

The AB task is designed to assess levels of, and changes in, semantic associational activation, that is, the extent to which a cue word leads to more remote associations in a semantic network. The emotional version of the task was previously found to be REM sleep dependent; thus, it was chosen for the current protocol. The task is scored in comparison to empirically determined norms for the typicality of associations given by participants in response to common emotion cue words [25,38].

On each administration of the AB task, two positive and two negative cue words, each randomly selected without replacement from a set of 16 emotion cue words, were presented. Participants were required to respond with the first three words that came to mind as being meaningfully associated with the presented cue word. Participants were given a maximum of 30 s to respond; a countdown clock on the screen displayed the time remaining to type their responses. The presentation software developed in-house with Inquisit [39] recorded all words typed by participants and recorded the total time taken by them to enter all three responses. RT was defined as the time in milliseconds between the start of the countdown clock to the third press of the Enter key (corresponding to submission of the third word association).

Participant responses were later scored by an experimenter using an empirically determined table of the three most common word associates given for each cue word [25]. Any participant response that was not an established common associate for the cue word was

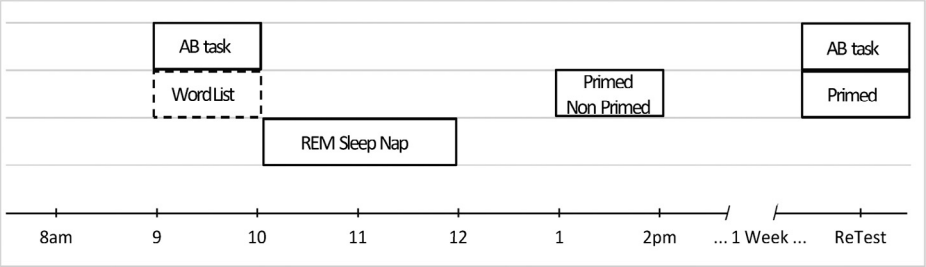

Fig. 1. Participants arrived at 8 am and completed the informed consent form. At 9 am, they completed an AB task and studied a word list. Between 10 am and 12 pm, they took a PSG-recorded nap and were awakened 10 min into REM sleep. One hour post awakening, they completed an AB task consisting of cue words they had studied before sleeping (Primed), along with novel cue words (NonPrimed). They returned 1 week later for a retest on the AB task and the Primed AB task.

considered to be atypical and given a score of 1. Accordingly, an AB score from 0 to 3 could be assigned to each cue word. AB scores for the two negative cue words of each task were then summed and converted to percentages, as were the scores for the two positive cue words.

2.4.1. Priming effect

Before sleeping, the participants were instructed to memorize a word list of four negative and four positive cue words, each randomly selected without replacement from the set of 16 cue words. These eight words were presented in randomized order, at 4 s/word, for three sequential presentations. The Primed AB tasks consisted of cue words drawn from this word list, whereas the NonPrimed task consisted of four completely new cue words drawn from the same set of 16.

AB scores for the NonPrimed and Primed tasks were first calculated as described previously to produce percentages. Subsequently, a Priming Effect was calculated by subtracting the NonPrimed from Primed scores for negative and positive cue words separately. Thus, positive scores indicate that the Primed cue words produced more uncommon associates than did the NonPrimed cue words; negative scores indicate the opposite.

2.4.2. Reaction time

For all AB tasks, RT measures were recorded as the total time taken for participants to give three responses to each cue word. An average time was calculated separately for the two negative cue words and two positive cue words of each AB task.

For the Priming Effect, differences in RT (Priming Effect RT) were calculated as the difference between NonPrimed and Primed response times (Priming Effect RT = Primed RT – NonPrimed RT).

2.5. Statistics

2.5.1. Hypothesis 1: AB

A group (NM, CTL) × cue type (negative, positive) × time point (Test and ReTest) analysis of variance (ANOVA) tested the hypothesis that NM sufferers would have lower AB scores than CTLs, particularly for negative cue words, which would be maintained over time. Specific *t*-test comparisons were done to examine between- and within-group differences.

2.5.2. Hypothesis 2: Priming Effect

A two-group (NM and CTL) × two-cue-type (negative and positive) × time-point (Test, ReTest) ANOVA with Priming Effect as the dependent measure tested the hypothesis that NM sufferers would have lower Priming Effects than CTLs, particularly for negative cue words; *t*-test comparisons were used to assess specific between- and within-group differences.

2.5.3. Hypothesis 3: AB RT

A group (NM, CTL) × cue type (negative, positive) × time point (Test, ReTest) ANOVA tested the hypothesis that NM sufferers would have slower RT overall, particularly for negative cue words, which would be consistent over time. Specific *t*-test comparisons were performed to determine between- and within-group differences.

2.5.4. Hypothesis 4: Priming Effect RT

A group (NM, CTL) × cue type (negative, positive) × time point (Test, ReTest) ANOVA with Priming Effect RT as the dependent measure tested the hypothesis that NM sufferers would have slower Priming Effect RT scores than CTLs, particularly for negative cue words, which would be consistent over time; *t*-test comparisons were used to assess specific between- and within-group differences.

Table 1
Sleep stage measures for naps of Nightmare and Control groups.

	NM	CTL	*t*	*p*
TST	82.11 ± 21.31	88.20 ± 27.86	–0.61	0.55
Sleep Efficiency	91.35 ± 7.99	90.09 ± 12.30	0.31	0.76
NR1 (min)	13.75 ± 6.38	14.60 ± 11.06	–0.24	0.81
NR2 (min)	38.36 ± 18.14	37.10 ± 20.56	0.16	0.88
NR3 (min)	13.14 ± 16.03	16.10 ± 12.08	–0.49	0.63
NREM (min)	65.25 ± 22.24	67.80 ± 27.62	–0.25	0.80
REM (min)	16.86 ± 8.28	20.40 ± 8.10	–1.04	0.31
NR1 (%)	18.14 ± 10.15	18.01 ± 15.16	0.02	0.98
NR2 (%)	46.53 ± 12.82	40.28 ± 12.53	1.19	0.25
NR3 (%)	14.13 ± 16.67	16.56 ± 12.48	–0.39	0.70
NREM (%)	78.81 ± 8.35	74.86 ± 13.87	0.87	0.39
REM (%)	21.19 ± 8.35	25.14 ± 13.87	–0.87	0.39
REM Efficiency	83.01 ± 17.76	95.84 ± 4.86	–2.21	0.04*
NREM in REM (min)	3.46 ± 4.33	1.05 ± 1.52	1.68	0.11
Wake in REM (min)	1.29 ± 4.24	0.15 ± 0.34	0.84	0.41
REM period (min)	21.61 ± 12.24	21.60 ± 9.36	0.00	1.00
# REM fragments	3.86 ± 3.08	2.70 ± 1.57	1.09	0.29
Sleep Latency (min)	10.54 ± 6.92	12.40 ± 12.56	–0.47	0.64
NR1 latency (min)	10.75 ± 6.22	11.35 ± 11.30	–0.17	0.87
NR2 latency (min)	19.50 ± 7.36	18.65 ± 15.58	0.18	0.86
NR3 latency (min)	41.82 ± 19.68	31.14 ± 15.06	1.22	0.24
REM latency (min)	40.39 ± 36.30	39.50 ± 24.15	0.07	0.95

Values presented as mean ± standard deviation. * Values differ; $p < 0.05$. TST, total sleep time; NREM, non-rapid eye movement; NR1, NREM stage 1; NR2, NREM stage 2; NR3, NREM stage 3; REM, rapid eye movement.

3. Results

3.1. Sleep structure

Participants slept for a target of 80 min of TST and were awakened 10 min into REM sleep. Four CTL participants were excluded for not sleeping or for waking up well before the target time had elapsed. The groups did not differ in the minutes of NREM sleep ($p = 0.80$), minutes of REM ($p = 0.31$), or TST ($p = 0.55$). However, the NM group had significantly lower REM efficiency ($t(22) = -2.21$, $p = 0.04$). Table 1 presents the means.

3.2. Associational breadth

Three CTL participants who did not sleep well were excluded from the sleep-dependent analyses (Priming Effect), but they were included in the AB analyses. One CTL participant did not sleep well and did not return for the 1-week follow-up, and was thus excluded from all analyses. Thus, a total of 13 CTL participants and 14 NM participants were included in the AB analyses, and 10 CTL and 14 NM participants in the Priming Effect analyses. Table 2 presents the means for all task types and groups.

Table 2
Scores of the associational breadth task for NM and CTL groups on all task types.

		NM	CTL
Associational Breadth	N	14	13
Test	Negative	0.86 ± 0.17	0.69 ± 0.21
	Positive	0.78 ± 0.18	0.63 ± 0.18
ReTest	Negative	0.86 ± 0.14	0.69 ± 0.21
	Positive	0.80 ± 0.17	0.58 ± 0.16
Priming Effect Tasks	N	14	10
Primed Test	Negative	0.77 ± 0.18	0.52 ± 0.27
	Positive	0.80 ± 0.18	0.67 ± 0.21
Primed ReTest	Negative	0.85 ± 0.15	0.63 ± 0.22
	Positive	0.83 ± 0.21	0.57 ± 0.14
NonPrimed	Negative	0.89 ± 0.15	0.65 ± 0.18
	Positive	0.76 ± 0.17	0.57 ± 0.16

Values presented as mean ± standard deviation.

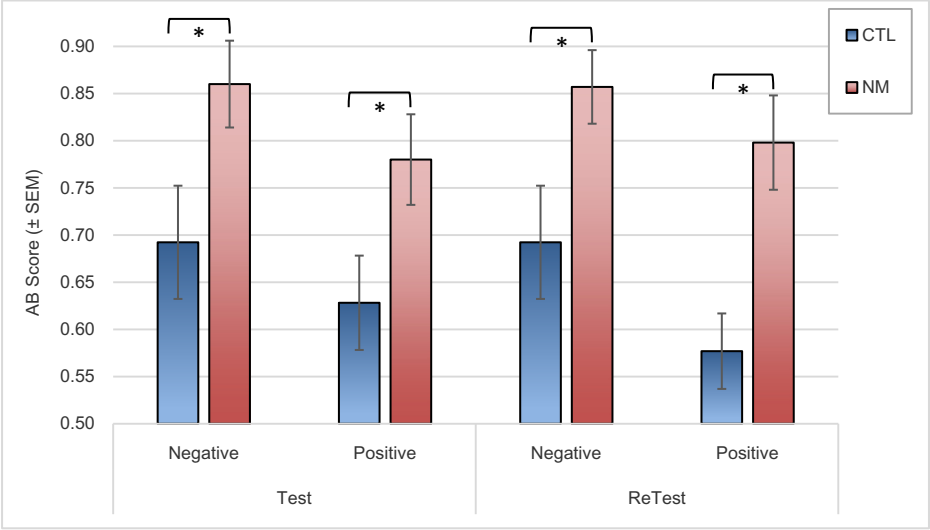

Fig. 2. Mean (±SEM) associational breadth (AB) score for both groups. The NM group had significantly higher scores for both negative and positive cue words, at both Test and ReTest. *Values differed significantly at $p < 0.05$.

A group (CTL, NM) × cue type (Negative, Positive) × time point (Test, ReTest) ANOVA for AB did not reveal a three-way interaction effect ($F(1, 25) = 0.35$, $p = 0.56$). A significant effect was noted for group ($F(1,25) = 17.91$, $p = 0.0003$), showing that the NM group had significantly higher AB scores at both Test and ReTest. There was also a significant effect for cue type ($F(1,25) = 10.06$, $p = 0.004$), indicating that negative AB scores were higher than positive AB scores. There was no main effect for time point, nor any other two-way interactions (all $p > 0.64$).

Post hoc independent samples t-test comparisons showed that the NM group had higher scores than the CTL group on both negative ($t(25) = 2.22$, $p = 0.04$) and positive ($t(25) = 2.09$, $p < 0.05$) AB, as well as higher scores than the CTL group on both negative ($t = 2.37$, $p = 0.03$) and positive AB at ReTest ($t(25) = 3.27$, $p = 0.003$; see Table 2 for means and Fig. 2 for comparisons). Within-group dependent t-tests did not reveal any significant differences between variables in the CTL group (all $p \geq 0.10$) or the NM group (all $p > 0.20$).

3.3. Priming effect

A group (CTL, NM) × cue type (Negative, Positive) × time point (Test, ReTest) ANOVA for the Priming Effect revealed a marginal three-way interaction effect ($F(1,22) = 2.68$, $p = 0.12$), a significant main effect for cue type ($F(1,22) = 8.04$, $p < 0.01$), with positive cue words producing higher AB scores than negative cue words, and a significant two-way interaction between cue type and time point ($F(1,22) = 5.21$, $p = 0.03$; see Fig. 3). These effects were clarified by post hoc within-group dependent samples t-tests which revealed that, for the CTL group, the positive Priming Effect was significantly higher than the negative Priming Effect at Test ($t(18) = 3.10$, $p = 0.01$), but equal at ReTest ($t(18) = 0.14$, $p = 0.89$). Further, the positive Priming Effect was significantly higher at Test than at ReTest ($t(18) = 2.32$, $p = 0.30$), and the negative Priming Effect tended to be lower at Test than at ReTest ($t(18) = -1.79$, $p = 0.09$). For the NM group, the positive Priming Effect was also higher than the nega-

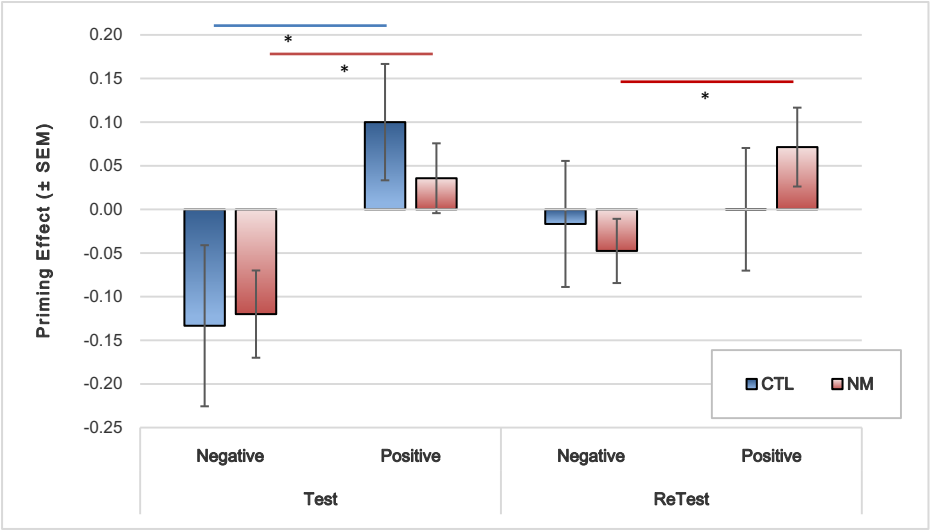

Fig. 3. Mean (±SEM) Priming Effects for positive and negative cue words. Both groups showed a significant Priming Effect for positive compared to negative cue types at Test; however, only the NM group maintained a significant Priming Effect at ReTest. * Values differ at $p < 0.05$.

Table 3
Reaction times (in seconds) on the associational breadth task for NM and CTL groups on all task types.

		NM	CTL
Associational Breadth Reaction Time	N	14	12
Test	Negative	13.82 ± 6.88	15.42 ± 8.60
	Positive	14.15 ± 7.79	13.18 ± 4.25
ReTest	Negative	14.30 ± 6.69	12.14 ± 3.29
	Positive	13.52 ± 5.42	13.89 ± 5.91
Priming Effect Reaction Time	*N*	14	9
Primed Test	Negative	16.67 ± 6.06	16.25 ± 6.11
	Positive	14.36 ± 6.86	14.25 ± 6.24
Primed ReTest	Negative	17.19 ± 6.27	13.09 ± 4.92
	Positive	15.96 ± 4.75	12.34 ± 4.69
NonPrimed	Negative	15.34 ± 5.46	11.62 ± 3.62
	Positive	14.85 ± 6.62	14.50 ± 6.10

Values presented as mean ± standard deviation.

tive Priming Effect at Test ($t(26) = 2.41$, $p = 0.03$), but in this group the effect was maintained at ReTest ($t(26) = 2.35$, $p = 0.04$). The positive and negative Priming Effects did not differ between Test and ReTest (both $p > 0.20$; see Fig. 3).

3.4. Associational breadth reaction time

RTs are presented in seconds as the average time taken by participants to type all three response words to negative and positive cue words. One CTL participant who allowed the time meter to run out for all task conditions (despite instructions to respond as quickly as possible) was excluded from the analyses. Table 3 displays the mean RTs for all task types and groups.

A group (CTL, NM) × cue type (Negative, Positive) × time point (Test, ReTest) ANOVA for RT did not reveal a significant three-way interaction effect ($F(1, 24) = 1.35 = 92$, $p = 0.18$). No other main effects or two-way interactions were significant (all $p > 0.54$). Post hoc *t*-tests revealed no between- or within-group differences (all $p > 0.22$).

3.5. Priming Effect RT

RTs for the Priming Effect were calculated as for the Priming Effect scores, that is, Primed RT – NonPrimed RT at both Test and ReTest. A group (CTL, NM) × cue type (Negative, Positive) × time point (Test, ReTest) ANOVA for Priming Effect RT did not reveal a significant three-way interaction effect ($F(1,21) = 0.07$, $p = 0.79$) and no significant effect for group ($F(1,21) = 0.34$, $p = 0.57$). However, it did reveal a significant main effect for cue type ($F(1,21) = 6.28$, $p = 0.02$), such that positive Priming Effect RTs were faster than negative Priming Effect RTs. Cue type interacted only marginally with group ($F(1,21) = 2.44$, $p = 0.13$); *t*-test comparisons showed this to be mainly due to the CTL group, which had faster RTs for positive than for negative cue types ($F(1,21) = 6.79$, $p = 0.02$), whereas the NM group had similar RTs for the two cue types ($F(1,21) = 0.57$, $p = 0.46$; Fig. 4).

There was also a trend for a main effect of time point ($F(1,21) = 3.28$, $p = 0.08$), with RTs decreasing from Test to ReTest. Again, although the group × time point interaction was not significant ($F(1,21) = 1.88$, $p = 0.18$), *t*-test comparisons revealed the trend to be due to the CTL group, which showed faster RTs at ReTest than at Test ($F(1,21) = 4.16$, $p = 0.05$), whereas the NM group showed no such improvement ($F(1,21) = 0.12$, $p = 0.73$).

4. Discussion

Although the provisional hypotheses that NM participants would demonstrate more restricted access to semantic emotional networks in the form of both lower AB task scores and longer RTs were not supported, the findings nonetheless reveal significant differences between NM and CTL groups. First, frequent NM sufferers were found to give more atypical responses on the AB task than did CTL participants. They had uniformly higher AB task scores, as indicated by higher scores on both the initial Test and the ReTest given 1 week later, and higher scores in response to both positive and negative cue words. That this difference was so ubiquitous suggests that

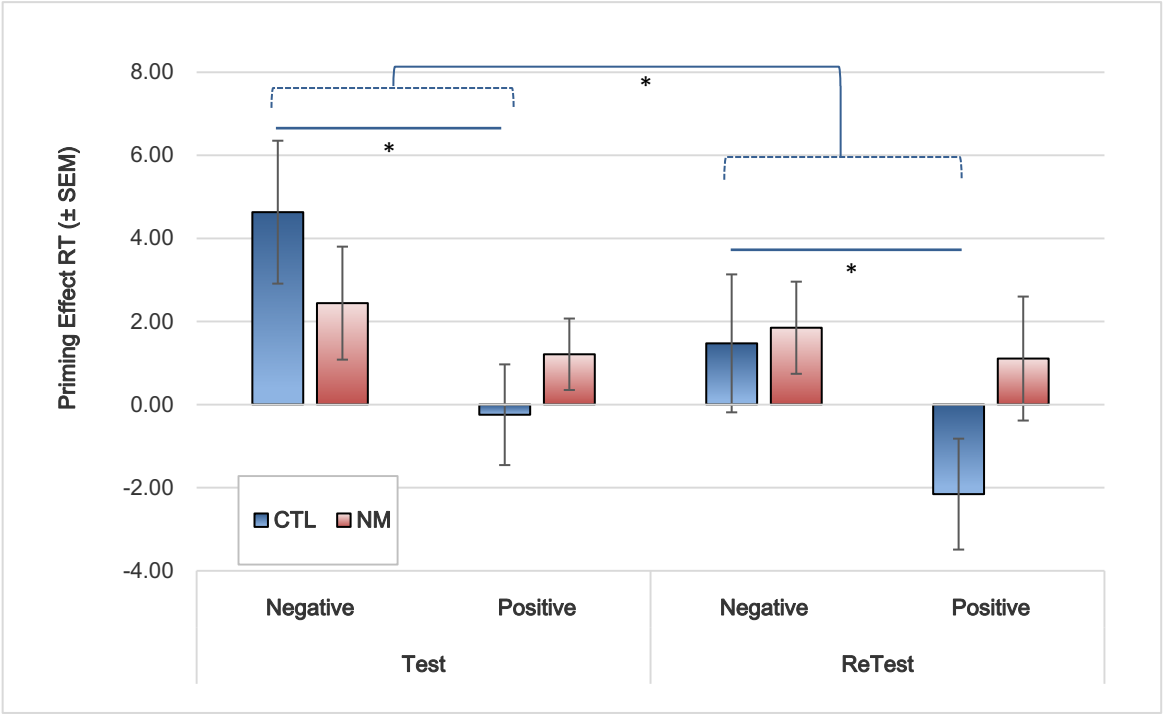

Fig. 4. Mean (±SD) Priming Effect RTs for positive and negative cue types. The CTL group showed significantly faster RTs for positive than for negative cue words at both Test and ReTest, with RTs at ReTest being significantly lower than those at Test. These differences were not seen for the NM group. *Values differed significantly at $p \leq 0.05$.

it may constitute a consistent trait associated with NM pathology. Further, the lack of differences between groups in RTs suggests that the NM participants' elevated associative breadth scores did not occur because they spent more time searching their associative nets for more remote associations; rather, the differences were likely due to automatic or habitual differences in associative access. These findings are in agreement with literature suggesting that NM participants benefit from fluid and broad associational abilities of the kind that promote artistic talent and creative thinking.

Second, NM participants showed immediate REM-dependent changes in AB that were similar to those of CTL participants, ie, relatively broad in response to positive cue words and restricted in response to negative cue words when tested immediately after their naps. This finding replicates and expands upon our previous finding of a selective positive Priming Effect for healthy college students after a morning nap [24]. However, at the 1-week ReTest, the NM group showed an enduring Priming Effect that disappeared in the CTL group. Concurrently, the NM group failed to show the improvement in RT at ReTest that was seen in the CTL group. It is possible that, for the CTL group, initial REM-dependent changes in associative access dissipated as the emotional cue words became either fully integrated into memory or fully stripped of their affective charge over time; one week later, the cue words benefited from strengthened consolidation and faster speed of access [40]. For the NM group, however, the initial REM-dependent changes in associative access did not dissipate, possibly because the cue words were not fully integrated into memory or their affective charge was not downscaled over the 1-week delay. In either case, the speed of accessing the words did not improve.

The differential effects of positive and negative emotions on associational access during waking state tasks are well established. For example, the induction of positive affect leads to the production of more broad semantic associations [30]; conversely, sadness leads to more constrained and specific item processing [41]. Our findings further validate these effects for priming stimuli and for processes linked to sleep. In particular, our priming task produced, as predicted, evidence of restricted AB in response to negative stimuli and expanded AB in response to positive stimuli. These consistent findings not only further validate the concept of affect-mediated semantic network access but also support our AB priming task as a valid measure of this differential effect.

However, our findings for the AB task without priming did not support an affect-mediated access effect; greater AB was noted in response to negative than to positive cue words in both the AB task before sleep and at the 1-week ReTest. This contrasts with the clear-cut differences in positive and negative stimuli in the AB priming task. The discrepancy may be due to a difference in processing modes for the baseline and priming tasks. Specifically, relatively automatic processing modes are mediated directly by emotional arousal, whereas controlled processing modes are mediated more by emotional valence [42]. Our baseline conditions may have triggered an automatic processing mode sensitive to arousal, as suggested by our findings that RTs did not differ across groups for the baseline AB tasks. In this case, an increased level of arousal, which presumably occurs for NM participants and in response to negative emotions, may covary with increased AB. Alternatively, the priming task may be considered a more controlled cognitive task sensitive to emotion valence, especially considering the effort required to memorize the priming word list. If so, in the priming condition stimulus valence may have exerted a differential (negative restricting, positive broadening) effect on AB. Further, primed RTs differed by valence, with faster responses occurring for positive than for negative cue words; this suggests that positive valence correlated with both increased breadth *and* speed of associative access.

That the priming effect for the CTL group dissipated over a 1-week interval supports existing claims that emotional memory integration is dependent on multiple sleep cycles. The "sleep to forget and sleep to remember" (SFSR) hypothesis suggests that over time REM sleep promotes strengthened consolidation of emotional memory episodes, while diminishing the emotional charge of such memories [40]. It is possible that the NM group is less efficient in this process, resulting in sustained Priming Effects one week later. This may be explained in several ways: heightened arousal at encoding may have led to a stronger and more resistant initial Priming Effect; lower REM efficiency could delay full integration; the experience of NMs during the 1-week interval could interfere with integration. In any case, such results imply that NM sufferers may suffer distress and altered emotional cognition for a longer period of time after an emotional event, such as a trauma or other adverse experience, a concept that corroborates existing findings of a correlation between a prior history of trauma or abuse and NM frequency [43,44].

In sum, findings support the hypothesis that NM sufferers have altered emotional cognition. During wakefulness, they display uncommon emotional associations, corresponding with anecdotal reports of heightened creativity and artistic expression. Following an initial REM nap, they show an emotion–word Priming Effect similar to that of CTLs, which was restricting for negative cue words and broadening for positive cue words. However, one week later, they continued to show this Priming Effect robustly, whereas the CTL group did not. This may mean that NM sufferers need more time to fully integrate emotional experiences into memory networks. Overall, the results supports the paradoxical notion of the NM sufferer as benefitting from an associational capacity that facilitates creative pursuits, while concurrently suffering from inadequate emotional memory integration and all the mental health consequences this may entail.

Conflict of interest

Michelle Carr, B.Sc., has no conflicts of interest to disclose and received a research grant from DSF/IASD.

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