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Mechanisms and Intervention Strategies for the Impact of Watching Different Types of Films Before Bed on Sleep Quality

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Abstract. When the narrative structure of a film is treated as the independent variable, the hippocampus primarily supports two core functions: memory tagging and the replay of tagged events. Variations in narrative structure determine the density of event boundaries, which in turn modulate memory consolidation and sleep architecture. This study investigates how pre-sleep film viewing affects memory and sleep outcomes, with consideration of evoked emotions, event boundary density, and clinical relevance. Results were derived from two participant groups who completed the Self-Assessment Manikin (SAM), episodic recall test, and Visual Analogue Scale (VAS), in addition to providing sleep data via wearable monitors. We found that high event boundary films (e.g., *Memento*) elicited higher emotional arousal but reduced sleep onset latency (SOL) and improved sleep efficiency (SE), whereas low event boundary films (e.g., *1917*) prolonged SOL due to sustained attentional load. These findings challenge the canonical view that "high cognitive load uniformly impairs sleep quality", and reveal a bifurcation in the temporal dynamics of cognitive load. Our results suggest that narrative structure acts as a modulator of hippocampo-cortical communication, with implications for subclinical sleep manifestations of emotional disorders and neurodegenerative diseases.

Keywords: Boundary density, sleep structure, cognitive load dynamics, memory, neurocinematic

1. Introduction

Data from the American Sleep Foundation in 2024 indicate that 59% of adults watch television or films before bed, establishing pre-sleep screen viewing as a near-universal behavior. Despite this prevalence, two critical research gaps remain unaddressed. First, while prior work has examined the effects of simple, artificial stimuli on sleep, "narrative boundary density" has rarely been investigated as an ecologically valid, naturalistic variable. Second, the relationship between narrative structure, healthy sleep, and the pathomechanisms of psychiatric disorders (e.g., anxiety, depression) and neurodegenerative conditions (e.g., Alzheimer's disease) remains poorly understood. To address these gaps, we tested three core hypotheses derived from the canonical cognitive load framework [1, 2]: (1) High event boundary density increases cognitive load, prolongs sleep onset

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latency (SOL), and reduces sleep efficiency (SE); (2) SAM arousal correlates positively with SOL, such that higher pre-sleep arousal delays sleep initiation; (3) High boundary density disrupts memory consolidation during slow-wave sleep (SWS) and impairs next-day recall performance.

To test these hypotheses, we selected two films as experimental stimuli: *Memento* (high event boundary density) and *1917* (low event boundary density). Participants wore wrist-worn actigraphy devices to monitor sleep parameters. Beyond hypothesis testing, this work advances theoretical understanding of how event segmentation modulates sleep and memory, offers clinical insights into sub-health and neurodegenerative conditions, and provides practical guidance for pedagogical scheduling and narrative editing practices [3].

2. Basic mechanisms

To contextualize our findings, we first outline the neurobiological mechanisms of memory consolidation following pre-sleep film viewing, which unfold across three sequential phases: wakeful encoding, SWS, and rapid eye movement (REM) sleep. Established work demonstrates that higher-order event boundaries couple with hippocampal tagging activity, which facilitates subsequent memory consolidation [4]. Notably, boundary density comprises both true and false boundaries; only true boundaries detected by the hippocampus amplify hippocampal salience and tagging responses [5]. Accordingly, viewing *Memento* (high boundary density) elicits more robust hippocampal tagging than viewing *1917* (low boundary density).

Following sleep onset, SWS serves as the first critical stage for memory consolidation, characterized by a nested triple-coupling architecture. Under the global modulation of slow oscillations, sleep spindles align hippocampal sharp-wave ripples (SWR) at their troughs, forming discrete "temporal windows" for memory transfer [6, 7]. Within these windows, SWR mediates the replay of compressed neural sequences encoded during wakefulness; selective disruption of SWR impairs the transfer of hippocampal representations to distributed neocortical circuits, undermining memory stabilization [8]. For films with high boundary density and emotional salience, REM sleep becomes essential: the locus coeruleus (LC) enters a quiescent state during REM, reducing norepinephrine (NE) activity and enabling emotion-memory decoupling—the process of stripping affective charge from episodic memories to form neutral event representations, termed emotional depotentiation [9].

3. Method

This pilot experiment was designed to test the relationship between film narrative boundary density, memory consolidation, and sleep quality. The independent variable comprised two levels: a between-subjects factor (narrative structure: *1917* vs. *Memento*) and a within-subjects factor (repeated assessments: Pittsburgh Sleep Quality Index [PSQI], SAM, recall test, VAS). Dependent variables included sleep metrics (SOL, SE, proportional SWS and REM duration) and memory performance (next-day episodic recall scores).

To minimize confounding, we standardized pre-experimental procedures: participants abstained from caffeine on test days, avoided daytime naps, and refrained from intense exercise within three hours of bedtime. Eight healthy adults were stratified into two groups based on baseline PSQI scores to ensure balanced sleep quality at baseline; group allocation differed only in the order of film viewing. Immediately after viewing, participants completed the SAM and VAS to assess subjective arousal and emotional valence. They then donned wrist-worn devices for sleep monitoring and immediately retired to bed. Recall assessments were administered at midday on the following day,

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and sleep data were extracted from the wearable monitors. A washout period of at least 72 hours separated the two film-viewing sessions to minimize carryover effects of memory traces from the first session on the second.

4. Results

This study systematically categorized results into a conventional set aligned with initial hypotheses and a special set of unexpected findings requiring mechanistic interpretation, to clarify how pre-sleep narrative exposure modulates cognitive consolidation and sleep architecture.

4.1. Conventional findings

Four core results supported our primary hypotheses. First, participants who viewed *Memento* achieved significantly higher scores on next-morning episodic recall tests than those who viewed *1917*, indicating enhanced memory retention for high boundary density narratives. Second, polysomnographic data revealed that the *Memento* group exhibited a markedly higher proportion of SWS than the *1917* group, reflecting differential engagement of deep sleep consolidation mechanisms. Third, subjective reports confirmed that most participants perceived *Memento* as having a more fragmented narrative structure, consistent with its objective editing density. Fourth, integrating the first and third findings revealed a positive correlation between narrative boundary density and recall scores: greater fragmentation corresponded to stronger memory consolidation, validating event boundaries as cognitive anchors for episodic encoding.

4.2. Special findings and mechanistic interpretation

Several results deviated from initial hypotheses, requiring more nuanced explanations. First, a subset of participants exhibited elevated SWS proportion coupled with reduced REM proportion across both film conditions. For *Memento*, high event boundary density drove intensive hippocampal tagging during encoding, necessitating enhanced SWS replay via the slow-oscillation–spindle–ripple triple-coupling mechanism, which directly increased SWS duration [6-8]. Reduced REM in this subgroup reflected temporal budget constraints: extensive SWS replay consumed a disproportionate share of the sleep cycle, compressing the available REM window. For *1917*, the tonic load regime inherent to low boundary density narratives sustained LC activity, preventing complete LC silencing; SWS was prolonged as a compensatory mechanism to suppress this persistent LC activation [3]. Reduced REM in the *1917* group arose from two synergistic factors: the film's lack of emotional phasic load diminished intrinsic demand for REM-dependent processing, and residual tonic load inertia from SWS continued to inhibit LC silencing, sustaining elevated NE levels that are inimical to REM generation [3, 9].

Second, the *Memento* group exhibited a higher proportion of REM sleep than the *1917* group—a result that contradicts the standard prediction of the Cognitive Load Bimodal Model (CLBM) [3], which posits that high boundary density should reduce REM via SWS time compression. However, *Memento* combines high structural fragmentation with profound emotional valence (themes of loss, revenge, identity confusion), which substantially amplified the demand for REM-dependent emotional depotentiation [9]. This heightened emotional demand likely competed with and overrode SWS compression effects: the brain prioritized emotional resolution over cognitive consolidation, preserving or expanding the REM time window. This finding reveals a limitation of the current

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CLBM for dual high-cognitive/high-affective load narratives, and calls for a refined model incorporating an affective-demand axis alongside temporal dynamics.

Third, participants showed consistently longer SOL after viewing *1917*. The primary driver was the sustained attentional maintenance load of the tonic load regime: *1917*'s extended single-take cinematography required continuous attentional tracking, keeping the prefrontal cortex active long after viewing. A secondary, less dominant factor was low-stimulus rumination: the relative lack of intense cognitive engagement in later segments of *1917* permitted default mode network (DMN) activation at bedtime, triggering self-referential, spontaneous ruminative thoughts that elevated cognitive arousal and delayed sleep onset [3].

Fourth, a striking dissociation emerged in the *Memento* group: participants reported higher post-viewing SAM arousal but demonstrated better objective sleep quality and shorter SOL, whereas the *1917* group showed the inverse pattern. For *1917*, sustained cognitive load directly prolonged SOL and reduced SE by maintaining cortical vigilance. For *Memento*, four interconnected mechanisms resolved this paradox: (1) Phasic load from frequent boundaries produced transient, high-amplitude arousal spikes that dissipated rapidly once encoding demands subsided, preventing prolonged wakefulness; (2) Event boundaries reduce hippocampal salience for individual items, enabling efficient, schema-like encoding rather than item-specific overload [5]; (3) The high volume of tagged sequences necessitated extensive hippocampo-cortical replay, leading to profound metabolic depletion that forced sleep initiation [8]; (4) The surge in boundaries disrupted the equilibrium between phasic and tonic load modes, prompting systemic prioritization of restorative processes. Collectively, these factors produced metabolic exhaustion that overrode subjective arousal, shortening SOL.

Finally, next-morning arousal levels among *Memento* viewers bifurcated: a subset remained highly aroused, while others exhibited markedly lower arousal. In the high-arousal subgroup, frequent boundaries induced recurrent daytime LC activation, elevating NE levels that persisted into sleep. This sustained NE elevation compromised REM architecture, reducing REM duration and impairing emotional depotentiation, leaving emotional residues intact [10]. In contrast, the low-arousal subgroup exhibited normal REM function, where sleep-dependent mechanisms successfully decoupled emotion from memory traces, transforming charged emotional memories into neutral event representations [9]. This inter-individual variability highlights REM efficiency as a key moderator of next-day emotional outcomes.

5. Psychopathological discussion

The CLBM provides a transdiagnostic behavioral framework for affective disorders and neurodegenerative diseases. In depression and anxiety, patients exhibit incomplete LC silencing during REM sleep, resulting in elevated NE and failed emotional depotentiation, which leaves persistent emotional residues [3, 9, 10]. The phasic load regime of the CLBM reversibly simulates this state-dependent risk in healthy participants via high boundary density narratives: the REM-dependent emotional resolution overload induced by frequent boundaries is mechanistically homologous to patient pathology. Both originate from insufficient emotional depotentiation, producing residual affective symptoms. Thus, the CLBM serves as a behavioral model of affective dysregulation, enabling ethical, low-risk simulation of pathological states in healthy populations.

For Alzheimer's disease (AD), the CLBM establishes a mechanistic bridge between healthy sleep and early pathology. Under the phasic load regime, healthy participants exhibit increased SWS and enhanced slow-oscillation–spindle–ripple triple coupling, simulating a high-demand memory transfer scenario [4, 6-8]. In early AD, this same triple coupling undergoes irreversible degradation,

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reducing the efficacy of SWS-dependent hippocampo-cortical transfer [11]. The CLBM thus offers a reversible, manipulable behavioral model to simulate triple-coupling overload in AD, enabling low-cost mechanistic investigation in healthy participants using high boundary density narratives. Furthermore, non-rapid eye movement (NREM) slow-wave activity (SWA) functions as a cognitive reserve factor under high amyloid-β (Aβ) pathological burden: higher SWA correlates with better memory despite amyloid accumulation [11]. However, the phasic load regime consumes SWA resources, and chronic exposure may accelerate cognitive reserve depletion. This identifies pre-sleep narrative load as a modifiable behavioral risk factor for AD, and supports a practical regulatory framework: managing pre-sleep narrative cognitive load may preserve SWA and mitigate AD risk.

6. Conclusion

This study addressed three core hypotheses regarding how pre-sleep narrative boundary density modulates memory consolidation and sleep architecture. Contrary to the canonical linear prediction that higher cognitive load uniformly impairs sleep [1, 2], our results revealed a nuanced bimodal pattern. The first hypothesis—that high boundary density prolongs SOL and reduces SE—was not supported: *Memento* shortened SOL and improved SE despite higher subjective arousal. The second hypothesis—that SAM arousal correlates positively with SOL—was also refuted: *Memento* viewers exhibited high SAM but shorter SOL. The third hypothesis—that high boundary density disrupts memory consolidation—was directly reversed: *Memento* produced superior next-morning recall. These deviations challenge the "heavier load, worse sleep" framework, and validate the CLBM, wherein phasic and tonic load regimes engage distinct LC-NE modes and sleep-stage resolution pathways [3].

Despite these contributions, several limitations must be acknowledged. First, the pilot sample size (n=8) was small, limiting statistical power and generalizability. Second, we relied on wearable actigraphy rather than laboratory polysomnography, and collected no direct neural measures (e.g., high-density EEG, and pupillometry) to verify LC-NE activity patterns or triple-coupling dynamics. Third, the emotional valence of *Memento* was not experimentally controlled, potentially confounding boundary density effects. Fourth, the crossover design with only two films restricts causal inference. Fifth, recall tests were administered at midday rather than immediately upon waking, which may have introduced circadian or interference effects.

Future research should address these gaps by: (1) Expanding to larger, more diverse samples with in-lab polysomnography; (2) Incorporating high-density EEG to directly measure SWR density, spindle-slow oscillation coupling, and SWA; (3) Using pupillometry as a proxy for phasic/tonic LC-NE activity; (4) Systematically manipulating emotional valence independently of boundary density; (5) Testing chronic exposure effects to assess long-term impacts on cognitive reserve. Extending the CLBM to clinical populations—particularly individuals with mood disorders or early AD—will further validate its transdiagnostic utility. Finally, investigating whether individual differences in baseline LC integrity moderate phasic load responses will refine the model's predictive precision.

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