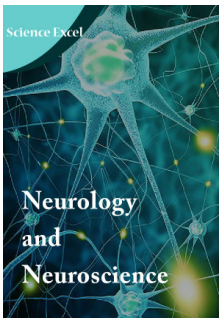
Neurology & Neuroscience

Opinion Article



The Limbic-Reticular Coupling Explanation of Paradoxical Anterior Thalamic Nuclei in Memory

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- Received Date: 15 June 2026
- Accepted Date: 23 June 2026
- Publication Date: 26 June 2026

Keywords

Anterior thalamic nuclei, familiarity, novelty, mammillothalamic tract, recall, theta wave

Abstract

From the limbic-reticular coupling theory proposed by Cai for memory, it is herein highlighted the anterior thalamic nuclei (ATN) mediating both familiarity and recall. For novelty consolidating both familiarity and recall, it relays theta low-frequency from supramammillary nucleus to septum, hippocampus and ATN, with novelty impaired by lesion of mammillary bodies or mammillothalamic tract (MTT) to ATN. For familiarity, it is impaired by lesions of posterior cingulate and retrosplenial cortices, which interconnect with ATN. Accordingly, it is explained that MTT lesion manifests recall impairment from the net effects of both impaired novelty from MTT to ATN and spared familiarity from related cortices to ATN.

Introduction

Cai proposed the limbic-reticular coupling theory, suggesting that the limbic hippocampus and amygdala regulate the subcortical mammillary bodies, septum, hypothalamus and habenula, and in turn regulate the four ascending reticular noradrenergic (NA), serotonergic (5-HT), dopaminergic (DA) and acetylcholinergic (ACh) systems to consolidate and recall declarative memories [1,2].

Warrington and Weiskrantz proposed the disconnection theory, suggesting that the diencephalic amnesia result from the disconnection of memory storage in temporal lobe and complex cognition in prefrontal cortex following lesions to the thalamus [3].

Clinical studies have not supported the orbital frontal cortex for declarative memory. It was reported that a patient with extensive bilateral orbitofrontal lesions exhibited no memory impairment [4]. An earlier report also indicated that large bilateral orbitofrontal lesions did not cause amnesia [5].

The anterior thalamic nuclei (ATN) and mammillothalamic tract (MTT) also correlate to amnesia from disconnection theory[3], but lie beyond the limbic-reticular coupling pathways. Herein, it is attempted to explain the amnesic lesions of ATN and MTT with the limbic-reticular coupling theory.

Familiarity and Recall

Aggleton divided the long-term declarative memory into two components as recall via hippocampus-MTT-ATN and familiarity via amygdala-mediodorsal thalamic nuclei [6]. Indeed, restricted MTT lesion only impaired recall but not familiarity [7]. However, discrepant from Aggleton's hypothesis, selective ATN lesions impaired both recall and familiarity [8,9].

It is not difficult to explain such discrepancy. On one aspect, familiarity was impaired in lesions of posterior cingulate cortex [10,11], including retrosplenial cortex [11], both of which interconnected with ATN [12], explaining the ATN function of familiarity. On another aspect, selective lesion of MTT, the main input from mammillary bodies to ATN [12], only impaired recall but not familiarity [7]. Taking these two aspects together, it explains that ATN lesions impaired both recall and familiarity [8,9].

Novelty, Familiarity and Recall

Recently, Cai applied novelty detection by hippocampal CA1 and amygdala to the limbic-reticular coupling theory for declarative memory consolidation via NA, 5-HT, DA and ACh, while the theta wave from medial temporal lobe and hippocampal CA3 coupled to ACh and DA for declarative memory recall [2].

Since novelty consolidating memory encoding via limbic-reticular coupling, it enhances both familiarity and recall. The hippocampal CA1 theta was shown necessary for encoding the novel input under the regulation by ACh from medial septum [13]. It was further demonstrated that the neurons of supramammillary nucleus regulated such rhythmical ACh neurons of medial septum

Citation: Cai ZJ. The Limbic-Reticular Coupling Explanation of Paradoxical Anterior Thalamic Nuclei in Memory. Neurol Neurosci. 2026;7(2):052.

[14]. Accordingly, the supramammillary nucleus and MTT mediate novelty, enhancing both familiarity and recall.

However, restricted MTT lesion only impaired recall but not familiarity [7]. The explanation herein is that, compared to the strong spared familiarity of ATN from posterior cingulate and retrosplenial cortices [10,11], the impaired familiarity from MTT lesion is negligible, so that grossly the MTT lesion manifested only impaired recall but not familiarity[7].

Conclusion

It is reconciled the effects of ATN and MTT lesions to the limbic-reticular coupling theory of declarative memory consolidation and recall.

Conflicts of Interest

The author declares no conflict of interest for this work.

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