



## Striatum–hippocampus balance: From physiological behavior to interneuronal pathology

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### ABSTRACT

In neurological disorders in which the cross-talk between striatal and hippocampal memory systems is affected, such as epilepsy, Down syndrome and Huntington's disease, cell-type specific alterations in synaptic plasticity lead to distinctive patterns causing functional imbalance between the two memory systems. Despite the complex network in which their neuronal activity is likely to be engaged, a common property of striatal and hippocampal neurons is to undergo bidirectional synaptic plasticity that relies on activity of interneurons and correlates with specific learning skills. As interneuronal dysfunction plays a primary role in the pathogenesis of these disorders, interneurons can be viewed as critical elements in neurophysiological substrates of such flexible relationships between these two memory systems.

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**Abbreviations:** DG, dentate gyrus; CA, cornu ammonis; LTP, long term potentiation; LTD, long term depression; GABA,  $\gamma$ -aminobutyric acid; MSNs, medium spiny neurons; LAIs, large aspiny cholinergic interneurons; PV, parvalbumin; FSIs, fast-spiking interneurons; Bsn, Bassoon; VPA, valproic acid; BDNF, brain-derived neurotrophic factor; NMDA, N-methyl-D aspartate; TrkB, tropomyosin-related kinase B; DS, Down syndrome; HD, Huntington's disease; ACh, acetylcholine.

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## 1. Relationships between striatum and hippocampus

### 1.1. Behavioral evidences for striatum–hippocampus interactions

Since the mid-1950s neurobiological studies have clearly demonstrated that the mammalian brain is organized in multiple memory systems (Packard and Knowlton, 2002; Squire, 2004; White and McDonald, 2002). The possibility to produce localized hippocampal damage in experimental animals allowed neuro-psychologists to develop early animal models of the human amnesic syndrome, and numerous studies, focusing on the pattern of spared and impaired learning that follows hippocampal system damage, led to the idea that non-hippocampal forms of learning could exist. Over the past two decades neuroanatomical and neurophysiological advances have increased our comprehension of neuronal systems that mediate non-hippocampal forms of learning and increasing evidence now indicates that the caudate nucleus (i.e. dorsal striatum) is part of an independent memory system that mediates non-hippocampal dependent memory.

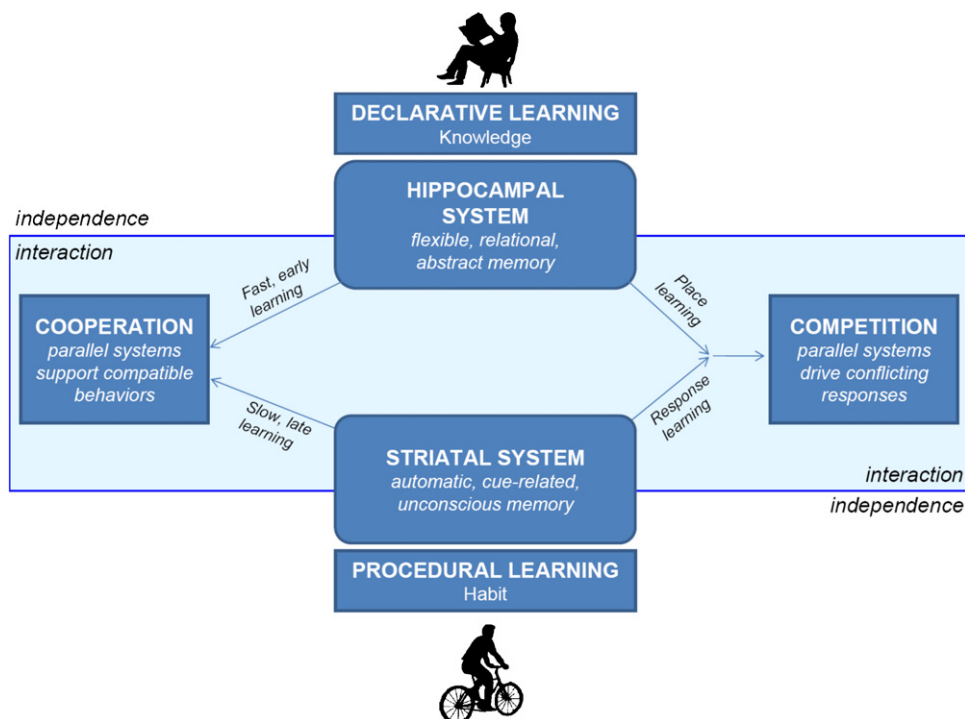
It is now accepted that the hippocampus is more important for remembering particular spatial locations defined by arrays of external cues (allocentric coordinates) (Morris et al., 1982) and that it is mainly involved in the formation of detailed cognitive ‘maps’ of the context in which learning occurs (place learning). On the other hand, dorsolateral striatum is more important for learning and choosing actions in body-centered (egocentric) coordinates (e.g., (Brasted et al., 1997; Cook and Kesner, 1988), and, therefore, it is engaged by repetitive stimulus–response (S–R) association (response learning).

Converging evidence from animal and human studies indicates differential roles of the hippocampal and the corticostriatal systems in memory and suggests that declarative and non-declarative memory systems may act independently. However,

hippocampal and striatal memory systems can operate in parallel as part of a dynamic system with the goal of optimizing behavior based on previous experience (Kim and Baxter, 2001; Packard and McGaugh, 1996; Poldrack and Packard, 2003) and the information contained in the two systems can either synergize or be incompatible (Fig. 1) (Atallah et al., 2004; McDonald et al., 2004; White and McDonald, 2002). In situations in which an adequate learned solution is provided by simultaneous activation of hippocampus and caudate, and the behavioral outputs of the two nuclei are compatible, the two memory systems can engage a cooperative interaction (White and McDonald, 2002). Evidence from a number of animal studies indicates that the maze task can be performed by using either a hippocampus-dependent “place” strategy or a striatum-dependent “response” strategy. When these two structures act cooperatively, early learning may be mediated by one system and, following extended training, the other system comes to lead learned behaviors (Packard and McGaugh, 1996). This shift in strategy has been suggested to reflect a reversible change in the leading system governing the behavioral response since the early strategy can still be unmasked by the other system inactivation (Packard and McGaugh, 1996).

However, many studies support the hypothesis of competitive interactions between multiple memory systems. In this case, activation of both systems produces conflicting influences over behavior and disabling one system would eliminate its competitive output increasing the influence of the other system on behavior. In fact, when a behavioral task forces an animal to make use of one kind of information over the other, inactivation of striatum increases use of a hippocampal-dependent “place” strategy, whereas inactivation of hippocampus increases use of a striatum-dependent “response” strategy (Packard and McGaugh, 1996).

Early studies in the field of experimental psychology provided evidence that an intact dorsal striatum is necessary for the



**Fig. 1.** Interactions between memory systems. Schematic diagram of the reciprocal relationships between procedural striatal-dependent system and declarative hippocampal-dependent system. The distinction between different memory systems operating in parallel has been proposed to serve independent functions: the hippocampal system supports rapid acquisition of experience and concepts (i.e. semantic understanding while reading a book), whereas striatal system guarantees slower trial-and-error acquisition of skills (i.e. ability to ride a bike). Alternatively, these two systems can interact to drive behaviors, and cooperation or competition can occur depending on the task considered. Cooperation is possible when both systems support compatible behaviors, whereas in tasks in which their simultaneous activation results in conflicting response, the two memory systems become competitive for the control of the behavioral output.

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