

MacKay, D.G. (2011). Hippocampus. In P. C. Hogan (Ed.). *The Cambridge Encyclopedia of the Language Sciences* (pp 357-359). Cambridge, England: Cambridge University Press.

HIPPOCAMPUS

The hippocampus is a bilaterally symmetric subcortical structure adjacent to the lateral ventricle in the medial **TEMPORAL LOBE (MTL)**. Researchers in 1968 reported dramatic **MEMORY** phenomena associated with hippocampal-MTL damage, and data reported from 1998 to 2006 have indicated parallel phenomena for many other aspects of cognition, including language. I first discuss patient H. M., the initial source of data for the hippocampal-memory and hippocampal-language links. I then discuss related patient groups and the theoretical significance of the hippocampal-language link.

Because of the unique and circumscribed nature of his 1953 surgery, H. M. is probably the most studied patient in the history of neuropsychology (Ogden and Corkin 1991): A neurosurgeon inserted thin metal tubes above the eyes, and via suction, removed parts of H. M.'s hippocampus and directly linked MTL structures. This operation greatly ameliorated H. M.'s life-threatening epilepsy, left H. M.'s neocortex virtually undamaged, and spared all neocortex with known links to language comprehension. However, the operation caused a *selective memory deficit*, with normal recall of information familiar to H. M. before his operation and used frequently since then, but impaired recall of information newly encountered after his operation and not massively repeated since then (see MacKay et al. 2007).

H. M. has **SENTENCE**-level language deficits that precisely mirror his memory deficits. D. G. MacKay et al. (2007) tested H. M.'s sentence-level comprehension in six tasks. In one task, participants identified the grammatical versus ungrammatical status of never previously encountered sentences that were either grammatical or ungrammatical (see **GRAMMATICALITY**). Here, H. M. responded with the correct answer reliably less often than controls matched for age, IQ, and education. This comprehension deficit impaired a wide variety of syntactic structures, including ones that memory-normal participants find easy to recall: H. M. exhibited equivalent comprehension deficits for easy- and difficult-to-recall sentences.

In a second task, H. M. again performed reliably worse than controls in identifying grammatical sentences as grammatical and in detecting, identifying, and repairing errors in sentences containing incorrect and misordered words. A third task required multiple-choice identification of who-did-what-to-whom in novel sentences. Here, H. M. identified the correct **THEMATIC ROLE** of sentence constituents reliably less often than controls. A fourth task required multiple-choice recognition of the appropriate interpretation for sentences containing novel **METAPHORS**. Here, H. M. chose the correct interpretation reliably less often than controls, and his errors indicated failure to recognize that the sentences were metaphoric. A fifth task required yes-no recognition of the appropriate interpretation for **AMBIGUOUS** sentences. Here, H. M. responded correctly less often than controls and sometimes responded "yes-and-no" despite repeated requests to respond "yes-or-no."

Consistent with several earlier results discussed next, H. M.'s ambiguity comprehension deficits were not due to memory overload associated with multiple meanings: In the ambiguity detection and description task of MacKay, Stewart, and Burke (1998), H. M. took much longer than controls to begin to describe the *first* of two meanings in ambiguous sentences, even when he never discovered the second meaning. H. M. also discovered both meanings without experimenter help less often than controls and often failed to understand meanings that the experimenter had just explained. Research isolated seven deficits in how H. M. *described* the sentence meanings: Grammatically impossible interpretations, misreadings reflecting failure to comprehend sentence-level meaning, errors in pronoun use (**ANAPHORA**), error correction failures, free associative responses, self-miscomprehensions,

and failures to follow experimenter requests for clarification. Research also indicated comprehension failure involving an *initial* meaning for sentences, ambiguous or not.

To summarize, in a wide range of tasks involving many fundamental aspects of sentence comprehension, H. M. exhibited deficits not caused by his memory problems (for corroborating evidence on H. M.'s comprehension deficits, see Corkin 1984; Lackner 1974; and Schmolck, Stefanacci, and Squire 2000). However, H. M.'s comprehension deficits were *selective* rather than across the board: Experiment six in MacKay et al. (2007) demonstrated that H. M. comprehended familiar words and phrases in isolation without deficit despite large deficits in comprehending *these same stimuli* when embedded within sentences. Besides demonstrating selectivity, these results indicated that H. M.'s deficits were not attributable to low motivation, to failure as a child to learn the meaning of the critical words and phrases, or to failure to understand and follow instructions for the task.

H. M. also exhibited significant *production* deficits when describing the meanings of familiar words that he comprehended without deficit in MacKay et al. (2007): Judges blind to speaker identity rated H. M.'s meaning descriptions as reliably more redundant, less coherent, less grammatical, and less comprehensible than those of controls. These findings replicated earlier results indicating deficits in H. M.'s production of novel or non-cliché sentences (see MacKay, Stewart, and Burke 1998). Again, however, H. M. exhibited *selective* production deficits that mirrored his memory deficits, for example, spontaneously producing *cliché* phrases such as "in a way" (familiar from before his surgery) without errors (*ibid.*).

H. M. also exhibited similar deficits and sparing in the seemingly simple task of reading sentences aloud (MacKay and James 2001): He produced abnormal pauses at major syntactic boundaries unmarked by commas in the sentences, but normal pauses at syntactic boundaries marked with commas, a prosodic marker that H. M. had learned prior to his operation. H. M. also produced abnormal pauses within unfamiliar phrases in the sentences, but normal pauses within frequently used phrases. These and other selective deficits indicated that he has difficulty with the process of reconstructing novel aspects of sentence structure when reading aloud.

H. M. also exhibited similar deficits and sparing in visual cognition: When detecting target figures hidden in concealing arrays, he performed reliably worse than controls for unfamiliar targets but not for familiar targets (MacKay and James 2000). In short, H. M. exhibits similar selective deficits in visual cognition, episodic memory, sentence-level comprehension, and sentence production when speaking and reading aloud; *impaired* processing of never previously encountered events, visual figures, phrases, and propositions; but *spared* processing of information familiar to him before his lesion and used frequently since then.

Why are these parallels important? One reason is that H. M. is not unique: Other patients with hippocampal-MTL damage exhibit identical parallels, reinforcing the links among hippocampus-MTL, language, and memory. For example, other amnesiacs exhibit deficits in detecting the two meanings in ambiguous sentences (Zaidel et al. 1995) and make errors resembling H. M.'s

in reading novel sentences aloud (Friedman 1996; MacKay and James 2001).

Second, these parallels are difficult to explain in current systems theories, in which independent systems process memory, language comprehension, language production, and visual cognition, and the hippocampus subserves only the memory system (see, e.g., Schmolck, Stefanacci, and Squire 2000). Under systems theories, hippocampal-MTL damage should yield memory deficits without deficits in other cognitive systems, and certainly without *parallel* deficits and *parallel* sparing across supposedly independent systems for sentence comprehension, sentence production, visual cognition, and episodic memory. These predictions have failed, and major attempts to rescue current systems theories from these failed predictions have likewise failed (see MacKay 2001, 2006; and MacKay, James, and Hadley 2008).

Third, a new theoretical framework known as binding theory (not to be confused with the *anaphoric* binding theory in linguistics; see Jackendoff 2003, 15) readily explains and, indeed, originally predicted the links between hippocampal-MTL damage and parallel deficits and sparing in memory, sentence-level language, and other aspects of cognition. Under binding theory, hippocampal-MTL damage impairs binding mechanisms for forming new internal representations in the cortex but does not affect mechanisms for activating already existing cortical representations (see, e.g., MacKay et al. 2007 and James and MacKay 2001 for important theoretical details regarding forgetting, frequency of use, and **AGING AND LANGUAGE**).

To illustrate in detail how binding theory explains his selective deficits, consider H. M.'s sentence production in a standard picture-description task requiring the incorporation of prespecified target words (MacKay et al. 2007): H. M. described the word-picture stimuli significantly less accurately and completely than eight controls, included fewer target words, and produced more incomplete sentences (e.g., lacking a subject or verb), violations of **AGREEMENT** rules, non sequiturs, and run-on sentences than the controls. Descriptions by H. M. (1a–2a) versus controls (1b–2b) for the same word-picture stimuli illustrate some of these differences.

(1a) *H. M. description*: Because it's wrong for her to be and he's dressed just as this that he's dressed and the same way.

(1b) *Control description*: Well, I think I'll take that one although it looks wrong.

(2a) *H. M. description*: I want some of that pie either some pie and I'll have some.

(2b) *Control description*: Uh, there are two people getting pie, but there's only one piece of blueberry pie left, and so, either one of them will have to have it.

Note that H. M.'s picture-description problems in 1a and 2a were *selective*: Unlike agrammatic **APHASICS**, H. M. did not produce **MORPHEMES** and **NONSENSE** words jumbled together into **MORPHOLOGICAL** salads (Jackendoff 2003, 264). Moreover, he produced frequently used units, such as "it's wrong," "to be," "the same way" (1a), "some of that," and

"I'll have some" (2a), without errors. Under binding theory, separately stored syntactic units and rules serve to activate already formed internal representations so that words and phrases become produced in the appropriate order. Because H. M.'s syntax-based activation mechanisms are intact and frequently used since his lesion, H. M. therefore produces familiar words, phrases, and propositions such as "it's wrong" and "I'll have some," without errors. However, he lacks already formed internal REPRESENTATIONS for propositions that he has used repeatedly before and after his lesion to describe the MacKay et al. (2007) word-picture stimuli. The word-picture stimuli, therefore, triggered familiar units that H. M. simply concatenated without forming complete, appropriate, and coherent utterances (see 1b, 2b).

In conclusion, the pressing problem for future research is to test new binding theory predictions for relations among brain, language, memory, and other aspects of cognition (see MacKay et al. 2007).

— Donald G. MacKay

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