

IN MEMORIAM

The Unforgettable Career of Suzanne Corkin

Bradley R. Postle^{1*} and Elizabeth Kensinger²

Over the course of the past half century, Suzanne Hammond Corkin (1937–2016) has made a remarkable number of contributions to our understanding of the neural bases of human cognition. She is, of course, best known for her career-long association with the amnesic patient H.M., eloquently summarized in her book *Permanent Present Tense: The Unforgettable Life of the Amnesic Patient, H.M.* (Corkin, 2013). From this association have come foundational insights into the functions of the hippocampus and surrounding tissue of the medial temporal lobe (MTL), and into the organization of memory. In addition to these contributions, her wide-ranging research has also explored the neural bases of perception and working memory, the interaction of emotion and memory, and the effects of aging and of age-related neurodegenerative disease on cognition. Throughout her celebrated career, she set a pioneering example for how to be successful as a woman in science.

Suzanne Hammond grew up in the Hartford, Connecticut. As circumstance would have it, she was a childhood friend of the daughter of the neurosurgeon, William Beecher Scoville. She was of high-school age, and perhaps just a few miles distant, when Scoville performed the fateful bilateral medial temporal lobectomy on Henry Molaison, a 27-year-old with intractable epilepsy. She was pursuing her B.A. at Smith College when Scoville and Milner published their initial report of H.M.'s case (Scoville and Milner, 1957). Shortly thereafter, she enrolled in the Clinical Psychology graduate program at McGill University, where she learned from and worked with some of the giants of neuroscience, including D.O. Hebb, Wilder Penfield, Herbert Jasper, Theodore Rasmussen, Robert Malmo, and her mentor Brenda Milner. She initially came to Milner with an interest in somesthesia, a topic on which she was to publish work carried out at the Montreal Neurological Institute (Corkin et al., 1964, 1970), as well as, several years later, with collaborators in Boston (Moore et al., 2000a,b). Most famously, however, it was also at this time that she began her work with H.M.

The 1957 report from Scoville and Milner had established “a special importance to the anterior hippocampus and hippocampal gyrus in the retention of new experience” (p. 21). This was a revolutionary

breakthrough in memory research. (Consider that, just a few years earlier, Lashley (1950) had noted that “I sometimes feel, in reviewing the evidence on the localization of the memory trace, that the necessary conclusion is that learning just is not possible. . . It is not possible to demonstrate the isolated localization of a memory trace anywhere within the nervous system” (p. 477–478).) Critically, H.M.'s profound memory impairment coexisted with an above-normal IQ, indicating that memory could be understood as dissociable from other high-level mental functions. Shortly thereafter, a second important conceptual breakthrough came with Milner's demonstration that H.M. could retain learning of a sensorimotor skill (mirror tracing) despite no consciously accessible memory of his prior experience with the testing apparatus (Milner, 1962). Corkin followed up on this finding, demonstrating preserved learning in H.M. when navigating a tactually guided maze (Corkin, 1965), performing motor skills (Corkin, 1968), and recognizing degraded visual images (Milner et al., 1968). The implication of these findings was that *memory*, itself, was not a unitary entity, but could be neurally dissociated into different classes. One—which came to be known as declarative or explicit—was dependent on the hippocampus and MTL, and others—which became known as nondeclarative, procedural, or implicit—were not (e.g., Cohen and Squire, 1980; Schacter, 1987).

After receiving her PhD in 1964, Suzanne Corkin moved to the laboratory of Hans-Lukas Teuber at MIT. During her first 15 years at MIT, she was the engine behind the Department of Psychology's Clinical Research Center, where brain-injured subjects were studied. Years later, it was noted that this facility was “so original [for a department of psychology] that even today it is more admired than emulated” (Gross, 1994) (p. 453). During this time, Corkin also played a critical role in the editing of *Neuropsychologia*, the era's leading journal of the neuroscientific study of human behavior. In 1977, Teuber's untimely death led to two developments that were without precedent at MIT. First, although she held a research staff (i.e., not faculty) position, Corkin became director of the human neuropsychology lab. Second, in 1981,

¹ Departments of Psychology and Psychiatry, University of Wisconsin–Madison, Madison, Wisconsin; ² Department of Psychology, Boston College, Massachusetts

*Correspondence to: Bradley R. Postle, Departments of Psychology and Psychiatry, University of Wisconsin–Madison, Madison, WI, USA. E-mail: postle@wisc.edu

Received 11 July 2016; Accepted for publication 19 July 2016.

DOI 10.1002/hipo.22618

Published online 20 July 2016 in Wiley Online Library (wileyonlinelibrary.com).

Suzanne Corkin was promoted directly from the position of Principal Research Scientist to Associate Professor (with tenure). All this at a time when fewer than 10% of faculty in MIT's School of Sciences were female (MIT Faculty Newsletter, 1999).

From 1980s onward, Suzanne Corkin's Behavioral Neuroscience Laboratory made seminal contributions to many domains of cognitive neuroscience research. One important focus continued to be the organization and boundary conditions of non-declarative memory. One experiment, building on Milner's original 1962 study, demonstrated that H.M. retained learning on the mirror tracing task for as long as a year (Gabrieli et al., 1993). In others, comparison of the performance of H.M. with that of patients with damage to regions other than the MTL was used to localize the neural systems supporting different kinds of memory. For example, patterns of "dissociations among structural-perceptual, lexical-semantic, and event-fact memory systems in amnesia, Alzheimer's disease, and normal subjects" (Gabrieli et al., 1994), and of a "double dissociation of memory capacities after bilateral occipital-lobe or MTL lesions" (Keane et al., 1995), indicated that different forms of nondeclarative memory can rely on, in these examples, heteromodal cortex of the temporal and parietal lobes (preferentially vulnerable to early stages of Alzheimer's disease), or on early stages of the visual system. Another study, showing that H.M.'s nondeclarative memory performance with the same stimulus material could be intact or impaired, depending on how his memory was tested, called attention to the importance of understanding the cognitive processes that are challenged by a memory task: pre-exposure to words that came into the language after 1965, and for which H.M. had therefore never established a lexical representation, facilitated the subsequent identification of these words under perceptually challenging conditions, but did not influence the subsequent completion of three-letter word stems (Postle and Corkin, 1998).

In parallel with the nondeclarative memory research, Corkin's group was also contributing importantly to our understanding of the impacts of neurodegenerative disease on sensory and cognitive functioning. In one line of work, they carried out exhaustive studies of perceptual functions (visual, auditory, and olfactory) in Alzheimer's disease (Cronin-Golomb et al., 1991, 1993, 1995; Kurylo et al., 1991, 1993, 1994a, 1994b; Mendola et al., 1995). Some of these studies have had important implications for clinical practice, such as the characterization of "cognitive test performance in detecting, staging, and tracking Alzheimer's disease" (Locascio et al., 1995). Another line of research explored cognitive deficits associated with Parkinson's disease (Corkin and Growdon, 1985; Cronin-Golomb et al., 1987, 1989, 1994; Ogden et al., 1987; Postle et al., 1997a,b; Sagar et al., 1988a,b; Sullivan et al., 1989; Locascio et al., 2003); later in her career, she expanded this line of research to examine how genetic polymorphisms relate to inhibitory ability in Parkinson disease (Ziegler et al., 2014).

All the while, continued careful research with H.M. indicated that his amnesia was global, meaning that it was equally profound regardless of the domain of information being tested,

or the sensory modality through which it was presented (Corkin, 1984). Intriguingly, however, some of Corkin's studies of H.M. also turned up surprising examples of preserved declarative memory. During two visits to the MIT lab, H.M. was able to draw, from memory, remarkably accurate floor plans of a house in which he lived, once while he was still living in it, and once 3 years after he had moved to a different home. Furthermore, he remembered the address at which this house was located. Corkin suggested that this "personal semantic information . . . [was] learned slowly over an extended period of time, presumably with the support of cortical structures" (p. 158), perhaps including parietal, retrosplenial, occipital, and infero-temporal elements of a spatial processing network (Corkin, 2002). In another series of studies, H.M. showed normal recognition memory for colored magazine pictures after delays of 10 min, 24 hr, 72 hr, 1 week, and 6 months (with the caveat that he studied each picture for 20 s, whereas control subjects studied each for 1 s) (Freed and Corkin, 1988; Freed et al., 1987). Corkin speculated that this may have reflected preserved familiarity judgments, perhaps supported by residual posterior perirhinal cortex, an idea supported by elevated fMRI activity in H.M.'s caudal MTL while viewing novel relative to repeated pictures (Corkin, 2002). Despite these demonstrations of better-than-expected performance on tests of declarative memory, careful assessment of the quality of H.M.'s memory for naturalistic information that he would have encountered repeatedly in his daily life indicated that any remembered information was likely to be highly impoverished. When given the first names for 35 individuals who had become famous after his surgery and asked to say any last name that came to mind, he generated 12 famous last names (e.g., Woody-Allen; Sophia-Loren). On a forced-choice recognition task, he could successfully discriminate postoperatively famous names from names pulled from a phonebook 87% of the time, yet he could provide information about the person's fame for only about one-third of those names (with most of these being individuals who became famous in the 1960s, O'Kane et al., 2004). These examples of new learning were exciting discoveries given H.M.'s dense amnesia. Notably, however, there is no evidence that this postmorbidly learned information could be accessed volitionally, nor that it could be combined flexibly with other information to create new declarative memories.

Suzanne Corkin was interested in, and contributed to, theoretical debates throughout her career. One noteworthy contribution in recent years has been to the question of whether the declarative memory functions of the hippocampus are limited primarily to the encoding and consolidation of information into long-term memory (LTM), as had been the prevailing view up through the 1990s (e.g., Squire and Bayley, 2007; Squire and Zola-Morgan, 1991), or whether this structure is also important for the retrieval of some aspects of remote memory, as stipulated by the more recently proposed Multiple Trace Theory (MTT, e.g., Nadel and Moscovitch, 1997, 1998). The results from a study of Corkin et al. suggested that, consistent with MTT, the retrieval of premorbidly experienced autobiographical episodes was dramatically impoverished in

H.M. and another amnesic patient (Steinvorth et al., 2005). Corroborating evidence was produced by a study of “Ecphory of autobiographical memories: An fMRI study of recent and remote memory retrieval” (Steinvorth et al., 2006). Ever the advocate of giving prominence to careful evaluation of the data, as recently as 2013, Corkin responded to critiques of these studies by stating that “Based on our experiments, I stand by the view, in agreement with the Multiple Trace Theory, that we need a functioning hippocampus to re-experience unique moments in our past, regardless of how long ago they were acquired” (Corkin, 2013).

Up to this point, much of the work that we have summarized has been in the neuropsychological tradition in which Suzanne Corkin was trained. Her career is also noteworthy, however, for the manner in which she embraced the methods of modern cognitive neuroscience. In two noteworthy examples that showcase her early adoption of neuroimaging methods, she and her collaborators were among the first to carry out fMRI studies characterizing the role of the hippocampal formation and MTL in LTM encoding (Stern et al., 1996), and she spearheaded the definitive (in vivo) anatomical characterization H.M.’s lesions (Corkin et al., 1997). This work was influential in expanding the focus of human LTM research from the hippocampus proper to the broader MTL system. Years later, upon H.M.’s death, Corkin oversaw in situ MRI scans at 3 T, followed by ex vivo scanning at ultra-high-resolution scanning at 7 T (Augustinack et al., 2014). The resultant images have provided the most detailed information to date on the extent of the lesions of this most-studied of neurological patients. Among the surprising findings from the 2014 study was that, contrary to the report from her earlier study (Corkin et al., 1997), H.M.’s medial temporal stem showed marked deterioration. Although it was unclear whether this pathology was due to the surgical resection or to age-related degeneration later in life, it afforded a revisiting of the question of a possible role for the temporal stem in supporting declarative memory functions (Horel, 1978; Zola-Morgan et al., 1982; Gaffan, 2001; Augustinack et al., 2014).

Given Suzanne Corkin’s career-long interest on the functions of the MTL, it is not surprising that she became interested in how the amygdala contributes to memory for emotional experiences. This research provided evidence that interactions between the amygdala and hippocampus support the encoding of high-arousal emotional experiences but not of lower-arousal ones (Kensinger and Corkin, 2004). This line of research also revealed the disruption of emotion-enhanced memory in patients with Alzheimer’s disease (Kensinger et al., 2002, 2004) and provided evidence that, despite emotion-enhanced memory in older adults (Kensinger et al., 2006), there were age differences in the contextual details remembered (Kensinger et al., 2002, 2005) and in the accuracy of memory discriminability for positive events (Piguet et al., 2008).

Suzanne Corkin and her collaborators utilized neuroimaging methods to great avail in many domains of study, including in the neurobiology of aging and of age-related neurodegenerative disease. She was a proponent of the importance of “functional

MRI for studying episodic memory in aging and Alzheimer’s disease” (Corkin, 1998) and later utilized structural as well as functional MRI methods to elucidate age-associated changes. This research emphasized the importance of assessing age-related changes in white matter tracts (Salat et al., 2005), with one study indicating that “cognition in healthy aging is related to regional white matter integrity, but not cortical thickness” (Ziegler et al., 2010) and another revealing that “white matter pathology isolates the hippocampal formation in Alzheimer’s disease” (Salat et al., 2010). Always enthusiastic about the knowledge that could be gained through new technological advances, her recent research reported the benefits of a multi-spectral structural magnetic resonance imaging method for measuring the volumes of the substantia nigra and basal forebrain in patients with Parkinson disease (Ziegler and Corkin, 2013; Ziegler et al., 2013).

The importance of Suzanne Corkin’s research contributions were recognized with an MERIT award from the National Institutes of Health and the Baltes Distinguished Research Achievement Award from the American Psychological Association, Division on Aging. The breath of research questions she addressed during the course of her career is truly remarkable and is a testament to her broad intellectual curiosity and to her advising style: She encouraged her trainees to find their intellectual passions and to let the data be their guide. Her laboratory was a vibrant, collaborative environment; she welcomed individuals with broad-ranging interests who shared her goal of using neuropsychological and neuroimaging methods to inform our understanding of the neural bases of human cognition. Her lineage of academic children, grandchildren, and great-grandchildren is impressive in number and noteworthy for the wide-ranging contributions of her trainees in their independent careers.

Through her daily comportment and accomplishments, Suzanne Corkin exemplified a wonderful model for how to be a successful woman in science. It is fitting that she received the Department of Brain and Cognitive Sciences Undergraduate Advising Award from MIT in 2011 and that her last public speech was at a Women in Science event at the 2016 meeting of the International Neuropsychological Society. Many testimonials to Suzanne Corkin, the mentor, as well as the scientist, are collected in a special issue of *The Journal of Cognitive Neuroscience* (2013, v. 25, issue 1) that marked her retirement. Together with her scientific accomplishments, her mentees and associates will recall with admiration and fondness her infectious joie de vivre, her passion for travel and exploration, and the aplomb with which she balanced a productive career with the raising of a warm, loving family. She will be missed.

REFERENCES

- Augustinack JC, van der Kouwe AJW, Salat DH, Benner T, Stevens AA, Annese J, Fischl B, Frosch MP, Corkin S. 2014. H.M.’s

- contributions to neuroscience: a review and autopsy studies. *Hippocampus* 24:1267–1286.
- Cohen NJ, Squire LR. 1980. Preserved learning and retention of pattern-analyzing skill in amnesia: Dissociation of knowing how and knowing that. *Science* 210:207–209.
- Corkin S. 1965. Tactually-guided maze learning in man: Effects of unilateral cortical excisions and bilateral hippocampal lesions. *Neuropsychologia* 3:339–351.
- Corkin S. 1968. Acquisition of motor skill after bilateral medial temporal-lobe excision. *Neuropsychologia* 6:255–265.
- Corkin S. 1984. Lasting consequences of bilateral medial temporal lobectomy: clinical course and experimental findings in H.M. *Seminars in Neurology* 4:252–262.
- Corkin S. 1998. Functional MRI for studying episodic memory in aging and Alzheimer's disease. *Geriatrics* 53:S13–S15.
- Corkin S. 2002. What's new with the amnesic patient H.M.? *Nature Reviews Neuroscience* 3:153–160.
- Corkin S. 2013. Permanent Present Tense. *The Unforgettable Life of the Amnesic Patient, H.M.* Basic Books.
- Corkin S, Amaral DG, Gonzalez RG, Johnson KA, Hyman BT. 1997. H.M.'s medial temporal-lobe lesion: findings from MRI. *The Journal of Neuroscience* 17:3964–3979.
- Corkin S, Growdon JH. 1985. Cognitive impairments in Parkinson's disease. In: Yahr MD, Bergmann KJ, editors. *Advances in Neurology: Volume 45: Parkinson's Disease*. New York: Raven Press. p 383–392.
- Corkin S, Milner B, Rasmussen T. 1964. Effects of different cortical excisions on sensory thresholds in man. *Transactions of the American Neurological Association* 89:112–116.
- Corkin S, Milner B, Rasmussen T. 1970. Somatosensory thresholds: contrasting effects of Rolandic and posterior parietal lesions. *Archives of Neurology* 23:41–58.
- Cronin-Golomb A, Corkin S, Growdon JH. 1987. Relational abilities in Alzheimer's disease and Parkinson's disease. *The Clinical Neuropsychologist* 1:298.
- Cronin-Golomb A, Corkin S, Growdon JH. 1989. Influence of set-shifting deficit on problem solving in Parkinson's disease. In: Wurtman RJ, Corkin S, Growdon JH, Ritter-Walker E, editors. *Alzheimer's Disease. Proceedings of the Fifth Meeting of the International Study Group on the Pharmacology of Memory Disorders Associated with Aging*. p 457–461.
- Cronin-Golomb A, Corkin S, Growdon JH. 1994. Impaired problem solving in Parkinson's disease: Impact of a set-shifting deficit. *Neuropsychologia* 32:579–593.
- Cronin-Golomb A, Corkin S, Growdon JH. 1995. Visual dysfunction predicts cognitive deficits in Alzheimer's disease. *Optometry and Vision Science* 73:168–176.
- Cronin-Golomb A, Corkin S, Rizzo JF, Cohen J, Growdon JH, Banks KS. 1991. Visual dysfunction in Alzheimer's disease: Relation to normal aging. *Annals of Neurology* 29:41–52.
- Cronin-Golomb A, Sugiura R, Corkin S, Growdon JH. 1993. Incomplete achromatopsia in Alzheimer's disease. *Neurobiology of Aging* 14:471–477.
- Freed DM, Corkin S. 1988. Rate of forgetting in H.M.: 6-month recognition. *Behavioral Neuroscience* 102:823–827.
- Freed DM, Corkin S, Cohen NJ. 1987. Forgetting in H.M.: A second look. *Neuropsychologia* 25:461–471.
- Gabrieli JDE, Corkin S, Mickel SF, Growdon JH. 1993. Intact acquisition and long-term retention of mirror-tracing skill in Alzheimer's disease and in global amnesia. *Behavioral Neuroscience* 107:899–910.
- Gabrieli JDE, Keane MM, Stanger BZ, Kjølgaard MM, Corkin S, Growdon JH. 1994. Dissociations among structural-perceptual, lexical-semantic, and event-fact memory systems in amnesia, Alzheimer's disease, and normal subjects. *Cortex* 30:75–103.
- Gaffan D. 2001. What is a memory system? Horel's critique revisited. *Behavioral Brain Research* 127:5–11.
- Gross CG. 1994. Hans-Lukas Teuber: A tribute. *Cerebral Cortex* This issue:451–454.
- Horel JA. 1978. The neuroanatomy of amnesia: A critique of the hippocampal memory hypothesis. *Brain* 101:403–445.
- Keane MM, Gabrieli JDE, Mapstone HC, Johnson KA, Corkin S. 1995. Double dissociation of memory capacities after bilateral occipital-lobe or medial temporal-lobe lesions. *Brain* 118:1129–1148.
- Kensinger EA, Anderson A, Growdon JH, Corkin S. 2004. Effects of Alzheimer disease on memory for verbal emotional information. *Neuropsychologia* 42:791–800.
- Kensinger EA, Brierley B, Medford N, Growdon JH, Corkin S. 2002. Effects of normal aging and Alzheimer's disease on emotional memory. *Emotion* 2:118–134.
- Kensinger EA, Corkin S. 2004. Two routes to emotional memory: distinct neural processes for valence and arousal. *Proceedings of the National Academy of Sciences, USA* 101:3310–3315.
- Kensinger EA, Krendl AC, Corkin S. 2006. Memories of an emotional and a nonemotional event: effects of aging and delay interval. *Experimental Aging Research* 32:23–45.
- Kensinger EA, Piquet O, Krendl AC, Corkin S. 2005. Memory for contextual details: effects of emotion and aging. *Psychology and Aging* 20:241–250.
- Kurylo DD, Corkin S, Allard T, Zatorre RJ, Growdon JH, editors. 1991. *Effect of Alzheimer's Disease on Basic and High-Order Auditory Capacities*. Cambridge, MA: Center for Brain Sciences and Metabolism Charitable Trust. 37–49 p.
- Kurylo DD, Corkin S, Allard T, Zatorre RJ, Growdon JH. 1993. Auditory function in Alzheimer's disease. *Neurology* 43:1893–1899.
- Kurylo DD, Corkin S, Dolan RP, Rizzo IIIJF, Parker SW, Growdon JH. 1994a. Broad-band visual capacities are not selectively impaired in Alzheimer's disease. *Neurobiology of Aging* 15:305–311.
- Kurylo DD, Corkin S, Growdon JH. 1994b. Perceptual organization in Alzheimer's disease. *Psychology and Aging* 9:562–567.
- Lashley KS. 1950. Search of the engram. *Symposium of the Society of Experimental Biology* 4:454–482.
- Locascio JJ, Growdon JH, Corkin S. 1995. Cognitive test performance in detecting, staging, and tracking Alzheimer's disease. *Archives of Neurology* 52:1087–1099.
- Locascio JJ, Growdon JH, Corkin S. 2003. Relation between clinical characteristics of Parkinson's disease and cognitive decline. *Journal of Clinical and Experimental Neuropsychology* 25:94–109.
- Mendola JD, Cronin-Golomb A, Corkin S, Growdon JH. 1995. Prevalence of visual deficits in Alzheimer's disease. *Optometry and Vision Science* 72:155–167.
- Milner B. 1962. Les troubles de la memoire accompagnant des lesions hippocampiques bilaterales. In: Passouant P, editor. *Physiologie de l'Hippocampe*. Paris: Centre National de la Recherche Scientifique. p 257–272.
- Milner B, Corkin S, Teuber H-L. 1968. Further analysis of the hippocampal amnesic syndrome: 14 year follow-up study of H.M. *Neuropsychologia* 6:215–234.
- Moore CI, Stern CE, Corkin S, Fischl B, Gray AC, Rosen BR, Dale AM. 2000a. Segregation of somatosensory activation in the human hand area using fMRI. *Journal of Neurophysiology* 84:558–569.
- Moore CI, Stern CE, Dunbar C, Kostyk SK, Gehi A, Corkin S. 2000b. Referred phantom sensations and cortical reorganization after spinal cord injury in humans. *Proceedings of the National Academy of Sciences, USA* 97:14703–14708.
- Nadel L, Moscovitch M. 1997. Memory consolidation, retrograde amnesia and the hippocampal complex. *Current Opinion in Neurobiology* 7:217–227.
- Nadel L, Moscovitch M. 1998. Consolidation and the hippocampal complex revisited: in defense of the multiple-trace model. *Current Opinion in Neurobiology* 8:297–300.
- O'Kane G, Kensinger EA, Corkin S. 2004. Evidence for semantic learning in profound amnesia: an investigation with patient H.M. *Hippocampus* 14:417–425.

- Ogden JA, Growdon JH, Corkin S. 1987. Impaired ability of patients with Parkinson's disease to shift conceptual set in visuospatial tasks. *International Journal of Neuroscience* 35:132.
- Piguet O, Connally E, Krendl AC, Huot JR, Corkin S. 2008. False memory in aging: effects of emotional valence on word recognition accuracy. *Psychology and Aging* 23:307–314.
- Postle BR, Corkin S. 1998. Impaired word-stem completion priming but intact perceptual identification priming with novel words: evidence from the amnesic patient H.M. *Neuropsychologia* 36:421–440.
- Postle BR, Jonides J, Smith E, Corkin S, Growdon JH. 1997a. Spatial, but not object, delayed response is impaired in early Parkinson's disease. *Neuropsychology* 11:1–9.
- Postle BR, Locascio JJ, Corkin S, Growdon JH. 1997b. The time course of spatial and object visual learning in early Parkinson's disease. *Neuropsychologia* 35:1413–1422.
- Sagar HJ, Cohen NJ, Sullivan EV, Corkin S, Growdon JH. 1988a. Remote memory function in Alzheimer's disease and Parkinson's disease. *Brain* 111:185–206.
- Sagar HJ, Sullivan EV, Gabrieli JDE, Corkin S, Growdon JH. 1988b. Temporal ordering and short term memory deficits in Parkinson's disease. *Brain* 111:525–539.
- Salat DH, Tuch DS, Hevelone ND, Fischl B, Corkin S, Rosas HD, Dale AM. 2005. Age-related changes in prefrontal white matter measured by diffusion tensor imaging. *Annals of the New York Academy of Sciences* 1064:37–49.
- Salat DH, Tuch DS, van der Kouwe AJ, Greve DN, Pappu V, Lee SY, Hevelone N, Zaleta AK, Growdon JH, Corkin S. and others. 2010. White matter pathology isolates the hippocampal formation in Alzheimer's disease. *Neurobiology of Aging* 31:244–256.
- Schacter DL. 1987. Implicit memory: History and current status. *Journal of Experimental Psychology: Learning, Memory and Cognition* 13:501–518.
- Scoville WB, Milner B. 1957. Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, and Psychiatry* 20:11–21.
- Squire LR, Bayley PJ. 2007. The neuroscience of remote memory. *Current Opinion in Neurobiology* 17:185–196.
- Squire LR, Zola-Morgan S. 1991. The medial temporal lobe memory system. *Science* 253:1380–1386.
- Steinvorth S, Corkin S, Halgren E. 2006. Ecphory of autobiographical memories: an fMRI study of recent and remote memory retrieval. *NeuroImage* 30:285–298.
- Steinvorth S, Levine B, Corkin S. 2005. Medial temporal lobe structures are needed to re-experience remote autobiographical memories: Evidence from two MTL amnesic patients, H.M. and W.R. *Neuropsychologia* 43:479–496.
- Stern CE, Corkin S, Gonzales RG, Guimaraes AR, Baker JR, Jennings PJ, Carr CA, Sugiura RM, Vedantham V, Rosen BR. 1996. The hippocampal formation participates in novel picture encoding: Evidence from functional magnetic resonance imaging. *Proceedings of the National Academy of Sciences, USA* 93:8660–8665.
- Sullivan EV, Sagar HJ, Gabrieli JDE, Corkin S, Growdon JH. 1989. Different cognitive profiles on standard behavioral tests in Parkinson's disease and Alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology* 11:799–820.
- Ziegler DA, Ashourian P, Wonderlick JS, Sarokhan AK, Prelec D, Scherzer CR, Corkin S. 2014. Motor impulsivity in Parkinson disease: associations with COMT and DRD2 polymorphisms. *Scandinavian Journal of Psychology* 55:278–286.
- Ziegler DA, Corkin S. 2013. New MRI biomarkers advance the characterization of Parkinson disease. *European Neurological Reviews* 8:85–89.
- Ziegler DA, Piguet O, Salat DH, Prince K, Connally E. 2010. Cognition in healthy aging is related to regional white matter integrity, but not cortical thickness. *Neurobiology of Aging* 31:1912–1926.
- Ziegler DA, Wonderlick JS, Ashourian P, Hansen LA, Young JC, Murphy AJ, Koppuzha CK, Growdon JH, Corkin S. 2013. Substantia nigra volume loss before basal forebrain degeneration in early Parkinson disease. *JAMA Neurology* 70:241–247.
- Zola-Morgan S, Squire LR, Mishkin M. 1982. The neuroanatomy of amnesia: The amygdala-hippocampus vs. temporal stem. *Science* 218:1337–1339.