tion in machine discovery. In J. Shrager & P. Langley (Eds.), Computational models of scientific discovery and theory formation (pp. 255–274). San Mateo, CA: Morgan Kaufmann.

Kurz, E.M. (1998). Representation, agency, and disciplinarity: Calculus experts at work. In M.A. Gernsbacher & S.J. Derry (Eds.), Proceedings of the Twentieth Annual Conference of the Cognitive Science Society (pp. 585–590). Mahwah, NJ: Erlbaum.

Kurz, E.M., & Tweney, R.D. (in press). Creating environments for cognition: An agentive perspective on scientific and mathematical thinking. In M. Oaksford & N. Chater (Eds.), Rational models of cognition. Oxford, England: Oxford University Press.

Langley, P.W., Simon, H.A., Bradshaw, G.L., &

Zytkow, J.M. (1987). Scientific discovery: Computational explorations of the discovery process. Cambridge, MA: MIT Press.

Mynatt, C.R., Doherty, M.E., & Tweney, R.D. (1978). Consequences of confirmation and disconfirmation in a simulated research environment. Quarterly Journal of Experimental Psychology, 30, 395–406.

Nersessian, N. (1992). How do scientists think? Capturing the dynamics of conceptual change in science. In R.N. Giere (Ed.), Minnesota Studies in the Philosophy of Science: Vol. XV. Cognitive models of science (pp. 3–44). Minneapolis: University of Minnesota Press.

Newell, A., & Simon, H.A. (1972). *Human problem solving*. Englewood Cliffs, NJ: Prentice-Hall.

Norman, D.A. (1988). The psychology of everyday things. New York: Basic Books.

Tweney, R.D. (1991). Faraday's notebooks: The active organization of creative science. *Physics Education*, 26, 301–306.

Tweney, R.D. (1992). Stopping time: Faraday and the scientific creation of perceptual order. *Physis: Revista Internazionale di Storia Della Scienza*, 29, 149–164.

Tweney, R.D., & Chitwood, S.C. (1995). Scientific reasoning. In S. Newstead & J.St.B.T. Evans (Eds.), Perspectives on thinking and reasoning: Essays in honour of Peter Wason (pp. 241–260). Hove, England: Erlbaum.

Wason, P.C., & Johnson-Laird, P.N. (1972). Psychology of reasoning: Structure and content. Cambridge, MA: Harvard University Press.

Traumatic Memory Is Special

Lynn Nadel and W. Jake Jacobs¹

Department of Psychology, University of Arizona, Tucson, Arizona

Does the brain represent and store memories for traumatic events differently than memories for everyday autobiographical events (cf. the June 1997 Special Issue of *Current Directions*)? Laboratory evidence is central to answering this question, and hence to understanding clinical trauma. An answer would provide a guide to how "recovered" memories should be interpreted, and would also

Recommended Reading

Jacobs, W.J., & Nadel, L. (1985). Stress induced recovery of fears and phobias. Psychological Review, 92, 512–531.

LeDoux, J.E. (1994). Emotion, memory and the brain. Scientific American, 270, 50–57.

Lupien, S.J., & McEwen, B.S. (1997). The acute effects of corticosteroids on cognition: Integration of animal and human model studies. *Brain Research Reviews*, 24, 1–27.

Sapolsky, R.M. (1998). Why zebras don't get ulcers: An updated guide to stress, stress-related diseases, and coping. New York: W.H. Freeman.

Schacter, D. (1996). Searching for memory: The brain, the mind, and the past. New York: Basic Books.

have implications for treating victims of trauma. In this article, we consider empirical data concerning the neurobiological nature of multiple memory systems, and how stress and trauma affect these systems, and then we briefly discuss the implications of these facts for the clinical issues.

EMPIRICAL DATA

On the basis of several decades of empirical work, most investigators distinguish between at least two types of memory (e.g., explicit and implicit; see Schacter & Tulving, 1994, for a variety of multiplememory-systems approaches). Going beyond a simple dichotomy, more recent research establishes that each major class of memory encompasses more than one form of memory, and the concomitant involvement of more than one underlying neural substrate. Consider explicit memory, which refers to any and all forms of recollection entering awareness. This sort of memory is most often associated with the medial temporal lobe, an

area of the brain that includes the amygdala, rhinal cortex, parahippocampal gyrus, and hippocampal formation. Although there has been a tendency to think of these structures as parts of a larger medial temporal lobe memory system, recent work indicates that each is responsible for different aspects of explicit memory. In the present context, it is particularly important to attend to these distinctions, because stress has differential impact on them.

• Amygdala. This structure is thought to be essential in memory for emotionally charged events. Studies in rats, monkeys, and humans have now shown that (a) damage to the amygdala interferes with learning about fearful or unpleasant stimuli (Adolphs, Tranel, Damasio, & Damasio, 1994; Davis, 1992; LeDoux, 1995; McGaugh, Cahill, & Roozendaal, 1996); (b) neurons in the amygdala of experimental animals are activated by stimuli with motivational or emotional import (e.g., Rolls, 1982); and (c) the human amygdala is activated when a person is exposed to emotion-provoking stimuli or events (Morris et al., 1996). By contrast, other regions in the medial temporal lobe are apparently not involved, in any general way, with such stimuli.

- Rhinal cortex. This structure is thought to be central to recognition memory, the process by which an organism determines it has, or has not, had prior experience with a particular stimulus or event. A prominent demonstration of this function concerns the laboratory task known as delayed matching (or nonmatching) to sample. In this widely used paradigm, the experimental subject is exposed briefly to a sample stimulus, and then after a variable delay, allowed to choose between the sample and another, new, stimulus. In the more commonly used nonmatching case, the subject must choose the new stimulus in order to receive reward. Monkeys, and rats, with damage to the rhinal cortex are severely impaired at this task at a wide range of delay intervals (Mumby & Pinel, 1994; Murray, Gaffan, & Flint, 1996; Zola-Morgan, Squire, Amaral, & Suzuki, 1989). By contrast, subjects with damage to the hippocampus or amygdala are either not impaired at all or impaired only under a narrow range of as yet poorly understood conditions (e.g., Gaffan, 1994). In addition to the evidence from such behavioral studies, electrophysiological analyses of the rhinal area have shown that its neuronal activity reflects recognition memory (e.g., Brown, 1996).
- Parahippocampal gyrus. This brain region is now thought to play an important role in some forms of spatial cognition. Thus, this area in humans is activated in circumstances in which individuals are thinking about moving around in space (e.g., Maguire, Frackowiak, & Frith, 1996). In people with damage to the parahippocampal region, learning about the spatial layout of a test environment is severely impaired (e.g., Bohbot et al., 1998).

• Hippocampal formation. This region has long been implicated in spatial learning and memory (O'Keefe & Nadel, 1978) and in memory for episodes (Kinsbourne & Wood, 1975; Milner, 1962). How best to characterize its precise role in memory function has been a matter of intense debate in recent years. Many investigators (Metcalfe & Jacobs, 1998; Moscovitch, 1995; Nadel, Willner, & Kurz, 1985; Squire, Cohen, & Nadel, 1984; Teyler & DiScenna, 1985) agree that the hippocampus plays a role "binding" together the elements of an episode, which themselves are represented in dispersed brain systems. That is, the hippocampus provides a mechanism by which disaggregated bits of information making up an episode can be kept in touch with one another (Jacobs & Nadel, in press). There is general agreement that the hippocampus is essential to this function for recent memories; its role in the retrieval of remote memories is a matter of considerable current debate (cf. Moscovitch & Nadel, 1998; Nadel & Moscovitch, 1997).

A key point of this proposal is that various aspects of an episode memory are represented and stored in dispersed brain modules (cf. O'Keefe & Nadel, 1978, p. 100). Also, each module interconnects with the hippocampal complex, so that the collection of representations of the features of an episode can activate within the hippocampal complex an ensemble encoding that episode. This creation of a hippocampal ensemble (or "cognitive map"; O'Keefe & Nadel, 1978) occurs rapidly, through the mechanism of long-term synaptic potentiation (a form of neural plasticity thought by many to underlie learning and memory) within the relevant hippocampal connections. An act of episode retrieval can be

accomplished in two ways: first, by activating the relevant hippocampal ensemble, which then activates dispersed extrahippocampal features, or second, by activating some subset of these dispersed features, which then activate the hippocampal ensemble. In both cases, the hippocampal component is essential to accurate reconstruction of the episodic memory.

STRESS AND MEMORY SYSTEMS

These distinctions among types of explicit memory, and their neural substrates, must be taken into account in any consideration of the ways in which stress affects memory. The data suggest that within physiological limits, stress enhances the function of the amygdala, and consequently strengthens those aspects of explicit memory subserved by this structure (cf. Metcalfe & Jacobs, 1998). The data also firmly establish that high levels of stress or the high levels of the hormone corticosterone (cortisol in humans) typically resulting from stress impair the function of the hippocampus, weakening or totally disrupting those aspects of spatial and explicit memory subserved by this structure. A number of studies, with both humans and animals, have demonstrated this now wellaccepted fact (e.g., Bodnoff et al., 1995; de Quervain, Roozendaal, & McGaugh, 1998; Diamond & Rose, 1994; Foy, Stanton, Levine, & Thompson, 1987; Luine, Villegas, Martinez, & McEwen, 1994). For example, Luine et al. (1994) induced stress levels of corticosterone in rats by restraining them in Plexiglas containers in their home cages for 6 hr/day for 21 days. When tested on an eight-arm radial maze, a widely used spatial memory task, these rats were impaired compared with nonstressed control rats (see also Kállai, Kóczán, Szabó, Molnár, & Varga, 1995; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Lupien et al., 1998, for related studies in humans).

Intriguingly, abnormally low levels of corticosterone, produced by removal of the adrenal glands, can also impair spatial learning (e.g., Conrad & Roy, 1995; Vaher, Luine, Gould, & McEwen, 1994). We, and others, have concluded that the relation between corticosterone and hippocampal function is U-shaped; that is, circulating levels of corticosterone within some optimal range yield normal function. Too little or too much corticosterone impairs function.

Thus, the laboratory data show that the relation between stress and the function of neural structures important for explicit memory is quite complex. Within a certain range, stress could enhance all forms of explicit memory, but high levels of stress could enhance some aspects of explicit memory while impairing others. And here is the critical point: When stress is high enough to impair the function of the hippocampus, resulting memories will be different from those formed under more ordinary circumstances. These empirical data suggest that memories of trauma may be available as isolated fragments rather than as coherently bound episodes (e.g., van der Kolk & Fisler, 1995). This hypothesis contrasts with the position espoused by Shobe and Kihlstrom (1997), who did not take into account the differential effects of stress on the various memory modules.

CLINICAL IMPLICATIONS

We (Jacobs & Nadel, in press) have argued that these differential effects of stress on the various components of episode memory account for several of the unusual features of memories formed under stress. Traumatic stress can cause amnesia for the autobiographical context of stressful events, but stronger than normal recall for the emotional memories produced by them. That such emotional hypermnesia may result from traumatic stress is consistent with early reports (e.g., Charcot, 1887; Janet, 1889). Even in the context of extensive autobiographical amnesia, intrusive emotions or images associated with the trauma (and related events) may appear (Jacobs, Laurance, Thomas, Luzcak, & Nadel, 1996). Intrusions appear in the context of grief, anxiety disorders, mood disorders, and dissociative disorders (syndromes involving disturbances in identity, memory, or consciousness; Brewin, Hunter, Caroll, & Tata, 1996; Gibbs, 1996; Horowitz, 1986; Howe, Courage, & Peterson, 1995), and can also be elicited in the laboratory (van der Kolk, 1994). What distinguishes these intrusive memory states is the absence of the time-and-place contextual information that typically characterizes autobiographical episode memory.

Van der Kolk and Fisler (1995) showed that after an initial phase when traumatic memories are experienced as fragmentary, an autobiographical memory eventually emerges. We have suggested that this emergence reflects a process of "inferential narrative smoothing," whereby disembodied fragments are knit together into a plausible autobiographical episode (Jacobs & Nadel, in press).

The present analysis suggests that at least some memories "recovered" during therapy should be taken seriously. Although such memories may contain emotional experiences accumulated across multiple stressful events, some of this emotional content could be veridical. The narratives associated with these memories are less likely

to be veridical in their entirety. These narratives may be composites of real fragments of experience and the emotions elicited by those experiences, filled out by tacit knowledge and logic available to the individual, and shaped by interlocutors such as friends or therapists.

Note

1. Address correspondence to Lynn Nadel, Department of Psychology, University of Arizona, Tucson, AZ 85721.

References

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*, 372, 669–672.

Bodnoff, S.R., Humphreys, A.G., Lehman, J.C., Diamond, D.M., Rose, G.M., & Meaney, M.J. (1995). Enduring effects of chronic corticosterone treatment on spatial-learning, synaptic plasticity, and hippocampal neuropathology in young and mid-aged rats. *Journal of Neuro*science, 15, 61–69.

Bohbot, V., Kalina, M., Stepankova, K., Spackova, N., Petrides, M., & Nadel, L. (1998). Spatial memory deficits in patients with lesions to the right hippocampus and to the right parahippocampal cortex. *Neuropsychologia*, 36, 1217– 1238.

Brewin, C.R., Hunter, E., Caroll, F., & Tata, P. (1996). Intrusive memories in depression: An index of schema activation? *Psychological Medi*cine, 26, 1271–1276.

Brown, M.W. (1996). Neuronal responses and recognition memory. Seminars in the Neurosciences, 8, 23–32.

Charcot, J.M. (1887). Lecons sur les maladies du système nerveux faites a la Salpêtrière [Lessons on the illnesses of the nervous system held at Salpetriere] (Vol. 3). Paris: Progres Medical en A. Delahye & Lecorsnie.

Conrad, C.D., & Roy, E.J. (1996). Dentate gyrus destruction and spatial learning impairment after corticosteroid removal in young and middle-aged rats. *Hippocampus*, 5, 1–15.

Davis, M. (1992). The role of the amygdala in fear and anxiety. Annual Review of Neuroscience, 15, 353–375.

de Quervain, D.J.-F., Roozendaal, B., & McGaugh, J.L. (1998). Stress and glucocorticoids impair retrieval of long-term spatial memory. *Nature*, 394, 787–790.

Diamond, D.M., & Rose, G.M. (1994). Stress impairs LTP and hippocampal-dependent memory. Annals of the New York Academy of Sciences, 746, 411–414.

Foy, M.R., Stanton, M.E., Levine, S., & Thompson, R.F. (1987). Behavioral stress impairs longterm potentiation in rodent hippocampus. Behavioral and Neural Biology, 48, 138–149.

Gaffan, D. (1994). Dissociated effects of perirhinal cortex ablation, fornix transection and amygdalectomy: Evidence for multiple memory systems in the primate temporal lobe. Experimental Brain Research, 99, 411–422.

Gibbs, N.A. (1996). Nonclinical populations in research on obsessive-compulsive disorder—A critical-review. Clinical Psychology Review, 16, 729–773.

- Horowitz, M.J. (1986). Stress response syndromes (2nd ed.). New York: Jason Aronson.
- Howe, M.I., Courage, M.I., & Peterson, C. (1995). Intrusions in preschoolers' recall of traumatic childhood events. Psychonomic Bulletin & Review, 2, 130–134.
- Jacobs, W.J., Laurance, H.E., Thomas, K.G.F., Luzcak, S.E., & Nadel, L. (1996). On the veracity and variability of recovered traumatic memory. *Traumatology*, 2(1) [On-line]. Available: http://rdz.stjohns.edu/trauma/traumaj. html.
- Jacobs, W.J., & Nadel, L. (in press). Neurobiology of reconstructed memory. Psychology of Public Policy and Law.
- Janet, P. (1889). L'automatisme psychologique. Paris: Alcan.
- Kállai, J., Kóczán, G., Szabó, I., Molnár, P., & Varga, J. (1995). An experimental study to operationally define and measure spatial orientation in panic agoraphobia subjects, generalized anxiety and healthy control groups. Behavioural and Cognitive Psychology, 23, 145–152.
- Kinsbourne, M., & Wood, F. (1975). Short-term memory processes and the amnesic syndrome. In D. Deutsch & J.A. Deutsch (Eds.), Short-term memory (pp. 258–291). New York: Academic Press.
- Kirschbaum, C., Wolf, O.T., May, M., Wippich, W., & Hellhammer, D.H. (1996). Stress- and treatment-induced elevations of cortisol levels associated with impaired declarative memory in healthy adults. *Life Sciences*, 58, 1475–1483.
- LeDoux, J.E. (1995). Emotion: Clues from the brain. Annual Review of Psychology, 46, 209–235.
- Luine, V., Villegas, M., Martinez, C., & McEwen, B.S. (1994). Repeated stress causes reversible impairments of spatial memory performance. *Brain Research*, 639, 167–170.
- Lupien, S.J., de Leon, M., de Santi, S., Convit, A., Tarshish, C., Nair, N.P.V., Thakur, M., Mc-Ewen, B.S., Hauger, R.L., & Meaney, M.J. (1998). Cortisol levels during human aging predict hippocampal atrophy and memory deficits. *Nature Neuroscience*, 1, 69–73.
 Maguire, E.A., Frackowiak, R.S.J., & Frith, C.D.

- (1996). Learning to find your way: A role for the human hippocampal formation. *Proceedings of the Royal Society of London*, 263, 1745– 1750.
- McGaugh, J.L., Cahill, L., & Roozendaal, B. (1996). Involvement of the amygdala in memory storage—Interaction with other brain systems. Proceedings of the National Academy of Sciences, USA, 93, 13508–13514.
- Metcalfe, J., & Jacobs, W.J. (1998). Emotional memory: The effects of stress on 'cool' and 'hot' memory systems. In D.L. Medin (Ed.), The psychology of learning and motivation: Vol. 38. Advances in research and theory (pp. 187– 222). San Diego: Academic Press.
- Milner, B. (1962). Les troubles de la memoire accompagnant des lesions hippocampiques bilaterales. In P. Passouant (Ed.), *Physiologie de l'hippocampe* (pp. 257–272). Paris: Centre National de la Recherche Scientifique.
- Morris, J.S., Frith, C.D., Perrett, D.I., Rowland, D., Young, A.W., Calder, A.J., & Dolan, R.J. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature*, 383, 812–815.
- sions. *Nature*, 383, 812–815.

 Moscovitch, M. (1995). Recovered consciousness:
 A hypothesis concerning modularity and episodic memory. *Journal of Clinical and Experimental Neuropsychology*, 17, 276–290.
- Moscovitch, M., & Nadel, L. (1998). Consolidation and the hippocampal complex revisited: In defense of the multiple-trace model. Current Opinions in Neurobiology, 8, 297–300.
- Mumby, D.G., & Pinel, J.P.J. (1994). Rhinal cortex lesions and object recognition in rats. Behavioral Neuroscience, 108, 11–18.
- Murray, E.A., Gaffan, E.A., & Flint, R.W., Jr. (1996). Anterior rhinal cortex and amygdala: Dissociation of their contributions to memory and food preference in rhesus monkeys. Behavioral Neuroscience, 110, 30–42.
- Nadel, L., & Moscovitch, M. (1997). Memory consolidation, retrograde amnesia and the hippocampal formation: A re-evaluation of the evidence and new model. Current Opinions in Neurobiology, 7, 217–227.

- Nadel, L., Willner, J., & Kurz, E.M. (1985). Cognitive maps and environmental context. In P. Balsam & A. Tomie (Eds.), Context and learning (pp. 385–406). Hillsdale, NJ: Erlbaum.
- O'Keefe, J., & Nadel, L. (1978). The hippocampus as a cognitive map. Oxford, England: Oxford University Press.
- Rolls, E.T. (1982). Neuronal mechanisms underlying the formation and disconnection of associations between visual stimuli and reinforcement in primates. In C.C. Woody (Ed.), Conditioning (pp. 363–373). New York: Plenum Press.
- Schacter, D.L., & Tulving, E. (1994). Memory systems 1994 (pp. 369–394). Cambridge, MA: MIT Press.
- Shobe, K.K., & Kihlstrom, J.F. (1997). Is traumatic memory special? Current Directions in Psychological Science, 6, 70–74.
- Squire, L.R., Cohen, N.J., & Nadel, L. (1984). The medial temporal region and memory consolidation: A new hypothesis. In H. Weingartner & E.S. Parker (Eds.), Memory consolidation: Psychobiology of cognition (pp. 185–210). Hillsdale, NJ: Erlbaum.
- Teyler, T.J., & DiScenna, P. (1985). The role of the hippocampus in memory: A hypothesis. Neuroscience and Biobeliavioral Reviews, 9, 377–389.
- Vaher, P., Luine, V., Gould, E., & McEwen, B. (1994). Effects of adrenalectomy on spatial memory performance and dentate gyrus morphology. *Brain Research*, 656, 71–76.
- van der Kolk, B. (1994). The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. Harvard Review of Psychiatry, 5, 253–265.
- van der Kolk, B., & Fisler, R. (1995). Dissociation and the fragmentary nature of traumatic memories: Overview and exploratory study. *Journal of Traumatic Stress*, 8, 505–525.
- Zola-Morgan, S., Squire, L.R., Amaral, D.G., & Suzuki, W.A. (1989). Lesions of perirhinal and parahippocampal cortex that spare the amygdala and the hippocampal formation produce severe memory impairment. *Journal of Neuroscience*, 9, 4355–4370.

Father Love and Child Development: History and Current Evidence

Ronald P. Rohner¹

Center for the Study of Parental Acceptance and Rejection, School of Family Studies, University of Connecticut, Storrs, Connecticut

Abstract

Six types of studies show that father love sometimes explains as much or more of the variation in specific child and adult outcomes as does mother love. Sometimes, however, only father love is statistically associated with specific aspects of offsprings' development and adjustment, after controlling for the influence of mother love. Recognition of these facts was clouded historically by the cultural construction of fatherhood and fathering in America.

Keywords

father love; paternal acceptance; parental acceptance-rejection theory

Research in every major ethnic group of America (Rohner, 1998b), in dozens of nations internationally, and with several hundred societies in two major cross-cultural surveys (Rohner 1975, 1986, 1998c; Rohner & Chaki-Sircar, 1988) suggests that children and adults everywhere-regardless of differences in race, ethnicity, gender, or culture-tend to respond in essentially the same way when they experience themselves to be loved or unloved by their parents. The overwhelming bulk of research dealing with parental acceptance and rejection concentrates on mothers' behavior, however. Until recently, the possible influence of father love has been largely ignored. Here, I

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.