

# Errata

In the BBS multiple book review of Donald Symons, *The Evolution of Human Sexuality*, BBS 3(2), 1980 the full reference was inadvertently omitted: Symons, D. 1979. *The Evolution of Human Sexuality*. New York: Oxford University Press.

The following commentary on Olton et al., "Hippocampus, Space, and Memory" BBS 2(3) 1979 appeared with some passages inadvertently transposed. The correct version appears here in its entirety.

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## Bait in arms: what happens when the wind blows?

Imagine a student taking a final examination in physiological psychology, and getting the following multiple-choice question: The hippocampus is important in what normal process or processes?

- a. exploring novel stimuli
- b. encoding
- c. associative learning
- d. consolidation
- e. spatial memory
- f. short-term memory
- g. recognition memory
- h. episodic memory
- i. working memory
- j. retrieval
- k. interference
- l. inhibition
- m. motivation
- n. cognitive mapping
- o. all of the above
- p. none of the above

The student mumbles, "That's what I asked myself last night. How can I answer this? There are too many variables involved for a simple choice: species, tasks, response measures, definition of 'process,' neuroanatomical complexity of the hippocampus. There can't be just one process, so it must be 'all' or 'none.' I'll pick 'o'."

Olton, et al., on the basis of their bait in arms experiments with hippocampectomized rats, presumably would *like* to choose alternative "i," although they leave open other possibilities and would *probably* choose "o." There has been such a voluminous and controversial literature on the functional significance of the hippocampus that a single explanation, attractive for its parsimony, would almost certainly tumble in the wind of empirical evidence. For example, take the evidence cited by Olton et al. that primate hippocampal function differs from that of rats, and evidence cited by Horel (1978) and others (e.g., Iversen 1977; Oscar-Berman, Sahakian and Wikmark 1976; Oscar-Berman and Zola-Morgan 1979a, b; Weiskrantz 1978) about lack of correspondence between nonhuman animal results and those obtained from human bitemporal-lobe patients. (Additional evidence and issues are reviewed by Kinsbourne and Wood 1975; Lhermette and Signoret 1976; Isaacson and Pribram 1975; Iversen 1977; Numan 1978; O'Keefe and Nadel 1978; Oscar-Berman and Zola-Morgan 1979a, b; Rozin 1976; and Weiskrantz 1978.)

However, parsimony is appealing, and just as I was intrigued by Kinsbourne and Wood's (1975) notion that human amnesics suffer from a reduced ability to use episodic memory in contrast to their intact semantic memory (Tulving 1972), my intrigue was sustained with the analogous dissociation of impaired working memory and normal reference proposed in Olton et al.'s target article to explain defects in hippocampectomized rats. My own work with human amnesics (alco-

holic Korsakoff patients) has shown certain abnormalities that are in agreement with Olton et al.'s data and hypothesis. Thus, Korsakoffs can learn to form strategies, but the individual strategies themselves are more frequently irrelevant than relevant to the solution of an ongoing problem (Oscar-Berman 1973); in a discrete-trials two-choice probability learning paradigm, Korsakoffs *eventually* learn to match responses to reinforcement contingencies, although their response-by-response patterns of behavior are somewhat abnormal (Oscar-Berman et al, 1976); Korsakoff patients are seriously impaired on delayed-response and DRL (differential reinforcement at low rates) tasks (Öberg and Oscar-Berman 1976; Oscar-Berman 1978; Oscar-Berman and Zola-Morgan 1979c), and their DRL impairment results from virtually countless errors of commission.

Despite these striking consistencies between the rat and the human data, however, other findings emphasize the limitations of the working memory explanation for many additional deficits observed in alcoholic Korsakoff patients. For example, Korsakoff patients show hyporeactive electrodermal arousal levels (Oscar-Berman and Gade 1979); they are abnormally slow in early processing stages of visual perception (Oscar-Berman, Goodglass, and Cherlow 1973); they have restricted selective attention (Oscar-Berman and Samuels 1977; Talland 1965), retarded associative learning ability (Oscar-Berman and Zola-Morgan 1979a, b), and decreased sensitivity to changing reinforcement rates (Heyman, Oscar-Berman, Bonner, and Ryder 1979). That the working memory hypothesis does not deal explicitly with these additional Korsakoff deficits is not totally disconcerting, however, because Korsakoffs are not rats, and their brain damage is not restricted to the hippocampal system. Although it is generally accepted that brain damage in alcoholic Korsakoff's syndrome includes the mammillary bodies of the hypothalamus (an essential connecting link in the hippocampal system) or the dorsomedial thalamic nuclei (Barbizet 1970; Brion 1969; Victor, Adams, and Collins 1971), or both, in fact, brain damage in chronic alcoholics and Korsakoff patients is widespread and quite diffuse (Barbizet 1970; Brion 1969; Cala, Jones, Mastaglia, and Wiley 1978; Kapur 1979; Victor, Adams, and Collins 1971; Victor and Banker 1978). Consequently the other deficits (arousal, processing, attention, etc.) may be a function of damage to areas outside the hippocampal system, to the effects of combined lesions of limbic system structures or nonlimbic (e.g., other thalamic) structures, to all of the above, or to none of the above. We just don't know yet.

In short, I am in agreement with Olton et al.'s final note of caution and skepticism about the generality of a parsimonious explanation based upon relatively few tested experimental memory paradigms especially when the data are consistent for only one species (unless the hippocampus is found not to serve homologous functions across the mammalian class). As it stands, Olton et al. have a good idea, but until we know more about the hippocampus, it may be trading accuracy for parsimony.

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