

# BRIEF COMMUNICATION

## A Method to Study Short-Term Memory (STM) in the Goldfish<sup>1</sup>

RALPH S. RYBACK<sup>2</sup>

*McLean Hospital and Harvard Medical School*

(Received 19 June 1975)

RYBACK, R. S. *A method to study short-term memory (STM) in the goldfish.* PHARMAC. BIOCHEM. BEHAV. 4(4) 489–491, 1976. – Twenty-one common goldfish (13–15.5 cm long) were randomly divided into alcohol (A) and nonalcohol (NA) groups and were trained in an alcohol solution of 400 mg/100 ml or in water, respectively. All alcohol fish were placed in an alcohol solution of 400 mg/100 ml for 3 hr before training in the same alcohol concentration. Fish were trained on a position discrimination task for 2 consecutive days. The door used for training was that opposite to each fish's spontaneous preference. Savings in relearning on Day 2 was taken as a measure of long term memory strength. Only fish which reached criterion on both days were immediately given 10 forced reversal trails in the opposite direction (i.e., a fish trained on right door was forced to choose the left door.) A and NA subjects were then tested after a 5 min (STM) delay, respectively, in a free choice situation for 10 trails (i.e., neither door was blocked). The results suggest that alcohol facilitates the STM of the forced reversal information.

Memory    Alcohol    *Carassius Auratus*

THE process of retention can be divided into 3 phases: (1) immediate memory, (2) short-term memory (STM), and (3) long-term memory (LTM) [21]. Moderate to excessive alcohol intake in man has been associated with a range of effects on memory including dissociative states [9, 10, 11, 15, 23], alcohol amnesia or blackout [6, 7, 19], and the Wernicke-Korsakoff Syndrome [5, 12, 22, 25]. A question that has created a good deal of controversy within the literature on amnesia in Korsakoff patients has been whether they have an intact or impaired STM. Warrington and her collaborators [1,26] have presented a great deal of evidence to suggest that the amnesia is a deficit in LTM, but that STM is essentially normal, while Cermak and Butters [4] have provided examples of patients who have LTM deficits combined with gross impairments in STM as well. A similar controversy has arisen around the effects of alcohol on STM in man. Goodwin *et al.* [8], Tamerin *et al.* [24] and Ryback [19] all found an impairment in STM in intoxicated alcoholics, while Mello [13] did not. Carpenter and Ross [3], Nash [14], Ryback *et al.* [18] have demonstrated disruption of visual STM in nonalcoholics with relatively low doses of alcohol. It is likely that the latter discrepant findings are in part related to a number of variables including the definition of STM used [7, 10, 21, 23], the type of subjects used, the amount of absolute alcohol consumed, and the type of STM test (e.g., verbal or nonverbal). Accordingly, an experimental method and/or

subject which might [16,17] better define some of these variables and test nonverbal "STM" and "LTM" would be helpful. Such a method in goldfish with some initial findings are presented.

### METHOD

Fishes were 21 common goldfish, 13–15.5 cm long, obtained from Ozark Fisheries (Stoutland, Missouri) from a group of 24 fish, of which 3 (2 NA and 1 A) failed to meet the criterion. The training apparatus, which involved a position discrimination task, was constructed in the shape of a trapezoid and was set in the center of a square shallow tank (20 cm deep with 75 cm sides). Fish entered the 5 cm wide apex of the trapezoid which had a 23 cm base and could exit through 1 of 2 opposing doors each placed in the sides 52 cm from the apex and 2.6 cm from the base. An incorrect choice was determined by the fish bumping into a glass barrier inserted behind the incorrect door. Fish were randomly divided into alcohol (A) and nonalcohol (NA) groups and were trained in an alcohol solution of 400 mg/100 ml or in water, respectively. All alcohol fish were placed in an alcohol solution of 400 mg/100 ml for 3 hr before training and relearning in the same alcohol concentration. The door used for training was that opposite to each fish's spontaneous preference (determined the day prior to training by an unimpeded run of 10 trials).

<sup>1</sup> This study was supported in part by General Research Support Grant FR05484.

<sup>2</sup> Now at NIAAA, Rm. 16105, 5600 Fishers Lane, Rockville, MD, 20852.

TABLE 1

PERFORMANCE OF NONALCOHOL (NA) AND ALCOHOL (A) FISH IN A 2 DOOR POSITION DISCRIMINATION TEST ON DAY 1 (LEARNING), DAY 2 (RELEARNING OR LTM) AND RETENTION (I.E., PREFERENCE FOR ORIGINAL CHOICE) AFTER A 5 MINUTE DELAY FOLLOWING 10 FORCED REVERSAL TRIALS.

Mean Score	Group Nonalcohol (NA)		Retention after delay	Group Alcohol (A)		Retention after delay
	Trials to criterion Day 1	Trials to criterion Day 2		Trials to criterion Day 1	Trials to criterion Day 2	
	7.0	2.0	8.2*	7.0	1.8	4.7

\* $p < 0.005$  (1 tail  $t$ -Test) for Group NA retention after delay (8.2) as compared to Group A (4.7).

Training proceeded as follows: (1) A criterion of 9 out of 10 correct choices was used and fish were allowed up to 20 trials (approximately ½ hr) daily for 2 consecutive days unless the fish began a criterial run before the fourteenth trial. Then up to 23 trials were allowed. Savings in relearning on Day 2 was taken as a measure of LTM strength; (2) Only those fish which reached criterion on both days were immediately given ten forced reversal trials in the opposite direction (i.e., a fish trained on right door was forced to choose the left door by covering up the trained or right door). These forced reversal trials took 15 min; (3) A and NA fish were then tested after a 5 min (STM) delay, respectively, in a free choice situation for 10 trials (i.e., neither door was blocked).

#### RESULTS

A and NA subjects reached criterion the first day with means of 4.5 and 4.1, respectively, for the mean number of trials before the criterion run began. The scores for these A and NA fish were 7.0 and 7.0 trials on Day 1 and 1.8 and 2.0 trials on Day 2, respectively (Table 1). NA and A fish returned to the first learned choice when tested in the free choice situation 5 min after the 10 forced reversal trials, with means of 8.2 and 4.7, respectively.

#### DISCUSSION

Some readers might interpret the work of Beritashvili [2] to be in conflict with the data presented. He found that if the time interval between raising a transverse partition and feeding was more than 10 sec that fish would not swim to the food but elsewhere. He also found that if an electrical stimulation was given during feeding that the fish would immediately swim away only to return after 10–12 sec. He called these findings "emotional memory in fish". Some readers might deduce that STM was therefore 8–12 sec in a fish. However, we have no information as to whether or not these fish were food deprived. Moreover, hunger is a drive which usually initially results in increased swimming behavior in fish. If a fish is not hungry it might attend to the raising of the partition for 8–10 sec consistent with the concept of IM and then swim away. If it

were actually hungry enough to feed and were shocked during feeding it would swim away but might return shortly not because of an impairment in memory but rather because of the conflicting drive of hunger. The latter (i.e., feeding versus pain or anxiety) has been used clinically as a prototypic model for systematic desensitization [27]. Finally, Beritashvili's work with conditioned-reflex memory in fish is not in conflict with the findings presented here.

These findings (Table 1) show that the present alcohol treatment did not adversely affect the rate of initial learning (Day 1) or of relearning or LTM (Day 2). However, the strength of STM is weaker in normal than in alcohol fish. It might be suggested that alcohol at this dose increases the decay rate of the previously learned task (Day 2), thus resulting in less reversion of alcohol subjects to the first learned choice 5 min after the forced reversal trials. This is not likely as neither original learning (Day 1) nor relearning or LTM (Day 2) was adversely affected. The tendency of alcohol fish, as compared with controls, not to revert to the previously learned choice 5 min after the forced reversal trials might suggest that alcohol (400 mg/100 ml) facilitated the STM of the forced reversal information. Of course it could be argued that alcohol facilitates learning of the forced reversal rather than the STM. However, it is interesting that several investigators with normal and alcoholic human subjects found impairment after alcohol in STM while IM and LTM were relatively unaffected [3, 8, 18, 19, 24]. Accordingly, it is possible that depending on the dose, alcohol may facilitate or inhibit STM [20]. Moreover, some pilot work (unpublished data) suggests that if alcohol fish are removed from the tank and returned 1 hr after the 10 conflictual trials, they revert to a mean score of 8.0 or the same (Table 1) as controls (8.2) in favoring the choice of the original door on which they were trained (LTM). Obviously, further research would necessitate the addition of various alcohol concentrations, and groups with varying time delay intervals after the 10 conflictual trials so that a gradient of STM to LTM alcohol effects could be delineated. Finally, this memory model could also be used to study the effect of chronic alcohol exposure (i.e., "Wernicke-Korsakoff" type syndrome) in the goldfish.

#### REFERENCES

1. Baddeley, A. D. and E. K. Warrington. Amnesia and the distinction between long and short-term memory. *J. verb. Learn. verb. Behav.* 9: 176–189, 1970.
2. Beritashvili, I. S. *Vertebrate Memory Characteristics and Origin*. New York: London: Plenum Press, 1971, pp. 92–96.
3. Carpenter, J. A. and B. M. Ross. Effect of alcohol on short-term memory. *Q. Jl Stud. Alc.* 26: 561–579, 1965.
4. Cermak, L. S. and N. Butters. The role of interference and encoding in the short-term memory deficits of Korsakoff patients. *Neuropsychologia* 10: 89–95, 1972.

5. Cermak, L. S., N. Butters and H. Goodglass. The extent of memory loss in Korsakoff patients. *Neuropsychologia* 9: 307-315, 1971.
6. Goodwin, D. W., J. B. Crane and S. B. Guze. Alcoholic blackouts: A review and clinical study of 100 alcoholics. *Am. J. Psychiat.* 126: 191-198, 1969.
7. Goodwin, D. W., J. B. Crane and S. B. Guze. Phenomenological aspects of the alcoholic blackout. *Br. J. Psychiat.* 115: 1033-1038, 1969.
8. Goodwin, D. W., E. Othmer, J. A. Halikas, and F. Freeman. Loss of short term memory as a predictor of the alcoholic "blackout." *Nature* 227: 201, 1970.
9. Goodwin, D. W., B. Powell, D. Bremer, H. Hoine and J. Stern. Alcohol and recall: State-dependent effects in man. *Science* 163: 1358-1360, 1969.
10. McNamee, H. B., N. K. Mellow and J. H. Mendelson. Experimental analysis of drinking patterns of alcoholics: Concurrent psychiatric observations. *Am. J. Psychiat.* 124: 1063-1069, 1968.
11. McQuire, M. T., J. H. Mendelson and S. Stein. Comparative psychosocial studies of alcoholic and non-alcoholic subjects undergoing experimentally induced ethanol intoxication. *Psychosom. Med.* 28: 13-26, 1966.
12. Meissner, W. W. Learning and memory in the Korsakoff syndrome. *Int. J. Neuropsychiat.* 4: 6-21, 1968.
13. Mello, N. K. Short-term memory function in alcohol addicts during intoxication. In: *Alcohol Intoxication and Withdrawal*, edited by M. M. Gross. New York: Plenum Publish, 1973, pp. 333-343.
14. Nash, H. *Alcohol and Caffein: A Study of Their Psychological Effects*, Springfield, Illinois: C. C. Thomas, 1962.
15. Overton, D. A. State-dependent learning produced by alcohol and its relevance to alcoholism: In: *The Biology of Alcoholism: Vol. II, Physiology and Behavior*, edited by B. Kissin and H. Begleiter. New York: Plenum Press, 1972, pp. 193-217.
16. Ryback, R. The use of goldfish as a model for alcohol amnesia in man. *Q. Jl Stud. Alc.* 30: 877-882, 1969.
17. Ryback, R., B. Percarpio, J. Vitale. Equilibrium and metabolism of ethanol in the goldfish. *Nature* 222: 1068-1070, 1969.
18. Ryback, R., J. R. Winert and J. Fozard. Disruption of short-term memory in man following consumption of ethanol. *Psychon. Sci.* 20: 353-354, 1970.
19. Ryback, R. S. Alcohol amnesia: Observations on seven drinking inpatient alcoholics. *Q. Jl Stud. Alc.* 31: 616-632, 1970.
20. Ryback, R. S. Facilitation on inhibition of learning and memory by alcohol. *Ann. N. Y. Acad. Sci.* 215: 187-194, 1973.
21. Shuttleworth, E. C. and C. E. Morris. The transient global amnesia syndrome; a defect in the second stage of memory in man. *Archs Neurol.* 15: 515-520, 1966.
22. Talland, G. A. *Deranged Memory*. New York: Academic Press, 1965.
23. Tamerin, J. S., S. Weiner and J. H. Mendelson. Alcoholics' expectancies and recall of experiences during intoxication. *Am. J. Psychiat.* 126: 1697-1704, 1970.
24. Tamerin, J. S., S. Weiner, R. Poppen, P. Steinblase and J. H. Mendelson. Alcohol and memory: Amnesia and short-term function during experimentally induced intoxication. *Am. J. Psychiat.* 127: 1659-1664, 1971.
25. Victor, M., G. A. Talland and R. D. Adams. Psychological studies of Korsakoff's psychosis: I. General intellectual functions. *J. nerv. ment. Dis.* 128: 528-537, 1959.
26. Warrington, E. K. and L. Weiskrantz. An analysis of short-term and long-term memory defects in man. In: *The Physiological Basis of Memory*, edited by J. A. Deutsch. New York: Academic Press, 365-395, 1973.
27. Wolpe, J. *Psychotherapy by Reciprocal Inhibition*. Stanford, Calif.: Stanford University Press, 1958, p. 71.