ORAL CONSUMPTION OF LECITHIN: ENHANCEMENT OF MEMORY AND LEARNING IN MAN

by

WAYNE MARSTON CLARK
B.A., University of the Pacific, 1979

A THESIS SUBMITTED IN PARTIAL FULFULLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF ARTS

in the Department
of
Psychology

ACCEPTED
FACULTY OF GRADUATE STUDIES
[redacted]
DEAN
DATE 5 Feb 1982

We accept this thesis as conforming
to the required standard

[redacted]
(Frank Spellacy, Ph.D.)

[redacted]
(Michael Corcoran, Ph.D.)

[redacted]
(William Dyson, M.D.)

ABSTRACT

Supervisor: Professor Frank Spellacy

[redacted]

Lecithin has been proposed as a possible cholinergic agonist and as such may be useful as a mechanism to improve memory and learning. This study looked at lecithin's effect on memory in two separate experiments, one using a single large dose of lecithin and the other using a week long period of lecithin consumption. Thirty university students participated in each study. They were randomly assigned to one of three groups: one group that received lecithin the first week (either for the entire week or a single large dose) and a placebo the next, one group that reversed this order, and a control group that received placebo both weeks. The subject's memory was measured by five memory and learning tests. A difference score was computed for each subject by comparing his/her scores on the two testing sessions. The average difference scores for each of the treatment groups were then compared to the control group. In neither study

did lecithin produce a significant effect on memory. The extraneous variables that may have produced this null effect are discussed. In general, this study does not support the previous suggestions in the literature that state that oral administration of lecithin may improve memory and learning.

Examiners:

[redacted]

(Frank Spellacy, Ph.D.)

[redacted]

(Michael Corcoran, Ph.D.)

[redacted]

(William Dyson, M.D.)

ACKNOWLEDGEMENTS

The author wishes to thank the members of my committee, Dr. W. Dyson and Dr. M. Corcoran, for their assistance and support. A special thanks goes to my chairman, Dr. F. Spellacy, for his involvement and support throughout my graduate training.

Further thanks are extended to Dan Seabaugh for raising the question; to Anthony Risser for his assistance; to my parents for their continued support; and to Tamira Anderson for her help in so many ways.

CONTENTS

LIST OF TABLES

INTRODUCTION

Our memory is an important, if not indispensable, part of our daily cognitive functioning. It comprises two basic events: the storage of new information (learning), and the retrieval of previously stored information. Memory is generally broken down into two parts: a short term memory (STM) encompassing events that have occurred in the previous 20 seconds, and a long term memory (LTM) for events occurring over 20 seconds in the past (Klatzky,1980). Given the importance of memory, it would be useful to discover a safe, orally administered substance that would improve memory both in memory-impaired clinical populations and in normal individuals. Although the neural mechanisms underlying memory are yet to be discovered, the cholinergic system (neurons that use the neurotransmitter acetylcholine, ACh) is believed to play an important role. This study examines the effects on memory of an safe, orally administered precursor to ACh, lecithin.

The role of acetylcholine in learning and memory has been supported by numerous studies that suggest that modifi-

cations of cholinergic synapses form the physiological basis of memory (Biederman,1970; Deutsch,1971; Deutsch & Rogers, 1978; Hamburg,1967; Karczmar,1975; Signorelli,1976; Squire,1970; Statens,1976). In these experiments examining long-term memory animals were first taught a behavior, e.g. to run into a particular alley of a Y maze. Then at intervals ranging from 30 minutes to 28 days the animals were retested after being given a cholinergic drug. Their performance on the behavior was then assessed to determine the drug's effect on retention of learned behavior. It was found that the influence of the drug varies with the type of drug used, with the dosage employed, and with the age (strength) of the memory. Drugs that increase ACh (cholinergics,e.g. physostigmine) improve the retrieval of behaviors learned more than three days before injection but tend to impair the retrieval of behaviors learned in the previous one to three days. Drugs that reduce or interfere with ACh (anticholinergics) almost always impair retrieval. Deutsch and Rogers (1979) suggested that the cholinergic amnesia was due to a synaptic blockade (a decreased ability to respond to stimulation) by excess acetylcholine, and that the anticholinergic amnesia was due to a lack of acetylcholine. In either case a role for acetylcholine in memory and learning is indicated.

Numerous studies on the effect of cholinergic drugs on various aspects of human memory have also been conducted. In general, anticholinergics (e.g. scopalamine, atropine) have been found to impair certain aspects of human memory. In a series of studies Drachman (1966,1971,1974,1977a,1977b,1978) looked at scopolamine's effect on immediate memory, memory storage, and retrieval from old storage in young normal subjects. The subjects were either given one mg. of scopolamine, treated with methscopolamine (to control for peripheral effects), or used as a control. In all of these studies scopolamine markedly interfered with the subject's ability to retrieve old memories. However, short-term memory, as measured by the digit-span test, was spared. Ostfeld and Aruguette (1962) found that scopolamine interfered with the subject's ability to recall 10 common objects or to repeat paragraphs. Ghoneim and MeWaldt (1975,1977) found that scopolamine impaired long term memory in normal subjects. The deficit appeared to be in the storage process, and the retrieval process was unaffected. Studies by Petersen (1977) also support the view that scopolamine interferes primarily with the acquisition of new information (learning). Additional research with scopolamine and other anticholinergics has produced a wide range of deficits in memory and learning (Crow, Grove & White, 1971; Safer & Allen,

1971; Sitaram & Weingartner, 1977). Thus, in normal human subjects centrally acting anticholinergic agents appear to produce marked impairment of memory storage, some impairment of retrieval, and little or no impairment of short-term memory.

The role of cholinergic agonists in human memory has also been investigated. These agonists work by inhibiting acetylcholinesterase, the enzyme that breaks down ACh, and thus they slow the destruction of ACh and increase the concentration of ACh at the synapse. Davis, Hollister, Overall, Johnson, and Train (1976) gave either varying dosages of physostigmine or a saline solution to young adults and observed the effects on LTM. Results indicated that the drug impaired memory in addition to producing adverse side effects. The experimenters concluded that all dosages used were too high and in a second study a smaller level was employed (Davis, Mohs, Tinklenburg, Pfefferbaum, Hollister, & Kopell, 1978). In this study subjects were able to recall more words on Buschke's (1973) selective reminding task (measures both storage and retrieval) under the drug condition, but only when tested at 80 minutes. This result appears to support Deustch's theory that only the older memories will be aided by a cholinergic agonist. A second agonist, arecoline, has also been shown to produce a similar

improvement in the number of words recalled (Sitaram, Weingartner, & Gillin, 1978). In addition it was found that the improvement produced by arecoline was inversely related to baseline performance, that is, the subjects who performed the worst in baseline showed the largest improvement. In a separate study Drachman and Leavitt (1974) gave one mg. of physostigmine to young normal subjects and found their LTM performances showed a trend toward improvement. In general it appears that small dosages of cholinergic agonists produce small increases in memory recall, particularly on recall tasks with a long delay period.

The results of the findings on cholinergic drugs and memory indicate that ACh does play an important role in memory and that there appears to be a critical level of ACh required, with either too much or too little producing a memory deficit. The maintenance of this optimal ACh level both in clinical disorders and in normal functioning indicates the need for a long acting, safe oral cholinergic agonist. Since both physostigmine and arecoline have short durations, approximately one hour, and for the most part are applied via injection, they appear to be ruled out. One substance that has recently been proposed as an answer is choline chloride.

Choline is the precursor to ACh. The reaction involves combining choline and acetylcoenzyme A in a process catalyzed by choline acetlytransferase (Ansell & Spanner, 1977). The brain is unable to make choline de novo and extracts it instead from the systemic circulation. There is evidence that choline is primarily taken up into the axon terminal buttons by a sodium-dependent high affinity system that is localized in the subcellular components that synthesize ACh. Choline uptake is linearly related to the plasma choline level, and the system is not saturated even at high plasma choline levels (Freeman, Choi, & Jenden, 1975; Haga & Noda, 1973; Yamamura & Synder, 1973). The plasma choline level therefore appears to determine how much choline is available for ACh synthesis.

Choline's ability to increase ACh concentrations was first demonstrated by Cohen and Wurtman (1975). These investigators administered choline intraperitoneally to rats and found this led to an increase in both the plasma choline level and the brain ACh level. The increase in plasma levels of choline (22 percent) is maximal 40 minutes after injection, is dose dependent, and lasts for up to 80 minutes. Other investigators found that the injection of choline in rats caused an increase of choline and ACh in the corpus striatum (Haubrich & Chippendale, 1977; Haubrich & Reid,

1972; Haubrich, Wang Clody, & Wedenking, 1975). Hirsch and Wurtman (1977) found similar results in the hippocampus. Comparable results have been obtained in mice (Hanin & Schuberth, 1974; Haurbrich, Reid, & Gillette, 1972), and in guinea pigs (Haubrich, Wang, & Weduking, 1974). Cohen and Wurtman (1976) have also shown that ACh concentrations in the brain and free blood choline concentrations vary with dietary (oral) choline consumption. They found a 45 percent increase in the brain ACh concentration when rats consumed 20 mg. of choline. Dietary choline's ability to increase plasma choline levels in man has also been studied (Aquilonious & Eckernas, 1975; Growdon, Cohen, & Wurtman, 1977). The increase in plasma choline levels were found to be directly proportional to the amount of choline ingested. The increase was maximal 90 minutes after ingestion. However, large individual differences were found in the increases in plasma levels. These findings indicate that synthesis of brain ACh can be stimulated in vivo by elevating the concentration of its precursor.

Other studies in rats have suggested that choline administration not only increases brain ACh levels but also increases the amount of ACh that is released (Ulus, Hirsch, & Wurtman, 1977; Ulus, Scally, & Wurtman, 1977; Ulus & Wurtman, 1976). These studies have found that the activity of

tyrosine hydroxylase (an enzyme in the dopamine system) is increased by the administration of choline. It has previously been demonstrated that substances that increase the amount of ACh being released also increase the activity of tyrosine hydroxylase (Roth, Salzman, & Nowycky, 1978). This indirectly indicates that more ACh is being released after administration of choline. However, the induction of tyrosine hydroxylase also occurs after a wide variety of drug manipulations (Raese, Patrick, & Barchas, 1976), so the finding does not constitute a proof.

Additional indirect evidence that choline can increase brain ACh levels in man comes from the numerous clinical studies of choline treatment for disorders thought to be caused by deficiencies in cholinergic mechanisms (Spillance, White, Goodhart, & Flack, 1977). Dietary administration of choline to patients with Alzheimers disease (presenial dementia) has produced moderate improvements in overall cognitive functioning (Boyd, White, Graham, & McQueen, 1977; Signoret, Whiteley, & Lhermitle, 1978; Smith Swash, & Baily, 1978). In general, the less advanced cases were the most responsive to treatment. Administration of choline to patients with Tardive Dyskinesia, a movement disorder thought to be caused by a cholinergic deficiency, has been largely successful (Davis, Berger, & Hollister, 1975;

Davis, Hollister, Barchas, & Berger, 1976; Growdon, Hirsch, & Wurtman, 1977). These double-blind controlled studies indicate that the administration of choline improves some symptoms of Tardive Dyskinesia. However, the small sample sizes of these studies leaves in doubt the conclusiveness of the findings. Taken together these neurochemical, behavioral, and clinical studies favor the theory that choline increases brain cholinergic activity.

If this theory is correct, choline should produce memory effects similar to those produced by cholinergic agonists, for example, physostigmine and arecoline. The findings to date have been discrepant. One group of investigators has found choline to improve long-term learning and memory in normals (Sitaram, Weingartner, Caine, & Gillin, 1978; Sitaram, Weingartner,& Gillin,1978,1979). Using an uncategorized serial learning test and Bushke's selective reminding task as dependent variables, the examiners compared the effects of 10 grams of oral choline to a placebo, with 10 subjects in each condition. Ninety minutes after consumption the performance on the two tasks was measured and compared to the subjects' baseline levels. The subjects then repeated the procedure on a separate day under the opposite condition. The results indicate that subjects took fewer trials to learn the serial learning task (10 words)

in the choline condition. For the selective reminding tasks the 12 words were classified as either high imagery (easy to visualize) or low. The results indicated that choline enhanced storage and recall of the low imagery words but not the high ones.

In contrast to these significant results are the findings of the Davis group (Davis, Mohs, Tinkleburg, Hollister, Pfefferbaum, & Kopell, 1980; Mohs, Davis, & Darley, 1979). In these studies subjects were tested on a digit span test STM, a 15-item word learning and retrieval task, and the selective reminding task. The choline was orally consumed four times daily (14 grams per day) for three days. The researchers found no significant difference between the choline subjects and controls on any of the tasks either in a normal group or in an elderly normal population. Finally, in patients with early Alzheimers Disease, Signoret, Whitely, and Lhermitle (1978) found improvements in long-term learning and recall as well as in daily functioning in patients taking oral choline.

There are many possible reasons for these discrepancies. First, the majority of subjects in the Sitaram studies were female; those in the Davis studies were all males. The dosages of choline, 10 grams 90 minutes before testing in the

Sitaram study and 14 grams given throughout the day in the Davis study, could have produced the differences due to the sensitive nature of the ACh balance suggested by Deutsch. There could have been an overdosage of choline in the Davis study, for example. Finally, the small sample sizes compared to a relatively large number of dependent measures makes any statements of significance questionable. The true effects of choline can only be determined through further replication research.

Choline therefore appears to have a possible application as an agent in memory and movement disorders, and as well it may provide a possible method of maintaining optimal ACh levels and improving memory in normals. Unfortunately, choline produces some adverse side effects. These include nausea, slght depression, and a marked fishy odor. This odor is thought to be caused by choline degradation in the intestine to yield trimethylamine. These side effects, particularly the odor, have been reported in most of the studies involving choline. These side effects along with a relatively short duration of action limit its clinical usefulness.

Lecithin (phosphatidyl choline), the normal dietary form of choline, has been proposed as a possible substitute

for choline. In lecithin, choline is bound to a phospholipid moiety. The normal metabolic fate of lecithin apparently differs from that of free choline. Lecithin appears to be resistant to enzymatic breakdown and consequently does not produce trimethylamine (Growdon, Gellenberg, Hirsch, & Wurtman, 1978). Nor do any of the other choline-induced side effects occur during lecithin consumption.

Lecithin's ability to increase ACh was first investigated by Hirsch and Wurtman in 1978. In this study rats that consumed a single meal of lecithin showed a significant increase in the concentration of free choline and ACh in their brain and adrenal gland. These investigators also looked at lecithin's ability to raise serum choline levels in man (Hirsch, Growdon, & Wurtman, 1978; Wurtman, Hirsch, & Growdon, 1977). Volunteers consumed either free choline or lecithin granules containing an equivalent amount of free choline base. Lecithin produced an increase in plasma choline that was greater than that under choline (an increase of 256 percent compared to 86 percent) and the elevation lasted far longer (12 hours compared to four). Houtsmaller (1979) gave normal subjects half the lecithin dose (10 grams) as in the previous studies. He found the increase in plasma choline levels to be comparable, but the duration of this increase was different, lasting six hours instead of

12. A comparison of the experiments suggest that plasma choline levels peak around .03 mmol. and that larger doses of lecithin only increase the time this level is maintained. This suggests that the critical variable in any lecithin study may be the interval since administration and not the dosage level. The ability of lecithin to produce a higher and longer-lasting plasma choline increase than that produced by choline may be due, in part, to lecithin's greater resistance to enzymatic degradation. An additional possibility for the increase follows from the theory that it is a lipid-bound form of choline that is carried in the blood to the brain (Ansell & Spanner, 1971; Freeman, Choi, & Jenden, 1975). It may therefore be easier for lecithin to enter the circulation directly. It should be noted that the technique used to measure choline levels first removes any bound lipids. Whether it was originally lecithin or free choline in the blood cannot be determined (Schea & Aprison, 1973).

Several clinical studies have examined lecithin's effectiveness in treating disorders that are thought to involve cholinergic deficiencies. Gelenberg, Wojeik, and Growdon (1979) looked at lecithin's ability to treat Tardive Dyskinesia. Either lecithin (105 grams) or choline was administered to patients in three daily dosages. All patients improved on both treatments with a tendency toward

greater improvement under lecithin. However, lecithin did not produce any of the adverse side effects that were seen with choline. Similar results have been found by Growdon, Gelenburg, Doller, Hirsch, and Wurtman (1978).

Barbeau (1978a,1978b,1978c,1979) has looked at lecithin as a treatment for a variety of movement disorders. In patients with Tardive Dyskinesia given 30 grams of lecithin daily there was a marked reduction in the observed abnormal movements. The patients indicated they felt a marked reduction in the underlying face and neck muscle tension and resulting movements. In 10 cases of Friedreich's ataxia there was an average of 35 percent improvement in movement performance scores when the patients ingested 30 grams of lecithin. In contrast to the above positive therapuetic results, lecithin was not found to be effective in Gilles de la Tourette's disease (a disorder characterized by involuntary verbal and motor tics) or in Huntington's Chorea. In general, these uncontrolled case studies offer possible support for lecithin's potential therapuetic application. More studies using a controlled double-blind approach should be undertaken.

Finally, Christie, Blackburn, Glen, Zeisel, Shering, and Yates (1979) have looked at lecithin treatment of

Alzheimer's disease. This disorder appears to be associated with a selective degeneration of central cholinergic neurons and produces a general dementia, particularly involving memory impairment. In this study 12 patients were maintained on either choline or lecithin alternately. Twenty-five grams of lecithin or 1.25 grams of choline were given four times daily orally to the subjects. There was no improvement seen in patients under the choline treatments; however, both the nurses' behavioral ratings and numerous memory/learning tests showed improvements in three of the less severely affected patients under lecithin. This improvement vanished when the subjects were under the choline condition. The authors suggest that lecithin treatments may at least slow down the dementing process. In a seperate study Etienne, Gauthier, Dastor, Gollier, and Ratner (1979) examined lecithin's treatment potential in seven patients in the early stages of Alzheimer's disease. The patients consumed between 25 and 100 grams of lecithin powder daily. Psychological tests showed no change in retrieval, constructional ability, or the ability to recognize faces. However, the new learning ability of three patients improved during the lecithin treatment as measured by the paired associate learning test from the Weschler memory scale. No side effects were reported under lecithin. The authors suggest

that these three cases may have been somehow less impaired and therefore better able to respond to increased ACh therapy. Although these two studies show some lecithin-related improvements, it may be that these are just chance findings given the small sample size and large number of dependent variables employed. Once again the studies need to be carefully replicated.

Overall, the clinical effectiveness of lecithin therapy is unclear. It appears to be helpful in some disorders, mainly in less advanced cases, but not in others. Although it is too early to make a definitive statement, the clinical and biochemical studies on lecithin indicate that it may increase ACh levels in the brain and improve memory in clinical disorders.

The application of lecithin as a cholinergic agonist with regard to memory in normals has not yet been examined. If lecithin does indeed increase the levels of ACh, a memory improvement similar to that seen with physostigmine in normals would be expected. In review these improvements were seen in delayed recall paradigms and for difficult (low imagery) words.

The present study looked at lecithin's effect on memory and learning as measured by five tests: a short-term

memory test, a selective reminding recall test, a paired associated learning test, a serial learning test, and a visual-spatial memory test. Although previous studies have not demonstrated a significant cholinergic effect on STM or visual-spatial memory, these two tests were included in an effort to sample all areas of normal human memory. Based on previous studies it was expected that lecithin would significantly increase the performance of normal subjects on these tasks as compared to a control group. Two experiments were undertaken. The first one looked at the effect of a single large dose of lecithin as measured six hours later; the second experiment examined the effects of a week long period of lecithin ingestion. Finally, it should be noted that this study was concerned with lecithin's potential effectiveness for day-to-day normal use. For this reason all lecithin preparations used were commercially available and all dosages were within the recommended level stated on the containers.

EXPERIMENT ONE

Method

Subjects

College student volunteers were recruited from four classes at the University of Victoria. An additional four university employees also volunteered for a total of 30 subjects, 20 females and ten males. The ages of the subjects ranged from 20-35 years with the average age 25. The subjects were randomly assigned to the three groups, two treatment groups and one control (see table one), by first randomly selecting three subjects and then randomly assigning each subject to one of the three groups. This produced ten subjects in each experimental condition.

Lecithin

The lecithin used was a Soya Lecithin Powder (20 percent phosphatidyl choline) produced by Trophic Canada LTD. All information regarding the composition of the lecithin preparation was obtained directly from the company without

independent verification. Since commercial preparations were used the purity may have varied from sample to sample. This study was interested in the effects of a relatively large single dose; therefore a level of thirty grams was used. This level is similar to that used in the Barbeau studies and it was the maximum recommended level stated on the container. These 30 grams (three tablespoons) were then mixed with an oatmeal muffin mix to yield 15 grams in each of two muffins. Ground corn meal was used as a placebo, with an equivalant amount (15 grams) mixed into each control oatmeal muffin. The lecithin and placebo muffins were found to be indistinguishable to naive testers. Mixing lecithin into baked goods was the method of consumption recommended by the manufacture; the heating process does not change lecithin's structure (information supplied by manufacturer).

Memory Tests

Five different memory tests were employed. Two forms of each test were constructed and were found to be equivalent in a pilot study. A brief description of each test follows; please see appendix one for an example of each test.

Bushke's restricted reminding task. A shortened form of Bushke's test (six trials compared to 12) was used, with Form One being a list of 20 animals and Form Two being a list of 20 foods. The list was first read to the subject with the subject repeating each item. At the end of the list they were then asked to recall as many of the items as possible. When a subject could not recall any additional items within a 15-second period, they were told the items they had not yet recalled. The subject was then asked to recall all the words, both the ones remembered the first time and the ones of which they needed to be reminded. This procedure was repeated for a total of six trials. A subject was only reminded of a word until it was recalled once; therefore, the later trials involved the subject recalling words without any additional reminders. Twenty minutes after the sixth recall trial the subject was asked to recall as many of the items as possible without any reminding; this constituted a seventh trial. A subject's score was determined by all the words recalled on the seven trials, a possible range of 0-140. According to Bushke (1973) this task tests long-term memory and recall.

Digit recall. The digit recall test taken from the Weschler memory test was used. Subjects were first asked to repeat a series of digits of increasing length. The longest

length list correctly recalled was the subject's digit forward score. The subjects were then asked to recall an increasing length series of digits backwards, with the longest series recalled being the subject's score. The digit forward and backward scores were then combined to yield the total digit test score.

Associate word learning test. In this test subjects attempted to learn 12 pairs of words. The words were chosen to have little or no easy associations between them. This was done to make the task more difficult than the standard Weschler associate learning task. The first time through, both words in each pair were read to the subject. After all 12 pairs had been given the first word in each pair was then read and the subject tried to recall its partner. The correct answer, if missed, was not given by the experimenter. This procedure (both words then recall of the partner) was repeated for a total of three trials unless the subject correctly recalled all the partners earlier. The score was the total number of correctly recalled pairs, with the subject receiving full credit on trials not given. This test was designed to measure associate learning.

Colored marker test. In this test the subjects were first shown a white board divided into 36 squares, six by six. Next, seven different colored markers were shown to the subjects. They were then instructed to shut their eyes and the seven markers were placed on seven different squares on the board. The subjects then opened their eyes for five seconds, looked at the pattern, then shut their eyes. Ten seconds later they were given the markers and told to recreate the pattern with the proper marker in the proper square. If the pattern produced by the subject was not correct, the procedure was repeated with the original pattern set up. This continued until the subject produced the correct pattern or for a maximum of ten trials. A subject's score was the number of trials taken to learn the pattern. This test was designed to measure visual spatial learning and memory.

Serial learning. In this test the subject learned a list of 14 unrelated words in order. First, the list was read to the subject; then he/she was asked to recall the list, starting from the beginning in correct order. When a subject recalled a word out of order or could not remember the next word, he/she was stopped and the entire list was re-read. This procedure continued until the subject recalled all the words. A subject's score was the number of trials taken to learn the list. This test was designed to measure serial learning and memory.

Procedure

Initial contact. In blocks of three the volunteers from the sign-up sheet were first contacted by phone to set up a convenient time in the morning. The purpose of the study was explained in greater detail and any questions were answered. The subjects were asked to come in for breakfast and not to consume any food before they arrived. These procedures were designed to control the amount of lecithin that might have been consumed by the subjects in their normal eating.

Double blind. In order to insure that neither the subject nor the experimenter knew what condition the subject was in, the muffins were previously placed in bags labeled one, two, or three by an assistant. The experimenter determined which subject received which bag but only the assistant knew the contents of each bag, to be revealed to the experimenter only after all testing of the subject was complete. It was hoped that by using the double-blind procedure the amount of experimenter bias would be reduced.

Consumption of lecithin. After first signing a consent-release form each subject consumed the two oatmeal muffins with 30 grams of either lecithin or placebo in them. Subjects were also offered juice and cereal if desired. They were then told to return in six hours for testing and not to

eat anything in the intervening interval. This six-hour interval was chosen based on the findings of Wurtman et al (1978) that serum choline is elevated at four hours and remains so for up to 12 hours depending on the dose. The six hour period was considered to be long enough for the maximum level to be reached but short enough to test the subject when the level is still high.

Memory testing. The subject then returned that afternoon and was tested on the five memory tests. An appointment was then set up for the following week and the entire procedure repeated. This one-week interval was considered to be more than adequate in terms of controlling a drug state carry-over since the longest period that lecithin produced elevations of serum choline was 12 hours. After the second testing session the subject was first asked to guess which condition he/she had been in as well as reporting any side effects. The subject's experimental condition was then revealed to both the subject and the experimenter. After any additional questions the subjects had were answered, they were thanked and invited to return later to find out the results of the study.

Design. The design used is summarized in Table One. As was described earlier the 30 subjects were randomly assigned to the three groups, giving 10 subjects per group. Group Three is of course the control group. Groups One and Two are counter-balanced with respect to treatment order. This helps to control for any treatment order confounding. In addition, this allows for a possible replication of the treatment effect in each of the two groups. Since one subject from each group was run each day, the groups are balanced with respect to testing days. In addition, five of the subjects in each group were tested on Form One of the tests first and the other half were given Form Two first. Therefore, the two forms, even though supposedly equivalent, are counter-balanced within each group with respect to week of testing. Each subject's scores on the five memory tests during testing session one were compared with his/her scores for session two. As such, each subject served as his/her own control since it was the difference in the two session scores that was important and not the overall level of performance. The difference scores for each subject were chosen instead of the raw score in order to try to reduce between-subject variability. For example, the scores on the colored marker test ranged from one to ten but the greatest within-subject difference was only four. Also, since a difference score

analysis does not directly compare performance between groups, it was hoped that some of the extraneous variables that influence memory, i.e., sex and age, would fail to produce confounding group differences because of the within-subject control.

Finally, the average difference scores for each group were compared to those seen in the control group.

Results

The average score for each group on each of the five tests is given in Table Two. Here a larger score on the first three tasks shows improvement, whereas on the Colored Marker Test and the Serial Learning Test a smaller score means improvement. The average difference scores for each are given in Table Three. These were calculated by subtracting the scores on the second week from the first. Here a negitive score on the first three indicates improvement, whereas a positive number on the Colored Marker Test and the Serial Learning shows an increased performance.

The first analysis was designed to partially test whether the groups were different at the start. As such it was a check on the success of the random assignment. A Multiple Analysis of Variance was run on the first week scores

of groups two and three (before any lecithin had been received). The Greatest Characteristic Root (GCR) test showed no significant group differences on the initial overall performance ($p=.88$). Although this procedure does not really measure the initial condition, only an equal placebo had been given to both groups. It was hoped that this would have affected the two groups equally. In addition, it was not possible to test the before-treatment level of Group One. It can only be assumed that the random assignment led to an equivalent group. This finding of no difference between the groups is important even when using difference scores. The groups must not be significantly different before a comparison of the difference scores can be undertaken.

The second analysis compared the difference scores of Group One and Group Three. The multivariate test indicated no significant between-group difference (GCR, $p=.44$); nor did any of the individual memory tests differ significantly between the two groups as measured by the univariate tests. Examination of Table Three shows that the group differences are not even in the expected direction. All five of the tests showed the lecithin group to perform better while they were in the placebo condition. Compared to the control group this second-trial increase was greater for the first three tests but less for the CMT and the SL tests. Therefore, even

including any possible learning effects, the first group showed better performance under placebo.

The difference between the second (placebo then lecithin) group and the control group was then analyzed. There was no significant between group difference (GCR p=.29) although the univariate tests showed the digit recall test to be significantly different in the two groups. However, since the multivariate F was not significant, this must be regarded as a chance finding. An examination of Table Three shows the results to be in the predicted direction, with the subjects higher while under lecithin on all five tests. In addition, on four out of five tests (not SL) this second-week increase was greater than that seen in the control group, which also showed a second-trial increase. However, in view of the nonsignificant between-group differences it appears that the increases seen under lecithin in the second group are probably due to the overall second trial increase seen in the control group.

As a final test to try to determine what effect, if any, lecithin produced, a nonparametric sign test was used. Each subject's scores on the two weeks were compared. If, for example, a subject did better on three out of five of the tests while taking lecithin, he/she would be considered

to have performed better under lecithin. Using this procedure it was found that ten subjects did better on lecithin, seven did worse, and three did the same. The probability of this pattern of occurrence is not significantly different from that due to chance ($p=.15$). With regard to sex or level of performance there was no clear pattern in the individuals who improved. Overall, the results indicate that the oral consumption of a single dose of lecithin does not significantly affect a subject's memory as measured by the five memory tests.

EXPERIMENT TWO

Method

Subjects

An additional 30 subjects were recruited from five classes at the University of Victoria. Their ages ranged from 19-39, with the average being 26. There were 17 females and 13 males in the sample. The subjects were randomly assigned to the three experimental groups as described in Experiment One.

Lecithin

The lecithin used was 3.6 (20 percent phosphatidyl choline) gram capsules manufactured by Swiss LTD. The subject received two capsules daily (7.2 grams total) for seven days (total 50.4 grams). This was the dosage recommended on the container and the 50-gram total corresponded to the amount of lecithin used in previous studies. Capsules filled with corn meal were used as a placebo. Although the placebo capsules were not matched to the lecithin capsules

in terms of color, the yellow color and texture of the corn meal corresponds to that of lecithin granules. It was hoped that subjects who had seen lecithin before would tend to identify with this yellow color (the lecithin capsules were in contrast dark brown). Later questioning of the subjects often revealed this color identification with the placebo to have occurred.

Memory tests

The two forms of the five tests were identical to those described earlier.

Procedure

After an initial phone briefing the subjects came to the lab to pick up a packet containing 14 capsules and to sign the release forms. At this time they were instructed to take one capsule that evening and then to take two capsules daily, morning and evening, for one week. They were then to take the morning capsule on the testing morning and come in and take the memory tests.

Once again, to insure that the experiment was double blind, an assisstant prepared sealed envelopes containing the capsules beforehand, the contents of which were unknown to the experimenter until completion of the testing.

After the first testing session the subject was given an additional 14 capsules and the procedure was repeated. The subject debriefing after the second testing session was similar to that used in Experiment One.

The design used was identical to that given in Table One. All counterbalancing procedures were also repeated from the first study.

The difference scores for each subject were then compared and the between-group difference scores were then tested.

Results

The average score for each group is presented in Table Four. Once again a higher score on the first three indicates improvement, whereas a lower score on the last two shows improvement.

The first analysis partially tested whether the groups were initially equivalent. The first two weeks of Groups Two and Three were compared. Using the Greatest Characteristic Root Test there was no overall multivariate between-group difference ($p=.43$).

The average group difference scores are presented in Table Five. Here once again a smaller score on the first three items indicates improvement, whereas on the last two a larger score does.

An analysis based on these difference scores was then undertaken. A MANOVA showed no significant difference between Group One and Three (GCR p=.4), nor were any of the univariate tests significant. An examination of Table Four shows that the subjects did better under lecithin in three out of five of the tests, although of course not significantly better than the controls.

The second analysis compared Groups Two and Three. Once again no significant difference was found overall (GCR p=.19), nor did any of the individual tests show significant differences. Examination of Table Four shows that overall the subjects in Group Two did better under lecithin in all five of the tests. Further, in every case this difference was greater than any second-trial increase seen in the control group. Due to the relatively large between-subject variations the difference was not significant and must be reported as a chance finding.

Finally, an examination of the subjects individually indicated that thirteen did better under lecithin and seven

did worse. Once again this was not significant (p=.07). Nor were there any significant effects related to sex or performance level. It therefore appears that a week-long period of lecithin consumption does not significantly influence memory.

Discussion

These results indicate that for the particular aspects of memory measured in this study lecithin did not produce any significant improvements. Based on a MANOVA on the five memory variables the changes seen under lecithin were not significantly different from those seen in the comparable period in the control group. Nor were the groups significanlty different on any of the individual tests. Although there appears to be a trend toward more of a lecithin effect in the second study, one should be careful not to overinterpret this trend. The differences seen were still nonsignificant and as such must still be regarded as findings due to chance. There are many possible reasons for failing to detect lecithin's effect on memory, if indeed it exists.

To begin with, one reason may be the dosage of lecithin used. As stated in the introduction, I was interested in lecithin's application as a commercially available die-

tary supplement. As such I did not feel it was appropriate to give either massive dosages or to obtain specially prepared pure lecithin samples, since neither of these options would be followed by an individual taking a lecithin supplement. It is also difficult to compare the dosages used in this study to those of previous studies. The dosages in previous studies ranged from 10 grams to 150 grams. An additional complication is the fact that commercial lecithin preparations vary from 10 to 40 percent in their composition of pure lecithin, with the rest being filler. In general, the dosage used in this study appears to be slightly less than the average used in prior studies. Whether different results would be obtained with higher dosages cannot be determined.

The length of time lecithin was consumed could also have been a critical variable. Although Wurtman et al (1977) showed the serum choline level to be raised within four hours, there is no guarantee that memory effects would occur in the same time course. It may be that a longer period of lecithin consumption (weeks or even months) would have produced stronger effects. However, the fact that memory effects were produced 90 minutes after the ingestion of choline (Sitaram et al, 1978) argues against this possiblity.

The population of subjects used may not have offered a chance for memory improvement. College students are generally a very bright sub-sample of the population. This may indicate that they are already functioning at their optimum ACh level. It may be that a wider sample of the normal population would have produced larger effects. Arguing against this point is the wide range of scores observed on the five memory tests. There appeared to be more than sufficient room for improvement in the majority of the cases.

However, the main problem with trying to demonstrate a lecithin effect is in trying to overcome the numerous extraneous variables that also influence memory. Some subjects being tested indicated that they were on medication, getting the flu, fighting a hangover,or in sleep deficit. All these factors could easily mask a lecithin effect or conversely produce a false effect depending upon when they occurred. In either case the variability within each subject would be increased, making it more difficult to produce a significant overall effect. Perhaps the best method of dealing with this problem is to greatly increase the number of subjects. However, time and monetary constraints made this impossible. Even if a study with a large sample did find a significant effect, it might only be a statistical one and not a clinical one. By this I mean for lecithin to be useful to the

individual it must produce an effect that can be seen in that individual, since he/she would not be concerned with a treatment that changes a group mean by a small amount.

As a final method of determining if there was any effect due to lecithin, the subjects were asked to guess which group they were in based on how they felt their memory had been in daily functions. Only 30 percent of the subjects guessed correctly, less than expected due to chance. It therefore appears that even for memory functions not measured in the five memory tests, lecithin did not produce any noticeable effects.

Lecithin's failure to produce an effect could be attributed to any of the above reasons or to an actual ineffectiveness. Therefore, the most definitive statement that can be made is that for the particular memory tests used under normal commercial dosages of lecithin, this study does not support lecithin's ability to influence memory.

It is recomended that future studies should examine lecithin's effectiveness in populations where ACh may not be at optimum levels, ie, in the elderly or learning disabled. Additional long-term studies using a wide variety of memory and cognitive tests must also be undertaken before lecithin should be forgotten.

REFERENCES

Ansell,G.,& Spanner,S. Studies on the origin of choline in the brain of the rat. Biochemical Journal, 1971, 122, 741-750.

Barbeau,A. Emerging treatments: replacement therapy with choline or lecithin in neurological diseases. Canadian Journal of Neurology Science, 1978, 5, 157-160.

Barbeau,A. Lecithin in neurological disorders. Neurology, 1978,28,358.

Barbeau, A. Phosphatidyl choline in neurological disorders. New England Journal of Medicine, 1978,299,200-201.

Barbeau, A. Lecithin in movement disorders. In A. Barbeau, J. Growdon, R. Wurtman (Eds.), Nutrition and the Brain, (Vol. 5) New York: Raven Press, 1979.

Biederman, G. Progress in Neurobiology, 1974,2,289-307.

Boyd,W.,White,J., Graham,J.,& McQueen,J. Clinical effects of choline in Alzheimers senile dementia. The Lancet, 1977,Oct.1,711.

Buschke, H. Selective reminding for analysis of memory and learning. Journal of Verbal Learning and Verbal Behavior, 1973,12,543-550.

Christie,J., Blackburn,A., Glen,S., Zeisel,A., Shering,A.,& Yates,C. Effects of choline and lecithin of CSF choline levels and on cognitive function in patients with presenile dementia of the alzheimer type. In A. Barbeau, J. Growdon, R. Wurtman (Eds.), Nutrition and the Brain, (Vol.5) New York: Raven Press, 1979.

Cohen, E.,& Wurtman,R. Brain ACh: control by dietary choline. Science, 1976,191,561.

Cohen, E.& Wurtman, R. Brain ACh: increase after systemic choline administration. Life Sciences, 1975,16,1095-1102.

Crow,T.,& GroveWhite,G. Differential effect of atropine and lyoscine on human learning capacity. British Journal of Pharmacology, 1972,44,140-144.

Davis, K. Berger,P.,Hollister,L. Choline for Tardive Dyskinesia. The New England Journal of Medicine, 1975,293,152.

Davis,K., Berger,P.,& Hollister,L. Deanol for tardive dyskinesia. American Journal of Psychiatry, 1977,134,807.

Davis, K., Hollister,L., Barchas,J.,& Berger,P. Choline in Tardive Dyskinesia and Huntington's Disease. Life Sciences, 1976,19,1507-1516.

Davis,K., Hollister,L.,Overall,J.,Johnson,A.,& Train,K. Physostigmine: effects on cognition and affect in normal subjects. Psychopharacology, 1976,51,23-27.

Davis,K., Mohs,R., Tinklenberg,J., Hollister,L., Pfefferbaum,A. Cholinomimetics and memory. Archives of Neurology, 1980,37,49-52.

Davis,K., Mohs,R., Tinklenberg,J., Pfefferbaum,A., Hollister,L.,& Kopell,B. Physostigmine: improvement of long-term memory processes in normal humans. Science, 1978,201,272-274.

Deutsch,J. The cholinergic synapse and the site of memory. Science, 1971,174,788-794.

Deutsch, J.,& Rogers,J. Cholinergic excitability and memory: animal studies and their clinical implications. In K.Davis, and P. Berger (Eds.), Brain Acetylcholine and Neuropsychiatric Disease, New York: Plenum Press,1978.

Drachman,D. Central cholinergic system and memory. In M. Lipton (Ed.) Psychopharmacology: a Generation of Progress, New York: Raven Press,1978.

Etienne,P.,Gauthier,S.,Dastoor,D., Collier,B.,& Ratner,J. Alzheimer's Disease: clinical effect of lecithin treatment. In A. Barbeau, J. Growdon, and R. Wurtman (Eds.) Nutrition and the Brain, New York: Raven Press,1979.

Freeman,J., Choi,R., & Jenden,D. Plasma choline: its turnover and exchange with brain choline. Journal of Neurochemistry, 1975,24,729-734.

Ghoneim,M., & MeWaldt, S. Effects of diazepam and Scopolamine on storage, retrieval, and organizational processes in memory. Psychopharmacology, 1975,44,257-262.

Ghoneim, M., & MeWaldt, S. Studies of human memory: diazepam, scopolamine, and physostigmine. Psychopharmacology, 1977,52,1-6.

Growdon, J., Cohen, E., & Wurtman, R. Effects of oral choline administration on serum and CSF choline levels in patients with Huntington's Disease. Journal of Neurochemistry, 1977,28,229-231.

Growdon, J., Gellenburg , J., Hirsch, M.,& Wurtman, R. Lecithin can suppress Tardive Dyskinesia. The New England Journal of Medicine, 1978,298,1029.

Growdon, J., Hirsch, M., & Wurtman, R. Oral choline administration to patients with Tardive Dyskinesia. The New England Journal of Medicine, 1978,297,525.

Hamburg, M. Retrograde amnesia produced by intraperitoneal injection of physostigmine. Science, 1967,156,973-974.

Hanin, I.,& Scheberth, J. Labelling of ACh in the brain of mice fed on a diet containing choline. Journal of Neurochemistry, 1974,23,819-824.

Haubrich, D.,& Chippendale,T. Minireview: regulations of acetylcholine synthesis in nervous tissue. Life Science, 1977,20,1465-1478.

Haubrich,D., Rand, W., & Gillette, J. ACh formation in mouse brain and effect of cholinergic drugs. Nature and New Biology, 1972,238,88.

Haubrich, D., Wang, P., & Weduking, P. Increase in rat brain ACh induced by choline or deanol. Life Science, 1975,17,975-980.

Haubrich, D., Wang, P., & Weduking,P. Role of choline in biosynthesis of ACh. Federation Proceedings, 1974,33a,477.

Hirsch, M., Growdon,J.,& Wurtman,R. Increase in hippocampal ACh after choline administration. Brain Research, 1977,125,383-385.

Hirsch, M., Growdon,J.,& Wurtman,R. Relations between dietary choline or lecithin intake, serum choline levels and various metabolic indices. Metabolism, 1978,27,953-960.

Hirsch, M.,& Wurtman, R. Lecithin consumption increases ACh concentration in rat brain. Science, 1978,202,225.

Houtsmuller, U. Metabolic fate of dietary lecithin. In A Barbeau, J. Growdon, and R. Wurtman (Eds.) Nutrition and the Brain, (Vol 5) New York: Raven Press, 1979.

Karczmar,A. Cholinergic influences on behavior. In P. Waser (Ed.) Cholinergic Mechanisms, New York: Raven Press, 1975.

Klatzky, R.L. Human Memory San Fransisco:Freeman and Co.,1980.

Lee, C., Yu,J.,& Turner, K. New England Journal of Medicine, 1976,295,937.

Mohs,R., Davis,K., & Darley,C. Cholinergic drugs and memory. In L. Poon (Ed.) Aging in the 1980's, Washington, D.C.: American Psychological Association,1980.

Ostfeld, A.,& Aruguete, A. Central nervous effects of hyoscine in man. Journal of Pharacology and Experimental Therapeutics, 1962,137,133-139.

Petersen, R. Scopolamine induced learning failures in man. Psychopharmacology, 1977,52,283-289.

Raese, J.,Patrick,R.,& Barchas,J. Phospholipid-induced activation of tyrosine hydroxylase from rat brain striatal synaptosomes. Biochemistry and Pharacology, 1976,25,2245-2250.

Roth, R. Salzman, P.& Nowycky, M. Impulse transmitter flow and short-term regulation of transmitter biosynthesis in central catecholaminergic neurons. In M Lipton and K. Killan (Eds.) Psychopharmacology: a Generation of Progress, New York:Raven Press,1978.

Safer,D.,& Allen,P. The central effects of scopolamine in man. Biological Psychiatry, 1971,3,347-355.

Schea, P.,& Aprison, M. An enzymatic method for measuring picomole quantities of ACh and choline in CNS tissue. Analytic Biochemistry, 1975,56,165-177.

Signoret, J., Whitely, A., & Lhermitle, F. Influence of choline on amnesia in early Alzheimer's Disease. The Lancet, 1978, Oct. 14, 837.

Sitaram, N., Caine, E., & Gillin, C. Choline: selective enhancement of serial learning and encoding of low imagery words in mam. Life Science. 1978, 22, 1555-1560.

Sitaram, N., & Weingartner, H. Human serial learning: enhancement with arecoline and choline and impairment with scopolamine. Science, 1977, 201, 275.

Sitaram, N., Weingartner, H., & Gillin, C. Choline chloride and arecoline: effects on memory and sleep in man. In A Barbeau, J. Growdon, & R. Wurtman (Eds.) Nutrition and the Brain, (Vol 5) New York: Raven Press, 1979.

Smith, C. Swash, M. & Baily, M. Choline therapy in Alzheimer's disease. The Lancet, 1978, Aug. 5, 318.

Squire, L. Physostigmine: effects on retention at different times after brief training. Psychonomic Sciences, 1970, 19, 49-50.

Stanes, M., Brown, C., & Singer, G. Effect of physostigmine on Y maze discrimination in the rat. Psychopharmacologia, 1976, 46, 269-276.

Ulus, I., Hirsch, M., & Wurtman, R. Trans-synaptic induction of adrenomedullary tyrosine hydroxylase activity by choline: evidence that choline administration can increase cholinergic transmission. Proceedings of the National Academy of Science, 1977, 74, 798-800.

Ulus, I. Scally, M. & Wurtman, R. Choline potentiates the trans-synaptic induction of adrenal tyrosine hydroxylase by release of ACh. Life Sciences, 1977, 21, 145-148.

Ulus, I., & Wurtman, R. Choline administration: activation of tyrosine hydroxylase in dopaminergic neurons of rat brain. Science, 1976, 194, 1061.

Whitehouse, J. The effects of physostigmine on discrimination learning. Psychopharmacologia, 1966, 9, 183-188.

Wurtman, R., Hirsch, M., & Growdon, J. Lecithin consumption raises serum free choline levels. The Lancet, 1977, July 9, 68.

Yamamura, H.,& Snyder, S. High affinity transport of choline into synaptosomes of rat brain. Journal of Neurochemistry, 1973,21,355-374.

TABLE 1

Design for both experiments.

Group	Week One	Week Two
1	XO	PO
2	PO	XO
3	PO	PO

X=lecithin, P=placebo, and O= testing

TABLE 2

Average group scores.

Week	Group	Bushke	Digit	ASSL	CMT	SerL.
1	1(L)	108.3	11.3	24.8	4.8	5.0
2	(P)	113	12.1	25.4	4.2	4.8
1	2(P)	104.8	11.2	26.1	6.1	5.7
2	(L)	108.5	12.5	26.4	4.0	5.0
1	3(P)	105.6	11.7	24.1	5.8	6.7
2	(P)	105.9	11.7	24.2	4.8	6.0

L=lecithin, P=placebo

TABLE 3

Average group difference scores.

Group		Bushke	Digit	ASSL	CMT	SerL.
1(LP)	mean	-4.7	-.8	-.6	.6	.2
1	SD	14.9	1.5	4.3	1.2	1.8
2(PL)	mean	-3.7	-1.3	-.3	2.1	.7
2	SD	12.9	1.1	3.9	2.2	2.1
3(P)	mean	-.3	0	-.1	1.4	1.3
3	SD	11.2	1.4	4.9	2.4	1.8

L=lecithin, P=placebo

TABLE 4

Average group scores experiment two.

Week	Group	Bushke	Digit	ASSL	CMT	SerL.
1	1(L)	102.5	11.9	25.9	4.3	5.7
2	(P)	98.3	12.2	24.6	4.5	5.6
1	2(P)	99.0	10.3	19.7	5.2	7.0
2	(L)	99.5	11.3	20.7	3.9	6.1
1	3(P)	92.8	11.3	24.8	5.3	5.7
2	(P)	90.8	11.1	24.8	4.3	6.4

TABLE 5

Average group difference scores experiment two.

Group		Bushke	Digit	ASSL	CMT	SerL.
1(LP)	mean	4.2	-.3	1.3	-.2	.1
1	SD	18.6	1.3	6.4	2.1	2.4
2(PL)	mean	-.5	-1.0	-1.0	1.3	.9
2	SD	15.5	2.5	6.2	2.2	1.3
3(PP)	mean	2.0	.2	0.0	1.0	-.7
3	SD	12.4	2.1	4.7	2.5	3.1

Appendix A

A

Test Forms, Consent Form, and Schedule Form.

Informed Consent

Title of Study: Oral Consumption of Lecithin: Enhancement of Short Term Memory and Learning

Subject's Name: _____

I, the undersigned, do understand the purpose of the above study. I further understand that the amount of lecithin consumed will be within the recommended daily level. I agree to participate in this study and hereby release both Wayne Clark and the University of Victoria from any responsibility concerning my participation.

_____ _____

Subject's Signature Date

I, the undersigned, have defined and explained this study to the volunteer.

_____ _____

Investigator's Signature Date

Subject:_____ Trial:_____

Group:_____ Date:___

BUSCHKE'S RESTRICTED REMINDING PROCEDURE I

WORD LIST	1	2	3	4	5	6	7	8	9	10	11	12	13
DOG													
FOX													
HORSE													
BUFFALO													
LION													
RHINOCEROS													
ELEPHANT													
ANTELOPE													
BEAR													
LAMB													
RAT													
RACCOON													
SHEEP													
LLAMA													
GOAT													
CHEETAH													
SQUIRREL													
BEAVER													
DONKEY													
TURTLE													

ADDITIONAL WORDS RECALLED:

L.T.W. :

T.R. :

7th Trial :

Intrusions :

Subject:_____ Trial:_____

Group:_____ Date:___

BUSCHKE'S RESTRICTED REMINDING PROCEDURE II

WORD LIST	1	2	3	4	5	6	7	8	9	10	11	12	13
CABBAGE													
LEMON													
EGG													
STRAWBERRY													
BREAD													
RADISH													
CHERRY													
AVOCADO													
SUGAR													
CHEESE													
OLIVE													
COOKIE													
APPLE													
BROCCOLI													
CAKE													
FRIJOLE													
LIME													
PLUM													
MILK													
BANANA													

ADDITIONAL WORDS RECALLED:

L.T.W. :

T.R. :

7th Trial :

Intrusions :

Associate Learning: Form 1

Subject:_____ Group:___ Date:_____

First Presentation	Second Presentation	Third Presentation
sail - source	cord - hook	mushroom - darling
mushroom - darling	glide - chain	apron - echo
jockey - sink	apron - echo	firm - boldness
firm - boldness	shade - rice	shade - rice
pale - rib	firm - boldness	pale - rib
cord - hook	press - dish	glide - chain
free - child	free - child	cord - hook
apron - echo	fog - pine	press - dish
fog - pine	pale - rib	free - child
press - dish	sail - source	fog - pine
glide - chain	jockey - sink	jockey - sink
shade - rice	mushroom - darling	sail - source

First Recall	Second Recall	Third Recall
firm - _____	press - _____	apron - _____
fog - _____	glide - _____	fog - _____
apron - _____	mushroom - _____	mushroom - _____
cord - _____	shade - _____	shade - _____
glide - _____	sail - _____	sail - _____
mushroom - _____	pale - _____	glide - _____
free - _____	free - _____	jockey - _____
press - _____	cord - _____	pale - _____
sail - _____	apron - _____	cord - _____
pale - _____	fog - _____	firm - _____
shade - _____	jockey - _____	press - _____
jockey - _____	firm - _____	free - _____
# Correct: ___	# Correct: _____	# Correct: _____
		Total # Correct: _____

Associate Learning: Form 2

Subject:_____ Group:____ Date: _____

First Presentation	Second Presentation	Third Presentation
plate - trail	clue - rescue	intent - contour
intent - contour	blank - bark	bacon - pot
slave - stopwatch	bacon - pot	tack - cast
tack - cast	shirt - ranch	shirt - ranch
violet - blunt	tack - cast	violet - blunt
clue - rescue	hay - ocean	blank - bark
speed - wake	speed - wake	clue - rescue
bacon - pot	dollar - odd	hay - ocean
dollar - odd	violet - blunt	speed - wake
hay - ocean	plate - trail	dollar - odd
blank - bark	slave - stopwatch	slave - stopwatch
shirt - ranch	intent - contour	plate - trail

First Recall	Second Recall	Third Recall
tack - _____	hay - _____	bacon - _____
dollar - _____	blank - _____	dollar - _____
bacon - _____	intent - _____	intent - _____
clue - _____	shirt - _____	shirt - _____
blank - _____	plate - _____	plate - _____
intent - _____	violet - _____	blank - _____
speed - _____	speed - _____	slave - _____
hay - _____	clue - _____	violet - _____
plate - _____	bacon - _____	clue - _____
violet - _____	dollar - _____	tack - _____
shirt - _____	slave - _____	hay - _____
slave - _____	tack - _____	speed - _____

Correct: _____ # Correct: _____ # Correct: _____

Total # Correct: _____

Serial Learning: Form 1

Subject:_____ Group:___ Date:_____

	1	2	3	4	5	6	7	8	9	10	11	12
sweet												
thumb												
shiver												
indent												
pond												
odor												
middle												
sunset												
blew												
cap												
mad												
pipe												
net												
history												

Checker Test: Form 1

Trial #	1	2	3	4	5	6	7	8	9	10
# Correct										

Serial Learning: Form 2

Subject:_____ Group:___ Date:___

	1	2	3	4	5	6	7	8	9	10	11	12
score												
doorbell												
sandal												
bush												
whirl												
figure												
humor												
circus												
tight												
brass												
pilot												
globe												
vacant												
grain												

Checker Test Form 2

Trial #	1	2	3	4	5	6	7	8	9	10
# Correct										

Subject _____ Group ___ Date _____

Digit Span Form 1

Digits Forward

5-8-2
6-4-3-9
4-2-7-3-1
6-1-9-4-7-3
5-9-1-7-4-2-8
5-8-1-9-2-6-4-7
2-7-5-8-6-2-5-8-4

6-9-4
7-2-8-6
7-5-8-3-6
3-9-2-4-8-7
4-1-7-9-3-8-6
3-8-2-9-5-1-7-4
7-1-3-9-4-2-5-6-8

Score:___

Digits Backward

2-4
6-2-9
3-2-7-9
1-5-2-8-6
5-3-9-4-1-8
8-1-2-9-3-6-5
9-4-3-7-6-2-5-8

5-8
4-1-5
4-9-6-8
6-1-8-4-3
7-2-4-8-5-6
4-7-3-9-1-2-8
7-2-8-1-9-6-5-3

Score: _____

Score: _____

Total Score: _____

Total Score : _____

Lecithin Study

Days

	1	2	3	4	5	6	7	
Morning	X							
Evening								

	8	9	10	11	12	13	14	15
Morning	Testing							
Evening								Testing X

Name _____

VITA

Surname: Clark Given Names: Wayne Marston
Place of Birth: Washington D.C. Date of Birth: Jan. 8, 1957

Educational Institutions Attended, with Dates Entering and Leaving:

University of Colorado	1975 to 1975
Oregon State University	1976 to 1976
University of the Pacific, CA	1976 to 1976
Colorado State University	1977 to 1977
University of the Pacific	1977 to 1979
University of Victoria	1979 to 1981

Degrees Awarded, with Dates and Names of Institutions:
B.A. (Honors) 1979 University of the Pacific

Honors and Awards:
Deans honor list U.O.P., 76,78,79
Phi Kappa Phi, National Honor Society
University of Victoria Fellowship, 79/80, 80/81

Publications:
Lag, Deficit, Delay, and Rate Constructs In Specific Learning Disabilities. Bulletin of the Orton Society: Special Edition. In Press.

PARTIAL COPYRIGHT LICENSE

I hereby grant the right to lend my thesis (the title of which is shown below) to users of the University of Victoria Library, and to make single copies only for such users or in response to a request from the library of any other university, or similar institution, on its behalf or for one of its users. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by me or a member of the University designated by me. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Title of Thesis:
Oral Consumption of Lecithin: Improvement of Memory and Learning In Man.

Author: [redacted]

Wayne Marston Clark
March 20, 1981