1975

Effect of protein-calorie malnutrition and partial social isolation on spatial delayed alternation performance in the developing rhesus monkey (Macaca mulatta)

Susan Paula Yeaton
The University of Montana

Recommended Citation

Yeaton, Susan Paula, "Effect of protein-calorie malnutrition and partial social isolation on spatial delayed alternation performance in the developing rhesus monkey (Macaca mulatta)" (1975). *Graduate Student Theses, Dissertations, & Professional Papers*. 5785.
https://scholarworks.umt.edu/etd/5785

THE EFFECT OF PROTEIN-CALORIE MALNUTRITION AND PARTIAL SOCIAL ISOLATION ON SPATIAL DELAYED ALTERNATION PERFORMANCE IN THE DEVELOPING RHESUS MONKEY (MACACA MULATTA)

By

Susan P. Yeaton

B.A., Vassar College, 1972

Presented in partial fulfillment of the
requirements for the degree of
Master of Arts
UNIVERSITY OF MONTANA
1975

Approved by:

David Strobel
Chairman, Board of Examiners

John M Stewart
Dean, Graduate School

Date June 23, 1975

7-1-75

Yeaton, Susan P., M.A., June, 1975

The Effect of Protein-Calorie Malnutrition and Partial Social Isolation on Spatial Delayed Alternation Performance in the Developing Rhesus Monkey (Macaca mulatta) (112 pp.)

Director: David A. Strobel DAS

The effects of protein-calorie malnutrition and partial social isolation on learning were studied in 16 juvenile rhesus monkeys. Diet and social environment were varied in a 2 x 2 factorial design with two repeated measures. The nutritional manipulation involved rearing from 120 days of age on either a high protein (25% casein by weight) or a low protein (3.5% casein by weight) diet. Social environments were varied by rearing either in group living cages or in individual housing (partial social isolation). Testing took place in a Wisconsin General Test Apparatus and involved daily presentations of 36 problems of spatial delayed alternation with correction (delay intervals of 10, 20, 30, 40, 50, and 60 seconds).

Significant differences between groups were found for both the environment and the diet effects across trials such that the high protein subjects performed better than the low protein subjects and isolates performed better than social animals. In addition, social animals were found to make more perseverative response errors than isolates. However, dietary and rearing conditions were not found to interact in producing their effects. Finally, a significant diet by delay effect was found such that high protein animals performed better than low protein animals on the shorter delay intervals.

ACKNOWLEDGMENTS

The author wishes to thank Dr. David Strobel (Chairman), Dr. James Walsh, Dr. John Means, and Dr. Donald Jenni (Zoology) for serving as members of her thesis committee. In addition, a number of individuals assisted in the testing of subjects: Charles Geist, Elise Lindvig, Lesley Lanham, Clare Huffman, and Gale Weiss.

This research was supported in part by grant number HD-04863-01 from the National Institute of Child Health and Human Development and by an experimental psychology traineeship funded by grant number MH-10986-08 from the National Institute of Mental Health.

TABLE OF CONTENTS

LIST OF TABLES

Table Page

In Text

In Appendix A

Table Page

In Appendix B

LIST OF FIGURES

Figure Page

In Text

In Appendix B

CHAPTER I

INTRODUCTION

While it has long been accepted that adequate nutrition is a requirement for normal growth and development, it has only been in recent years that malnutrition, especially protein-calorie malnutrition (PCM), has been recognized as an important world health problem. The term protein-calorie malnutrition was proposed by the Joint FAO/WHO Expert Committee on Nutrition (World Health Organization, 1962) to describe a number of clinical syndromes, such as kwashiorkor, marasmus, and famine edema, resulting from diets deficient in protein and/or calories. While there has been some disagreement over the classification of PCM disorders (World Health Organization, 1971), kwashiorkor is generally found in young children with inadequate protein intake and is characterized by edema, growth retardation, muscle wasting with retention of subcutaneous fat, and lowered serum albumin, serum lipase, and cholesterol levels (Jelliffe and Welbourn, 1963). Marasmus is found in children whose diets provide an insufficient number of calories and consequently results in growth retardation, muscle wasting without retention of subcutaneous fat, and near normal serum albumin, serum lipase, and cholesterol levels (Jelliffe and Welbourn, 1963).

Only rough approximations of the prevalence of such severe forms of malnutrition are available, but it has been estimated that between 0% and 7.6% of the world's children, less than five years of age, suffer from either kwashiorkor or marasmus (Bengoa, 1970). However, a much larger portion of children suffer from moderately inadequate diets, with estimates varying from country to country between 4.4% and 43.1% of children less than five years old (Bengoa, 1970). Similarly, a recent summary limited to data collected in Africa, Asia, and the Americas has reported that severe PCM ranged between 0.5% and 8%, and that moderate PCM ranged from 4% to 43% (World Health Organization, 1972). Thus, it is clear that moderate PCM is much more prevalent and is potentially a more important problem from both world health and socio-economic points of view than are severe forms of PCM.

Nevertheless, most studies of malnutrition have been based on animals subjected to severe dietary regimens, or on human data obtained from children hospitalized because of severe PCM. Such studies have revealed permanent alterations in physical development and have suggested that malnutrition may cause long term alterations in behavior and mental development. Essentially two models have been developed to interpret the results of such studies. The more popular model, the brain damage model, assumes that malnutrition brings about metabolic or structural changes in the central nervous system which result in various behavioral deficiencies characteristic of brain damage, such as hyperactivity, inter-sensory learning deficits, and poor motor control. The idea of vulnerable periods of central nervous system development is central to this model. Thus, it has been hypothesized that if a developmental process is restricted at the time of its fastest rate of growth, the ultimate extent of development will be restricted. Such a model predicts that normal development cannot be achieved even following rehabilitation, that the effect cannot be obtained in adults, and that the severity of restriction necessary to produce a given deficit decreases as the time of fastest rate of growth is approached (Dobbing, 1968).

Numerous studies have reported such permanent structural and physiological changes following PCM in the rapidly developing brain. Reductions have been reported in gross brain weight in rats (Winick and Noble, 1966), pigs (Dickerson, Dobbing, and McCance, 1967), and in children (Stoch and Smythe, 1963; Brown, 1965). Similarly, reductions have been reported in the total number of cells in the brain, both in rats (Winick and Noble, 1966; Culley and Lineberger, 1968) and in children (Winick and Rosso, 1969; Winick, 1972), and decreased myelination has been noted in rats (Dobbing, 1964; Dobbing and Widdowson, 1965; Culley and Mertz, 1965; Benton, Moser, Dodge, and Carr, 1966), and in pigs (Dickerson, Dobbing, and McCance, 1967; Dobbing, 1968). In summary, ample evidence is available to conclude that PCM leads to permanent changes in the central nervous system. However, such changes have not been demonstrated to be directly responsible for the behavioral abnormalities associated with malnutrition.

More recently, an environmental deficiency model has emerged to explain such abnormalities. This model hypothesizes that malnutrition prevents an organism from adequately interacting with its environment (Levitsky and Barnes, 1972; Strobel, 1972; Zimmermann, Steere, Strobel, and Hom, 1972). Thus, it has been suggested that the apathy and listlessness associated with severe PCM may lead to a partial breakdown between the organism and its environment, creating a situation analogous to sensory or perceptual deprivation (World Health Organization, 1972). While such a model predicts alterations in the ability of the organism to learn from its environment or to be stimulated by it, the environmental deficiency model should not be confused with an environmental deprivation or isolation approach to the abnormalities reported in malnutrition research. Thus, while the effects may be indistinguishable, the environmental deficiency model hypothesizes that they are the result of a deficiency on the part of the organism to successfully interact with its environment, while an environmental deprivation approach is concerned with the effects resulting from living in a less than optimal environment.

Such environmental deprivation has been a major confounding variable in field studies of human PCM. Many studies have lacked adequate controls for various biological and socio-economic differences between control and experimental groups. Often there are differences between groups in housing, sanitation, water supply, and exposure to parasitic and infectious diseases (World Health Organization, 1967). Underprivileged children frequently face unstable homes, poor child-rearing practices, and deficits in environmental stimulation. In severe cases of malnutrition, a schism between the expected pattern of reciprocal stimulation between mother and child has been reported (Pollitt, 1972). Additionally, many studies have been based on children hospitalized for severe cases of PCM, but the effects of separation trauma and institutionalization on development (Spitz, 1965; Provence and Lipton, 1962; Yarrow, 1964; Skeels, 1966; Eichenwald and Fry, 1969; Yaktin and McLaren, 1970) have not been considered.

Stoch and Smythe (1963, 1967, 1968) studied 20 South African children who were severely malnourished (marasmus) during the first two years of life. Control subjects were matched for age, sex and socio-economic level but were from more stable homes than the malnourished subjects. After seven and eleven years, scores for the malnourished subjects fell below their controls on a number of psychological tests, including full scale, verbal, nonverbal, vocabulary, and pattern completion. The authors concluded that deficits in visuomotor and pattern perception could suggest organic brain damage or may have been the result of decreased receptivity to external stimuli during the sensori-motor period of development. However, Stoch and Smythe have been criticized for failure to control for the disparate family settings of their subjects.

Cravioto and Robles (1965) studied 20 children (0-30 months old) who were hospitalized during rehabilitation from kwashiorkor. While lower IQs, especially decreased languaged development (Gesell schedules), were found in all age groups, older subjects improved during hospitalization while younger subjects (less than 6 months) failed to improve. Non-nutritional factors were not taken into account but a majority of children were reported to have parents who were either illiterate or of low scholastic achievement (Cravioto, 1968).

In a later cross-sectional study, Cravioto, DeLicardie, and Birch (1966) compared intersensory functioning between upper and lower quartiles of height for age in a rural population of Guatemalan children. It was assumed that children in the lowest quartile of height for age would be most likely to have experienced early malnutrition. To control for small stature as a result of a maturational lag, children of the same ages and with equivalent height differences were selected from an upperclass urban population (assuming that such children never experienced malnutrition). Data on parental stature and on the social, economic and educational status of the families of these children were also collected. While differences in height were not associated with differences in ability in the urban population, in the rural sample, the shorter children were found to have lowered intersensory integrative ability. The authors concluded that it was more likely that the inadequate intersensory integrative performance and low stature in the rural children were a result of malnutrition than that malnutrition (low stature) and poor intersensory development were independent results of general subcultural differences (Cravioto, 1968).

Champakam, Srikantia, and Gopolan (1968) studied 19 Indian children (8 to 11 years old) who had been rehabilitated from infantile kwashiorkor, and found them to be inferior to controls (matched for age, sex, religion, caste, socio-economic status, and family size) on a number of psychological tests. The greatest differences were found in the areas of abstract and perceptual abilities, however, the differences between groups tended to decrease as the age of the subjects increased.

Hansen, Freesemann, Moodie, and Evans (1971) tested 40 children who had a history of kwashiorkor (9 to 10 years previously). Controls were siblings of similar age and only slight differences were detected between experimental and control groups (New South African Individual Scale). However, while the controls did not exhibit the classical signs of kwashiorkor, it is probable that they suffered nutritional deficiencies similar to their ex-kwashiorkor siblings.

Monckeberg (1968) determined intelligence quotients for 14 Chilean children, 3 to 6 years old at the time of testing, who were hospitalized for marasmus during the first year of life. The mean IQ of these children was found to be significantly below the average for Chilean preschool children of low socio-economic class. Monckeberg reported that the best development was usually in the personal-social area, while language was most retarded. He concluded that although nutritional conditions improved, malnutrition during the first months of life caused long term brain damage (at least up to the sixth year of life). However, Monckeberg did not take into account the effects of social factors within the lower socio-economic groups, nor the effects of hospitalization on development.

Cabak and Najdanvic (1965) determined intelligence quotients for 36 Serbian children, 7 to 14 years old, who had experienced marasmus in early childhood. Only a mild degree of retardation was found, with half of the sample falling within the normal range for children in nearby communities. However, Cabak and Najdanvic apparently made no attempt to control for differences in family settings.

Brockman and Ricciuti (1971) studied 20 Peruvian children (11.8-43.5 months old) hospitalized for severe PCM (marasmus). Nineteen controls were matched for age, sex, socio-economic status, and were selected from day care centers with conditions similar to the rehabilitation center. The malnourished group was found to be inferior to the control group on ten sorting tasks, and failed to show improvement following twelve weeks of rehabilitation. The authors concluded that the lower sorting scores did not appear to be due to less frequent contact with the objects nor to a lack of interest, but rather to an inability to discriminate the similarities and differences among the objects. However, they cautioned that failure to improve after three months is not a sufficient basis to infer permanent retarding effects.

Canosa, Salomon, and Klein (1972) have carried out two studies of five and six year old children recruited from a nutritional rehabilitation day care center in rural Guatemala. In the first study, 20 children who had recovered from severe malnutrition were compared with a control group of 10 reportedly well-nourished children. The control group was selected from siblings of children who had attended the rehabilitation center and was matched for father's occupation, parental education, living conditions, and family structure. The rehabilitated children were found to be inferior to controls on four psychological tests (memory for sentences, memory for digits, memory for incidental learning, and memory for intentional learning) which had in common the need for close attention and short term recall. (There were no differences between groups on two tests of matching.)

The experimenters felt that rather than intellectual factors, the malnourished children were deficient in either short term recall or motivation and attention.

The second study involved 11 well-nourished and 17 previously malnourished children selected on the same criteria as in the preceding study. No differences between groups were found on memory for sentences, for digits, for incidental or intentional learning. However, the groups did differ on tests of memory for visual designs and a cube tapping test. It was suggested that these results reflected differences in attention or task concentration rather than differences in cognitive ability or short term memory. Furthermore, since language development tests are sensitive to social class differences and since no differences in language development were found between experimental and control groups,it was felt that the test differences could not be attributed to sampling bias, but that these differences were directly related to health and nutritional variables.

Finally, Cobos (1972) presented pairs of malnourished (mild and severe) and well-nourished siblings in the poverty areas of Bogata, Columbia, with a battery of psychological tests. Using a multiple regression technique, he found that even after social variables had been accounted for, nutritional factors had an impact on the psychological test scores. However, Cobos pointed out that a definite causal relationship had not been demonstrated since the effect may have been mediated through an unknown mechanism, such as unexplored social factors.

In summary, while human field studies of PCM are suggestive of a causal relationship between PCM and permanent psychological deficits, many investigators feel that such a relationship has not been indisputably demonstrated (Cravioto, DeLicardie, and Birch, 1966; Coursin, 1967; Dobbing, 1968; Monckeberg, 1968; Frisch, 1970; Barnes, 1972; Cobos, 1972; Klein, Habicht, and Yarbrough, 1972). The numerous factors which contribute to a subject's score on a psychological test have served as confounding variables in most studies. Often groups have been equated for general socio-economic status but factors within a socio-economic level, especially family stability and mother-infant relationships, have not been assessed. These factors are perhaps best controlled by the use of paired siblings. However, without the use of longitudinal studies, it is difficult to determine both the nutritional conditions experienced by the siblings during their critical periods of development and the differences in family relations during those periods. Thus, Cobos (1972) has suggested that food deprivation early in development alters the mother-child interaction by interfering with the mother's ability to satisfy the basic needs of the child. He further maintained that the inability of the family to protect the child from food deprivation implies some degree of impairment of the function of the family which will affect the child's developmental processes. Further, reports based on studies of severe malnutrition are limited since it is difficult to assess the effects of hospitalization and separation trauma, and the contribution of infectious diseases which often accompany malnutrition. They are further limited by the applicability of their findings to the much more prevalent cases of moderate PCM.

Animal research provides a unique opportunity to control for many of the variables that interfere with human PCM research. The experimenter is free to manipulate numerous factors in the social and physical environments of his subjects. He maintains control over the experimental diets, and the age of onset, duration, and severity of PCM imposed. With the use of littermates, genetic variation can be minimized. However, interpretations drawn from animal research are limited by the extent that human PCM disorders are mimicked in animals, by the limited behavioral repertoires of the animals, and in the extent that inferences can be made from the animal to the human condition.

Rats are widely used in PCM research because they are inexpensive to acquire and maintain, and they develop rapidly. However, it is difficult to produce a kwashiorkor-like syndrome in rats without forced feeding (Bradfield, 1968). Barnes, Moore, Reid, and Pond (1967) have been successful in producing a kwashiorkor-like syndrome with a high-calorie, low-protein diet. Protein deficient rats are anemic and have fatty livers, while calorie deficient rats have near normal hemoglobin levels. Rats experiencing PCM are small for age, exhibit loss of muscle, and loss of appetite (Widdowson, 1968).

Rats that experience early PCM exhibit a number of behavioral abnormalities. They are inferior [in this paper, the use of the terms "inferior" and "superior" refer only to relative efficiency on a given task] on maze learning tasks (Griffiths and Senter, 1954; Barnes, Cunnold, Zimmermann, Simmons, McLeod, and Krook, 1966; Barnes, 1968;

Zimmermann and Wells, 1971; Wells, Geist, and Zimmermann, 1972). However, studies by Geist (1973) suggest that the inferior performance may be due to motivational factors. Variations in activity and exploratory behavior have also been reported as a consequence of early PCM (Lat, Widdowson, and McCance, 1960; Guthrie, 1968; Frankova and Barnes, 1968a; Barnes, 1968), perhaps indicating variations in emotionality and over-sensitivity to the environment in malnourished rats. Thus, Levitsky and Barnes (1970) noted greater mobility in an open field by previously malnourished rats and greater percentage reduction in mobility following a loud noise. In addition, decreased exploratory activity has been reported in malnourished rats in the presence of novel objects (Zimmermann and Zimmermann, 1972). An increased emotionality has been reported in a shock avoidance paradigm, where rehabilitated rats acquired the conditioned avoidance response at the same rate as controls, but were delayed in adapting to extinction (Frankova and Barnes, 1968b), and increased response rates have been found in a Sidman avoidance paradigm (Barnes, 1972). Finally, Frankova (1973) has reported that during suckling, malnourished rats are more dependent on their mothers, show no social grooming, and are more aggressive. When later tested in the presence of a partner, low protein rats responded with inhibited exploratory activity and withdrawal, while their high protein controls showed increased activity and approach behaviors.

In recent years, a number of similarities between the effects of early PCM and early environmental isolation, such as increased emotionality, physiological effects on growth and development, and the existence of critical periods early in development leading to long term behavioral effects, have been noted and have led to the suggestion that they may affect behavior through the same mechanism (Levitsky and Barnes, 1973). This similarity has led several investigators to engage in research concerned with the combined effects of PCM and early experience. Frankova (1968) sought to determine the extent to which the effects of early PCM could be altered by environmental stimulation. The dietary manipulation involved raising infant rats in litters of 4, 9, 13, or 17 animals. Half of the litters received stimulation (handling) while half were undisturbed for the first 90 days of life. Between 90 and 110 days, the general level of activity was low in the unstimulated groups, declining as litter size increased, while stimulation caused the greatest activity in litters of 9 and 13. Thus, the effect of early dietary restriction on the activity of adult rats was appreciably modified by early stimulation.

Levitsky and Barnes (1972) extended their studies of nutritional and environmental interactions to include rats that were isolated at weaning. High and low protein groups were produced by feeding mother rats either high or low protein diets while nursing and continuing the pups on those diets for four weeks after weaning, followed by ten weeks of control diets. Early experience was varied by creating normal, stimulated or isolated environments for the pups. While the environmental conditions had no effect on rate of growth, differences between groups were found on number of behavioral measures. Significant increases in activity in an open field were found in isolated and malnourished groups, along with a significant interaction effect. While no differences were noted in social behavior in the well-nourished rats, stimulated malnourished rats made more following responses and more fighting responses than malnourished isolates. Finally, while there was no effect of early malnutrition with stimulation in tendency to explore a new environment, the interaction of malnutrition and isolation produced a large decrement in exploratory behavior. In summary, with the exception of fighting, whatever effect was produced by early malnutrition was exaggerated by isolation. Similarly, Wells, Geist, and Zimmermann (1972) found the greatest number of errors in maze performance when rats were simultaneously subjected to both dietary and environmental deprivation.

In many ways, the results from studies of early PCM in pigs have paralleled the findings in rats. Malnourished pigs display low serum proteins, fatty liver, edema, low weight, and decreased food consumption (Platt, 1968; Barnes, 1968; Barnes, Reid, Pond, and Moore, 1968). They are more emotional (Barnes, Moore, and Pond, 1970), exhibit decreased exploratory activity and inferior shuttle box escape avoidance (Barnes, Moore, Reid and Pond, 1968; Barnes, 1968). While there are no differences between PCM and control animals in the acquisition of a conditioned response, low protein pigs are more resistant to extinction (Barnes, 1967, 1968; Barnes, Moore, Reid, and Pond, 1967). They have been found to be inferior in the acquisition and extinction of a conditioned avoidance response (Barnes, Moore, and Pond, 1970), and were inferior and more emotional in a self-shaping procedure (Barnes, Moore, Reid, and Pond, 1968; Barnes, Reid and Pond, and Moore, 1968)

While behavioral deficits have been demonstrated in rats and pigs as the result of early PCM, there is some question as to the validity of extrapolation of experimental findings from lower animals to humans. However, data derived from two species of the same taxonomic order is often comparable (Kerr and Waisman, 1968), and has certainly been a factor in the choice of primates for behavioral research. Rhesus monkeys have been subjected to a variety of learning tasks and the ontogenetic development of their learning abilities delineated (Zimmermann and Torrey, 1965). Maximum intellectual improvement is not obtained prior to sexual maturity (Zimmermann and Torrey, 1965) and it has been suggested that learning is probably involved in the organization of their social behavior (Mason, 1961). In addition, their slow growth and development makes possible long periods of observation in the laboratory, permitting experimental research during the period of nutritional deprivation.

While several investigators failed to obtain the classical signs of kwashiorkor in rhesus monkeys subjected to early protein restriction (Kerr and Waisman, 1968; Ordy, Samorajski, Zimmermann, and Rady, 1966), such signs have been reported as a result of tube feeding techniques (Deo, Sood, Ramalingaswami, 1965; Ramalingaswami and Deo, 1968). Physiologically, the rhesus monkey responds to protein deficiency with decreased weight gain, decreased levels of total serum protein, serum albumin, serum cholesterol, fatty liver, and atrophy of the gastrointestinal tract (Ordy,Samorajski, Zimmermann, and Rady, 1966; Ramalingaswami and Deo, 1968; Geist, Zimmermann, and

Strobel, 1972).

Rhesus monkeys subjected to low protein diets exhibit a number of behavioral abnormalities. Kerr and Waisman (1968) characterized such monkeys as inactive, retarded in social development, and showing little interest in their surroundings. While Hillman and Riopelle (1971) reported that adult rhesus monkeys deprived of protein did not demonstrate a preference for high protein foods, Peregoy, Zimmermann, and Strobel (1972) found that low protein monkeys were able to discriminate between high and low protein diets and preferred high protein foods. However, Pettus, Geist, and Schultz (1974) determined that this preference did not persist following rehabilitation. While Geist, Zimmermann, and Strobel (1972) found no differences in activity levels between high and low protein monkeys, low protein monkeys have been shown to display decrements in curiosity and manipulative responses in chain pulling (Zimmermann and Strobel, 1969; Strobel and Zimmermann, 1972), and in puzzle solving tasks (Strobel and Zimmermann, 1971) when compared with controls. However, with the introduction of food reward, low protein animals manipulated the puzzle at least as much as high protein animals and showed sharper declines in responding during extinction (Aakre, Strobel, Zimmermann, and Geist, 1973). Thus, it appears that low protein monkeys have higher levels of food motivation, and therefore are more sensitive to the absence or presence of food. Greater interest in food than objects has been reported by Peregoy, Zimmermann, and Strobel (1972). Wise, Zimmermann, and Strobel (1973) and Wise and Zimmermann (1973b) found that in food competition measures of social dominance, low protein monkeys were superior to controls. In addition, Wise and Zimmermann (1973a) found low protein monkeys to have lower shock threshold than normal monkeys.

The reaction of low protein monkeys to objects has also been investigated. Peregoy, Zimmermann, and Strobel (1972) demonstrated that high protein animals accepted a greater number of toys than low protein animals, but Pettus, Geist, and Schultz (1974) found that this effect did not persist following rehabilitation. Zimmermann, Strobel, and Maguire (1970) found that while low protein monkeys were superior to controls on a learning set task with familiar objects, when new stimuli were introduced, they became highly emotional and tended to avoid the novel stimuli. Similarly, Strobel and Zimmermann (1972) found that the introduction of novel objects in a free operant chain manipulation situation led to decreased rates of manipulation in low protein monkeys, while high protein controls showed increased performance. Finally, Strobel (1972) found that high protein monkeys trained to shuttle to the top of a vertical tunnel, made more responses when novel stimuli were suspended from the top of the apparatus, while the low protein groups showed a decrement in response to objects. Furthermore, in a study of disinhibition of delay, low protein animals differed from controls by displaying increased responsiveness in the presence of novel stimuli.

The behavior of low protein monkeys in social situations has also been investigated. Zimmermann and Strobel (1969) found that low protein monkeys behaved apathetically toward other monkeys and showed a predominance of self-directed activities. In addition, Zimmermann, Steere, Strobel, and Hom (1972) noted that low protein animals engaged in less sexual behavior, less play and less grooming than high protein controls. They were characterized as aggressive and displaying a lack of reciprocal responsiveness in social situations. Furthermore, studies of social dominance suggest an instability of dominance relationships among low protein animals. Thus, while they are less dominant than controls in a shock avoidance paradigm (Wise and Zimmermann, 1973b) and in aggressive interactions in the social room (Wise, Zimmermann, and Strobel, 1973), they become more dominant in food competition situations (Wise and Zimmermann, 1973b; Wise, Zimmermann, and Strobel, 1973).

High and low protein monkeys have been tested on a variety of learning tasks in an effort to determine the effects of PCM on mental development. Zimmermann (1973) found no differences on an object discrimination task or on reversal learning for a group of year old monkeys tested before and one month after being placed on low protein diets. In addition, using monkeys subjected to early PCM, no differences between high and low protein groups were found in learning set formation, both for oddity (Zimmermann, Geist, and Strobel, 1973) and for object discrimination learning (Stoffer and Zimmermann, 1973). When low protein animals were tested on 100 6-trial problems of object discrimination learning set with six repetitions, low protein animals were found to be superior to high protein controls on both performance on the first trial of every problem, a measure of long term memory, and on the remaining trials, a measure of learning set formation. However, the differences between groups disappeared following rehabilitation (Zimmermann, 1969b). Similar results were obtained by Zimmermann, Strobel, and Maguire (1970). High and low protein groups were also tested on delayed response, which is considered to be a measure of short term memory, and no significant differences were found (Zimmermann, Geist, Strobel, and Cleveland, 1973). In summary, protein malnutrition was not found to effect test performance on delayed response, object discrimination, reversal learning, learning set (object and oddity), or long and short term memory.

However, low protein animals have been found to be inferior on other tasks. Zimmermann (1973) tested high and low protein groups of monkeys on object discrimination and reversal learning in which the objects were mounted on masonite plaques to produce a stimulus-response discontinuity. Such discontinuity makes discrimination problems more difficult for rhesus monkeys to learn. While there were no differences between groups in learning the discriminations, the low protein group was found to be inferior to controls on the reversal learning task, a task which differentiates the learning capacities of higher primates (Rumbaugh and Pournelle, 1966). Thus, although Zimmermann found no differences in reversal learning when the monkeys touched the objects, low protein monkeys were inferior on the same task with stimulus-response discontinuity.

In another experiment designed to investigate the effects of stimulus response discontinuity, both the location and size of the discriminative stimulus were varied in a learning set paradigm. The discriminative cue occupied either the center or periphery of a grey plaque and varied in total area from 5% to 100% of the plaque. Thus, in the case of central cues, as the size of the cue decreased, the discontinuity between where the monkey placed his fingers and the locus of the cue increased. While the high and low protein groups did not differ in the ability to learn the original problem, the low protein animals were inferior on the reversal problems as the area of the central cue decreased (Strobel, 1972; Strobel, Geist, Zimmermann, and Lindvig, 1974).

Stimulus-response discontinuity was investigated in two further studies (Strobel, 1972). The conditional learning paradigm involved the placement of a card containing either a square or a triangle between two identical plaques which covered the food wells. The square or triangle indicated the position of the food reward on any given trial. The low protein groups were found to be significantly inferior to the control groups in learning this task. The hidden and embedded figures experiment involved teaching the monkeys to discriminate between a square and a triangle and then testing them on transfer of training to the hidden and embedded figures problems. While the high protein groups scored initally superior and improved across trials, the low protein animals did not respond significantly above chance.

Finally, the high and low protein monkeys were tested on a patterned strings task. This can be described as a reward-directed task since the reward is visible, attached to the far end of one string. In both the parallel and pseudocrossed patterns, the monkey can obtain the reward by pulling the string nearest to the reinforcement, and no differences were found between high and low protein groups. However, on the crossed pattern, the monkey must select the string opposite the reinforcement. Thus, the crossed pattern created spatial discontinuity between the response locus and the reinforcement, and on this pattern, low protein animals were found to be much inferior to their high protein controls (Zimmermann, Geist, and Strobel, 1973; Zimmermann, Geist, Strobel, and Cleveland, 1973).

In summary, while there are a number of learning and perceptual tasks in which low protein monkeys perform as well as controls, they have displayed inferior performance on a group of tests involving a discontinuity between stimulus and response, such as object reversal with objects mounted on plaques, central stimulus reversal learning, conditional discrimination learning, and embedded and hidden figures discrimination, or a discontinuity between response and reinforcement (patterned strings, crossed pattern). These tasks have in common the need for an ability to localize and select critical cues from the environment, i.e., they make increased demands on the attentional processes of the organism.

The suggestion that protein malnourished animals may suffer from attentional deficiencies, receives support from a study of human PCM by Klein, Gilbert, Canosa, and DeLeon (1969). While they found no differences in discrimination learning between children who suffered early PCM and controls, malnourished children were found to be inferior on tasks which made increased demands on their attentional processes, such as rapid tapping or embedded figures. Since these children could perform adequately when the tapping sequence was slowed down and could solve embedded figures problems once the embedded figure was pointed out, the deficiency can be considered attentional rather than one of mental capacity _per se_.

In summary, the high incidence of protein-calorie malnutrition throughout the world has led many investigators to study the long term effects of nutritional deprivation. In a series of animal and human studies, PCM early in development has been shown to result in a number of structural changes within the central nervous system. In addition, numerous human field studies of early PCM have suggested a causal relationship between PCM and long term psychological deficits. However, there are a number of uncontrolled variables in such human field studies, mostly socio-economic factors, which have tended to confound studies of nutritional deprivation with environmental deprivation. Consequently, there have been a series of studies involving rats and pigs which have demonstrated behavioral deficits and abnormalities as a result of early nutritional deprivation, as well as interactions between nutritional and environmental deprivation, but such studies are limited to the extent that inferences can be drawn to the human condition. As a compromise between experimental control over nutritional and environmental variables and the validity of extrapolation to the human condition, rhesus monkeys have been selected for PCM research.

Malnourished monkeys have been observed during the period of nutritional deprivation and have displayed a variety of abnormal behaviors. They are highly emotional, tend to avoid novel stimuli, and their behavior in social situations resembles that of isolated monkeys.

However, in a variety of learning tasks, including many rather difficult discriminations (such as learning set), malnourished monkeys have performed as well as controls. The only discrimination tasks which have demonstrated deficient performance by low protein monkeys have involved stimulus-response discontinuity and have been interpreted as indicating an attentional deficit in protein malnourished monkeys. If low protein monkeys display inferior performance on tasks which require the ability to localize and select critical cues from the environment, then they may also experience difficulty when confronted with tasks in which the critical cue is not even present in the external environment. Delayed alternation is such a task, that is, in delayed alternation the critical cue as to the correct response is the animal's response on the preceding problem. Thus, the purpose of the present experiment is to determine if protein malnourished monkeys perform differently than controls on delayed alternation, a task which requires them to attend to their own past behavior.

Rationale

It has been suggested that the inferior performance of low protein monkeys on tasks involving stimulus-response discontinuity may be due to an attentional deficiency (Strobel, 1972; Strobel, Geist, Zimmermann, and Lindvig, 1974). In the various experimental procedures employed to demonstrate this defect, the critical cue has been present in the environment and the protein malnourished animal has been interpreted as failing to locate or select the appropriate cue. Spatial delayed alternation provides another method for separating the critical cue from the response locus since in the case of delayed alternation, the cue to alternate comes from inside the animal (French, 1965). By using a correction procedure, whereby each trial is rerun until the animal makes the correct response, the adoption of a simple "shift" strategy is adequate for solution of the problem. In this case, the critical cue becomes the animal's last response, for instance, if his last response was to the left, the next response should be to the right. Thus, such a task requires only that the animal adopt a shift strategy in reference to his preceding response. If the malnourished monkey is fixated on the locus of response, as suggested by the stimulus-response discontinuity experiments, then he should prove inferior on a delayed alternation task. Assuming that problems with longer delays make increased attentional demands on the animal, then the performance of low protein monkeys can be expected to drop relatively more rapidly than that of high protein monkeys as the length of the delay interval increases. In addition, since no differences between high and low protein monkeys have been found on the delayed response task (Zimmermann, Geist, Strobel, and Cleveland, 1973),in which the critical, reinforced cue is given by the experimenter, deficient performance by low protein monkeys on delayed alternation cannot be interpreted as due simply to defective short term memory.

In addition to expecting inferior performance on the delayed alternation task by low protein monkeys, a similar effect may be anticipated with respect to environmental deprivation. Several investigators have noted similarities between protein malnourished animals and those subjected to early environmental deprivation (Zimmermann, Steere, Strobel, and Hom, 1972; Levitsky and Barnes, 1972, 1973). Mason (1968) has characterized monkeys deprived of early social experience as displaying abnormal postures and movements, poor integration of motor patterns, defective social communication, and motivational disturbances, including increased emotionality and excessive fearfulness. Zimmermann, Steere, Strobel, and Hom (1972), noting similar abnormalities in malnourished monkeys, have suggested that since low protein monkeys tend to be neophobic and avoid social interaction, they may suffer from self-imposed stimulus deprivation and perhaps can be considered functional social isolates. Similarly, Levitsky and Barnes (1972) have proposed two mechanisms in an effort to account for the behavioral effects of malnutrition, i.e., that malnutrition may change an animal's experience or perception of the environment, rendering him less capable of receiving or integrating environmental information; or malnutrition may produce behavior, such as extreme food orientation, that is incompatible with the incorporation of environmental information. Such models suggest that the same or very similar mechanisms may be involved in producing the behavioral abnormalities seen with early malnutrition and early environmental deprivation. If this is the case, malnutrition and environmental deprivation can be expected to interact in producing their behavioral effects. In order to test such a model, diet and environmental stimulation should be varied in a factorially designed experiment.

Finally, Strobel (1972) and Strobel, Geist, Zimmermann, and Lindvig (1974) reported that the various reinforcers used with low protein monkeys and slightly deprived high protein monkeys (raisins or high protein diet for high protein monkeys and sugar coated cereal for low protein monkeys) have equal incentive value. Assuming that response rate is directly related to food motivation, no significant differences in response latency between diet groups are expected.

CHAPTER II

METHOD

Subjects and Nutritional Treatment

Twelve laboratory-born rhesus macaques (Macaca mulatta) were housed individually with their mothers for the first 90 days of life in wire cages measuring 76.2 x 76.2 x 76.2 cm. The animals were then separated and placed individually into 47.0 x 61.0 x 48.3 cm wire cages. During the first day post separation, the infant monkeys were maintained on milk formula diet (Blomquist and Harlow, 1961), provided every two to four hours. On subsequent days, bottles of formula were placed in bottle holders on a wire ramp and given to the infants ad libitum in their home cages. By 120 days of age all animals were weaned to solid food which contained 25% casein by weight as the sole source of dietary protein, according to procedures detailed by Zimmermann (1969a).

Three additional infant rhesus monkeys were reared from birth with their mothers in group living cages at the Davis Regional Primate Research Center. At 112 days of age, the animals were separated from their mothers, shipped by air to the laboratory, and were received the same day. The monkeys were immediately housed in individual wire cages identical to those of the laboratory-born infants and placed on the same milk formula diet. Weaning to solid food containing 25% protein was completed by 120 days of age for all animals.

A fourth infant macaque was reared from birth to 141 days of age with its mother in an individual cage at the Davis Regional Primate Research Center. At this time the animal was separated from its mother and shipped by air to the laboratory. The infant monkey was immediately housed in an wire cage identical to those described above. Being past the age at which milk is required during weaning, the animal was provided with the 25% protein diet.

At 120 days of age, one group of animals (LP-SOC), comprised of one laboratory-born female and three males from the Davis colony, was placed in a 146.1 x 71.8 x 182.9 cm cage and was provided with continuous social enrichment in the form of group living. The animals were provided with a low protein diet containing 3.5% casein by weight (3.3% of calories), but isocaloric with respect to the 25% high protein diet (23.6% of calories supplied by casein). A control group of socially enriched laboratory-born monkeys (HP-SOC), consisting of two males and two females, was placed in a group living cage identical to that of the LP-SOC group at 120 days of age and was maintained on the 25% protein diet given during weaning. A third group of laboratory-born rhesus monkeys (LP-ISO), comprised of one female and three males, was housed in individual wire cages measuring 76.2 x 76.2 x 76.2 cm and maintained under this condition of partial social isolation beginning at 120 days of age. The animals were given an identical 3.5% low protein diet as that of the LP-SOC group. A control group (HP-ISO) of three laboratory-born monkeys and one infant from the Davis colony which had been reared individually with its mother, consisting of one female and three males, was provided with identical conditions of partial social isolation as that of the LP-ISO group. At 120 days of age, however, the animals were maintained on the 25% high protein diet. At the time of testing, all animals were between two and four years of age. For convenience, each group will be referred to by either the low protein or high protein diet, as well as by the environmental rearing conditions, i.e., LP-SOC, HP-SOC, LP-ISO, and HP-ISO groups. Details of the composition and preparation of the various diets, as well as the *ad libitum* feeding procedure, are presented in Geist, Zimmermann, and Strobel (1972).

Experimental Histories

The LP-ISO group was tested on a variety of curiosity and manipulation tasks including chain pulling (Strobel and Zimmermann, 1972), home cage activity (Geist, Zimmermann, and Strobel, 1972), and puzzle solving (Strobel and Zimmermann, 1971). In addition, all groups were tested for food competition dominance (Wise, Zimmermann, and Strobel, 1973; Wise and Zimmermann, 1973b), food preferences (Peregoy, Zimmermann, and Strobel, 1972), competitive and social dominance (Wise and Zimmermann, 1973b; Wise, Zimmermann, and Strobel, 1973), and visual exploration (Zimmermann and Strobel, 1969). The animals received minimal experience in the Wisconsin General Test Apparatus (WGTA) consisting of shaping and 50 problems of learning set. Social experience was provided to all monkeys two to four times each week in a 243.8 x 228.6 x 203.2 cm playroom with animals of equivalent age and diet conditions. Whereas the LP-SOC and HP-SOC groups began social experience at 120 days of age, such experience was delayed in the LP-ISO and HP-ISO groups until 485 days of age in order to maximize the effects of early partial social isolation.

Apparatus

Delayed alternation problems were presented in a WGTA which consisted of a cage, form board, and table. The form board was a 38.1 x 22.9 cm tray on wheels with food wells located 26.7 cm apart. The tray moved along a track, the length of the 66.0 x 70.5 cm table. Animals placed in the cage were separated from the form board and table by means of vertical iron bars and a movable opaque partition of Masonite. In order to measure the response latency of each animal, a pair of photocells was located 3.8 cm from the partition and at a height of 5.1 cm. The photocells were connected to a photorelay and then to a Hunter timer. Moving the form board to within 3.8 cm of the animal activated the timer, and following each response, removing the tray from within reach of the animal terminated the latency measurement operation. A one-way viewing screen masked the experimenter from view at the other end of the table. The stimuli were identical pairs of 8.9 x 8.9 cm wooden blocks painted "smoke" grey. Reinforcements were sugar-coated pieces of cereal for the low protein animals and raisins or pieces of the 25% protein diet for the high protein monkeys. These differential reinforcers have previously been found to have nearly the equivalent incentive value for the different diet groups previously tested in the WGTA (Strobel, 1972; Strobel, Geist, Zimmermann, and Lindvig, 1974).

Procedure

Pretraining. Because of the minimal experience of all animals with the procedures employed in testing in the WGTA, pretraining was essential, both to familiarize each monkey to the WGTA itself, and to acquaint the animals with the operations involved in responding and with the stimulus objects. Pretraining followed the paradigm of delayed response, albeit with only a single zero second delay period. Each animal was given 36 trials per day until a criterion of 32 correct responses was achieved for two consecutive days. A trial consisted of a single presentation of a stimulus pair and one object was reinforced on each trial. With the opaque partition raised allowing the animal full view of the stimulus objects, a reinforcement was placed by the experimenter in one food well and covered by the correct member of the pair, while the incorrect member of the pair covered the empty food well. A trial was begun immediately by moving the form board within reach of the animal and allowing the response of pushing aside one of the stimulus objects. However, in order to maintain the concept of a discrete trial in which only one response is contained within a given trial (French, 1965), a trial was concluded after a single response regardless of whether or not it was correct. A trial was concluded when the form board was removed from within reach of the monkey and the opaque partition was lowered following the response. The position of the reinforcement was randomized according to a modified Gellermann series (Lester, 1966).

Spatial delayed alternation. Each animal was presented with 36 delayed alternation problems each day. Six problems were presented for each delay interval of 10, 20, 30, 40, 50, and 60 seconds comprising the daily testing session. A problem consisted of multiple presentations of the stimulus pair (trials) employed during pretraining until the correct stimulus member was selected and a food reinforcement was obtained. The experimenter placed a reinforcement into both food wells underlying the stimulus pair on the first presentation of the objects for each delay interval. The opaque partition was lowered to prevent the animal from viewing the process of reinforcement placement. The first presentation of the stimuli was initiated by raising the partition and moving the form board within reach of the monkey. The animal, being presented with two identical objects, responded to a preferred side by pushing aside a member of the stimulus pair. Since both objects covered reinforcement, the animal always secured a reward. On the second and remaining five problems for each delay interval, the correct stimulus object (covering reinforcement) alternated systematically from side to side. Thus, if on problem n, in which both objects covered reinforcement, the animal selected reinforcement from the right member of the stimulus pair, the correct member on problem n + 1 to problem n + 5 was left, right, left, right, left, respectively. Conversely, if an animal selected reinforcement from the left stimulus object on problem n, the correct object on problem n + 1 to problem n + 5 was right, left, right, left, right, respectively. However, a given problem did not necessarily contain only one response (trial). For, if on problems n + 1 to n + 5 an incorrect response was made by the animal, repeated presentations of the stimulus pair were given, with reinforcement remaining on the unchosen side, until a correct response was obtained and reinforcement was secured. Hence, in following such a correctional procedure, each problem terminated in a correct response, after which alternation was resumed (French, 1965). A given presentation of the stimulus pair was concluded when the opaque partition was lowered following a response by the animal. All delay intervals were presented each day for six problems and were ordered across six days according to a six by six Latin Square design for each of the animals. The present investigation of spatial delayed alternation continued for at least 35 days for each of the groups and until evidence for stability of performance was demonstrated by means of a curve-fitting procedure.

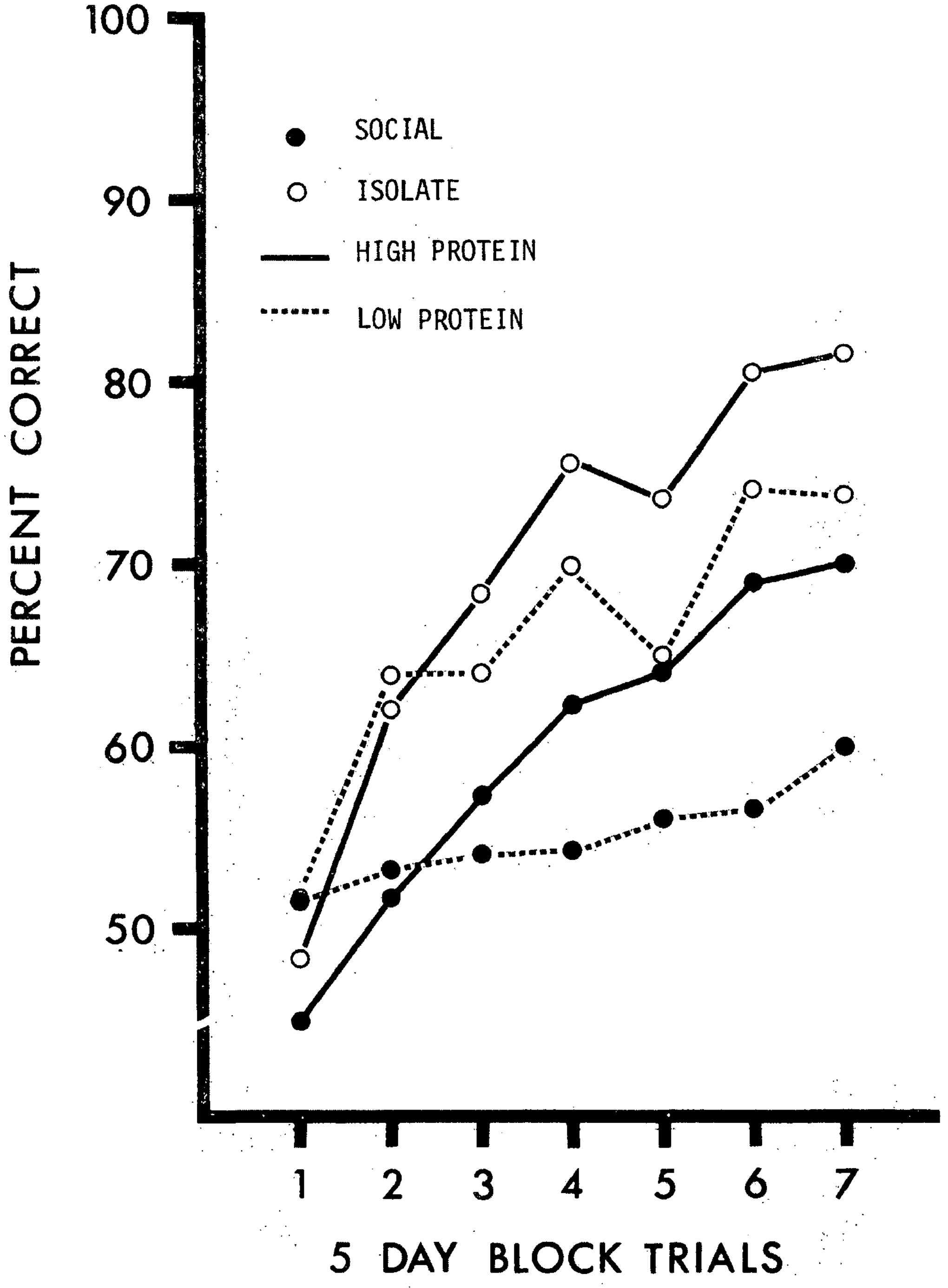
CHAPTER III

RESULTS

Daily percent correct responses (100 times the number of correct responses divided by the sum of the correct and incorrect responses) were calculated for each animal and were averaged across days and animals to produce 5 day block means for each group. Beginning 30 days following the onset of spatial delayed alternation testing, a curve fitting procedure was initiated in order to determine if stability of performance had been attained. The procedure involved prediction of the next 5 day block mean by way of extrapolation from the best parabolic fit of all existing data. Groups were tested until such a time as the obtained 5 day block mean value was greater than or equal to the predicted value and less than or equal to the absolute average deviation between the previously obtained values and the best parabolic fit of those scores. The HP-SOC and LP-SOC groups were tested for 45 days and HP-ISO and LP-ISO groups were tested for 35 days.

Figure 1 presents a summary of percent correct responses across 5 day blocks for all groups. From Figure 1, it is evident that while all groups improved across trials (5 day blocks), they progressed at different rates. Thus, while the HP-SOC group was initially inferior to the LP-SOC group, it rapidly improved and soon surpassed the

Figure 1. Performance across 5 day block trials (HP-SOC, LP-SOC, HP-ISO, and LP-ISO)



LP-SOC group. Similarly, the HP-ISO group, though initially inferior, quickly surpassed the LP-ISO group. (The dip in performance during the testing of the isolate groups at 5 day block number 5 occurred in conjunction with the introduction of a new experimenter.) Additionally, it is evident that the performance of the social groups was consistently inferior to that of the isolate groups.

A 2 x 2 x 6 x 7 fixed effect analysis of variance (n = 4) was performed on the percent correct response data. As such, the analysis was composed of two diets (high and low protein), two environments (social and isolate), six delay intervals (10, 20, 30, 40, 50, and 60 seconds), and seven, 5 day blocks (the maximum number of blocks for which all groups were tested). A summary of this analysis of variance is presented in Table 1 of Appendix A.

As a result of this analysis of variance, the main effect of environment was found to be significant ($p < .01$). The overall mean percent corrent for the social groups was 57.4 while that for the isolate groups was 68.0 (Table 1). The main effect for diet was in the expected direction, 64.9% correct for the high protein groups as opposed to 60.5% correct for the low protein groups. However, it failed to reach significance ($p = .091$). The effect of diet x environment was not significant.

Performance for all groups combined improved across 5 day block trials ($p < .001$) (Figure 2). The environment x 5 day block trials interaction was also significant ($p < .01$) as seen in Figure 3. Thus, while both social and isolate groups initially responded at near chance levels, the isolate groups improved more rapidly than the

Table 1

Main Effects

GROUP	PERCENT CORRECT ALL RESPONSES	PERCENT CORRECT FIRST RESPONSES
Social	57.4	51.1
Isolate	68.0	60.9
High Protein	64.9	58.5
Low Protein	60.5	53.4

Figure 2. Performance across 5 day block trials (averaged for all groups)

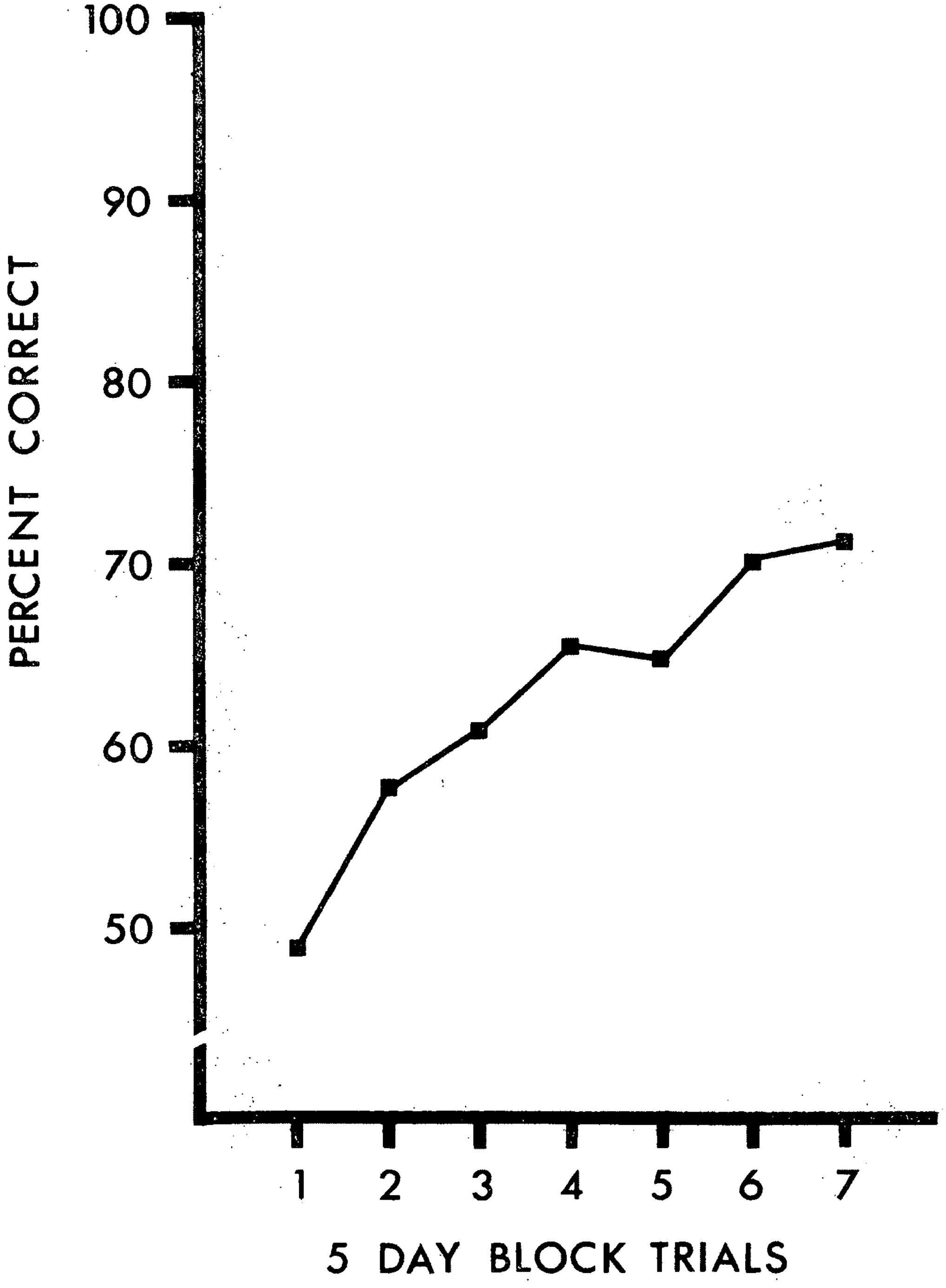
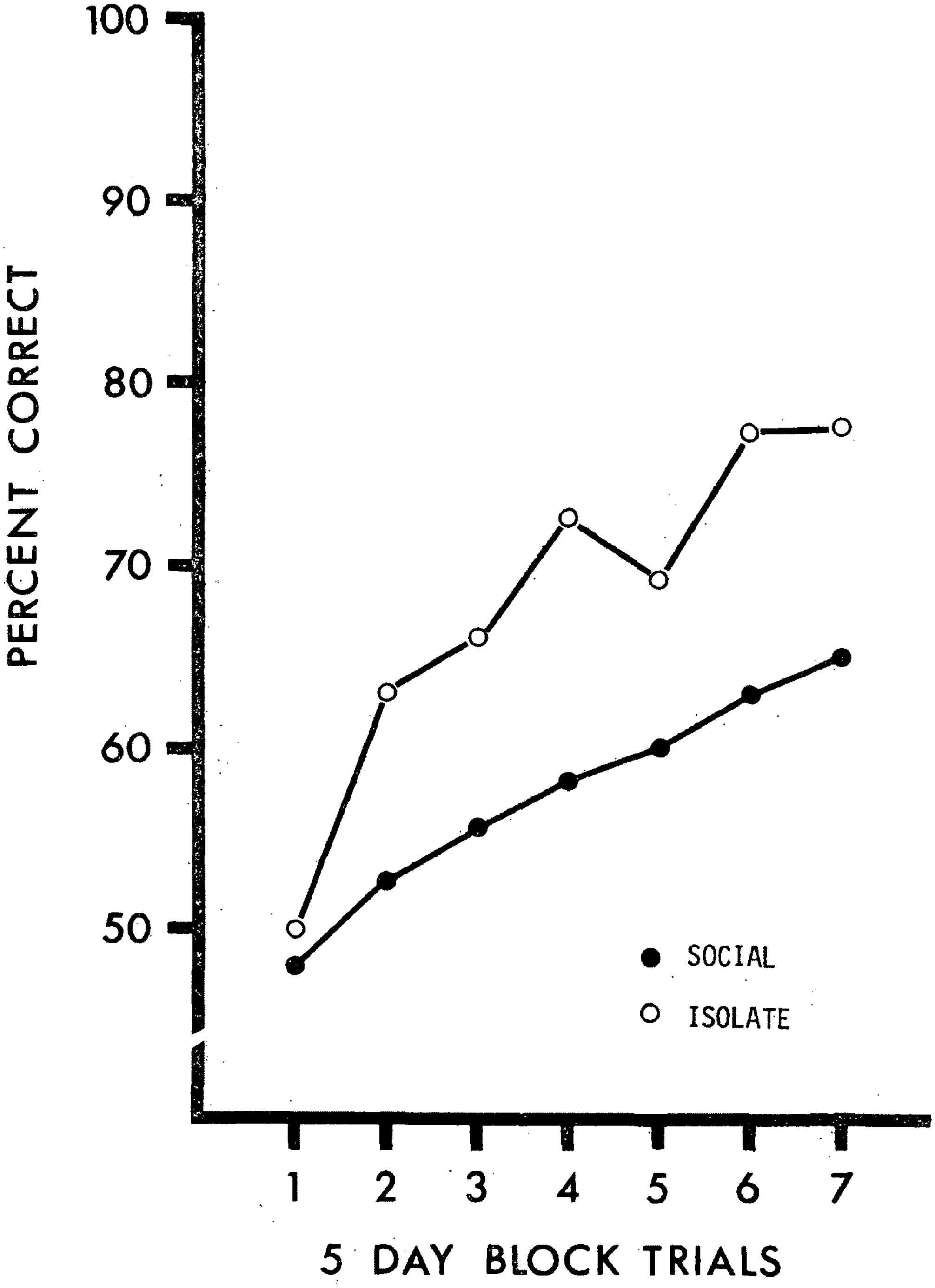


Figure 3. Performance across 5 day block trials (social versus isolate)



social groups and maintained their superiority across blocks. Additionally, the diet x 5 day block trials interaction was also significant ($p < .001$). Thus, while the low protein groups initially performed slightly better than the high protein groups, the high protein groups rapidly surpassed the low protein groups and continued to improve their performance at a faster rate (Figure 4). The diet x environment x 5 day block trials interactions was not found to be significant.

In addition, a significant effect ($p < .05$) due to length of the delay interval was found such that performance decreased slightly from short to long delay intervals (Figure 5). Finally, the diet x delay interaction was significant ($p < .01$). Thus, in Figure 6 it can be seen that high protein groups performed better than low protein groups on the short delay intervals but this difference between groups disappeared at the longer delay intervals. None of the other interactions were found to be significant.

Although the social and isolate groups were tested for an unequal number of days (45 and 35 days, respectively), the above analysis of variance was based on only 35 days of data, i.e., the maximum number of days for which both groups were tested. Since the social groups were tested for 45 days, additional information is contained in an analysis of variance for the social groups based on 45 days of data. A summary of this analysis of variance is contained in Table 2 of Appendix A and for comparison an analysis of variance based on only the isolate groups is contained in Table 3 of Appendix A. The results from these analyses generally follow the same trend as found with the overall analysis of

Figure 4. Performance across 5 day block trials (high protein versus low protein)

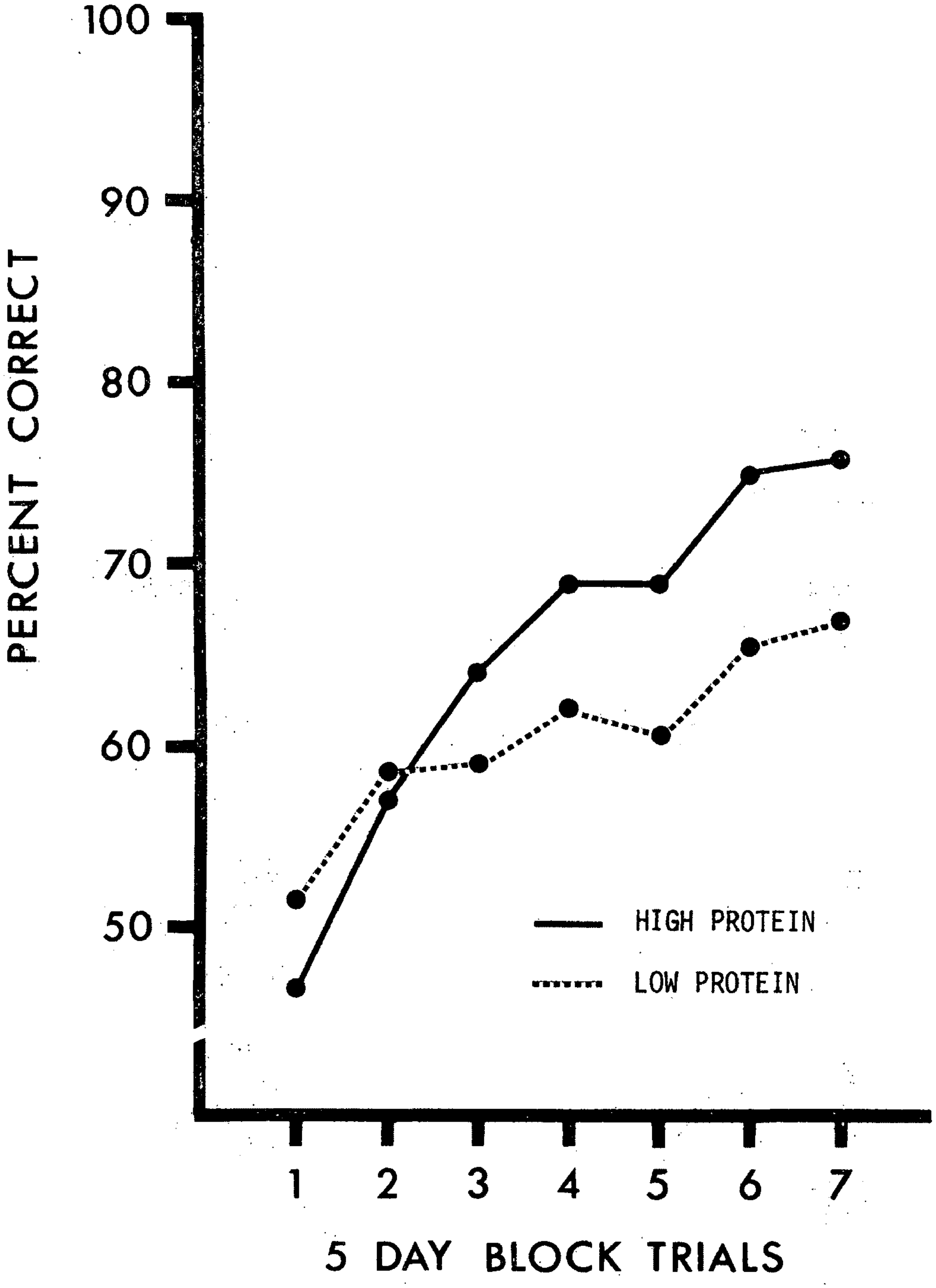


Figure 5. Performance across delay (averaged for all groups)

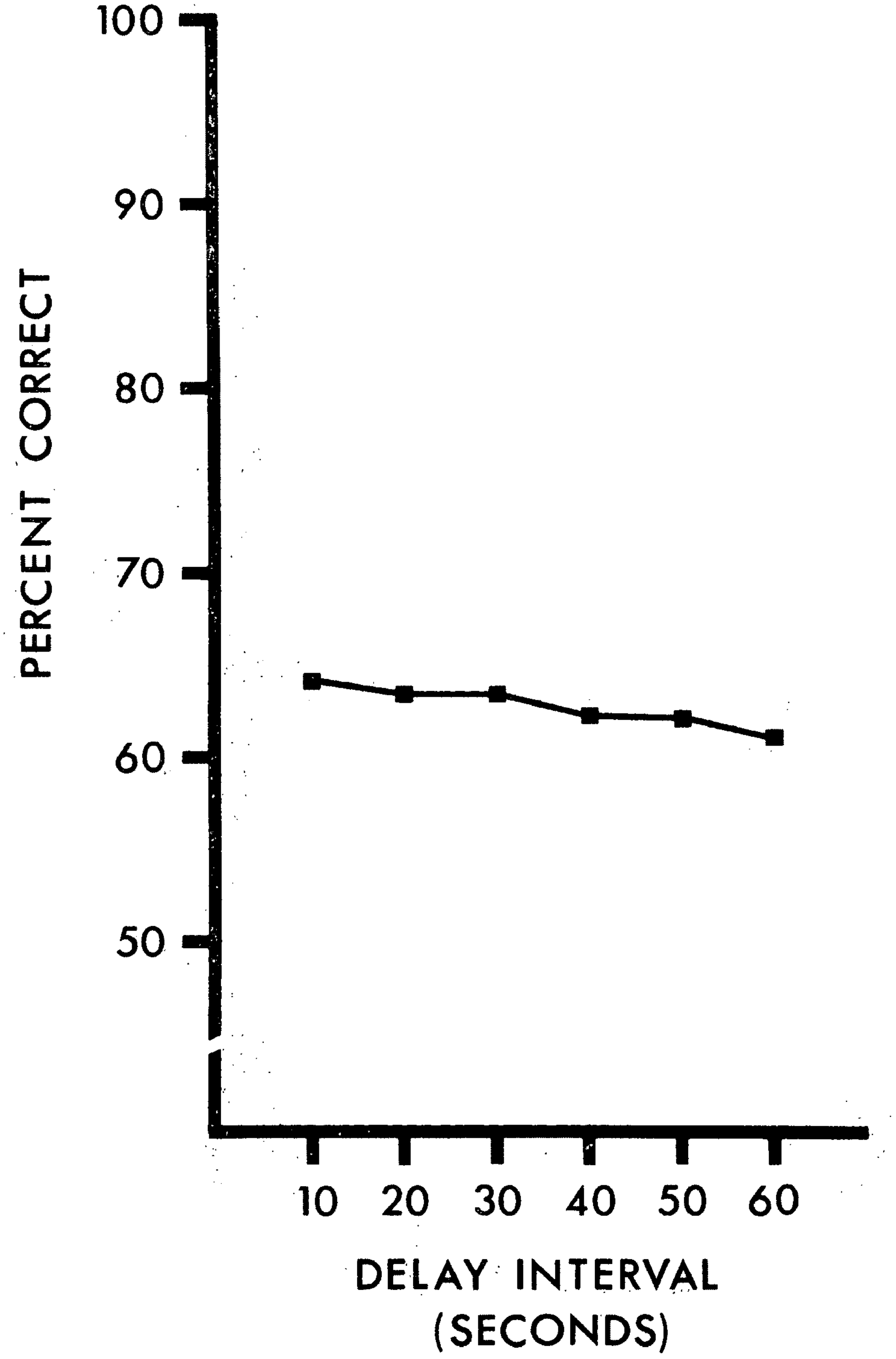
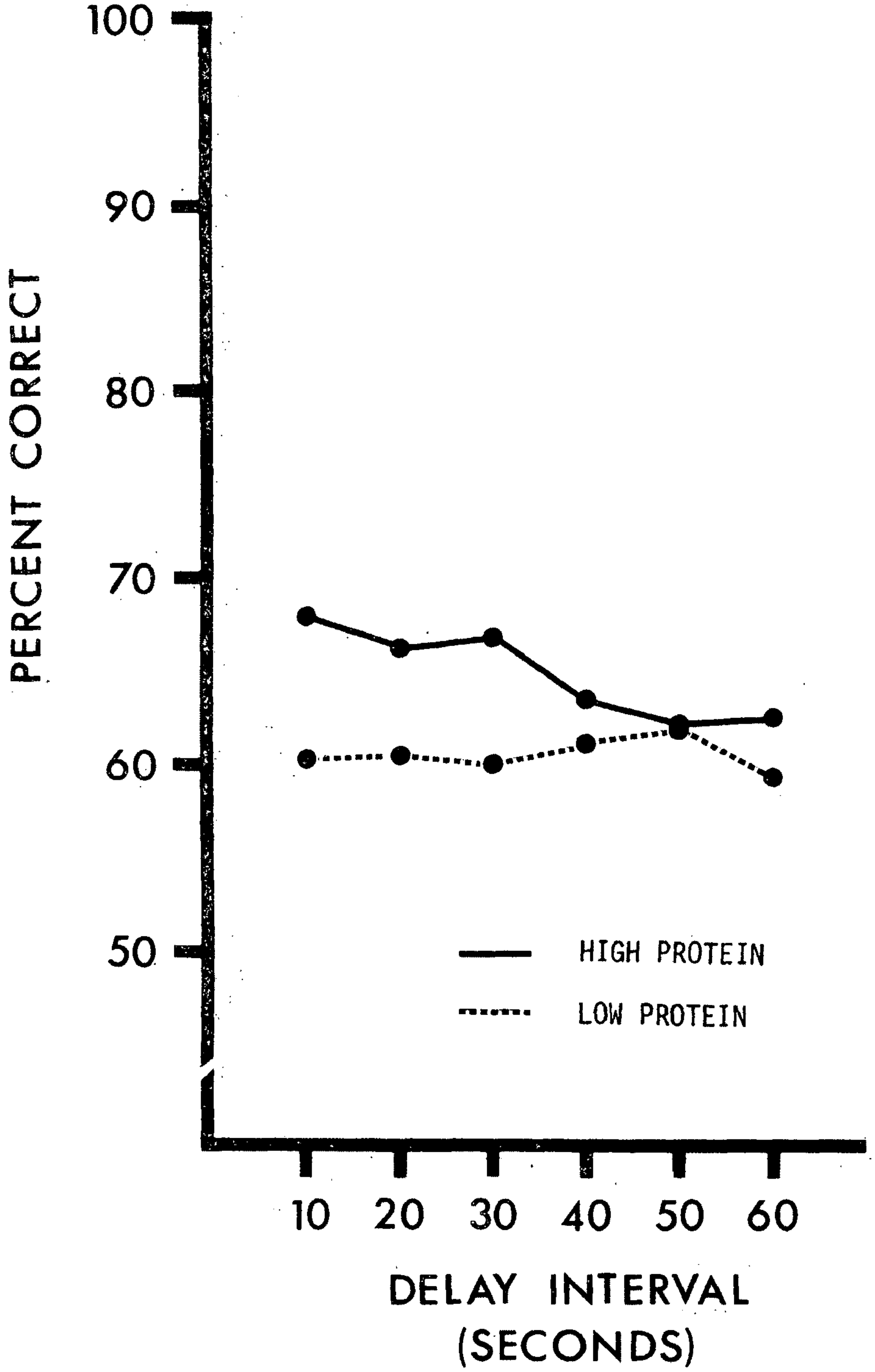


Figure 6. Performance across delay (high protein versus low protein)



variance based on all four groups (Table 1 of Appendix A). However, in the analysis based on only the social groups, the diet main effects reaches significance ($p < .05$), such that HP-SOC animals obtained a mean of 63.9% correct while the LP-SOC group averaged only 56.1% correct. The effects of performance across 5 day blocks and the diet x 5 day block interaction follow the same pattern as in the overall analysis of variance, however, the diet x 5 day block interaction reaches significance only in the social groups comparison ($p < .001$). Similarly, the delay and the diet x delay effects follow the same pattern as in the combined analysis but reach significance only for the isolate groups ($p < .05$).

The above analyses considered percent correct based on all the responses an animal made to a given problem, including correction trials. It is also possible to analyze the percent correct data as a function of the initial response to a given problem. Such a procedure excludes all correction trials from the analysis and considers an animal's performance on a given problem to be correct or incorrect on the basis of his first response to that problem. The summary for such an analysis of variance based on first responses only is presented in Table 4 of Appendix A. In general, the results appear very similar to the analysis contained in Table 1 of Appendix A, with the exception that the effects due to delay and to diet x delay fail to reach significance.

The main effect of environment was found to be significant ($p < .01$) with means of 51.1% correct for the social groups and 60.9% correct for the isolate groups. These values are considerably lower than those obtained from the analysis of variance considering all responses, i.e., 57.4% correct for the social groups and 68.0% correct for the isolate groups (Table 1). Similarly, the main effect for diet was in the expected direction, 58.5% correct for the high protein groups and 53.4% correct for the low protein groups, but failed to reach significance ($p = .072$). Again these values are somewhat below those obtained in the analysis of variance based on all responses, i.e., 64.9% for high protein groups and 60.5% correct for the low protein groups (Table 1). A similar trend is found in Figures 7, 8, and 9 representing percent correct across 5 day blocks ($p < .001$), the environment x 5 day block interaction ($p < .001$), and the diet x 5 day block interaction ($p < .01$). When compared with Figures 2, 3, and 4 (based on all responses) these curves are of remarkably similar shape and slope, but the initial points are somewhat below chance (50%). In addition, all points are consistently lower on the graphs summarizing first responses only.

Tables 5 and 6 of Appendix A contain summaries of the analysis of variance based on first responses for social groups and for isolate groups, respectively. These analyses follow the same general trend as in Tables 2 and 3 (considering all responses) with the exception that the delay and diet x delay effects fail to reach significance and all points are lower.

The difference between the analyses based on all responses and those based on initial responses only is the inclusion of data obtained from the correction trials. The use of the correction procedure allows an animal to make more than one error to a given problem, that is, it

Figure 7. Performance across 5 day block trials--first responses (averaged for all groups)

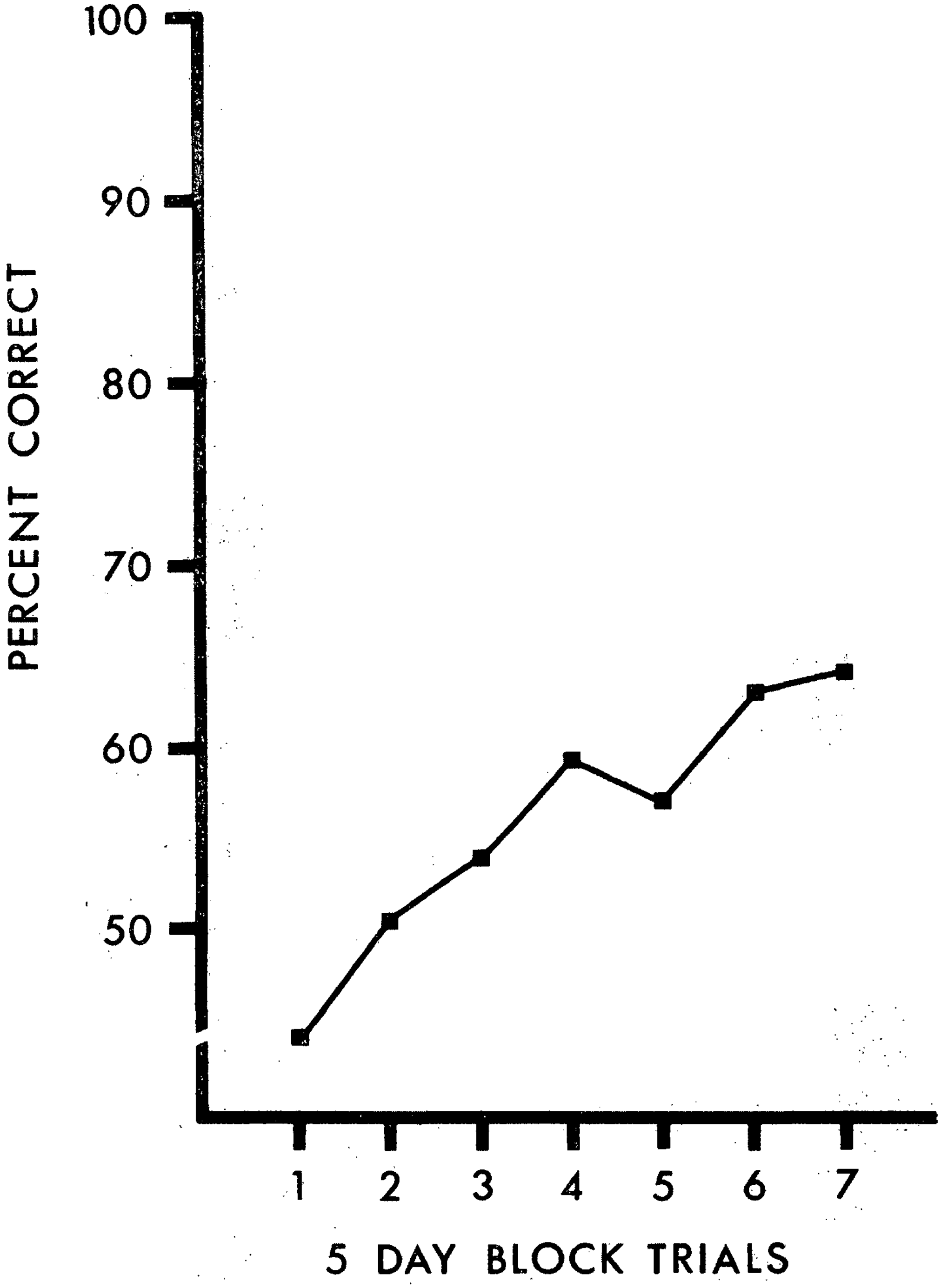


Figure 8. Performance across 5 day block trials--first responses (social versus isolate)

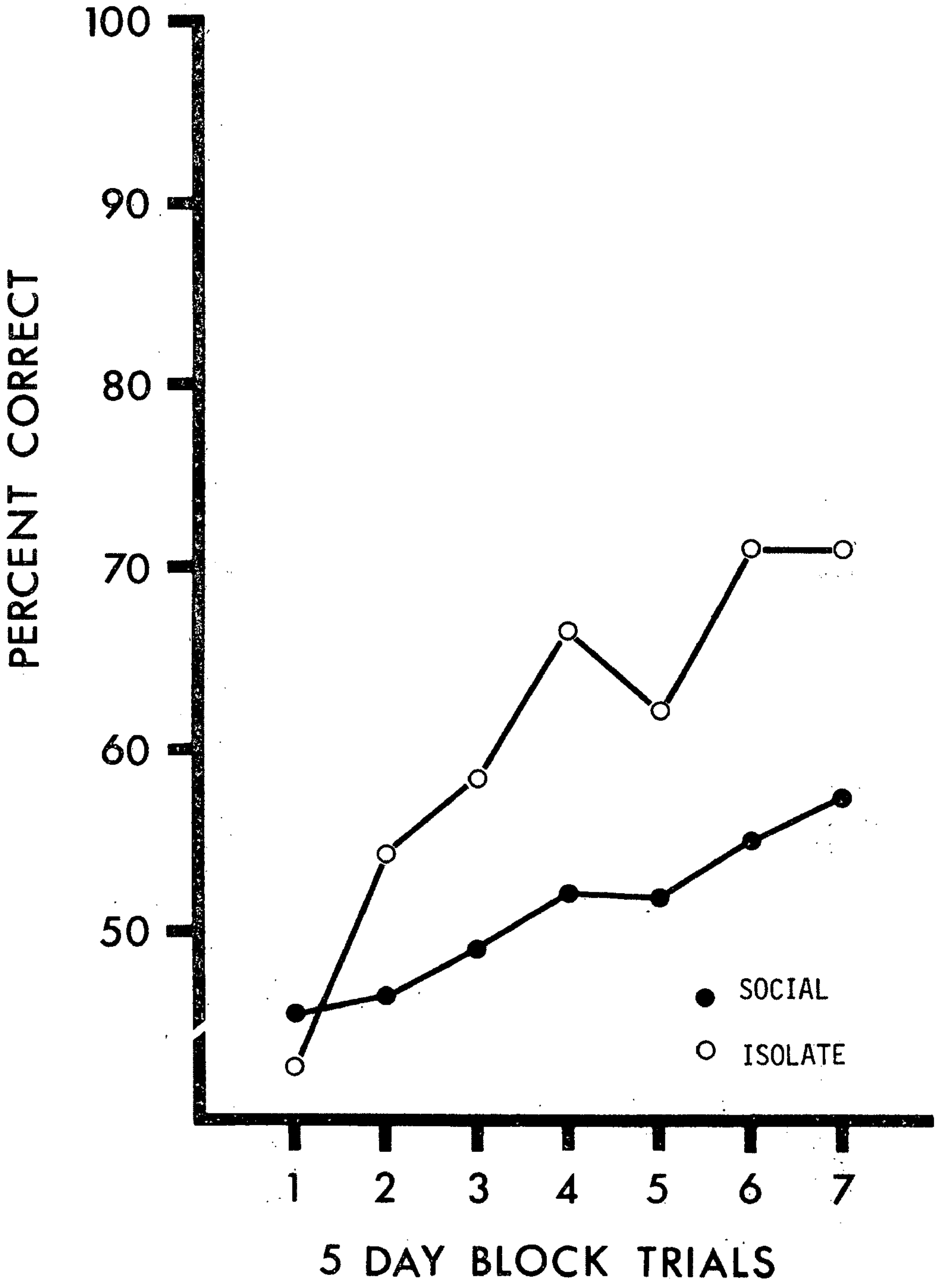
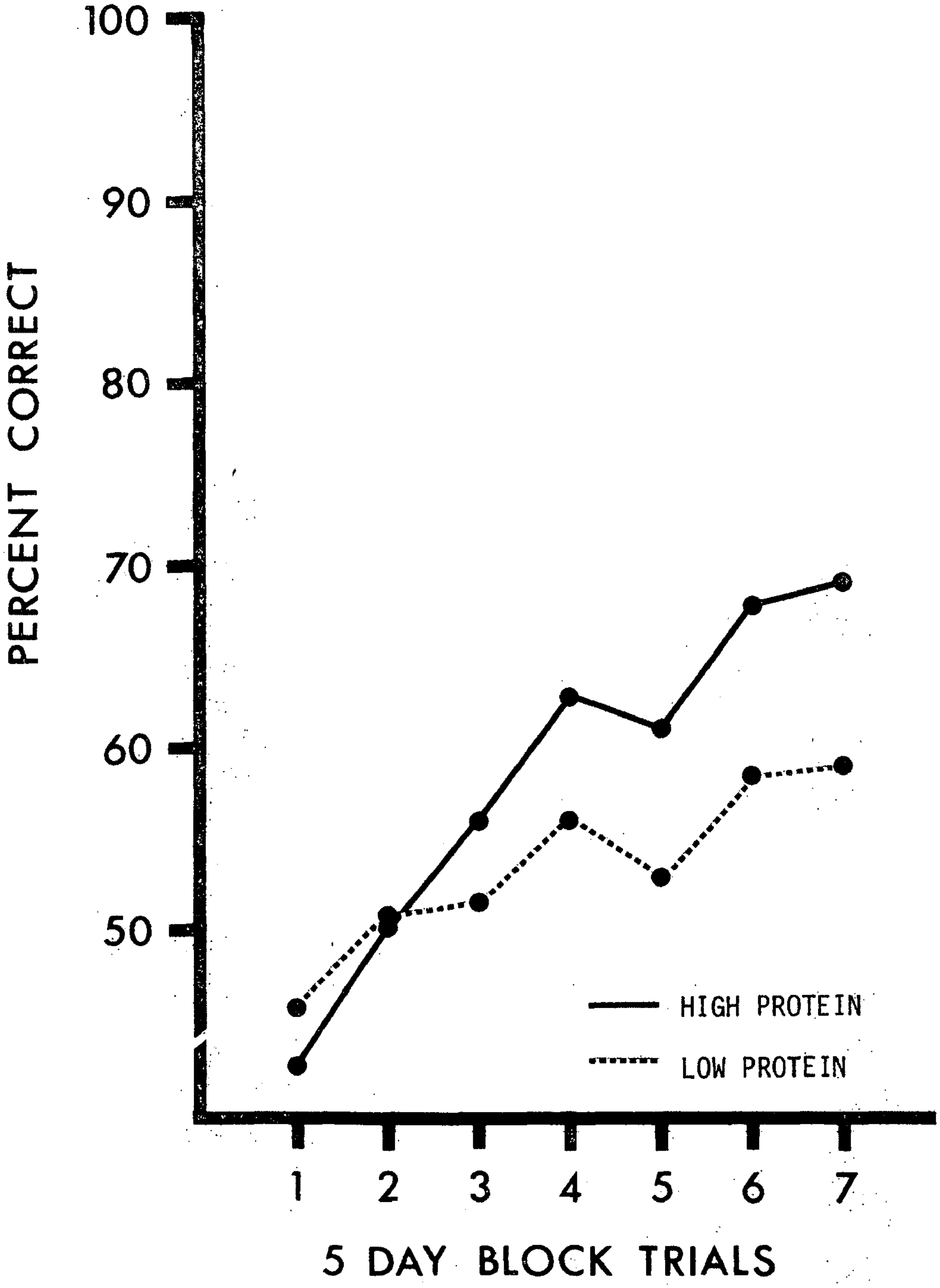


Figure 9. Performance across 5 day block trials--first responses (high protein versus low protein)



allows him to make perseverative response errors until he reaches the correct solution. Thus, it was thought possible that there might be a difference between the groups with respect to the relative frequency of perseverative response errors.

A chi square analysis of perseverative response errors (frequencies of 1, 2, 3, 4, or more than 4 errors to a given problem) in the HP-SOC, LP-SOC, HP-ISO, and LP-ISO groups was performed for each delay interval. As can be seen in Table 7 of Appendix A, the analysis of differential frequency of perseverative response errors across groups was highly significant for all delay intervals. The data was then recombined in order to investigate the possibility of differences resulting from the social versus the isolate groups and from high protein as compared with low protein groups. As can be seen in Table 8 of Appendix A, the differences between social and isolate groups were highly significant across all delays while none of the comparisons involving high and low protein groups reached significance (Table 9 of Appendix A). Therefore, the significant effects found in the comparison of all four groups (Table 7, Appendix A) can be interpreted as resulting from the social animals making proportionally more perseverative response errors than isolates.

Response latencies, the time interval from the introduction of the form board until the animal has responded, were recorded for all trials. A 2 x 2 x 6 x 7 fixed effect analysis of variance (diet x environment x delay interval x 5 day block trials) was performed on this data and is summarized in Table 10 of Appendix A. Neither the diet nor the environment main effects were significant. The environment x 5 day block trials interaction was significant ($p < .01$) and is represented in Figure 10. It can be seen that the social groups' response latency was initially quite low, increased rapidly until the fourth 5 day block, and dropped back down to an intermediate level. In contrast, the isolate groups initially displayed long response latencies, but these rapidly dropped to low values. The length of the delay interval also showed a significant response latency effect ($p < .001$). As can be seen in Figure 11, response latency increased from a low to a moderate level as delay intervals increased. In addition, the environment x delay interaction was significant ($p < .05$). In Figure 12 it is evident that in the social groups, response latency increased from moderate to high values as the length of the delay interval increased as opposed to the isolate groups where response latency remained low across delay intervals, increasing only slightly across delay intervals. A very similar trend is found in the diet x delay interval interaction ($p < .01$). As can be seen in Figure 13, response latency increased from moderate to high values as the length of the delay interval increased in the low protein groups, and while remaining low, increased slightly across delay intervals in the high protein groups.

A reciprocal transformation, which is often used with time data, was performed on the response latency data. An analysis of variance based on this transformed data is presented in Table 11 of Appendix A, and was not found to change the significance levels of any of the effects.

Figure 10. Response latency across 5 day block trials (social versus isolate)

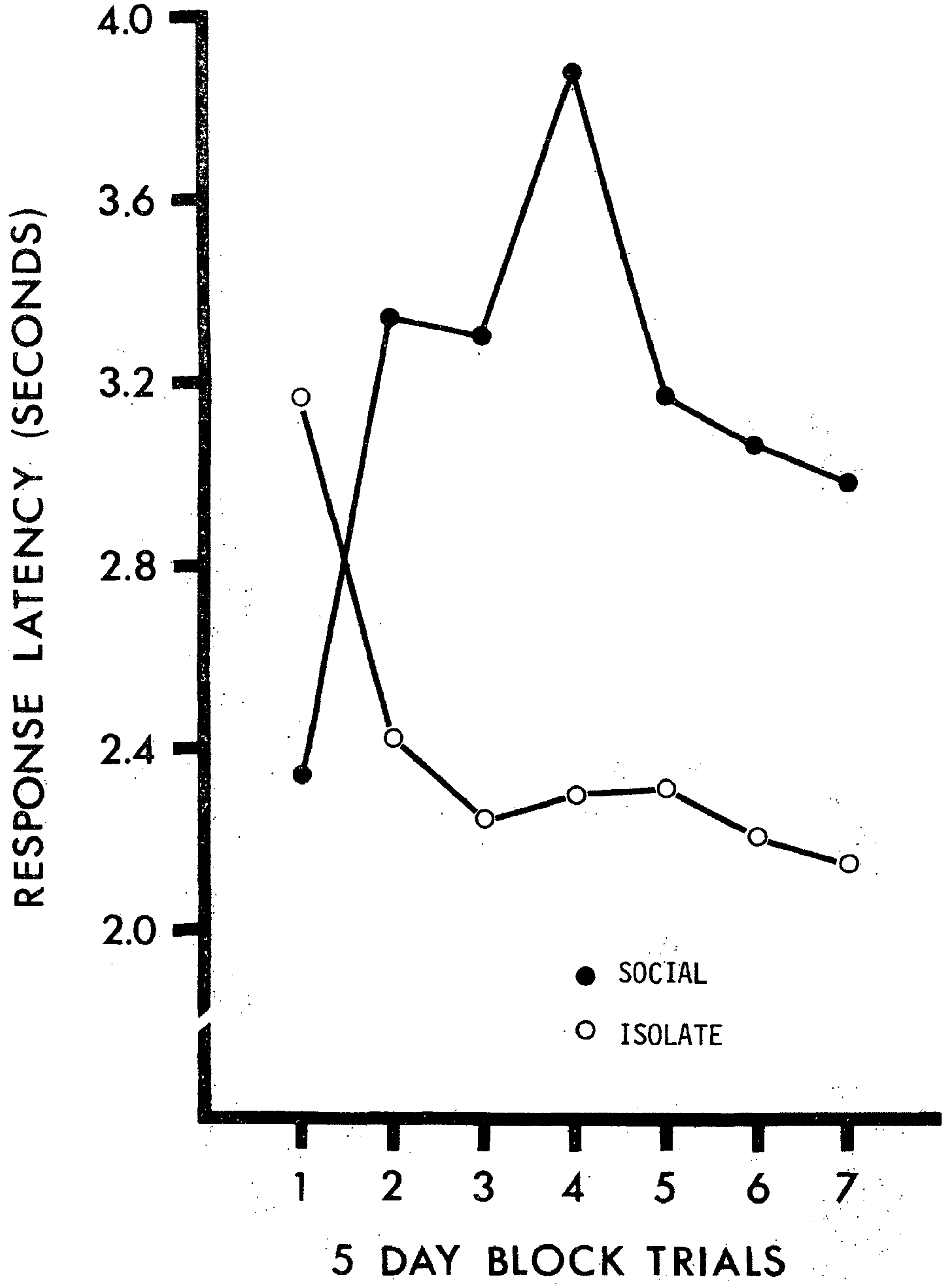


Figure 11. Response latency across delay
(averaged for all groups)

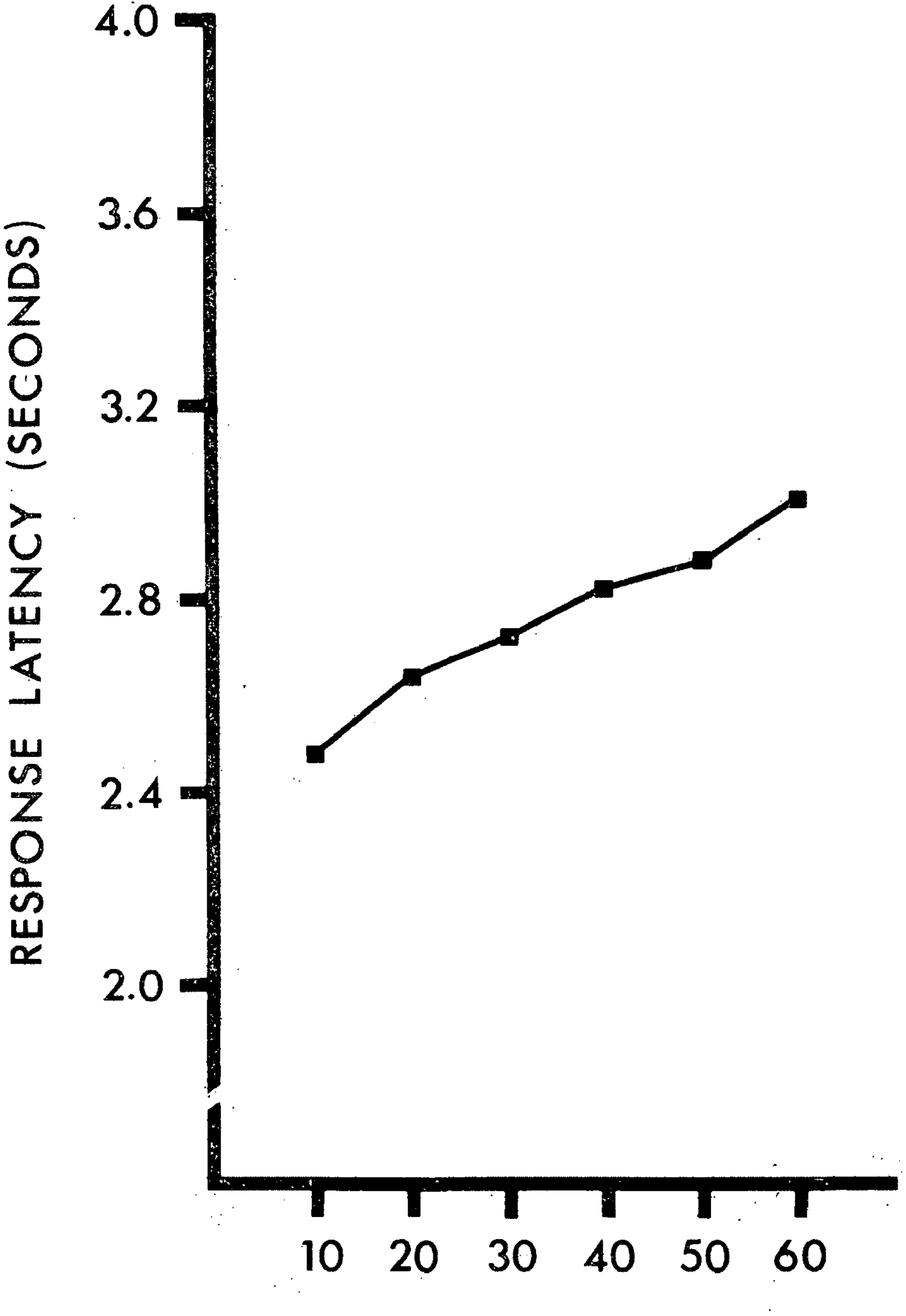


Figure 12. Response latency across delay (social versus isolate)

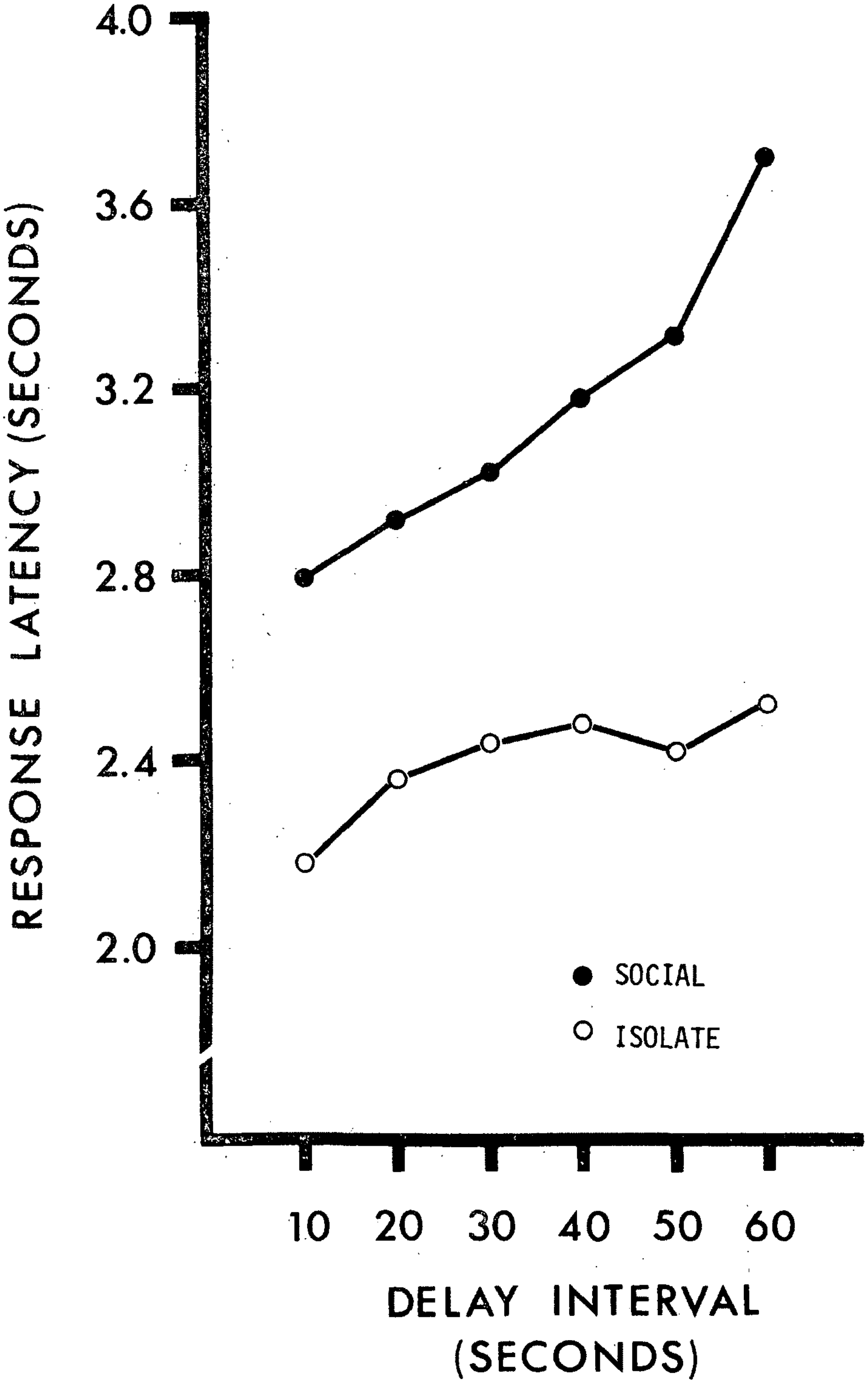
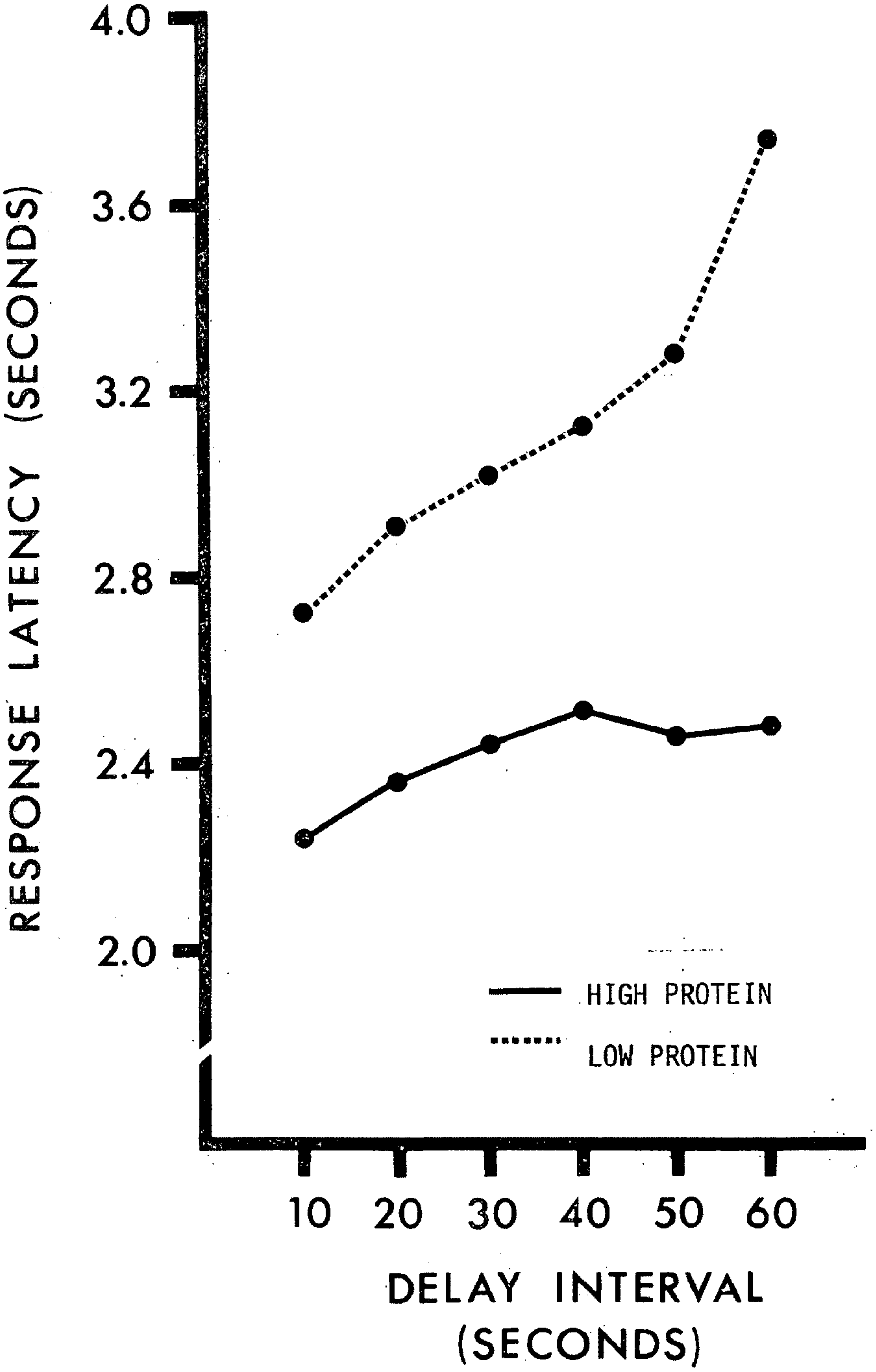


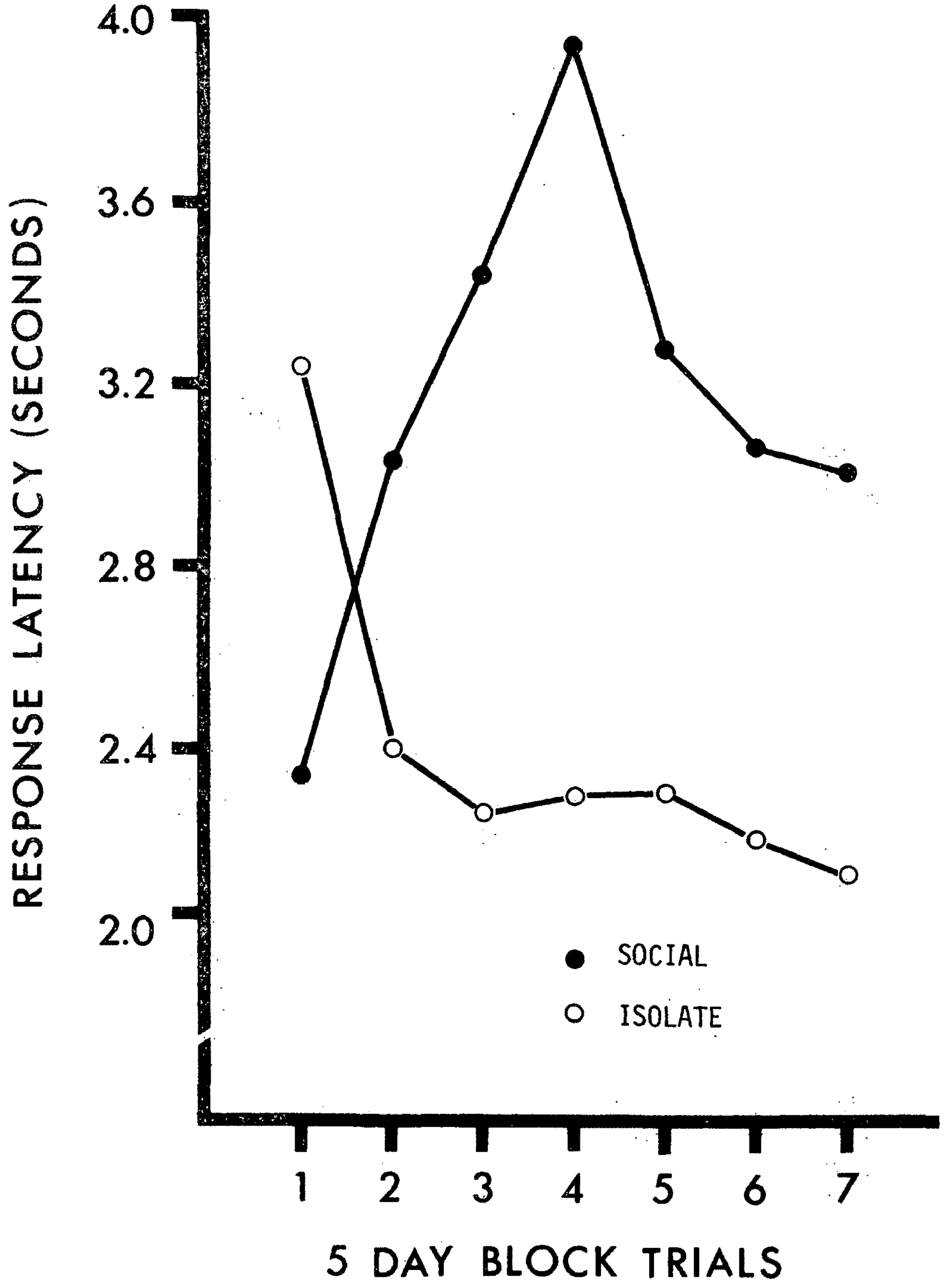
Figure 13. Response latency across delay
(high protein versus low protein)



A separate analysis of variance of response latencies was performed for the social groups based on 45 days of data (Table 12 of Appendix A) and for comparison, an analysis of the isolate groups alone is included in Table 13 of Appendix A. These analyses follow the same general trend as the overallanalysis (Table 10, Appendix A) with the exception that the 5 day blocks effect reached significance in the isolate groups comparison, and diet x delay interval interaction failed to reach significance in either analysis.

Finally, since the number of responses at each delay interval varies, the daily mean response latency is not equivalent to the average of the response latencies for the delay intervals. In other words, the response latencies for the various delay intervals must be weighted in order to obtain the daily mean response latency. The data from these weighted means was analyzed by means of a 2 x 2 x 7 (diet x environment x 5 day block trials) analysis of variance and is summarized in Table 14 of Appendix A. Only the environment x 5 day block trials interaction was significant ($p < .001$) and as can be seen in Figure 14, this interaction is very similar to that seen in Figure 10, based on unweighted means.

Figure 14. Mean response latency across 5 day block trials (social versus isolate).



CHAPTER IV

DISCUSSION

The Diet Effect

As predicted, a general improvement across 5 day block trials on the delayed alternation task was found ($p < .001$). All groups improved, but they progressed at different rates and eventually reached different levels of performance. However, while differences between groups were anticipated, the hypothesis that the high protein groups would perform better than the low protein groups is not supported by a significant main effect for diet. Thus, while the diet effect was in the expected direction, 64.9% correct for high protein as opposed to 60.5% correct for low protein animals, it failed to reach significance ($p = .091$). A similar trend was noted in the analysis of first response data, i.e., the main effect for diet, while in the expected direction, failed to reach significance ($p = .072$). (The importance of first response data will be discussed in more detail later.)

However, the failure of the diet main effect to reach significance can be easily explained with reference to the diet by 5 day block trials interaction. As can be seen in Figure 4 for overall percent correct ($p < .001$) and in Figure 9 for percent correct first responses ($p < .01$), the effect of diet varied across trials such that, while initially inferior to the low protein groups, the high protein groups quickly improved, surpassing the low protein groups by the third 5 day block and maintaining their superiority for the remainder of the experiment. This type of interaction, where the effects cross each other, acts to lessen the main effect. Thus, the performance of the high protein groups on this task is distinct from that of the low protein groups when viewed with respect to performance across trials, but the superiority of the high protein groups tends to be obscured due to their initial inferiority when only the main effect is considered.

This initial inferiority of the high protein groups and the general tendency to perform at below chance (50% correct) levels (Figures 4 and 9) at the first 5 day block may at first seem puzzling, but is not difficult to explain when the experimental paradigm is considered. Thus, it is reasonable to assume that when first confronted with the delayed alternation task, the animal will respond according to the law of effect. In other words, following reinforcement, the animal will tend to repeat his previous response (win-stay). (Sutherland and Mackintosh [1971] maintain that while there is evidence that rhesus monkeys and chimpanzees do develop strategies, there is no need to appeal to a win-stay, lose-shift strategy when simple operations of reinforcement and nonreinforcement are adequate to explain the data.) However, efficient performance on delayed alternation requires the adoption of a win-shift, lose-shift strategy, that is, correct performance on delayed alternation with correction requires that an animal respond counter to the law of effect. Thus, to the extent that an animal recalls his previously reinforced response and adopts a win-stay strategy, his initial performance will tend to be below chance. Therefore, for the first 5 day block, the general tendency to respond at below chance levels, especially immediately following reinforcement (Figure 9), can be seen as the result of an experimental paradigm that requires the animal to respond counter to the law of effect.

In addition, it is evident from Figures 4 and 9 that this tendency toward less than chance performance at the first 5 day block is greater in the high protein animals. One possible explanation for this closer to chance performance in the low protein groups is that the low protein groups are content to respond at chance levels, especially when confronted with a difficult problem (Strobel, 1972). However, since they eventually respond at above chance levels, this explanation seems inadequate. Alternatively, it is possible that both the high protein and the low protein groups initially adopt a win-stay, lose-shift strategy, but that the low protein groups are relatively less efficient in applying this strategy due to a failure to attend to their previous response. Thus, while it has been demonstrated that low protein monkeys are able to remember the location of a reward when the critical cue is provided by the experimenter as in delayed response (Zimmermann, Geist, Strobel, and Cleveland, 1973), it was argued earlier that they may experience difficulty in attending to their own past behavior. If low protein monkeys do have difficulty in attending to their own past behavior, they will experience difficulty in applying a win-stay strategy with reference to that behavior. As a result, their scores should deviate less from chance than those of the high protein groups.

Such an argument could also account for the inferior performance of the low protein groups across the remaining 5 day blocks (Figure 9). As group performance rises above 50% correct, it can be assumed that the monkeys have abandoned their original and never reinforced win-stay hypothesis and have adopted a win-shift hypothesis. Again, such a strategy requires the animal to attend to his preceding reinforced response and to shift with reference to that response. If the low protein animal experiences difficulty in attending to his last response, he will be less efficient at shifting with reference to that response and therefore should perform less well than high protein animals using a win-shift strategy. Alternatively, it is possible to argue that low protein animals are simply slower to adopt the win-shift strategy and consequently perform less well than the high protein animals. Such an argument by itself fails to account for the different first 5 day block data points.

It should be noted that the above discussion deals mainly with the first response percent correct data. This emphasis is intentional and is due to the fact that in the case of a correct response (win), that is, following reinforcement, the animal must learn to change his strategy from win-stay to win-shift; whereas following an incorrect response, both the experimental design and the basic laws of reinforcement and nonreinforcement require a lose-shift strategy. Assuming that lose-shift also requires attending to past behavior, low protein animals would be expected to perform less well than high protein animals following an incorrect response. Trial-to-trial response patterns will be discussed in more detail later, but differential performance following an incorrect response does not appear to be the case, as high and low protein animals seem to perform equally well following an error. In other words, there were no differences between diet groups with reference to perseverative response errors (Table 9, Appendix A). Therefore, it seems reasonable to assume that the failure of low protein animals to attend to their previous behavior following reinforcement is directly associated with obtaining reinforcement. It may be that low protein animals are so disrupted when they receive reinforcement that they fail to remember or attend to the response that led to reinforcement. It will be recalled that Levitsky and Barnes (1972) suggested as one of several possible mechanisms to account for the behavioral effects of malnutrition, that malnutrition may produce behavior, such as extreme food orientation, that is incompatible with the incorporation of environmental information. Such an interpretation seems to be compatible with the hypothesized disruptive effect of food reinforcement on attending to one's own behavior. If such food orientation is solely responsible for the differential performance between high and low protein animals following reinforcement, then it would be expected that such differences between groups would disappear in experimental paradigms which do not use food as a reinforcer.

Finally, the significant ($p < .05$) main effect of delay intervals (Figure 5) is perhaps best viewed with respect to the significant ($p < .01$) diet by delay interaction (Figure 6). Thus, it appears that whatever advantage high protein animals have on the shorter delays, this advantage is lost at the longer delay intervals. Also, it is of importance to note that the performance of low protein monkeys does not fall off at the longer delays and may even tend to improve slightly. It was previously suggested that low protein monkeys are suffering from attentional deficit and it was assumed that problems with longer delays make increased attentional demands on the animal. Therefore, it was predicted that the performance of the low protein monkeys would fall off relatively more rapidly than the performance of high protein monkeys as the length of the delay interval increased. Since this prediction was found to be incorrect, it is appropriate to examine the underlying assumptions, and since the arguments have already been discussed with reference to an attentional deficit and the support for this notion presented, an evaluation of the assumption that longer delays are more difficult is in order.

As can be seen in Figure 5, while the delay effect is significant ($p < .05$), it is slight, suggesting that longer delay intervals are only slightly more difficult than are shorter delays. Little support with respect to this finding comes from the standard delayed alternation studies since they tend to use only one delay interval, generally 5 seconds (Rosvold and Delgado, 1956; Rosvold, Mishkin, and Szwarcbart, 1958; Mishkin, 1957; Pribram and Tubbs, 1967; Stamm, 1964; Abplanalp and Mirsky, 1973). However, Riopelle and Churukian (1958) reported that on a visual discrimination learning task, performance did not vary sharply as a function of intertrial interval (10, 30, 60, and 120 second intervals). Therefore, it seems likely that the failure to find the predicted trend with reference to the diet by delay interaction was a result of incorrectly assuming that 60 second delay intervals would be considerably more difficult for rhesus monkeys than 10 second delays.

It may be possible to explain the obtained diet by delay interaction with reference to the relative rate of reinforcement for the different delay intervals. Since the main effect for diet was not significant with reference to response latencies, it can be assumed that low protein monkeys and slightly deprived high protein monkeys are approximately equally motivated with respect to obtaining food reward. However, while on the average, equally motivated, food deprived high protein animals and ad lib low protein animals may respond slightly differently to changes in the relative rate of reinforcement. Thus, as the rate of reinforcement decreases as a result of longer intertrial delay intervals, high protein animals may become slightly less motivated to perform, causing a slight drop in their performance, while low protein animals with their extreme food orientation, may maintain or even slightly increase their motivation to perform, thus maintaining their level of performance.

Finally, it is worth noting that the delayed alternation performance in this study is generally lower than is usually reported in the literature. However, there are several differences between this study and the classical delayed alternation with correction studies. First, in the standard paradigm a 5 second delay interval has normally been used (Rosvold and Delgado, 1956: Mishkin, 1957; Rosvold, Mishkin, and Szwarcbart, 1958; Pribram, Mishkin, Rosvold, and Kaplan, 1952; Pribram and Tubbs, 1967; Abplanalp and Mirsky, 1973) although Stamm

(1964) used 7 seconds. Secondly, only one delay interval per study was used and therefore only on the initial trial for a given day were both boxes baited. While the longer delay intervals in the present study may have served to lower overall performance, Figure 5 suggests that this was not the case since within the 10 and 60 second range, the length of the delay interval had only a slight effect. However, it is possible that the shape of this function is quite different in the 0 to 10 second range. However, it seems more likely that baiting both boxes 6 times instead of once per day was largely responsible for the relatively low performance levels. This provision was originally included in order to provide an opportunity for the monkeys to make a shift with reference to their pattern of responding. However, since the only signal for the opportunity to make a response shift was a change in delay interval, the monkeys may have been unaware of this opportunity to shift their response pattern. Assuming that rhesus monkeys form hypotheses while problem solving, this provision probably had a deleterious effect on performance. Thus, on 5 out of 36 trials per day, a monkey engaging in hypothesis testing was likely to receive incorrect information concerning his hypothesis.

The Environment Effect

In addition to diet affecting performance on the delayed alternation task, it was predicted that a significant environment main effect would be found such that the socially enriched groups would perform better than the isolate monkeys. Such a prediction was based on the several studies investigating diet by environment interactions in rats which reported that the effects of malnutrition were exaggerated by environmental deprivation (Frankova, 1968; Levitsky and Barnes, 1972; Wells, Geist, and Zimmermann, 1972), along with a knowledge of the severe social and emotional disturbances which characterize socially deprived monkeys (Mason, 1968). Noting the behavioral similarities between protein-malnourished animals and those subjected to early environmental deprivation, several investigators have suggested that very similar mechanisms may be involved in producing these effects (Levitsky and Barnes, 1973; Zimmermann, Steere, Strobel, and Hom, 1972). Thus, it seemed reasonable to assume that socially deprived monkeys would perform less well than enriched animals on the delayed alternation task.

However, this was not found to be the case. While a significant main effect for environment was found ($p < .01$), it was in a direction opposite to that predicted. Thus, the overall percent correct for isolates was 68.0 while that for social animals was only 57.4. A similar trend is present when only first response data is considered ($p < .01$). As can be seen in Figure 3 for overall percent correct, and in Figure 8 considering first responses only, the isolate groups are consistently and strikingly superior to the social animals.

While there is considerable literature concerning social and emotional abnormalities following environmental and social deprivation, there has been relatively little research on how social rearing conditions affect learning ability and much of this literature is confusing. For instance, Rowland (1964a, b) totally isolated infant monkeys for 6 or 12 months and tested them during and following isolation on both discrimination learning and learning set formation. While he found no significant differences in performance between isolates and controls, the rearing conditions of control animals were not specified. Since this research was conducted in the Harlow laboratory, the so-called control animals were probably partial social isolates. Such a finding would be consistent with the data of Griffin and Harlow (1966) in which no significant differences in learning set formation were found between partial social isolates and monkeys totally isolated from birth to 3 months. The difficulty with this research and that of Harlow, Schiltz, and Harlow (1969) which reported some differences between groups, is that total isolates are being compared with controls which are in fact partial social isolates. (Partial social isolates are raised in individual cages from birth where they can see and hear but not touch other monkeys.) Since the environment main effect in the present study (Figures 3 and 8) was found between partial social isolates and socially enriched subjects, the above studies are of little explanatory value. However, Harlow, Harlow, Schiltz, and Mohr (1971) reported the results of a test battery administered to 6 and 9 month total social isolates (isolated for the first 6 or 9 months of life), socially enriched monkeys (reared in a nuclear family housing apparatus), and controls (once again, partial social isolates). While the control animals seem to be of little interest to Harlow in this study, they are in fact the most consistently superior group. The first test of the battery involved 20 discrimination problems of 25 trials each. While the results of this study failed to reach significance ($p < 0.1$), the trend was for total isolate and control monkeys to perform better than enriched animals. Similarly, when tested on 600 problems of 6 trial learning set, the control and isolate groups were found to be significantly superior to the enriched groups ($p < .05$). When tested for 1,800 trials of 0 and 5 second delayed response, the control animals were found to be superior to the isolate and enriched groups ($p < .05$). Similarly, when tested on multiple delayed response (5, 10, 20, and 40 second), the trend ($p < 0.1$) was for control to be superior to isolate and enriched animals. Only when tested on oddity learning set, were enriched animals found to be superior to isolates and controls ($p < .05$). Thus, on four out of five of the tests in this battery, there was at least a trend for control animals (partial social isolates) to demonstrate superior performance. However, Harlow, concerned mainly with the enriched versus total isolate comparisons, failed to note this trend and concluded that although early environment may greatly alter emotional and personality variables, it has little or no effect on learning or intellectual variables.

In summary, in Harlow et al. (1971), which appears to be the only study which compares learning ability between socially enriched and partially socially isolated monkeys, the trend was for superior performance by the partial social isolates (controls). Therefore, the finding in the present study, that partial social isolates outperformed enriched monkeys on the delayed alternation task, is not inconsistent with the findings reported by Harlow et al. (1971).

While there is little information on how rearing environments affect learning in monkeys, there is even less data which might help to explain this phenomenon. Isolates are frequently described as emotional and fearful, but Harlow et al. (1971) maintained that their adaptation sequence greatly alleviates this problem and it is interesting to note that when presented with what Harlow described as a somewhat emotionally taxing task (0 and 5 second delayed response), it was his enriched monkeys who made totally inadequate responses while reportedly threatening the experimenter and ignoring the problem. During delayed alternation testing a similar phenomenon was noted. Socially enriched animals were relatively emotional, that is, they tended to run in circles and to call frequently, or alternatively, to engage in a variety of quiet but competing behaviors such as grooming or playing with the sawdust in the floor of the cage. As can be seen in Figure 10, such animals also tended to exhibit long response latencies. In contrast, partial social isolates tended to be quiet, less active, and responded more readily. Thus, the socially enriched monkeys appeared to be more emotionally aroused during WGTA testing than did the isolates.

Harlow et al. (1971) attributed the behavior of the enriched monkeys during 0 and 5 second delayed response testing to latent insecurity and hostility as a result of being removed from the security of friends and family, and there is some experimental evidence for a sort of separation anxiety occurring in rhesus monkeys. For instance, Willott and McDaniel (1974) studied the reaction of pairs of juvenile rhesus monkeys to threat of separation and reported that the animals displayed disturbance, especially pacing, increased activity, distress vocalizations, and threats. In addition, several investigators have studied the affects occurring during peer separation in juvenile rhesus monkeys (Erwin, Mobaldi, and Mitchell, 1971; McKinney, Suomi, and Harlow, 1972; Erwin, Brandt, and Mitchell, 1973) and have generally reported increases in locomotion, coo-screeching (Erwin, Mobaldi, and Mitchell, 1971), and stereotyped movements. Therefore, it seems likely that socially enriched monkeys are stressed when removed from their home environments and placed in the WGTA.

In addition to undergoing peer separation, there are several additional factors which might add to the stress of socially enriched monkeys in the WGTA test situation. For instance, it was more difficult to persuade the social monkeys to enter the transport cage and occasionally the WGTA, and therefore, social animals may be more aroused when they arrive at the test situation. In addition, social monkeys are not accustomed to being confined in a small cage as are the isolates and may find such confinement to be stressful. Finally, while isolates spend much of their time sitting quietly in their cages, social animals live in a more active environment and may find delay intervals especially disturbing.

In summary, the socially enriched monkeys displayed relatively more emotional behavior during WGTA testing than did isolates and several reasons have been offered to account for this behavior in terms of stress due to changes in the social and physical environment which would not be expected to influence the behavior of partial social isolates to the same degree.

Such an emotionality argument can also be used to explain the perseverative response data. It will be recalled that since a correction procedure was employed in delayed alternation testing, when the animal made an incorrect response to a problem, that problem was presented repeatedly until he made the correct response. The perseverative response analysis was performed because it appeared to the experimenter during the course of testing, that there was a tendency in social animals to respond with chains of errors. While it is possible, as Harlow et al. (1971) suggested, that enriched monkeys under stress simply ignored the problem, this argument does not seem terribly compelling. Harlow (1959a) suggested a better answer. Thus, he reported the results from testing 12, 30, and 50 month rhesus monkeys on the Hamilton Perseverance Test. In this test, the animal was presented with four boxes having spring-loaded lids and was allowed four trials in order to find the food reward which was contained in one of the boxes. An error in this paradigm was defined as making an additional response to an unrewarded box after lifting the lid previously during the problem. There was a tendency for 30 and 50 month monkeys to make many fewer perseverative errors of this type than did year old monkeys. Thus, an alternative explanation for the perseverative response errors found in the delayed alternation performance is that enriched animals under the stress of testing in the WGTA, tended to exhibit a more juvenile response pattern.

In summary, on the delayed alternation task, socially enriched animals were found to make more first response errors and more perseverative response errors than did partial social isolates. This finding receives support from a general trend evident in the study by Harlow et al. (1971). Due to general observations of their behavior during testing and the evidence for emotional responses during peer separation (Erwin, Mobaldi, and Mitchell, 1971; McKinney, Suomi, and Harlow, 1972; Erwin, Brandt, and Mitchell, 1973), it has been suggested that this inferior performance of socially enriched monkeys during delayed alternation testing in the WGTA is a result of emotional behavior due to relatively large changes in the social and physical environment of these animals. Thus, it seems likely that these differences between enriched and isolate groups would fail to occur if the animals were tested in their home cages.

The Diet by Environment Interaction

Noting the similarities between the emotional and social abnormalities seen in protein malnourished monkeys and those subjected to early social deprivation, Zimmermann, Steere, Strobel, and Hom (1972) suggested that low protein monkeys might be considered functional social isolates. Thus, it was proposed that similar mechanisms might be involved in producing the behavioral effects seen in malnutrition and early social deprivation. It was therefore hypothesized that social deprivation and protein malnutrition would interact in the delayed alternation experiment. However, as can be seen in Table 1 of Appendix A, no such interactions were found. The answer to this somewhat puzzling situation was probably given by Harlow et al. (1971) when they emphasized a difference between measuring the effects of social deprivation on emotional stability and measuring the effects on learning ability *per se*.

The studies which have reported interactions between rearing and diet conditions, have tended to deal with this first measure.

Thus, Frankova (1968) and Levitsky and Barnes (1972), both working with rats, reported significant diet by environment interactions with such measures as activity; tendency to explore a new environment, and social behavior. Similarly, Elias and Samonds (1974), working with cebus monkeys, reported significant diet by environment interactions for exploratory behavior and activity. Finally, Wells, Geist, and Zimmermann (1972) found a significant diet by environment interaction in rats tested on maze performance. It should be obvious that all these studies capitalize on the excessive fearfulness and neophobia of low protein and isolates animals. Thus, Harlow et al. (1971) emphasized that if an investigator was interested in evaluating learning capacity in socially deprived animals, he should be careful to provide extensive adaptation to the test situation and should avoid designs that involve pain or shock, or which tend to be emotionally disturbing as would variants of an open field (including Hebb Williams mazes). Thus, although diet by environment interactions have been reported, they have tended to measure emotionality rather than learning <u>per se</u>.

In the present experiment, all groups received extensive adaptation to the test situation and were familiar with the test stimuli as a result of pretraining. Under such conditions, the effects of excessive fearfulness in the test situation and neophobia in isolates and low protein monkeys should have been minimized. However, while Harlow has suggested that the fearfulness of isolates habituates as a result of extensive experience with the test situation, this has not been shown to be the case with "separation anxiety." Thus, when the behavior of separated rhesus juveniles was recorded during two days (Erwin, Mobaldi, and Mitchell, 1971; and Erwin, Brandt, and Mitchell, 1973) or two weeks of peer separation (McKinney, Suomi, and Harlow, 1972), the behavioral effects were found to persist throughout separation. In addition, Willott and McDaniel (1974) would not have been able to obtain their results, which required conditioning subsequent separation to the appearance of a transport cage, if fear of separation habituated rapidly as a result of repeated separations. Thus, there is the suggestion that while fear of novelty (characteristic of low protein and early social isolates) habituates, this does not appear to be the case with respect to "separation anxiety" (characteristic of social animals). In the present study, the experimenter took precautions to eliminate emotional behavior in the isolate and low protein monkeys but was unaware that such a factor might be operating in social animals. Such emotional behavior was quite likely responsible for the lowered performance of enriched animals. (Since low protein animals tend to behave as functional social isolates in a variety of circumstances, the effect of separation on the LP-SOC group would be expected to be attenuated.) Additional evidence for the suggestion that emotional behavior did not account for the lowered performance of the low protein groups is provided by the failure to find differences in the distribution of perseverative response errors as a result of diet.

In summary, while diet by environment interactions have been reported in the literature, such that the effects of malnutrition have been enhanced by social isolation, they have been found in studies which tend to measure heightened emotional behavior, a characteristic of both these syndromes. The failure to find a significant interaction between diet and environment in the present study tends to support Harlow's argument that there are differences between measuring the emotional stability and the learning capacity of isolates. Furthermore, there is the suggestion that there are two mechanisms involved in producing the behavioral abnormalities associated with protein malnutrition. Thus, it may be that one mechanisms produces its effects by creating an animal that is to some degree a functional social isolate, while a second mechanism, perhaps involving an attentional deficit (possibly in the brain) acts to product a learning deficit.

Response Latencies

Strobel, Geist, Zimmermann, and Lindvig (1974) and Strobel (1972) reported that the various reinforcers used with low protein monkeys and slightly deprived high protein monkeys, i.e., raisins or high protein diet for the high protein groups and sugar coated cereal for the low protein groups, have equal incentive values. Thus, assuming that response rate is directly related to food motivation, it was hypothesized that no significant diet effects for response latencies would be found. As predicted, the diet main effect and the diet by 5 day block trials interaction were not significant. In order to provide evidence that the superior performance of high protein animals on delayed alternation could not be attributed to increased motivational levels as a result of food deprivation, the HP-ISO group was placed on _ad lib_ feeding and tested for an additional 5 days at the end of the experiment. Response latencies increased during this period, from an average of 2.0 seconds to 5.1 seconds while performance dropped 11%. Thus, there seems to be some evidence for the assumption that response latency is a measure of motivation to perform and that the diet groups were approximately equated with respect to this measure throughout the experiment. However, it should be noted that these findings do not agree with those of Gross. Gross (1963) tested a group of rhesus monkeys on 3 or 5 second delayed alternation performance and reported that deprivation level did not alter performance and in addition, that latency and performance were not significantly correlated. However, Gross used 2, 26, or 50 hours of deprivation and did not have an ad lib group. Therefore, it is difficult to make comparisons between his findings and those found under ad lib conditions, or to evaluate the effect of deprivation level on the acquisition of delayed alternation.

As previously mentioned, a significant ($p < .01$) environment by 5 day block trials effect was found for response latencies (Figure 10) and the relatively long response latencies of the social animals were attributed to their participation in competing behaviors. The rapid drop in response latency across 5 day blocks demonstrated by the isolate groups may correspond to habituation of fearfulness.

In addition a significant environment by delay effect ($p < .05$) was found such that both groups took longer to respond as the delay interval increased (Figure 12). There was a general, although slight tendency for performance to decline as the delay interval increased (Figure 5), however, it is not possible to determine form correlational data if performance declined as a result of longer response latencies (essentially lengthening the delay interval) or if longer response latencies were the result of more difficult problems (longer delay intervals).

Trial-to-Trial Response Patterns

In beginning a discussion of trial-to-trial response patterns, it is perhaps helpful to review some of the data analyses which have been previously mentioned. Figures 1 through 6 refer to analyses performed on overall percent correct response data. In such analyses, overall percent correct responses was defined as 100 times the number of correct responses divided by the sum of the correct and incorrect responses. Since a correction procedure was employed in this experiment, the daily number of incorrect responses, and therefore, the divisor in the above calculation was variable. However, this is not the case when first response data (Figures 7 through 9) is considered since analyses based on initial responses to a given problem exclude information based on correction trials. The analysis of perseverative response errors was employed to evaluate the data obtained from correction trials.

It can be assumed that if the probability of a correct response were constant across trials, then the overall percent correct response data would be equivalent to the percent correct of first responses. However, this was not found to be the case. That is, percent correct of first responses was consistently less than overall percent correct. This finding has led to a consideration of trial-to-trial response patterns.

Since percent correct of first responses is less than overall percent correct, it must be concluded that the probability of an error on a first response trial is in general greater than the probability of an error on a correction trial. This conclusion follows from the fact that by definition, the primary difference between first response trials and correction trials is that first response trials have a correct response immediately preceding them while correction trials have an error preceding them.

A tendency for this greater probability of a correct response following an error than following a correct response has been noted by several investigators of discrimination learning in rhesus monkeys. Moss and Harlow (1947) provided animals with one or two training trials consisting of presentation of a single positive (rewarded) or negative (non-rewarded) stimulus object prior to testing, and reported higher test trial discrimination performance following non-reward than rewarded training. Variations of this procedure have produced similar results as reported by Harlow and Hicks (1957) and King and Harlow (1962). (However, Miles [1965] failed to obtain this effect in squirrel monkeys.) In all these cases, the tendency for the probability of a correct response to be less following reward was attributed to a response shift error, i.e., the tendency of the monkey to investigate an unexplored stimulus object. This so-called error factor is discussed by Harlow (1959b) along with three additional error factors, differential cue (the tendency to respond to the position previously yielding food rather than to the object), stimulus perseveration (the tendency for the animal to make repetitive responses to the incorrect object, presumably as a result of stimulus preference), and position habit. However, position habit is quickly overcome in primate learning, and stimulus perseveration, differential cue, and response shift are only appropriate explanations for an error when two different stimulus objects are present, as in learning set, but not delayed alternation. Furthermore, Bowman and Takemura (1966) have demonstrated a response shift type of phenomenon in a situation where neither stimulus object had previously been displaced.

Thus, while the above studies provide support for the finding that the probability of a correct response is greater following an error than following a correct response, the explanation in terms of a response shift (as a tendency to investigate non-displaced objects) cannot be used to explain this phenomenon in the delayed alternation data. As was previously suggested, it may be that reinforcement is sufficiently disruptive to monkeys as to disturb their performance on the following trial or conversely, an error immediately following reinforcement may serve to focus attention more sharply.

While it is difficult to offer an explanation for the above general phenomenon in the delayed alternation data, when the trend across perseverative errors is considered, the results are even more puzzling. Since the above studies were based on discrete trial data (learning set), perseverative response errors could not occur in the sense that they can with a correction procedure. Within the framework of a correction procedure, the probability of perseverative errors can be determined. Thus, as can be seen in Appendix B, Figure 1 for group data and Appendix B, Table 1 for individual subjects, there is not only a trend for the probability of an error to decrease following a single error, but this trend reverses with perseverative errors (apparently tending toward chance). When individual subjects are considered, and using the probability of an error following a correct response as a reference, it can be seen that approximately 3/4 of the subjects follow the trend of a decreased probability of an error following a single error and increased probability of an error following a second or third (perseverative) error. Therefore, it appears that whatever the advantage of committing a single error immediately following reinforcement, it is not maintained for perseverative errors.

Frontal Lobe Damage

It was previously suggested that the deficits in learning ability or attention demonstrated by the low protein monkeys might be associated with an actual brain deficit. There are several parallels between low protein monkeys and those with frontal lobe lesions which should be mentioned as they may suggest a possible mechanism for the attentional deficits seen in protein malnutrition. First, deficits in delayed alternation performance following frontal lobe lesions are commonly reported in the literature (Mishkin, 1957; Stamm, 1964; Pribram and Tubbs, 1967). These deficits seem to be specific to the frontal lobes, other neocortical regions not being essential for the task (Thompson, 1967). However, lesions in rhinencephalic regions and the caudate nucleus produce similar effects (Rosvold and Delgado, 1956; Rosvold, Mishkin, and Szwarcbart, 1958). Thus, both monkeys with frontal lobe lesions and monkeys reared on low protein diets showed deficits in delayed alternation performance. Secondly, Strobel

(1972) and Strobel, Geist, Zimmermann, and Lindvig (1974) reported inferior performance of low protein monkeys on tasks involving stimulus-response discontinuity. Likewise, French (1962) and Riopelle and Churukian (1958) reported that frontal monkeys experienced difficulties on tasks involving stimulus-response discontinuity. In addition, while the frontal lobe is often considered to be involved in short term memory, Riopelle and Churukian (1958) found that the visual discrimination learning of the frontal monkey was neither increased nor decreased sharply as a function of intertrial intervals of 10, 30, 60, and 120 seconds, a finding which again parallels the results obtained in the present experiment. Finally, diet effects were not found to be significant with reference to perseverative response errors in the present study. Similarly, perseverative response errors were not found to be significant in frontal monkeys performing a sequential task which was similar to delayed alternation in that no cue was constantly related to reward and the correct response was contingent only on previous action (Pinto-Hamuy and Linck, 1965).

Thus, there appear to be several similarities between the inferior performance seen in monkeys with frontal lobe lesions and those reared on low protein diets which may tend to suggest frontal lobe damage in low protein monkeys. However, monkeys with frontal lobe lesions also display inferior delayed response performance, an effect not found with lesions in other areas of the cortex (Thompson, 1967). Zimmermann, Geist, Strobel, and Cleveland (1973) did not find a deficit in delayed response performance for monkeys placed on the experimental diets at 210 days of age. Since the monkeys in the present experiment were placed on the experimental diets much earlier, at 120 days of age, the delayed response study should probably be replicated with animals experiencing the earlier dietary manipulation. However, while there are several similarities between frontal and low protein animals, it seems unlikely that the effects of protein malnutrition will be found to be simply analogous to those reported with frontal lobe lesions.

CHAPTER V

SUMMARY

Protein-calorie malnutrition in rhesus macaques has been shown to result in a variety of behavioral abnormalities. However, when PCM is encountered in the human condition, it often occurs within the context of not only dietary, but also general environmental deprivation. Therefore, it is of importance to investigate how environmental deprivation and PCM interact to produce their effects. In the present study, the effects of nutritional and environmental deprivation on rhesus monkeys were investigated in a factorially designed experiment.

Sixteen infant rhesus monkeys were separated from their mothers at approximately 90 days of age and housed individually. At 120 days of age, these infants were divided into four experimental groups (n = 4): high protein social, low protein social, high protein isolate, and low protein isolate. The environment manipulation involved rearing either in group living cages or individually (partial social isolation). Nutrition was varied by feeding either a diet consisting of 25% casein by weight (23.6% of calories) or an isocaloric diet containing only 3.5% casein (3.3% of calories).

All groups were between two and four years of age when they were adapted to the Wisconsin General Test Apparatus, pretrained in order to overcome position preferences, and tested on a spatial delayed alternation with correction task. Significant differences were found both for environment and diet effects across 5 day blocks such that high protein subjects performed better across blocks than low protein subjects and isolates performed better than socially enriched animals. No interactions between diet and environment were found.

The diet effect was interpreted as suggesting an attentional deficit due to malnutrition, while the environment effect was attributed to increased emotionality during testing in the WGTA. The failure to find a significant diet by environment interaction suggests that these factors may produce their behavioral effects through different mechanisms.

REFERENCES

Aakre, B., Strobel, D. A., Zimmermann, R. R., and Geist, C. R. Reactions to intrinsic and extrinsic rewards in protein-malnourished monkeys. Perceptual and Motor Skills, 1973, 36, 787-790.

Abplanalp, J. M., and Mirsky, A. F. Electroencephalographic correlates of delayed-alternation and visual discrimination learning in rhesus monkeys. Journal of Comparative and Physiological Psychology, 1973, 85, 123-131.

Barnes, R. H. Experimental animal approaches to the study of early malnutrition and mental development. Federation Proceedings, 1967, 26, 144-147.

Barnes, R. H. Behavioral changes caused by malnutrition in the rat and pig. In D. C. Glass (Ed.), Environmental Influences. New York: Rockefeller University Press, 1968, pp. 52-60.

Barnes, R. H. Experimental studies in animals: Physiologic and behavioral correlates of malnutrition. In W. M. Moore, M. M. Silverberg, and M. S. Read (Eds.), Nutrition, Growth and Development of North American Indian Children. Department of Health, Education, and Welfare Publication No. (NIH) 72-26, Washington, D. C., 1972, pp. 121-128.

Barnes, R. H., Cunnold, S. R., Zimmermann, R. R., Simmons, H., MacLeod, R. B., and Krook, L. Influence of nutritional deprivations in early life on learning behavior of rats as measured by performance in a water maze. Journal of Nutrition, 1966, 89, 399-410.

Barnes, R. H., Moore, A. U., and Pond, W. G. Behavioral abnormalities in young adult pigs caused by malnutrition in early life. Journal of Nutrition, 1970, 100, 149-155.

Barnes, R. H., Moore, A. U., Reid, I. M., and Pond, W. G. Learning behavior following nutritional deprivations in early life. Journal of the American Dietetic Association, 1967, 51, 34-39.

Barnes, R. H., Moore, A. U., Reid, I. M., and Pond, W. G. Effect of food deprivation on behavioral patterns. In N. S. Scrimshaw and J. E. Gordon (Eds.), Malnutrition, Learning, and Behavior. Cambridge, Massachusetts: Massachusetts Institute of Technology Press, 1968, pp. 203-218.

Barnes, R. H., Reid, I. M., Pond, W. G., and Moore, A. U. The use of experimental animals in studying behavioral abnormalities following recovery from early malnutrition. In R. A. McCance and E. M. Widdowson (Eds.), Calorie Deficiencies and Protein Deficiencies. Boston: Little, Brown and Company, 1968, pp. 277-285.

Bengoa, J. M. Recent trends in public health nutrition. Protein-calorie mlanutrition. In Nutrition, Proceedings of the Eighth International Congress, Prague, September, 1969. Amsterdam: Excerpta Medica, 1970.

Benton, J. W., Moser, H. W., Dodge, P. R., and Carr, S. Modification of the schedule of myelination in the rat by early nutritional deprivation. Pediatrics, 1966, 38, 801-807.

Blomquist, A. J., and Harlow, H. F. The infant rhesus monkey program at the University of Wisconsin primate laboratory. Proceedings of the Animal Care Panel, 1961, 11 57-64.

Bradfield, R. B. Changes in hair associated with protein-calorie malnutrition. In R. A. McCance and E. M. Widdowson (Eds.), Calorie Deficiencies and Protein Deficiencies. Boston: Little, Brown and Company, 1968, pp. 213-220.

Brockman, L. M., and Ricciuti, H. N. Severe protein-calorie malnutrition and cognitive development in infancy and early childhood. Developmental Psychology, 1971, 4, 312-319.

Brown, R. E. Decreased brain weight in malnutrition and its implications. East African Medical Journal, 1965, 42, 584-595.

Bowman, R. E., and Takemura, K. The response shift phenomenon in discrimination learning set. Psychonomic Science, 1966, 4, 31-32.

Cabak, V., and Najdanvic, R. Effect of undernutrition in early life on physical and mental development. Archives of Disease in Childhood, 1965, 40, 532-534.

Canosa, C. A., Salomon, J. B., and Klein, R. E. The intervention approach: The Guatemala study. In W. M. Moore, M. M. Silverberg, and M. S. Read (Eds.), Nutrition, Growth and Development of the North American Indian Children. Department of Health, Education, and Welfare Publication No. (NIH) 72-26, Washington, D. C., 1972, pp. 185-199.

Champakam, S., Srikantia, S. G., and Gopalan, C. Kwashiorkor and mental development. American Journal of Clinical Nutrition, 1968, 21, 844-852.

Cobos, F. Malnutrition and mental retardation: Conceptual issues. In Lipids, Malnutrition and the Developing Brain. Ciba Foundation Symposia. New York: Elsevier, 1972, pp. 227-246.

Coursin, D. B. Relationship of nutrition to central nervous system development and functions: Overview. Federation Proceedings, 1967, 26, 134-138.

Cravioto, J. Nutritional deficiencies and mental performance in childhood. In D. C. Glass (Ed.), Environmental Influences. New York: Rockefeller University Press, 1968, pp. 3-51.

Cravioto, J., DeLicardie, E. R., and Birch, H. G. Nutrition, growth and neurointegrative development: An experimental and ecologic study. Pediatrics, 1966, 38, 319-372.

Cravioto, J., and Robles, B. Evolution of adaptive and motor behavior during rehabilitation from kwashiorkor. American Journal of Orthopsychiatry, 1965, 35, 449-464.

Culley, W. J., and Lineberger, R. O. Effect of undernutrition on the size and composition of the rat brain. Journal of Nutrition, 1968, 96, 375-381.

Culley, W. J., and Mertz, E. T. Effect of restricted food intake on growth and composition of preweanling rat brain. Proceedings of the Society for Experimental Biology and Medicine, 1965, 118 233-235.

Deo, M. G., Good, S. K., and Ramalingaswami, V. Experimental protein deficiency: Pathological features in the rhesus monkey. Archives of Pathology, 1965, 80, 14-23.

Dickerson, J. W. T., Dobbing, J., and McCance, R. A. The effect of undernutrition on the postnatal development of the brain and cord in pigs. Proceedings of the Royal Society (Biol.), 1967, 166, 396-407.

Dobbing, J. The influence of early nutrition on the development and myelination of the brain. Proceedings of the Royal Society (Biol.), 1964, 159, 503-509.

Dobbing, J. Vulnerable periods in developing brain. In A. N. Davison and J. Dobbing (Eds.), Applied Neurochemistry. Oxford: Blackwell Scientific Publications, 1968, 287-316.

Dobbing, J., and Widdowson, E. M. The effect of undernutrition and subsequent rehabilitation on myelination of rat brain as measured by its composition. Brain, 1965, 88, 357-366.

Eichenwald, H. F., and Fry, P. C. Nutrition and learning. Science, 1969, 163, 644-648.

Elias, M. F., and Samonds, K. W. Exploratory behavior and activity of infant monkeys during nutritional and rearing restriction. American Journal of Clinical Nutrition, 1974, 27, 458-463.

Erwin, J., Brandt, E. M., and Mitchell, G. Attachment formation and separation in heterosexually naive pre-adult rhesus monkeys (Macaca mulatta). Developmental Psychobiology, 1973, 6, 531-538.

Erwin, J., Mobaldi, J., and Mitchell, G. Separation of rhesus monkey juveniles of the same sex. Journal of Abnormal Psychology, 1971, 78, 134-139.

Frankova, S. Nutritional and psychological factors in the development of spontaneous behavior in the rat. In N. S. Scrimshaw and J. E. Gordon (Eds.), Malnutrition, Learning, and Behavior. Cambridge, Massachusetts: Massachusetts Institute of Technology Press, 1968, pp. 312-323.

Frankova, S. Effect of protein-calorie malnutrition on the development of social behavior in rats. Developmental Psychobiology, 1973, 6, 33-43.

Frankova, S., and Barnes, R. H. Influence of malnutrition in early life on exploratory behavior of rats. Journal of Nutrition, 1968a, 96, 477-484.

Frankova, S., and Barnes, R. H. Effect of malnutrition in early life on avoidance conditioning and behavior of adult rats. Journal of Nutrition, 1968b, 96, 485-493.

French, G. M. Spatial discontiguity in monkeys with lesions of the frontal cortex. Science, 1962, 135, 728-729.

French, G. M. Associative problems. In A. M. Schrier, H. F. Harlow, and F. Stollnitz (Eds.), Behavior of Nonhuman Primates, Vol. 1. New York: Academic Press, 1965, pp. 167-209.

Frisch, R. E. Present status of the supposition that malnutrition causes permanent mental retardation. American Journal of Clinical Nutrition, 1970, 23, 189-195.

Geist, C. R. Performance of rats raised on low protein diets in a water maze. Paper presented at Proceedings of Montana Psychological Association, 1973.

Geist, C. R., Zimmermann, R. R., and Strobel, D. A. Effect of protein-calorie malnutrition on food consumption, weight gain, serum proteins, and activity in the developing rhesus monkey (Macaca mulatta). Laboratory Animal Science, 1972, 22, 369-377.

Griffin, G. A., and Harlow, H. F. Effects of three months of total social deprivation on social adjustment and learning in the rhesus monkey. Child Development, 1966, 37, 533-547.

Griffiths, W. J., and Senter, R. J. The effect of protein deficiency on maze performance of domestic Norway rats. Journal of Comparative and Physiological Psychology, 1954, 47, 41-43.

Gross, C. G. Effect of deprivation on delayed response and delayed alternation performance by normal and brain operated monkeys. Journal of Comparative and Physiological Psychology, 1963, 56, 48-51.

Guthrie, H. A. Severe undernutrition in early infancy and behavior in rehabilitated albino rats. Physiology and Behavior, 1968, 3, 619-623.

Hansen, J. D. L., Fressemann, C., Moodie, A. D., and Evans, D. E. What does nutritional growth retardation imply? Pediatrics, 1971, 47, 299-313.

Harlow, H. F. The development of learning in the rhesus monkey. American Scientist, 1959a, 47, 459-479.

Harlow, H. F. Learning set and error factor theory. In S. Koch (Ed.), Psychology: A Study of a Science. New York: McGraw-Hill, 1959b, pp. 492-537.

Harlow, H. F., Harlow, M. K., Schiltz, K. A., Mohr, D. J. The effect of early adverse and enriched environments on the learning ability of rhesus monkeys. In L. E. Jarrard (Ed.), Cognitive Processes of Nonhuman Primates. New York: Academic Press, 1971, pp. 121-148.

Harlow, H. F., and Hicks, L. H. Discrimination learning theory: Uniprocess vs. duoprocess. Psychological Review, 1957, 64, 104-109.

Harlow, H. F., Schiltz, K. A., and Harlow, M. K. Effects of social isolation on the learning performance of rhesus monkeys. Proceedings of the Second International Congress of Primatology, 1969, 1, 178-185.

Hillman, N. M., and Riopelle, A. J. Acceptance and palatability of foods by protein-deprived monkeys. Perceptual and Motor Skills, 1971, 33, 918.

Jelliffe, D. B., and Welbourn, H. F. Clinical signs of mild-moderate protein-calorie malnutrition of early childhood. In G. Blix (Ed.), Mild-Moderate Forms of Protein-Calorie Malnutrition. Uppsala, Sweden: Almqvist and Wiksells, 1963, pp. 12-29.

Kerr, G. R., and Waisman, H. A. A primate model for the quantitative study of malnutrition. In N. S. Scrimshaw and J. E. Gordon (Eds.), Malnutrition, Learning, and Behavior. Cambridge, Massachusetts: Massachusetts Institute of Technology Press, 1968, pp. 240-249.

King, J. E., and Harlow, H. F. Effect of ratio of trial 1 reward to nonreward on the discrimination learning of macaque monkeys. Journal of Comparative and Physiological Psychology, 1962, 55, 872-875.

Klein, R. E., Gilbert, O., Canosa, C. A., and DeLeon, R. Performance of malnourished children in comparison with adequately nourished children. Paper presented at the Annual Meeting of the American Association for the Advancement of Science, December, 1969.

Klein, R. E., Habicht, J. P., and Yarbrough, C. Some methodological problems in field studies of nutrition and intelligence. In D. J. Kallen (Ed.), Nutrition, Development and Social Behavior. Department of Health, Education, and Welfare Publication No. (NIH) 73-242, 1973, pp. 61-75.

Lat, J., Widdowson, E. M., and McCance, R. A. Some effects of accelerating growth. III. Behavior and nervous activity. Proceedings of the Royal Society (Biol.), 1961, 153, 347-356.

Lester, D. A note on Gellerman series. Psychological Reports, 1966, 18, 426.

Levitsky, D. A., and Barnes, R. H. Effect of early malnutrition on the reaction of adult rats to aversive stimuli. Nature, 1970, 225, 468-469.

Levitsky, D. A., and Barnes, R. H. Nutritional and environmental interactions in the behavioral development of the rat: Long term effects. Science, 1972, 176, 68-71.

Levitsky, D. A., and Barnes, R. H. Malnutrition and animal behavior. In D. J. Kallen (Ed.), Nutrition, Development and Social Behavior. Department of Health, Education, and Welfare Publication No. (NIH) 73-242, 1973, pp. 3-16.

Mason, W. A. The effects of social restriction on the behavior of rhesus monkeys. III. Dominance tests. Journal of Comparative and Physiological Psychology, 1961, 54, 694-699.

Mason, W. A. Early social deprivation in nonhuman primates: Implications for human behavior. In D. C. Glass (Ed.), Environmental Influences. New York: Rockefeller University Press, 1968, pp. 70-101.

McKinney, W. T., Suomi, S. J., and Harlow, H. F. Repetitive peer separations of juvenile age rhesus monkeys. Archives of General Psychiatry, 1972, 27, 200-203.

Miles, R. C. Discrimination-learning sets. In A. M. Schrier, H. F. Harlow, and F. Stollnitz (Eds.), Behavior of Nonhuman Primates. New York: Academic Press, 1965, pp. 51-95.

Mishkin, M. Effects of small frontal lesions on delayed alternation in monkeys. Journal of Neurophysiology, 1957, 20, 615-622.

Monckeberg, F. Effect of early marasmic malnutrition on subsequent physical and physiological development. In N. S. Scrimshaw and J. E. Gordon (Eds.), Malnutrition, Learning, and Behavior. Cambridge, Massachusetts: Massachusetts Institute of Technology Press, 1968, pp. 269-278.

Moss, E. M., and Harlow, H. F. The role of reward in discrimination learning in monkeys. Journal of Comparative and Physiological Psychology, 1947, 40, 333-342.

Ordy, J. M., Samorajski, T., Zimmermann, R. R., and Rady, P. M. Effects of postnatal protein deficiency on weight gain, serum proteins, enzymes, cholesterol, and liver ultrastructure in a subhuman primate (Macaca mulatta). American Journal of Pathology, 1966, 48, 769-791.

Peregoy, P. L., Zimmermann, R. R., and Strobel, D. A. Protein preference in protein-malnourished monkeys. Perceptual and Motor Skills, 1972, 35, 495-503.

Pettus, J. P. Geist, C. R., and Schultz, G. E. Recovery from malnutrition: Food preference and neophobia. Perceptual and Motor Skills, 1974, 38, 767-773.

Pinto-Hamuy, T., and Linck, P. Effect of frontal lesions on performance of sequential tasks by monkeys. Experimental Neurology, 1965, 12, 96-107.

Platt, B. S. Experimental protein-calorie deficiency. In R. A. McCance and E. M. Widdowson (Eds.), Calorie Deficiencies and Protein Deficiencies. Boston: Little, Brown and Company, 1968, pp. 237-248.

Pollitt, E. Behavioral correlates of severe malnutrition in man. In W. M. Moore, M. M. Silverberg, and M. S. Read (Eds.), Nutrition, Growth, and Development of North American Indian Children. Department of Health, Education, and Welfare Publication No. (NIH) 72-26, Washington, D.C., 1972, pp. 151-166.

Pribram, K. H., Mishkin, M., Rosvold, H. E., and Kaplan, S. J. Effects on delayed response performance of lesions of dorsolateral and ventromedial frontal cortex of baboons. Journal of Comparative and Physiological Psychology, 1952, 45, 565-575.

Pribram, K. H., and Tubbs, W. E. Short-term memory, parsing, and the primate frontal cortex. Science, 1967, 156, 1765-1767.

Provence, S., and Lipton, R. C. Infants in Institutions. New York: International Universities Press, 1962.

Ramalingaswami, V., and Deo, M. G. Experimental protein-calorie malnutrition in the rhesus monkey. In R. A. McCance and E. M. Widdowson (Eds.), Calorie Deficiencies and Protein Deficiencies. Boston: Little, Brown and Company, 1968, 265-275.

Riopelle, A. J., and Churukian, G. A. The effect of varying the intertrial interval in discrimination learning by normal and brain-operated monkeys. Journal of Comparative and Physiological Psychology, 1958, 51, 119-125.

Rosvold, H. E., and Delgado, J. M. R. The effect on delayed-alternation performance of stimulating or destroying electrical structures within the frontal lobes of the monkey's brain. Journal of Comparative and Physiological Psychology, 1956, 49, 365-372.

Rosvold, H. E., Mishkin, M., and Szwarcbart, M. K. Effects of subcortical lesions in monkeys on visual-discrimination and single-alternation performance. Journal of Comparative and Physiological Psychology, 1958, 51, 437-444.

Rowland, G. L. The effects of total social isolation upon learning and behavior of rhesus monkeys. Dissertation Abstracts, 1964a, 1364-1365.

Rowland, G. L. The effects of total social isolation upon the behavior of rhesus monkeys. Unpublished doctoral dissertation, University of Wisconsin, 1964b.

Rumbaugh, D. M., and Pournelle, M. E. Discrimination-reversal skills of primates: The reversal/acquisition ratio as a function of phyletic standing. Psychonomic Science, 1966, 4, 45-46.

Skeels, H. M. Adult status of children with contrasting early life experiences: A follow-up study. Monographs of the Society for Research in Child Development, 1966, 31, 1-65.

Spitz, R. A. The First Year of Life. New York: International University Press, 1965.

Stamm, J. S. Retardation and facilitation in learning by stimulation of frontal cortex in monkeys. In J. M. Warren and K. Akert (Eds.), The Frontal Granular Cortex and Behavior. New York: McGraw-Hill, 1964, pp. 102-125.

Stoch, M. B., and Smythe, P. M. Does undernutrition during infancy inhibit brain growth and subsequent intellectual development? Archives of Disease in Childhood, 1963, 38, 546-552.

Stoch, M. B., and Smythe, P. M. The effect of undernutrition during infancy on subsequent brain growth and intellectual development. South African Medical Journal, 1967, 41, 1027-1030.

Stoch, M. B., and Smythe, P. M. Undernutrition during infancy, and subsequent brain growth and intellectual development. In N. S. Scrimshaw and J. E. Gordon (Eds.), Malnutrition, Learning and Behavior. Cambridge, Massachusetts: Massachusetts Institute of Technology Press, 1968, pp. 278-288.

Stoffer, G. R., and Zimmermann, R. R. Air-blast avoidance learning sets in protein-malnourished monkesy. Behavioral Biology, 1973, 9, 695-705.

Strobel, D. A. Stimulus change and attentional variables as factors in the behavioral deficiency of malnourished developing monkeys (Macaca mulatta). Doctoral Dissertation, University of Montana, 1972.

Strobel, D. A., Geist, C. R., Zimmermann, R. R., and Lindvig, E. K. Cue-locus--A factor in the behavioral deficiency of the developing protein-malnourished monkey (Macaca mulatta). Behavioral Biology, 1974, 10, 473-484.

Strobel, D. A., and Zimmermann, R. R. Manipulatory responsiveness in protein malnourished monkeys. Psychonomic Science, 1971, 24, 19-20.

Strobel, D. A., and Zimmermann, R. R. Responsiveness of protein deficient monkeys to manipulative stimuli. Developmental Psychobiology, 1972, 5, 291-296.

Sutherland, N. S., and Mackintosh, N. J. Mechanisms of Animal Discrimination Learning. New York: Academic Press, 1971.

Thompson, R. F. Foundations of Physiological Psychology. New York: Harper and Row, 1967.

Wells, A. M., Geist, C. R., and Zimmermann, R. R. Influence of environmental and nutritional factors on problem solving in the rat. Perceptual and Motor Skills, 1972, 35, 235-244.

Widdowson, E. M. The place of experimental animals in the study of human nutrition. In R. A. McCance and E. M. Widdowson (Eds.), Calorie Deficiencies and Protein Deficiencies. Boston: Little, Brown and Company, 1968, 225-234.

Willott, J. F., and McDaniel, J. Changes in the behavior of laboratory-reared rhesus monkeys following the threat of separation. Primates, 1974, 15, 321-326.

Winick, M. Neurological correlates of malnutrition in animals and man. In W. M. Moore, M. M. Silverberg, and M. S. Read (Eds.), Nutrition, Growth and Development of North American Indian Children. Department of Health, Education, and Welfare Publication No. (NIH) 72-26, Washington, D.C., 1972, pp. 139-150.

Winick, M., and Noble, A. Cellular response in rats during malnutrition at various ages. Journal of Nutrition, 1966, 89, 300-306.

Winick, M., and Rosso, P. The effect of severe early malnutrition on cellular growth of human brain. Pediatric Research, 1969, 3, 180-184.

Wise, L. A., and Zimmermann, R. R. Shock thresholds of low- and high-protein-reared rhesus monkeys. Perceptual and Motor Skills, 1973a, 36, 674.

Wise, L. A., and Zimmermann, R. R. The effects of protein deprivation on dominance measured by shock avoidance competition and food competition. Behavioral Biology, 1973b, 9, 317-329.

Wise, L. A., Zimmermann, R. R., and Strobel, D. A. Dominance measurements of low and high protein reared rhesus macaques. Behavioral Biology, 1973, 9, 77-84.

World Health Organization Technical Report Series, Joint FAO/WHO Expert Committee on Nutrition, 1962, No. 245.

World Health Organization Technical Report Series, Joint FAO/WHO Expert Committee on Nutrition, 1967, No. 377.

World Health Organization Technical Report Series, Joint FAO/WHO Expert Committee on Nutrition, 1971, No. 477.

World Health Organization Technical Report Series, Joint FAO/WHO Expert Committee on Nutrition, 1972, No. 485.

Yarrow, L. J. Separation from parents during early childhood. In M. L. Hoffman and L. W. Hoffman (Eds.), Review of Child Development Research, Vol. 1. New York: Russell Sage Foundation, 1964, pp. 89-136.

Yaktin, U. S., and McLaren, D. S. The behavioural development of infants recovering from severe malnutrition. Journal of Mental Deficiency Research, 1970, 14, 25-32.

Zimmermann, R. R. Early weaning and weight gain in infant rhesus monkeys. Laboratory Animal Care, 1969a, 19, 644-647.

Zimmermann, R. R. Effects of age, experience, and malnourishment on object retention in learning set. *Perceptual and Motor Skills*, 1969b, *28*, 867-876.

Zimmermann, R. R. Reversal learning in the developing malnourished rhesus monkey. *Behavioral Biology*, 1973, *8*, 381-390.

Zimmermann, R. R., Geist, C. R., and Strobel, D. A. Behavioral deficiencies in protein deprived monkeys. Kittay Foundation Conference, 1973.

Zimmermann, R. R., Geist, C. R., Strobel, D. A., and Cleveland, T. J. Attention deficiencies in malnourished monkeys. *Symposia of the Swedish Nutrition Foundation*, 1973, *12*, 115-126.

Zimmermann, R. R., Steere, P. L., Strobel, D. A., and Hom, H. L. Abnormal social development of protein malnourished rhesus monkeys. *Journal of Abnormal Psychology*, 1972, *80*, 125-131.

Zimmermann, R. R., and Strobel, D. A. Effects of protein malnutrition on visual curiosity, manipulation, and social behavior in the infant rhesus monkey. *Proceedings of the 77th Annual Convention of the American Psychological Association*, 1969, pp. 241-242.

Zimmermann, R. R., Strobel, D. A., and Maguire, D. Neophobic reactions in protein malnourished infant monkeys. *Proceedings of the 78th Annual Convention of the American Psychological Association*, 1970, pp. 197-198.

Zimmermann, R. R., and Torrey, C. C. Ontogeny of learning. In A. M. Schrier, H. F. Harlow, and F. Stollnitz (Eds.), *Behavior of Nonhuman Primates*, Vol. 2. New York: Academic Press, 1965, pp. 405-447.

Zimmermann, R. R., and Wells, A. M. Performance of malnourished rats on the Hebb-Williams closed field maze learning task. *Perceptual and Motor Skills*, 1971, *33*, 1043-1050.

Zimmermann, R. R., and Zimmermann, S. J. Responses of protein malnourished rats to novel objects. *Perceptual and Motor Skills*, 1972, *35*, 319-321.

APPENDIX A

Table 1

Summary of the Analysis of Variance:
Environment x Diet x Block x Delay
(HP-SOC, LP-SOC, HP-ISO, LP-ISO)
Percent Correct

Source[1]	SS	df	MS	F
E	18,655.9	1	18,655.9	18.9**
D	3,259.1	1	3,259.1	3.3
ED	26.3	1	26.3	0.0
S(ED)	11,822.7	12	985.2	
B	33,775.6	6	5,629.3	46.1***
EB	2,708.8	6	451.5	3.7**
DB	4,616.0	6	769.3	6.3***
EDB	361.9	6	60.3	0.5
S(ED)B	8,796.0	72	122.2	
I	782.9	5	156.6	2.4*
EI	350.4	5	70.1	1.1
DI	1,081.7	5	216.3	3.4**
EDI	206.0	5	41.2	0.6
S(ED)I	3,868.6	60	64.5	
BI	937.1	30	31.2	0.6
EBI	1,185.3	30	39.5	0.8
DBI	1,843.2	30	61.4	1.2
EDBI	1,448.5	30	48.3	1.0
S(ED)BI	18,302.9	360	50.8	

*p less than .05
**p less than .01
***p less than .001

[1] E=environment
D=diet

[1] B=5 day block trials
I=delay interval

Table 2

Summary of the Analysis of Variance: Diet x Block x Delay (HP-SOC versus LP-SOC) Percent Correct

Source[1]	SS	df	MS	F
D	6,536.8	1	6,536.8	8.4*
S(D)	4,673.2	6	778.9	
B	19,450.4	8	2,431.3	24.2***
DB	6,842.2	8	855.3	8.5***
S(D)B	4,817.8	48	100.4	
I	1,086.9	5	217.4	2.2
DI	177.4	5	35.5	0.4
S(D)I	2,959.7	30	98.7	
BI	2,026.3	40	50.6	0.9
DBI	2,066.7	40	51.7	0.9
S(D)BI	13,485.2	240	56.2	

*p less than .05
**p less than .01
***p less than .001

[1] D=diet
B=5 day block trials
I=delay interval

Table 3

Summary of the Analysis of Variance:
Diet x Block x Delay (HP-ISO versus LP-ISO)
Percent Correct

Source[1]	SS	df	MS	F
D	1,350.1	1	1,350.1	0.9
S(D)	8,880.5	6	1,480.1	
B	26,694.3	6	4,449.1	29.7***
DB	1,535.5	6	225.9	1.7
S(D)B	5,399.5	36	150.0	
I	697.9	5	139.6	2.8*
DI	824.8	5	165.0	3.3*
S(D)I	1,499.1	30	50.0	
BI	932.3	30	31.1	0.8
DBI	1,875.1	30	62.5	1.5
S(D)BI	7,414.8	180	41.2	

*p less than .05
**p less than .01
***p less than .001

[1] D-diet
B=5 day block trials
I=delay interval

Table 4

Summary of the Analysis of Variance:
Environment x Diet x Block x Delay
(HP-SOC, LP-SOC, HP-ISO, LP-ISO)
Percent Correct - First Responses

Source[1]	SS	df	MS	F
E	16,284.0	1	16,284.0	14.5**
D	4,260.2	1	4,260.2	3.8
ED	1,020.1	1	1,020.2	0.9
S(ED)	13,444.7	12	1,120.4	
B	29,465.5	6	4,910.9	26.4***
EB	5,880.1	6	980.0	5.3***
DB	3,819.3	6	636.5	3.4**
EDB	813.9	6	135.6	0.7
S(ED)B	13,398.7	72	186.1	
I	198.4	5	39.7	0.3
EI	504.1	5	100.8	0.8
DI	1,299.4	5	259.9	2.0
EDI	404.5	5	80.9	0.6
S(ED)I	7,761.6	60	129.4	
BI	2,347.1	30	78.2	0.9
EBI	2,666.7	30	88.9	1.0
DBI	3,148.1	30	104.9	1.2
EDBI	2,700.3	30	90.0	1.0
S(ED)BI	31,399.1	360	87.2	

*p less than .05
**p less than .01
***p less than .001

[1] E=environment
D=diet

[1] B=5 day block trials
I=delay interval

Table 5

Summary of the Analysis of Variance: Diet x Block x Delay (HP-SOC versus LP-SOC) Percent Correct - First Responses

Source[1]	SS	df	MS	F
D	4,693.9	1	4,693.9	10.3*
S(D)	2,732.9	6	455.5	
B	13,042.5	8	1,630.3	9.4***
DB	9,714.1	8	1,214.3	7.0***
S(D)B	8,333.8	48	173.6	
I	828.7	5	165.7	1.0
DI	574.1	5	114.8	0.7
S(D)I	5,097.3	30	169.9	
BI	4,825.9	40	120.6	1.4
DBI	2,909.9	40	72.7	0.8
S(D)BI	21,348.0	240	89.0	

*p less than .05
**p less than .01
***p less than .001

[1] D=diet
B=5 day block trials
I=delay interval

Table 6

Summary of the Analysis of Variance:
Diet x Block x Delay (HP-ISO versus HP-ISO)
Percent Correct - First Response

Source[1]	SS	df	MS	F
D	4,725.0	1	4,725.0	2.4
S(D)	11,895.9	6	1,982.6	
B	29,979.3	6	4,996.6	20.6***
DB	1,074.0	6	179.0	0.7
S(D)B	8,750.1	36	243.1	
I	357.4	5	71.5	0.6
DI	839.3	5	167.8	1.5
S(D)I	3,328.7	30	111.0	
BI	1,507.0	30	50.2	0.6
DBI	3,751.7	30	125.1	1.5
S(D)BI	15,125.3	180	84.0	

*p less than .05
**p less than .01
***p less than .001

[1] D=diet
B=5 day block trials
I=delay interval

Table 7

Perseverative Responses
(HP-SOC, LP-SOC, HP-ISO, LP-ISO)

	df	Chi Square
10 Second Delay	12	57.95***
20 Second Delay	12	57.03***
30 Second Delay	12	38.08***
40 Second Delay	12	27.50**
50 Second Delay	12	41.48***
60 Second Delay	12	49.20***

*p less than .05
**p less than .01
***p less than .001

Table 8

Perseverative Responses: Social-Isolate

	df	Chi Square
10 Second Delay	4	32.28***
20 Second Delay	4	38.61***
30 Second Delay	4	28.42***
40 Second Delay	4	15.92**
50 Second Delay	4	31.17***
60 Second Delay	4	41.67***

*p less than .05
**p less than .01
***p less than .001

Table 9

Perseverative Responses: High Protein-Low Protein

	df	Chi Square
10 Second Delay	4	3.95
20 Second Delay	4	6.83
30 Second Delay	4	1.80
40 Second Delay	4	2.17
50 Second Delay	4	2.48
60 Second Delay	4	0.70

Table 10

Summary of the Analysis of Variance:
Environment x Diet x Block x Delay
(HP-SOC, LP-SOC, HP-ISO, LP-ISO)
Response Latency

Source[1]	SS	df	MS	F
E	95.8	1	95.8	1.9
D	87.6	1	87.6	1.7
ED	10.0	1	10.0	0.2
S(ED)	604.6	12	50.4	
B	16.5	6	2.7	0.9
EB	80.1	6	13.3	4.4**
DB	37.1	6	6.2	2.0
EDB	20.8	6	3.5	1.1
S(ED)B	220.5	72	3.1	
I	25.5	5	5.1	8.4***
EI	8.1	5	1.6	2.7*
DI	11.7	5	2.3	3.8**
EDI	5.1	5	1.0	1.7
S(ED)I	36.5	60	0.6	
BI	19.5	30	0.6	0.9
EBI	21.6	30	0.7	1.0
DBI	16.7	30	0.6	0.8
EDBI	17.7	30	0.6	0.8
S(ED)BI	248.6	360	0.7	

*p less than .05
**p less than .01
***p less than .001

[1] E=environment
D=diet

[1] B=5 day block trials
I=delay intervals

Table 11

Summary of the Analysis of Variance:
Environment x Diet x Block x Delay
(HP-SOC, LP-SOC, HP-ISO, LP-ISO)
Response Latency (Reciprocal Transformation)

Source[1]	SS	df	MS	F
E	0.0205	1	0.0205	0.0
D	4.2130	1	4.2130	1.9
ED	1.2733	1	1.2733	0.6
S(ED)	26.5974	12	2.2164	
B	0.1648	6	0.0274	0.7
EB	1.0208	6	0.1701	4.1**
DB	0.4226	6	0.0704	1.7
EDB	0.1563	6	0.0261	0.6
S(ED)B	2.9754	72	0.0413	
I	0.2233	5	0.0446	9.6***
EI	0.0626	5	0.0125	2.7*
DI	0.0872	5	0.0174	3.7**
EDI	0.0149	5	0.0030	0.6
S(ED)I	0.2793	60	0.0046	
BI	0.1317	30	0.0044	1.2
EBI	0.0848	30	0.0028	0.8
DBI	0.0549	30	0.0018	0.5
EDBI	0.1352	30	0.0045	1.2
S(ED)BI	1.2889	360	0.0036	

*p less than .05
**p less than .01
***p less than .001

[1] E=environment
D=diet

[1] B=5 day block trials
I=delay interval

Table 12

Summary of the Analysis of Variance:
Diet x Block x Delay (HP-SOC versus LP-SOC)
Response Latency

Source[1]	SS	df	MS	F
D	88.9	1	88.9	0.9
S(D)	607.7	6	101.3	
B	71.2	8	8.9	1.8
DB	53.0	8	6.6	1.3
S(D)B	243.4	48	5.1	
I	38.7	5	7.7	5.8***
DI	9.6	5	1.9	1.4
S(D)I	39.8	30	1.3	
BI	41.2	40	1.0	0.9
DBI	44.5	40	1.1	1.0
S(D)BI	270.1	240	1.1	

*p less than .05
**p less than .01
***p less than .001

[1] D=diet
B=5 day block trials
I=delay interval

Table 13

Summary of the Analysis of Variance:
Diet x Block x Delay (HP-ISO versus LP-ISO)
Response Latency

Source[1]	SS	df	MS	F
D	19.2	1	19.2	1.0
S(D)	114.1	6	19.0	
B	35.4	6	5.9	8.3***
DB	9.2	6	1.5	2.2
S(D)B	25.4	36	0.7	
I	4.0	5	0.8	4.3**
DI	0.9	5	0.2	1.0
S(D)I	5.6	30	0.2	
BI	3.8	30	0.1	1.1
DBI	3.3	30	0.1	0.9
S(D)BI	21.4	180	0.1	

*p less than .05
**p less than .01
***p less than .001

[1] D= diet
B=5 day block trials
I=delay interval

Table 14

Summary of the Analysis of Variance:
Environment x Diet x Block
(HP-SOC, LP-SOC, HP-ISO, LP-ISO)
Mean Response Latency

Source[1]	SS	df	MS	F
E	15.8	1	15.8	1.7
D	14.3	1	14.3	1.5
ED	1.5	1	1.5	0.2
S(ED)	111.2	12	9.3	
B	3.0	6	0.5	1.0
EB	15.0	6	2.5	5.0***
DB	5.2	6	0.9	1.7
EDB	3.5	6	0.6	1.2
S(ED)B	36.0	72	0.5	

*p less than .05
**p less than .01
***p less than .001

[1] E-environment
D=diet
B=5 day block trials

APPENDIX B

Figure 1. Probability of an error

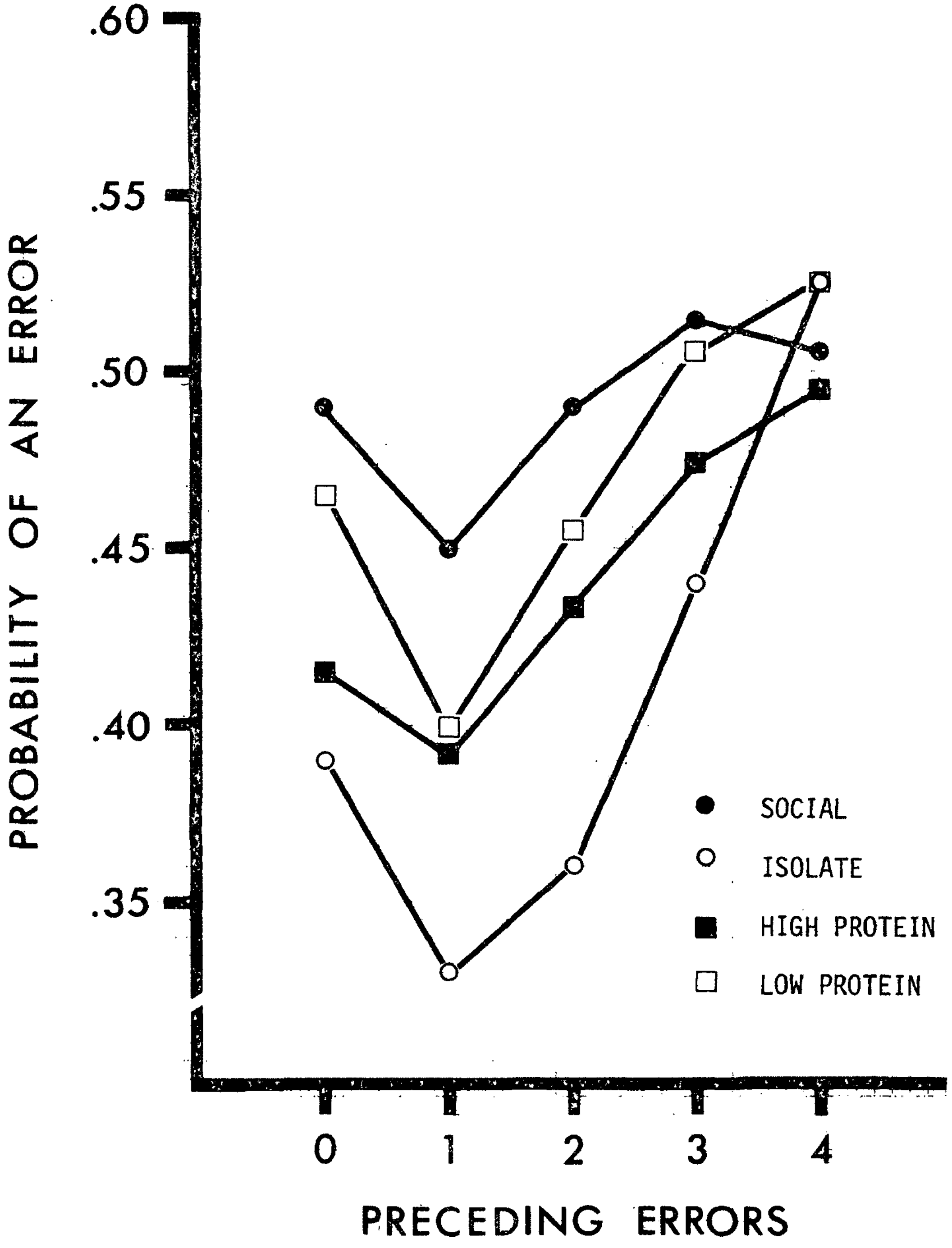


Table 1

Probability of an Error

	After a Correct Response	After 1 Error	After 2 Errors	After 3 Errors	After 4 Errors
LP-ISO					
19	.48	.35	.42	.49	.50
20	.42	.22	.34	.47	.47
21	.42	.35	.36	.48	.59
22	.38	.25	.23	.30	.29
HP-ISO					
31	.37	.31	.36	.51	.68
32	.37	.32	.35	.44	.58
34	.44	.44	.44	.42	.41
7217	.24	.36	.42	.32	.67
LP-SOC					
36	.50	.53	.54	.52	.61
7245	.53	.43	.47	.53	.42
7329	.46	.56	.58	.54	.55
7333	.48	.45	.46	.50	.51
HP-SOC					
26	.48	.47	.48	.60	.50
27	.52	.48	.47	.54	.45
29	.44	.32	.41	.43	.62
30	.46	.37	.49	.37	.34