5-1-1978

Prenatal Zinc Deficiency and Stress in the Rat

Michael Charles Rowe

PRENATAL ZINC DEFICIENCY AND

STRESS IN THE RAT

by
Michael Charles Rowe

Bachelor of Arts, University of Minnesota, Duluth, 1972

Master of Arts, University of North Dakota, 1974

A Dissertation

Submitted to the Graduate Faculty

of the

University of North Dakota

in partial fulfillment of the requirements

for the degree of

Doctor of Philosophy

Grand Forks, North Dakota

May
1978

Prenatal Zinc Deficiency and

Stress in the Rat

Michael Charles Rowe, Ph.D.

The University of North Dakota, 1978

Faculty Advisor: Professor Edward Halas

The studies presented below concern the effects of stress upon prenatal zinc deficient adult rats. One group of dams was fed a zinc deficient diet from the morning of day 14 to the morning of day 19 of the pregnancy. A second group was given the same amount of food as was eaten by their zinc deficient counterpart (pair-fed) plus water containing 50 ppm zinc, thus they were not zinc deficient but did experience starvation. A third group was fed the diet ad libitum and given the zinc-supplemented water.

Starting at 150 days of age three offspring from each of the above nutritional groups, either all male or female, were placed under stress. The stress consisted of 48 hours of food deprivation followed by 72 hours in an executive animal apparatus. This method was developed by Rice (1963) and modified to its present form by Weiss (1971). Measures of stress include: 1) weight loss during the 48 hours of food deprivation and during the 72 hours in the executive animal apparatus; 2) water consumption, wheel turns and shocks received while in the executive animal apparatus; and 3) gastric pathologies (total length and number were measured from scaled photographs of the fixed stomachs).

Zinc deficient males had less lower stomach pathology than normal rats. It was suggested that this group was culled at birth in a way that left the group less prone to lower stomach pathology. Abnormally high mortality rates observed in zinc deficient litters could make this possible.

Among executive and yoke stress groups, normally nourished _S_s had fewer upper stomach pathology than either pair fed or zinc deficient _S_s.

Wheel turning and shock rates of zinc deficient rats were dramatically higher than those of normal rats. This effect was most pronounced in the females and during the first two trial blocks. The upper stomach pathology, wheel turning and shock rate findings are yet further demonstrations of performance and physical impairments resulting from malnutrition, in particular zinc deficiency.

Raw data is not appended because of the physical bulk that would be required. Data in any reasonable format will be furnished upon request by the author.

This Dissertation submitted by Michael Charles Rowe in partial fulfillment of the requirements for the Degree of Doctor of Philosophy from the University of North Dakota is hereby approved by the Faculty Advisory Committee under whom the work has been done.

(Chairman)

Dean of the Graduate School

Permission

Title PRENATAL ZINC DEFICIENCY AND STRESS IN THE RAT

Department PSYCHOLOGY

Degree DOCTOR OF PHILOSOPHY

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Date 4-24-1978

ACKNOWLEDGEMENTS

I would like to acknowledge the cooperation and help of the members of my dissertation committee, Dr. James Antes, Dr. William Cornatzer, Dr. Ralph Kolstoe, and Dr. Harold Sandstead. I am especially grateful to Dr. Edward Halas for procuring financial support for me at the U.S.D.A. Human Nutrition Laboratory and for chairing my committee, which in this case was no easy job. I am also indebted to Ms. Ruth Smith for her logistical support.

DEDICATION

I would like to dedicate this work to the members of the Druid Tree Farm, especially its newest member, my wife Susie.

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ABSTRACT

The studies presented below concern the effects of stress upon prenatal zinc deficient adult rats. One group of dams was fed a zinc deficient diet from the morning of day 14 to the morning of day 19 of the pregnancy. A second group was given the same amount of food as was eaten by their zinc deficient counterpart (pair-fed) plus water containing 50 ppm zinc, thus they were not zinc deficient but did experience starvation. A third group was fed the diet *ad libitum* and given the zinc-supplemented water.

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Zinc deficient males had less lower stomach pathology than normal rats. It was suggested that this group was culled at birth in a way

that left the group less prone to lower stomach pathology. Abnormally high mortality rates observed in zinc deficient litters could make this possible.

Among executive and yoke stress groups, normally nourished Ss had fewer upper stomach pathology than either pair fed or zinc deficient Ss.

Wheel turning and shock rates of zinc deficient rats were dramatically higher than those of normal rats. This effect was most pronounced in the females and during the first two trial blocks. The upper stomach pathology, wheel turning and shock rate findings are yet further demonstrations of performance and physical impairments resulting from malnutrition, in particular zinc deficiency.

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CHAPTER I

INTRODUCTION AND REVIEW OF THE LITERATURE

It has been estimated that 5% of the adult population of the United States will be afflicted with a peptic ulcer at one time during their lifetime (Allen, 1959). Certain subpopulations have even higher incidence of peptic ulcers; doctors and business executives 10%, foremen 9%, transport drivers 8%, construction workers 7%, clerks 6% (data came from Great Britain where 3% of the adult population were estimated to become afflicted with peptic ulcers at some time during their lifetime, Tonkin, 1957).

Part One: Physiological Mechanisms of Ulceration

Peptic ulcers result from hydrochloric acid penetrating the entire thickness of the mucous membrane that lines the inner surface of the stomach. This mucous membrane under normal conditions is continually renewing itself; when the membrane is penetrated the muscular wall of the stomach is made vulnerable to digestion by the gastric juices. It is usually the case that hydrochloric acid is secreted only prior to or during eating to help in the digestion process. During digestion, the acid is diluted from a pH of about 1.0 (Harper, 1973) by the food. In the case of the peptic ulcer something has gone astray and the

gastric juices digest the protective nucous membrane. Peptic ulcer etiologies have been subdivided into two areas. Extrinsic causes result from ingestion of materials that mechanically, thermally or chemically harm the mucous membrane, or failure of a diet to supply the essential nutrients for the mucous membrane. Intrinsic causes arise from bodily functions that interfere with the normal mucous membrane regeneration or cause excess acid to be secreted.

Mechanical irritants consist of hard or fibrous foods that work similar to sandpaper, physically removing contacted areas of the mucous membrane. Ice-cold or boiling-hot foods serve as thermal irritants by providing an environment that is minimally suitable for the regeneration of the mucous membrane. Materials that, like the gastric juices, are capable of digesting the protective mucous membrane are classified as chemical irritants (Tonkin, 1957). It has also been shown that vitamins in general, but especially vitamin C are essential for mucous membrane regeneration (Dann, 1942). Vitamin C serves as an intercellular binder which increases tensile strength of healing wounds (Bourne, 1944).

Intrinsic etiologies may be subdivided into three nonexclusive areas; metabolic poisons, inadequate blood supply, and increased amounts of acid. Metabolic poisons create a poor environment for regeneration of the mucous membrane. Blood carries nutrients to the mucous membrane; when the blood supply to the mucous membrane is interrupted cell starvation results. Increased acid secretion has been shown to result from histamine's stimulation of the cells that secrete the stomach acid (Allen, 1959) or by central nervous system involvement via the vagus nerve (Harper, 1973).

Of interest in physiological psychology would be brain lesion studies. It has been demonstrated that lesions of the crura cerebelli, corpora quadrigemina, pons, floor of the fourth ventricle, ala cinirea (Pomorski, 1892) and of the cerebral pendunicles, optic thalamus and ammons horns (Preuschen, 1894; Ivy, 1920) of rabbits caused gastric hemorrhaging and ulcers. It thus seems evident, as in intrinsic and extrinsic etiologies, that there does not seem to be one specific brain area but a wide variety of areas that if damaged or disrupted will result in gastric pathologies.

Ulcers are not commonly observed in healthy animals (Table 1) with the exception of sea lions. The sea lions in the Schroeder and Wegeforth (1935) study were observed to ingest small sharp stones, whether by accident or on purpose it was not hypothesized. When a group of sea lions was kept off their occasional stone snack, no new ulcers developed and the old ulcers healed.

In summary, the development of gastric ulcers seems to be a sensitive indicator of a wide variety of physiological stressers; because of the low normal incidence of ulcers in laboratory animals, deviation from this incidence rate should indicate the presence of stress.

Part Two: Behavioral Mechanisms of Ulceration

A wide variety of behavioral methods have been used to foment ulcers. These methods will be briefly summarized before an analysis of the variables studied and correlated with gastric ulceration is begun.

TABLE 1[1]

Normal Incidence of Ulcers in Laboratory Animals

Animal	N	Incidence of Ulcers	Observers	
Dog	271	0	Turck	(1906)
Dog and Cat	200	0	Mann	(1916)
Dog (healthy)	1000	0	Ivy	(1919)
Dog	100	0	Battaglia	(1927)
Dog	1000	0.8%	Overgaard	(1934)
Dog	150	0	Keller	(1936)
Dog	55	0	Volini	(1938)
Rat	200	8%	Singer	(1913)
Rat	1189	0	McCarrison	(1913)
Rat	75	0	Howes	(1936)
Guinea Pig	1000	0	Smith	(1933)
Monkey	50	0	Hoff	(1935)
Monkey	300	0	Watts	(1935)
Rabbit	100	0	Beazell	(1936)
Swine (ill)	754	2.4%	Kernkamp	(1945)
Swine	1000	0	Ivy	(1926-1930)
Sea Lions	46	100%	Schroeder & Wegeforth	(1935)

[1]from Ivy, Grossman and Bachrach, 1950

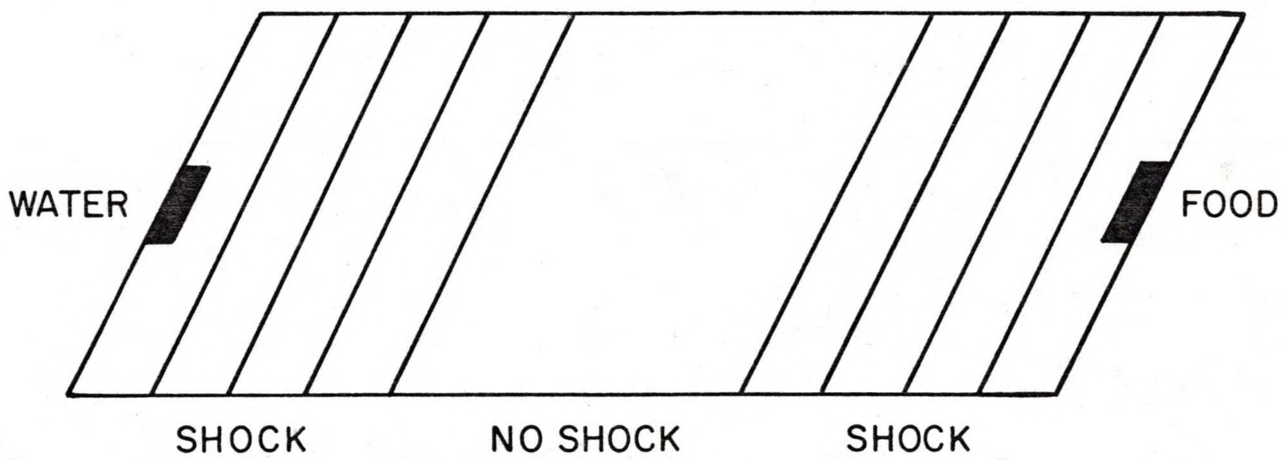
Methods and Apparati

Stern, Winokur, Eisenstein, Taylor and Sly (1960) and Ader (1965) established that immobilization for eighteen hours in a wire mesh cocoon was sufficient to produce ulcers in rats. Immobilization of rats for twenty-four hours in a body cast (Mikhail, 1969) or in a wire mesh cocoon (Lovibond, 1969), in the presence of a CS previously conditioned to shock has been shown to produce gastric ulcers. Groups that received the CS had a higher incidence of ulcers than did control groups that were merely immobilized. Weinstein and Driscoll (1972) and Ader and Plaut (1968) immobilized food deprived rats in wire mesh cocoons, while Weiss (1968 and 1970) and Price (1972) employed tail shocks on immobilized rats to produce ulcers in rats. Although not all of the above authors divulged their method of getting animals into their immobilization apparati, the ones that did stated that a light anaesthesia was helpful.

An apparatus that has come to be known as the Sawrey Weisz apparatus consists of a rectangular box with the floor divided into thirds (Figure 1). The Ss are generally permitted to eat and drink, shock free, for one hour every 24 to 48 hours. This is a long term procedure, lasting at least two weeks with some experimenters reporting experiments of thirty days in length. The floor shock usually varied from 1.0 to 2.5 mA. A control group for food and water deprivation (consisting of Ss that received food and water in amounts and in temporal yoking with experimental group Ss, but did not receive shocks or have the food-shock

Fig. 1. The Sawrey-Weisz Apparatus

SAWREY – WEISZ APPARATUS



conflict) had a significantly lower proportion of Ss with ulcers (Sawrey & Weisz, 1956). The difference between groups was significant at the $p<0.03$ level; indicating that it was not the temporary food and water deprivation but the approach-avoidance conflict that was critical for ulcer formation. Combinations of hunger, thirst, shock and the conflict between shock with food and water acquisition were systematically studied for their relative potency as ulceratives (Sawrey, Conger & Turell, 1956). It was observed that the conflict was the strongest ulcerative factor. Three Chi Squares were computed using a 2 X 2 design (presences vs. absence of ulcers in a S by presence or absence of the experimental treatments Hunger, Shock and Thirst) and it was found that the Hunger vs. no Hunger Chi Square was significant at the $p<0.001$ level; with the food deprived group having a higher proportion of Ss with ulcers; that the Shock vs. no Shock Chi Square was significant at the $p<0.05$ level, with the shocked group having a higher proportion of Ss with ulcers; and that the Thirst vs. no Thirst Chi Square was not significant at the $p<0.05$ level, although the water deprived group tended to have a higher proportion of Ss with ulcers than the non water deprived group. Because of an incomplete array of experimental condition combinations the same Chi Square could not be used to evaluate the conflict-no conflict dimension (Table 2).

Others using the Sawrey and Weisz apparatus include: Conger, Sawrey and Turrel (1958); Ader, Tatum and Beel (1960); Ader, Beels and Tatum (1960); Sawrey and Long (1962); J. Sawrey and W. Sawrey (1966). This method seems to be successful in producing ulcers, but because of

TABLE 2

Results from the W. Sawrey, Conger and Turrell Experiment

Conditions[1]	% of Ss with Ulcers	# of Ulcers, Total per Group	Weight loss (in grams)
c h s t	76	434	99.4
nc h s t	30	44	79.8
nc h s nt	40	26	75.1
nc h ns t	0	0	54.1
nc nh s t	0	0	42.9
nc h ns nt	20	4	54.6
nc nh ns t	0	0	25.4
nc nh s nt	0	0	10.7
nc nh ns nt	0	0	29.2 (gained)

[1]c=conflict, h=hunger, s=shock, t=thirst, n=not present

its long duration it may be prohibitive for some designs (critical age variables, drug studies in which the effect of the drug may change with repeated administrations, and other variables that could possibly change during an experiment that lasts up to 30 days). An experiment that runs for a long time also introduces a greater chance of equipment failure as was noted by W. Sawrey and Weisz (1956).

The third major group of apparati consists of variations of escape-avoidance or operant bar pressing situations with integrated shock. Pare (1962) used a cage consisting of three parts; on one side of the cage the S could bar press for food, on the other side the S could bar press for water, and in between the two sides the floor was

wired for shock. The lever pressing was programmed to reinforce only when the S alternated presses from the water to the food bar, thus increasing the exposure to the conflict and number of times the S received shock. This procedure was followed for eight hours per day for four weeks and resulted in erosions and blood clots but in no actual ulcers.

Pare (1972) shocked a normal Skinner box bar pressing situation at different times. He found that more ulcers were produced when the S was shocked while eating than while approaching the food, or while the food was being delivered, or while the S was lever pressing. The distribution of the four above experimental conditions were compared with regards to proportions of Ss that received ulcers in a 2 X 4 Chi Square. The distribution was significant at the $p<0.05$ level, indicating that stress, in the form of a shock, during feeding produces ulcers.

Seligman (1968) and Seligman and Meyer (1970) modified a Skinner box food reinforced bar pressing procedure by presenting CSs to one group that signalled an up coming shock, while another group was shock yoked with the first and did not receive a CS prior to the shock. The groups that did receive warnings (predictable shock groups) developed fewer ulcers than the yoked groups. In the first study none of the predictable shock group developed ulcers whereas 75% of the non-signalled group developed ulcers. In the second study, the signalled group averaged only 2.0 ulcers per S compared with 9.25 ulcers per S for the non-signalled group. Lever pressing was suppressed in the non-signalled shock groups; the difference between signalled and non-signalled groups were significant at the $p<0.001$ level in both studies.

Lockard (1963) and Gliner (1972) used two-way shuttle boxes. On one side the S would receive a CS prior to shock, while on the other side the S would receive no warning of the shock. The experimenters found that Ss who had a choice would stay on the signalled shock side, the difference between number of trials started on the signalled side compared with the non-signalled side was significant at the $p<0.001$ level; and that the signalled shock Ss developed fewer ulcers, the difference between signalled vs. non-signalled groups was significant at the $p<0.05$ level, respectively.

Rice (1963) designed a wheel turning escape-avoidance procedure. Moot, Cebulla and Crabtree (1970) used wheel turning escape-avoidance in conjunction with bar pressing for food. Weiss (1971a, b) also used wheel turning escape-avoidance, and Weiss (1971b) minimally punished the coping escape-avoidance response by a shock shorter than the UCS. This increased the ulcer producing ability of the situation by hypothetically giving the S an insolvable and thus highly stressful problem.

Weiss (1968), studying coping, provided one group of Ss with the escape-avoidance behavior of jumping on to an elevated platform, whereas another group could not escape and received a shock yoked to a partner S from the first group. Ulcers were not evaluated, but the group which could escape or avoid lost less weight and defecated less than the yoked group. The differences between groups concerning the above measures were significant at the $p<0.001$ and $p<0.02$ level respectively.

Hamilton and Katske (1973) perceivably advanced experimental ulcerative technology with a slightly modified two-way platform avoidance apparatus. The changes include: removal of the partitions that

normally divide the shuttle box into safe and shocked sides, thus adding ambiguity to the situation; omitting the CS that normally cues the avoidance behavior; running the entire experiment in the dark; and including a VI 60 second shock delivered to both sides of the apparatus for two seconds, thus providing an insolvable shock escape situation. Shocks used were 0.8 mA constant current delivered to floor grids. The escapable shocks were alternated from side to side on a FI 40 second schedule. Experiments on both Sprague-Dawley and hooded male rats produced ulcers in 100% of the Ss (n=16 for both strains) in only one hour. This procedure does not seem to be as effective as claimed from information gathered at the U.S.D.A. Human Nutrition Laboratory in Grand Forks, North Dakota.

Warning vs. No Warning

Several researchers have studied, with slight variation in design, the question of whether a warning prior to a shock will reduce the stress of the shocked situation. W. Sawrey (1961) had two groups of rats; one group received shocks which were preceded by CS's (light), while the other group received shocks and the same CS's but the UCS and CS were not paired. The group that received the signalled shocks had fewer ulcers than the unsignalled shock group. The difference between groups was significant at the $p<0.05$ level.

Pare (1964) ran three groups; one group, a control group, did not receive a shock or a CS; another group received a shock but did not receive a CS prior to it; the third group received both the CS and the shock. The experiment ran for 22 hours per day over a 23 day period.

While this procedure produced only two ulcers in a total of 24 rats, weight loss differences among groups were significant at the $p<0.01$ level, indicating that the tone made the event more stressful. Results contradicting Pare (1964) seem to be the rule rather than the exception. Seligman and Meyer (1970) found that signalling a shock reduced the stressfulness of the situation, as measured by number of ulcers developed. Weiss (1970) using signalled and non-signalled shock on immobilized Ss; Mezinskis, Gliner and Shemberg (1971) and Price (1972) using a blinking light CS with immobilized and free rats respectively; Seligman (1968) using four different CS's (white light, green light, tone above white noise and dimming of white noise), unanimously found a reduced incidence of ulcers in the groups that received signalled shocks compared to the various non-signalled groups. Also in agreement with the above results would be the earlier mentioned studies by Lockard (1963) and Gliner (1972) where, they found that Ss given a choice preferred the signalled avoidance situation to a non-signalled escape situation and that signalled Ss developed fewer ulcers.

In summary, the safety-signal hypothesis mentioned by Seligman and Meyer (1970) seems to adequately explain the above results. This safety-signal hypothesis briefly states that the Ss that received a signal prior to a shock are only under stress during the CS-UCS interval while the other non-signalled Ss are under stress during the entire duration of the experiment.

Shock Levels

Although there are a wide range of interexperiment differences among shock levels--few people have systematically studied the differences resulting from high and low shock intensities. Seligman and Meyer (1970) found that Ss who received a 1.4 mA shock developed more ulcers and erosions than did Ss who received only a 0.6 mA shock. The difference between group means was 4.125 gastric pathologies per S (high shock Ss averaged 5.625 pathologies per S while low shock Ss averaged only 1.5 pathologies per S). Gliner (1972) found that Ss who received a 2.5 mA shock developed significantly more ulcers than a group that received only a 0.5 mA shock; the difference between groups was significant at the $p<0.01$ level. The higher the shock intensity the more stress in a given situation seems to be both empirically and conceptually defensible.

Quantification of Ulcers and Other Physiological Measures

Several methods have been used to quantify ulcers; among them have been, percent of Ss having ulcers vs. percent of Ss not having ulcers, total number of ulcers, total length of ulcers, total area of ulcers and tank ordering stomachs on degree of healthiness. Lovibond (1969) looked at both ulcerated area and total number of ulcers; he found the measures to be directly proportional. Weiss (1970) found average number of ulcers per S to be a stronger discriminator than percent of Ss having ulcers which in turn was a stronger discriminator than total length of ulcers. Weiss (1971a), contradicting his previous finding, found a difference between total length of ulcers and total number

of ulcers, with total length of ulcers giving a greater difference between experimental groups. Pare (1964) photographed stomachs and had judges independently rank three stomach photographs at a time (one from each of the designs three experimental treatments--the three Ss that had been run concurrently and were matched by body weight). The judges did not know from which group each photograph had come and ranked on a three-point scale from healthiest to least healthy stomach. Little else has been done or mentioned concerning intercorrelations between the above mentioned quantifying methods; it seems that all methods are of similar reliability, and their respective validities can only be guessed at.

W. Sawrey and Long (1962) measured weight loss and found it positively correlated to number of ulcers. He then covaried out the variance that was accounted for by weight loss to take a refined look at inter-strain and sex differences. Ader, Tatum and Beels (1960); Weiss (1968, 1970); and Price (1972) have also measured weight loss and found it positively correlated with ulcer production or amount of stress encountered.

Plasma corticosterone levels increase with stress (Weiss, 1970, 1971a, b). The within group variances were quite high (Weiss, 1971a), which would necessitate large sample sizes. Related to the increased plasma corticosterone levels would be increased adrenal cortex weights, as corticosterone is a product of the adrenal cortices. Selye (1946) and Price (1972) have found that adrenal cortex weights, in relation to total body weights, increased with increasing degrees of stress. The analysis of adrenal cortex weights would be very difficult in small

animals because of the size of the adrenals and their inseparability from nearby tissues.

Other physiological measures that have been taken include: defecation (Weiss, 1968), which was significantly higher in the high stress group of his experiment. The difference between groups was significant at the $p<0.02$ level. Heart rate (Lovibond, 1969), which did not differ significantly between groups. Anal body temperature (Weiss, 1970), which was significantly higher under the experiments' high stress condition; the difference between groups was significant at the $p<0.01$ level.

Strain, Sex, Age, Weight and Level of Starvation

Sines (1959) selectively bred Sprague-Dawley rats for a high incidence of stomach ulcers. The first generation of rats was placed in body casts and deprived of food and water for 48 hours. The *Ss* were anaesthetized, their stomachs injected with air, and illuminated from the side. Gastric lesions appeared as dark blue areas. This technique was validated by sacrificing thirteen randomly chosen *Ss*; decisions made by the air injection inspection were correct over 90% of the time according to the dissections. The recovered ulcer susceptible *Ss* were then intrabred and their matured offspring placed in similar immobilization. The second generation's stomach's were surgically inspected as before, and the recovered ulcer susceptible *Ss* were again intrabred. The third generation that resulted was immobilized as before and random *Ss* were sacrificed. The results appear in Table 3.

TABLE 3

Ulcer Susceptible Bred Rats

Generation	Percent of Ss having Ulcers Male	Female	
1	58	68	(would represent normal base rate for Sprague-Dawley rats on this apparatus)
2	79	87	
3	96	87	

Sines, Cleeland and Adkins (1963) later studied the behavior of rats similar to the above described third generation ulcer susceptible rats. They were slower straight alley traversers (with food as a reinforcer), showed no difference in Y maze learning time, and were faster two-way shuttle avoidance learners than the average Sprague-Dawley laboratory rat.

W. Sawrey and Long (1962) studied differences in ulcer susceptibility among four strains of rats and between sex using the Sawrey and Weisz apparatus. After covarying out differences in weight loss among the four strains and between sexes, they found the adjusted means which are given in Table 4. They did not give their unadjusted group means which perhaps would have been even of greater interest.

Weinstein and Driscoll (1972) immobilized wild rats (Rattus norvegicus) using several different combinations of food deprivation and immobilization duration. Generally, it was found that food deprivation of 48 hours or longer in conjunction with 24 hours or longer of

TABLE 4

Adjusted Mean Number of Ulcers in the W. Sawrey and Long Experiment

Sex	Long-Evans	Wister	Sprague-Dawley	Nebraska Hoods
Male	16.69	15.89	16.74	1.68
Female	14.78	- 0.38	11.57	5.48

immobilization produced the largest incidence of ulcers, whereas deprivation for 24 hours or less in conjunction with less than 24 hours of immobilization was insufficient to produce ulcers.

J. Sawrey and W. Sawrey (1966) found that rats 164 days old and over 410 grams in weight had more ulcers than younger, lighter rats using the Sawrey and Weisz apparatus. The 164-day old rats were all over 410 grams and were compared with 84-day old 330-350 gram rats and 124-day old 370-390 gram rats in this confounded age-weight design.

Social Factors

Several social factors have been assessed for possible connection to the ulcerative process. Stern, Winokur, Eisenstein, Taylor and Sly (1960) and Ader (1965) found that albino rats reared in isolation from other rats were less prone to ulcers from immobilization. Stern et al. (1960) found that the difference between group and isolated housed Ss, as measured by percent of Ss developing ulcers, to be significant at the $p<0.001$ level; Ader's study did not find significant differences

between group and individually housed Ss but his trends were in the same direction as Stern et al.

Conger, W. Sawrey and Turrel (1958) and J. Sawrey and W. Sawrey (1966) found that group tested Ss developed fewer ulcers than did individually tested Ss using the Sawrey and Weisz apparatus. Sawrey and Sawrey found differences in the number of ulcers between group and individually tested Ss at the $p<0.001$ level.

Ader, Beels and Tatum (1960) removed the mother from the pups intermittently at different periods for a total of 24 hours for all groups. The matured pups were then tested in the Sawrey and Weisz apparatus and the pups whose mother was periodically removed during the first eleven days of life had a higher incidence of ulcers than a group whose mother was removed periodically during days 11 through 21 or a control group that was not disturbed. The difference among groups, concerning number of ulcers, was significant at the $p<0.01$ level.

Ader, Tatum and Beels (1960) found that the earlier the pups were removed from their mothers the more ulcers they received following placement in the Sawrey and Weisz apparatus. The earliest the pups were removed from their mother was at the age of fifteen days, these pups had the most ulcers, pups removed after the age of twenty-two days had the lowest number of ulcers.

Ader (1965) studied the effects of early life handling and latter life incidence of ulcers following immobilization. He found that Ss that received three minutes of handling from birth to the age of twenty-one days had fewer ulcers than unhandled controls, who in turn had fewer ulcers than Ss who received three minutes of a 0.2 mA shock during their

first twenty-one days of life. The difference in percent of Ss ulcered was not significant between the handled and control groups, but these two groups were both significantly different from the shocked group ($p<0.05$).

Social variables are seldom controlled or acknowledged in the physiological psychology research area, but as evidenced above, they are highly potent variables deserving of attention.

Part Three: Zinc Deficiency and Statement of Experimental Hypothesis

Skin lesions, along with hyperkeratinized esophageal, retarded weight gain, alopecia, retarded genital maturation, abnormal estrous cycle and increased mortality have been commonly observed in zinc deficient rats (Day & McCollum, 1940; Follis, Day & McCollum, 1941; Hurley, 1969; Whitenack, Luecke & Whitehair, 1970). Zinc deficiency has also been shown to retard the healing and decrease tensile strength of surgical and thermal wounds to the skin of rats (Sandstead, Lanier, Shephard & Gillespie, 1970; Sandstead & Shephard, 1968), and also to retard healing of skin wounds in cows (Miller, Morton, Pitts & Clifton, 1965). Zinc deficient rats have also been shown to be more emotional than control Ss as measured in the open field (Caldwell & Oberleas, 1969; Caldwell, Oberleas, Clancy & Prasad, 1970). Halas and Sandstead (1975) found that adult male rats who had been zinc deficient during their last trimester of gestation were poorer avoidance learners than their controls. The data showed a near normal learning rate during the first day and then a deterioration of performance that became more conspicuous as

the experiment progressed for the zinc deficient Ss. The control groups showed normal learning over trials. The data were interpreted as indicating an inability of the zinc deficient rats to tolerate stress. For a more complete review of the behavioral and physiological zinc deficient literature see Hanlon (1974), Peterson (1974), and Rowe (1974).

The healing process of gastric ulcers has been likened to the healing process of cutaneous wounds (Ivy, Grossman & Bachrach, 1950).

Evidence has been cited indicating that zinc deficient Ss are more emotional, tolerate stress less, have retarded wound healing, and many other pathological signs and symptoms. None of the above studies have taken direct measures of stress tolerance or effects. If the above relationships are correct the zinc deficient Ss, it is hypothesized, should show slower healing and more numerous ulcers than control Ss when placed under stress.

The Weiss (1971b) method was chosen to study the possible effects of prenatal zinc deficiency on stress in the adult rat. This method was chosen because of its close relationship to the human executive stress situation; the quantity of other behavioral data that could be measured (weight loss, water consumption, wheel turns, and shocks received); and its relative reliability in producing ulcers (several pilot studies with immobilization procedures and the Hamilton and Katske procedure (1973) yielded very inconsistent results). Sex, prenatal diet, stress condition were chosen as independent variables.

CHAPTER II

METHODOLOGY

Subjects

Thirty timed pregnant Long-Evans rats were received from the Charles River Breeding Labs.[1] Twenty of the dams were bred on January 27, 1975 and ten were bred on January 28, 1975. On the morning of February 10, day 14 of their pregnancy, the group of twenty dams was randomly divided into two dietary conditions; ten dams into the zinc-deficient (ZD) group and ten dams into the ad libitum (AL) group. On the morning of February 11, day 14 of their pregnancy, the remaining group of ten dams was randomly and individually yoked to dams in the ZD group for feeding; thus, they were a pair-fed (PF) group.

The ZD dams were fed ad libitum on a sprayed egg white diet described by Luecke, Olmam and Baltzer (1968) containing less than 1.00 ppm zinc (Table 5) and given deionized water. The PF dams were fed an amount of the diet that was equivalent to that eaten by their ZD counterpart on the previous day and given water containing 50 ppm of zinc in the form of zinc chloride. The AL dams were fed the zinc deficient diet ad libitum and given the zinc supplemented water. On the twentieth day of pregnancy all dams were taken off the zinc deficient diet and given access to unlimited amounts of rat chow and tap water. The dams' weights

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[1]Charles River Breeding Laboratories, 251 Ballardvale Street, Willmington, Massachusetts.

TABLE 5

The Zinc Deficient Diet[1]

Formula	g/kg
Egg White Solids, Spray Dried	200.00
Dextrose, Hydrate, Technical	630.108
Fiber, Nonnutritive	30.00
Oil, Corn	100.00
Salt Mix (see below)	
Vitamin Mix (see below)	
Salt Mix	
Calcium Carbonate ($CaCO_3$)	9.94405
Calcium Phosphate Dibasic ($CaHPO_4.2H_2O$)	3.1489
Cobalt Chloride ($CoCl_2.6H_2O$)	0.00185
Curpric Sulfate ($CuSO_4.5H_2O$)	0.00945
Ferric Citrate ($FeC_6H_5O_7.5H_2O$)	0.911542
Magnesium Sulfate ($MgSO_4.7H_2O$)	3.38106
Manganese Sulfate ($MnSO_4.H_2O$)	0.008791
Potassium Iodide (K1)	0.026518
Potassium Phosphate Dibasic ($K_2HPO_4.3H_2O$)	14.0044
Sodium Chloride (NaCl)	5.55198
Vitamin Mix	
Biotin	0.004
B_{12} (0.1% in Mannitol) Vitamin	0.020
Calcium Pantothenate	0.016
Choline Chloride	1.5
Folic Acid	0.0005
Menadione	0.00033
Niacin	0.025
Pyridoxine HCl	0.004
Riboflavin	0.006
Thiamine HCl	0.01
Inositol	1.00
	units/kg
Vitamin A Palmitate	10,000.000 IU
Vitamin D_2	1,250.000 IU
Vitamin E Acetate	110.000 IU

[1]This diet was obtained from General Biochemicals of Chagrin Falls, Ohio. It is a modified TDF1305 with 1g/kg of inositol added in place of chlorotetracycline.

were recorded daily from day 14 of their pregnancy to day 21 after delivering; while, food consumption was monitored daily without exception.

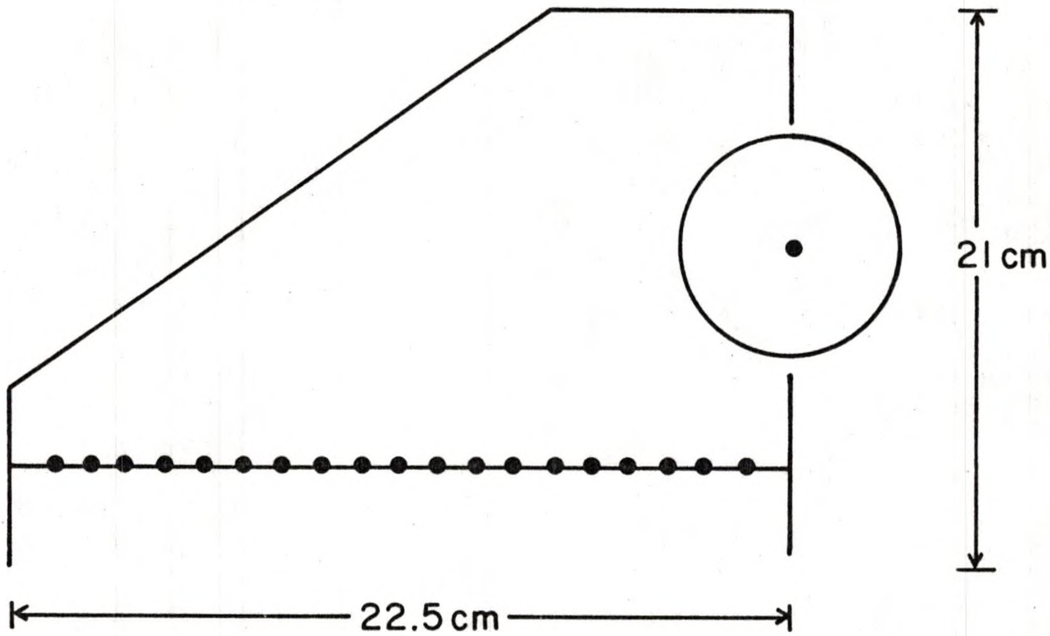
During the experimental dietary conditions the dams were caged in plexiglass cages with plastic bottom grates. The cages were housed in an air conditioned, filtered, constant temperature room with an automatic 12 hour light 12 hour dark lighting system.

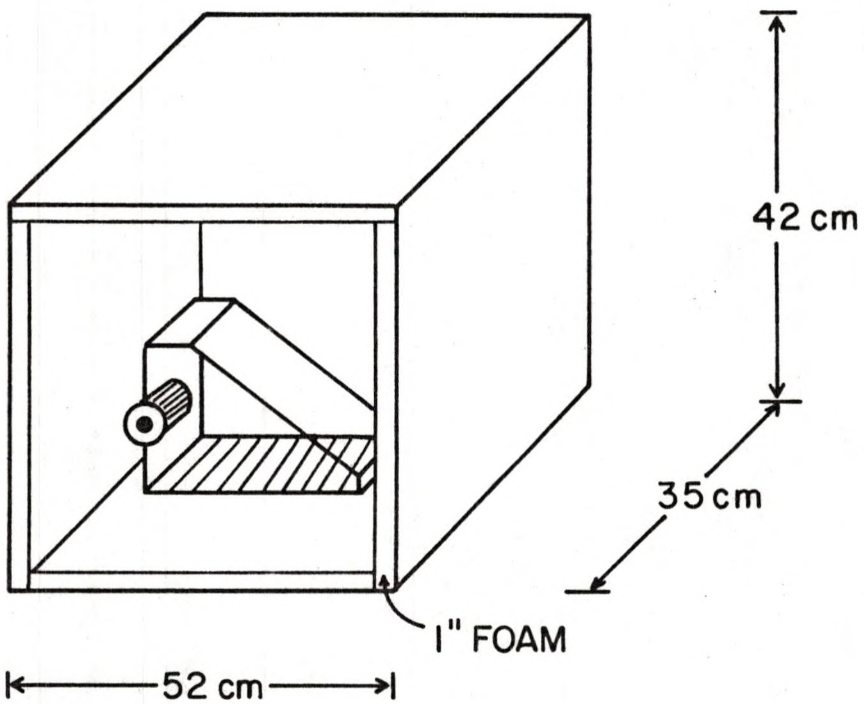
The pups were born after 21 to 22 days of gestation. At the age of two days all litter sizes were balanced so that each dam suckled nine pups; when substitution was needed it was done from the same dietary condition. At 21 days of age the pups were weaned to lab chow and tap water, and placed in stainless steel cages with two or three same sex Ss per cage. The litter weights were monitored daily starting at birth to the age of 24 days and thereafter every four days to the age of 40 days. The stress procedure was begun when the Ss had reached the age of 150 days.

Apparatus

Plexiglass avoidance-escape cages (Figure 2) were isolated in light-proof wooden cubicles lined with at least one inch of foam rubber sound proofing. Blowers supplied fresh air and white noise to each cubicle through three centimeter rubber hosing. The floor bars of these cages were 0.3 centimeters in diameter stainless steel rods set 1.3 centimeters center to center. Alternate bars were connected in parallel to similar polarity, thus the Ss received a shock by shorting this circuit across their feet or any other part of their body in contact with

Fig. 2. Stress apparatus and soundproof cubicles


21 cm
22.5 cm


42 cm
35 cm
1" FOAM
52 cm

opposite polarity bars (Figure 3, circuit diagram). Each pair of executive and yoked cages were wired in series, thus the current delivered to each cage of the pair was equal. The wheel at the tall end of the cage, when turned one revolution, operated a micro switch via a cam mounted on the axle of the wheel. This micro switch in turn could produce avoidance, escape, and conflictual shock. The CS's were produced by a 12 volt incandescent light and a Sonalert model SC628 tone generator mounted in each cubicle. The tone generator was pulsed four times per second and emitted a 2900 Hz tone, while the light was on constantly during the CS. The water bottles, manufactured by Scientific Prototype Company,[1] were of a design that limited spillage and were of about 51 milliliters in volume.

Procedure

The experimental stress procedure was executed in triplicate for each of the three apparatus groups. Each diet group was assigned to an apparatus group by a random cycle that produced equal exposure of the diet groups to the three apparatus groups. Within the diet groups each animal was randomly assigned to one of the three stress conditions (Executive, Yoked or Control). The executive (EXEC) S received a 20 second CS and could avoid or escape a 10 second shock by turning his or her wheel one revolution. Turning the wheel one revolution at any time reset the inter-trial interval clock. The Yoked S received a CS and a shock in yoke with his executive. The Control S received only the CS in

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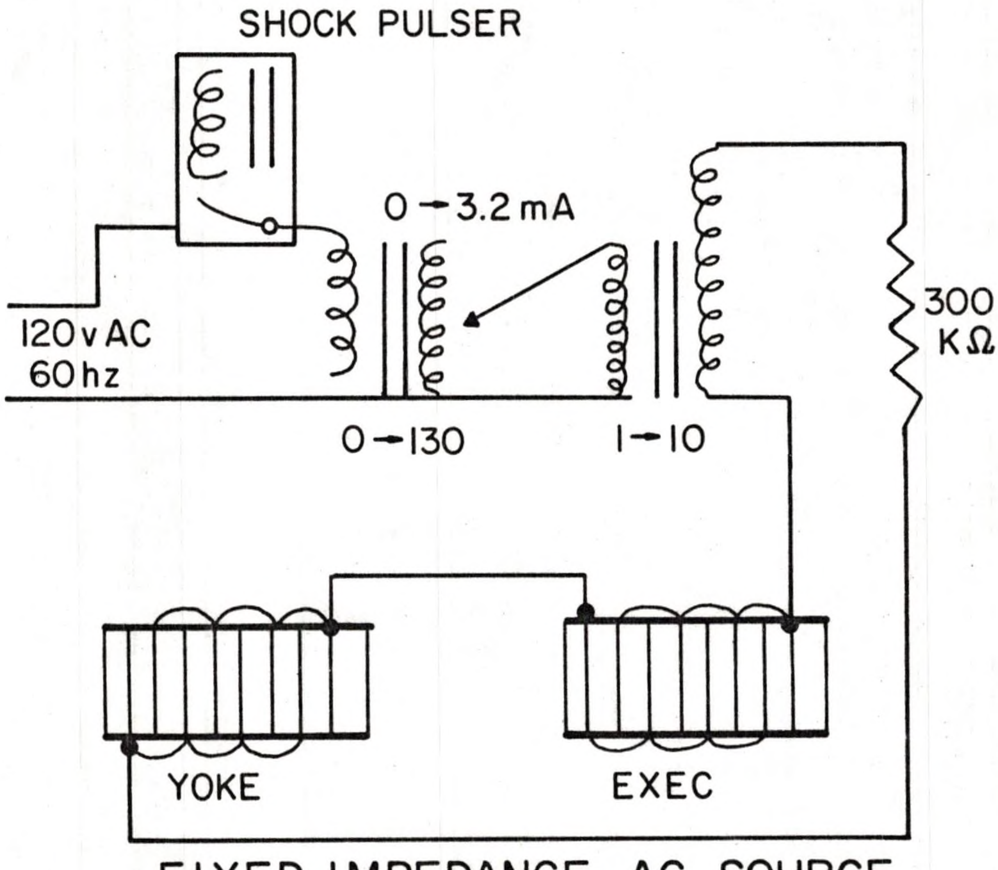
[1]Scientific Prototype Company, 615 West 131 Street, New York, New York 10027, circa 1974.

Fig. 3. The shock generator



FIXED IMPEDANCE AC SOURCE

yoke with his executive. Thus, nine same sex animals, three Ss from each diet group were run concurrently in the three experimental stress groups. The procedure was repeated twenty times thus, there were 180 Ss, sixty from each diet condition used.

Nine of the previously described Ss were weighed and then food deprived for 48 hours. At the end of the 48 hours the Ss were again weighed and then placed into their assigned experimental stress chamber to begin the wheel turning acquisition phase.

Wheel Turning Acquisition Phase

The purpose of this phase was to facilitate rapid acquisition of wheel turning avoidance-escape by the executive Ss, to monitor both the executive and yoked Ss' tolerance to the shock, and to monitor the equipment for proper functioning. An optimum shock level for avoidance-escape learning of 0.8 mA was used in conjunction with a 40 second inter-trial interval. The CS came on 20 seconds before the shock. The executive and yoked Ss were shocked for 10 seconds or until the executive S turned the wheel one revolution. This phase was in effect for 30 minutes; the first ten minutes of which the apparatus doors were kept open to closely observe the avoidance-escape acquisition and the Ss' reaction to the shock; the last twenty minutes of this phase the apparatus' doors were closed and the Ss were monitored on the equipment that recorded shocks received and wheel turns.

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Avoidance-escape Phase

This phase gave a measure of learning over two consecutive twelve hour periods. Again a twenty second CS and a ten second shock were used, but with a three minute inter-trial interval. Turning the executive wheel one revolution reset the inter-trial interval clock and terminated the CS or shock if they were being presented. The shock level was set a 1.6 mA during the first twelve hours and 2.2 mA during the second twelve hours. Wheel turns and number of shocks presented were recorded at the end of each of the twelve hour blocks.

Conflictual Coping Shock Phase

Directly on completion of the 24 hour Avoidance-escape phase the 48 hours of Conflictual-coping shock phase began. This phase had identical time parameters of the CS, inter-trial interval, and shock, to the avoidance-escape phase. In addition, a conflictual shock was presented after each revolution of the executives' wheels. This conflictual shock was a 0.2 second pulse at the same level as that of the avoidance-escape shock. The avoidance-escape shocks were 2.8 mA during the first twelve hours and were raised to 3.2 mA for the remainder of the experiment.

Total water consumption during these three phases was determined by refilling the water bottle to its original level with a 30 cc syringe. When it became evident that a S's water would not last twelve hours until the next check, a measured additional amount of water was injected into the water bottles. All Ss were checked at twelve hour intervals to make sure they had water, were alive, equipment was

properly functioning, etc.; when possible they were also checked at times between these twelve hour checks.

Sacrificing

All animals were weighed and as rapidly as possible individually sacrificed by decapitation. On removal the stomach was opened along the greater curvature, washed under tap water, flattened out by pinning to a cork board, and submerged on the cork board in a neutral, buffered, 10% formalin solution. The stomachs were unpinned from the cork board after about one hour. Photographs of the stomachs were taken at least one month from the time they were placed in the formalin solution (this was done as it has been observed that stomach pathologies photograph more clearly when allowed to become completely fixed. All photographs contained an identification number (these were non-repeating random numbers from 1 to 200), and a metric ruler calibrated in millimeters. From the photographs measurement of length and number of gastric pathologies could be made easily. Panatomic-X by Kodak was used for the negatives, exposed at F-11 for 1/15 of a second (a total of 1200 watts of incandescent lighting was set at 45 degrees from the horizontal, 60 centimeters from the stomach). Kodak Medalist print paper F-3 was used for the 12.7 by 17.8 centimeter prints.

The cages were soaked and scrubbed in a solution of a hospital disinfectant, rinsed, and air dried between each group of nine _S_s.

CHAPTER III

RESULTS

The results most relevant to the experiment--water consumption, stomach pathologies, weight loss during the food deprivation and during the wheel turning periods, wheel turning rate, and shock rate data--will be initially stated in a tabular format and then the various analysis of variance assumptions will be tested. Internal comparisons where necessary will be computed using a least squared difference procedure (Klecka, Nie & Hull, 1975). Since the degrees of freedom for these contrasts are large, the deviations are given in z scores rather than t scores. In addition to the classical course of statistical analysis of the data, group means that stand out from other group means (those of twice the magnitude of their immediate peers) will be mentioned. It is hoped that this procedure will minimize the chances of overlooking important differences that may exist, but because of the particular design did not stand out statistically.

Water Consumption

The consumption of water (measured in mls.) is summarized in Table 6. As is noticed, the within group variances are heterogeneous, generating a Hartley's F-max = 19.61 ($p<0.05$ with $n=10$ and $k=18$). Because of the heterogeneity of within group variances, the data were

TABLE 6

Water Consumption

Stress	Sex	Nutrition Zinc Deficient (ZD)	 Pair Fed (PF)	 Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)*	Female	100.30	112.80	167.20	126.78	mean
		38.41	76.26	92.79		var.
	Male	177.50	152.40	192.30	174.07	mean
		66.02	68.02	103.56		var.
	Totals	138.90	132.60	179.75	150.43	mean
Yoke*	Female	97.50	106.80	115.50	106.60	mean
		31.49	44.96	55.81		var.
	Male	147.20	126.00	172.30	148.50	mean
		76.34	44.72	62.96		var.
	Totals	122.35	116.40	143.90	127.55	mean
Control (Cont)*	Female	35.10	22.90	31.50	29.83	mean
		43.84	8.63	19.56		var.
	Male	29.30	41.00	24.10	31.47	mean
		13.45	29.05	5.28		var.
	Totals	32.20	31.95	27.80	30.65	mean
All**	Female	77.63	80.83	104.23	87.73	mean
	Male	118.00	106.47	129.57	118.01	mean
	Totals	97.82	93.65	117.15	102.87	mean

*n=10 per cell
**n=30 per cell

transformed using a natural log function, log(x+1), see Table 7. This transform removed the problem of heterogeneity of within group variances, F-max = 3.05 (p=ns., F-max = 11.00 is needed at the 0.05 level--its value was derived by algebraic extrapolation of the function). Analysis of variance was computed on the transformed data (Table 8). Two effects

TABLE 7

Log of Water Consumption

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)*	Female	4.555	4.541	4.989	4.690	mean
		0.372	0.664	0.556		var.
	Male	5.117	4.929	5.139	5.06	mean
		0.398	0.501	0.525		var.
	Totals	4.84	4.74	5.06	4.88	mean
Yoke	Female	4.536	4.593	4.682	4.60	mean
		0.366	0.460	0.383		var.
	Male	4.875	4.780	5.092	4.92	mean
		0.532	0.396	0.390		var.
	Totals	4.71	4.69	4.89	4.76	mean
Control (Cont)*	Female	3.273	3.116	3.330	3.24	mean
		0.690	0.358	0.574		var.
	Male	3.338	3.581	3.201	3.37	mean
		0.386	0.541	0.226		var.
	Totals	3.31	3.35	3.27	3.31	mean
All**	Female	4.12	4.08	4.33	4.18	mean
	Male	4.44	4.43	4.48	4.45	mean
	Totals	4.28	4.26	4.41	4.32	mean

*n=10 per cell
**n=30 per cell

TABLE 8

Analysis of Variance Table for Log of Water Consumption

Source	Df	MS	F-test
Sex	1	3.299	14.534***
Nutr	2	0.380	1.672
Stress	2	45.963	202.514***
Sex x Nutr	2	0.184	0.809
Sex x Stress	2	0.223	0.984
Nutr x Stress	4	0.234	1.031
Sex x Nutr x Stress	4	0.277	1.222
Error Term	162	0.227	

***$p<0.001$

achieved the $p<0.05$ level of significance, the SEX and STRESS main effects. The male Ss consumed more water than the female Ss did. The EXEC and YOKE group means did not differ ($z = 1.38$); both of these groups had significantly higher means than that of the CONT group's ($z = 18.05$ and $z = 16.67$ respectively, $p<0.001$ for both).

Lower Stomach Pathology Length

Length (measured in mm. across the longest extent and summed within Ss) of pathologies in the lower portion of the stomach are listed in Table 9. For stomach pathology data the N was reduced to 175 rather than 180. This resulted from a combination of loss of negatives and cropping of prints by a local film developer that left five unidentifiable. For these data, heterogeneity of within group variances was again a problem, Hartley's F-max = 39.14 (k=18 and n=10), and again a log

TABLE 9

Lower Stomach Pathology Length

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)**	Female	4.278*	4.970	6.550	5.27	mean
		7.627	6.674	9.470		var.
	Male	0.822*	6.150	5.480	4.15	mean
		0.932	12.167	5.965		var.
	Totals	2.55	5.56	6.02	4.71	mean
Yoke**	Female	6.510	4.180	3.589*	4.76	mean
		12.676	6.605	3.367		var.
	Male	0.750	2.490	3.144*	2.13	mean
		1.181	4.604	4.147		var.
	Totals	3.63	3.34	3.37	3.44	mean
Control (Cont)**	Female	1.030	0.544*	0.630	0.74	mean
		1.018	0.324	0.380		var.
	Male	0.760	0.950	0.530	0.75	mean
		0.909	0.913	0.607		var.
	Totals	0.90	0.75	0.58	0.74	mean
All***	Female	3.94****	3.23***	3.59****	3.59	mean
	Male	0.78****	3.20	3.05	2.34	
	Totals	2.36	3.21	3.32	2.96	mean

*n=9 rather than 10
**n=10 (generally) per cell
***n=30 per cell (generally)
****n=29 per cell

transformation, log (x+1), was implemented (Table 10). This transformation reduced the F-max to 6.67, a nonsignificant level, and analysis of variance was used with the transformed data (Table 11). The STRESS main

TABLE 10

Log of Lower Stomach Pathology Length

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	1.036	1.352	1.268	1.22	mean
		1.060	0.951	1.274		var.
	Male	0.501	1.136	1.432	1.02	mean
		0.439	1.171	1.032		var.
	Totals	0.77	1.24	1.35	1.12	mean
Yoke	Female	1.313	1.064	1.220	1.20	mean
		1.070	1.075	0.881		var.
	Male	0.438	0.828	0.984	0.75	mean
		0.459	0.834	0.984		var.
	Totals	0.88	0.95	1.10	0.97	mean
Control (Cont)	Female	0.616	0.417	0.464	0.50	mean
		0.431	0.191	0.235		var.
	Male	0.476	0.597	0.366	0.48	mean
		0.417	0.367	0.350		var.
	Totals	0.55	0.51	0.42	0.49	mean
All	Female	0.99	0.94	0.98	0.97	mean
	Male	0.47	0.87	0.93	0.75	mean
	Totals	0.73	0.90	0.96	0.86	mean

*For n's see Table 9

TABLE 11

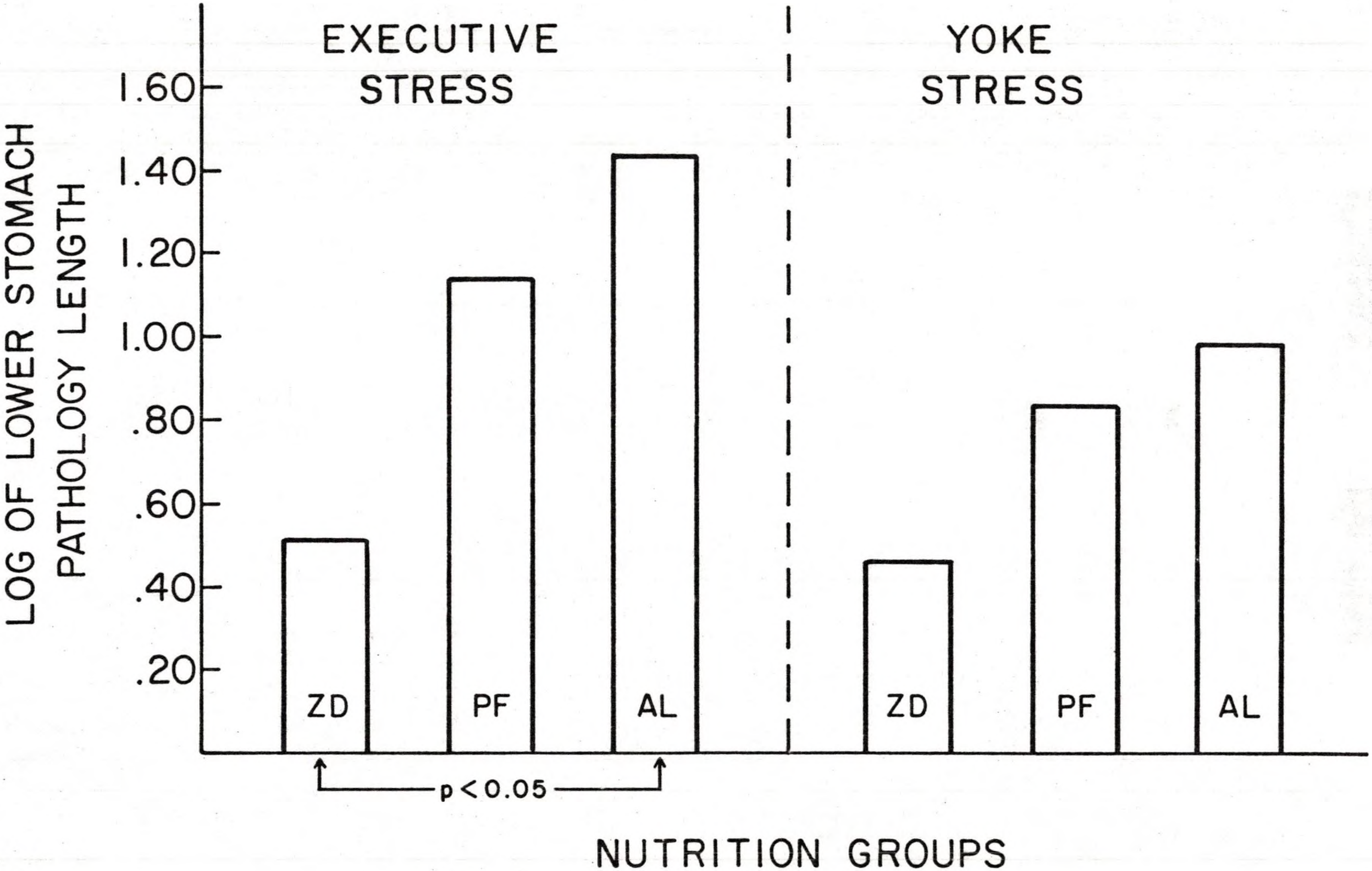
Unweighted Means Analysis of Variance Table for Log of Lower Stomach Pathology Length

Source	Df	MS	F-test
Sex	1	2.132	3.207
Nutr	2	0.756	1.197
Stress	2	6.370	9.581***
Sex x Nutr	2	0.946	1.423
Sex x Stress	2	0.680	1.023
Nutr x Stress	4	0.698	1.050
Sex x Nutr x Stress	4	0.222	0.335
Error Term	157	0.665	

***$p<0.001$

effect was significant at the $p<0.001$ level. This main effect consisted of significant differences between EXEC and CONT group means ($z = 4.23$ and $p<0.001$ with the EXEC mean being higher) and between YOKE and CONT group means ($z = 3.22$ and $p<0.01$ with the YOKE mean being higher); the EXEC and YOKE group means did not differ significantly ($z = 1.01$). The SEX main effect was close to being significant ($p = 0.076$), with the female group being somewhat higher than that of the males. ZD male EXEC and YOKE Ss seemed to demonstrate a degree of protection from the stress treatments having much lower group means than other YOKE and EXEC male groups (Figure 4). When these means were submitted to z-tests the ZD EXEC male group's mean was significantly lower than that of the AL EXEC male group's mean ($z = 2.54$, $p<0.05$).

Fig. 4. Zinc deficient (ZD) male protection concerning lower stomach pathology length


EXECUTIVE STRESS
YOKE STRESS
LOG OF LOWER STOMACH PATHOLOGY LENGTH
1.60
1.40
1.20
1.00
.80
.60
.40
.20
ZD
PF
AL
ZD
PF
AL
p < 0.05
NUTRITION GROUPS

Lower Stomach Pathology Number

The number of lower stomach pathologies is given in Table 12. Although the within group variances are not heterogeneous (F-max = 4.71, k=18 and n=10), many of the groups have somewhat skewed distributions. A log transformation, log (x+1) of the raw data (Table 13), yielded data that were neither of heterogeneous within group variances or seriously skewed. The log transformed data were analyzed using analysis of variance and the results are shown in Table 14. It can be seen that the STRESS main effect was significant ($p<0.05$). The EXEC and CONT group means were significantly different ($z = 2.64$ and $p<0.01$ with the EXEC mean higher), while the EXEC and YOKE group means and the YOKE and CONT group means were not significantly different ($z = 1.60$ and 1.04 respectively). Two other effects were near the $p = 0.05$ level, namely the SEX main effect ($p = 0.067$) and the NUTR by SEX interaction ($p = 0.063$). The SEX main effect has the female's group mean somewhat higher than that of the male group mean. The means involved in the NUTR by SEX interaction are graphed in Figure 5. As can be seen, this interaction consists of a break of trend for the SEX main effect for the PF Ss; the female PF Ss did not have more pathologies than did the male PF Ss.

Upper Stomach Pathology Length

Table 15 contains the group means of the upper stomach pathology length data. Many group means are less than their standard deviations indicating skew, also Hartley's F-max = 31.18 (k=18 and n=10) indicating heterogeneity of within group variances. The heterogeneity of within

TABLE 12

Lower Stomach Pathology Number*

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	8.778	7.200	11.200	9.06	mean
		9.418	5.350	11.970		var.
	Male	4.778	9.500	7.800	7.36	mean
		4.177	10.947	5.789		var.
	Totals	6.78	8.35	9.50	8.21	mean
Yoke	Female	8.600	7.500	7.889	8.00	mean
		6.059	6.311	7.044		var.
	Male	3.500	7.000	5.222	5.24	mean
		2.593	6.307	4.684		var.
	Totals	6.05	7.25	6.56	6.62	mean
Control (Cont)	Female	4.600	3.444	5.800	4.62	mean
		2.716	1.509	3.360		var.
	Male	3.000	5.800	3.000	3.93	mean
		3.127	3.521	2.539		var.
	Totals	3.80	4.62	4.40	4.27	mean
All	Female	7.33	6.05	8.30	7.22	mean
	Male	3.76	7.43	5.34	5.51	mean
	Totals	5.54	6.74	6.82	6.37	mean

*For n's see Table 9

TABLE 13

Log of Lower Stomach Pathology Number*

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	1.911	1.892	2.012	1.94	mean
		.956	0.723	1.125		var.
	Male	1.488	1.941	1.947	1.79	mean
		0.825	0.932	0.749		var.
	Totals	1.70	1.92	1.98	1.87	mean
Yoke	Female	2.123	1.784	1.761	1.89	mean
		0.523	0.993	1.125		var.
	Male	1.335	1.763	1.574	1.56	mean
		0.654	0.904	0.757		var.
	Totals	1.73	1.77	1.67	1.72	mean
Control (Cont)	Female	1.624	1.434	1.741	1.60	mean
		0.473	0.374	0.730		var.
	Male	1.153	1.825	1.163	1.38	mean
		0.706	0.430	0.753		var.
	Totals	1.39	1.63	1.45	1.49	mean
All	Female	1.89	1.70	1.84	1.81	mean
	Male	1.33	1.84	1.56	1.58	mean
	Totals	1.61	1.77	1.70	1.69	mean

*For n's see Table 9

TABLE 14

Unweighted Means Analysis of Variance Table for Log of Lower Stomach Pathology Number

Source	Df	MS	F-test
Sex	1	2.380	3.776
Nutr	2	0.411	0.658
Stress	2	2.085	3.336*
Sex x Nutr	2	1.806	2.889
Sex x Stress	2	0.127	0.203
Nutr x Stress	4	0.183	0.293
Sex x Nutr x Stress	4	0.324	0.518
Error Term	157	0.625	

*$p<0.05$

Fig. 5. Log of lower stomach pathology number: Sex by nutrition group interaction

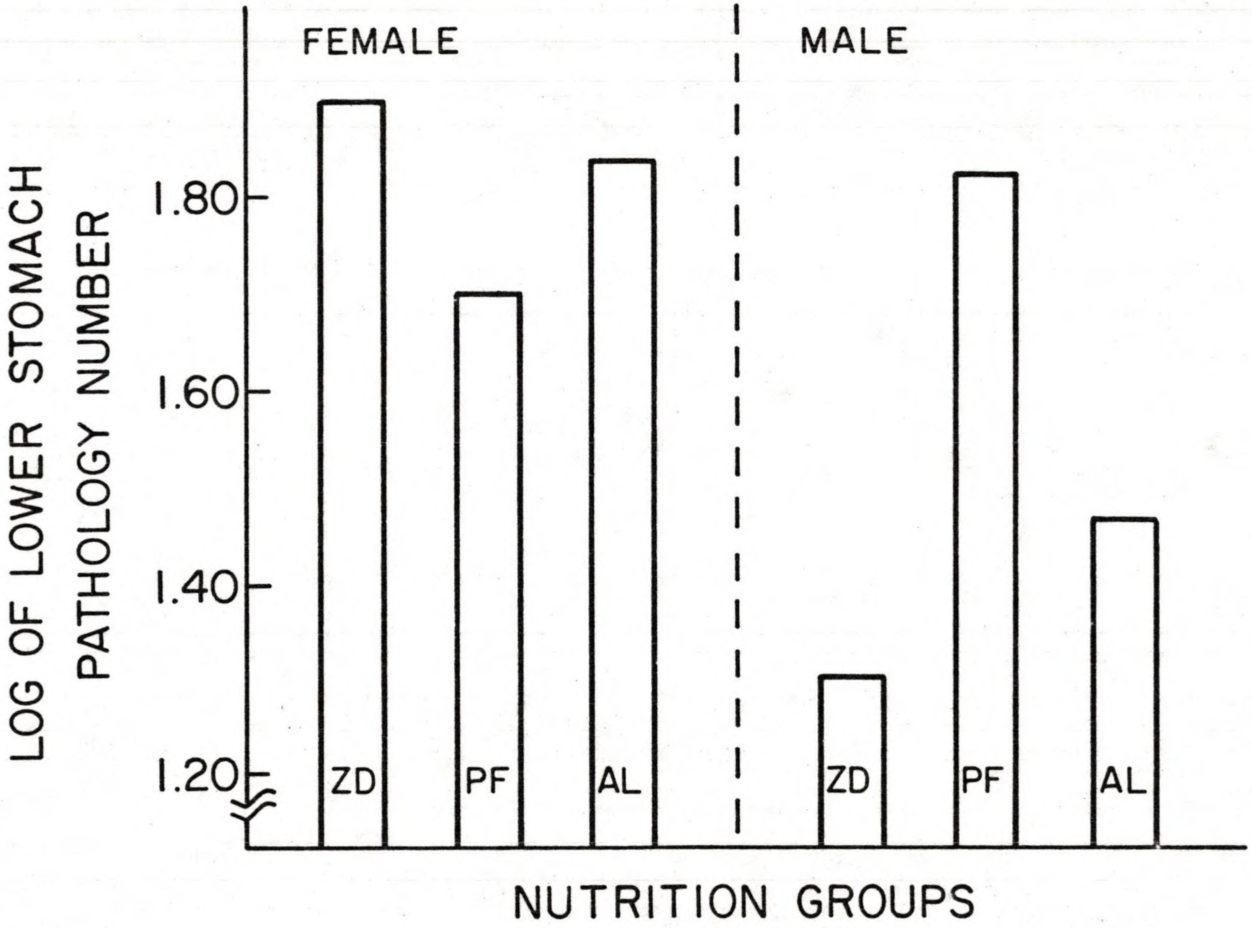

FEMALE
MALE
LOG OF LOWER STOMACH PATHOLOGY NUMBER
1.80
1.60
1.40
1.20
ZD
PF
AL
ZD
PF
AL
NUTRITION GROUPS

TABLE 15

Upper Stomach Pathology Length*

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	1.467	1.600	0.330	1.13	mean
		1.721	2.424	1.009		var.
	Male	0.889	0.340	0.290	0.51	mean
		1.668	0.560	0.491		var.
	Totals	1.18	0.97	0.31	0.83	mean
Yoke	Female	0.430	1.800	0.067	0.77	mean
		0.921	2.758	0.166		var.
	Male	0.960	1.480	0.856	1.10	mean
		1.842	2.086	1.956		var.
	Totals	0.70	1.64	0.46	0.93	mean
Control (Cont)	Female	0.090	2.344	2.370	1.60	mean
		0.129	4.022	3.942		var.
	Male	1.320	1.640	1.180	1.38	mean
		1.619	1.643	1.755		var.
	Totals	0.71	1.99	1.78	1.49	mean
All	Female	0.66	1.91	0.92	1.17	mean
	Male	1.06	1.15	0.76	1.00	mean
	Totals	0.86	1.53	0.85	1.08	mean

*For n's see Table 9

group variances problem was remedied (F-max = 8.61) via log transformation, log (x+1), although the degree of skew was not reduced (Table 16). Analysis of variance was computed on this not perfect but improved data (Table 17). None of the analysis of variance effects were significant at the $p<0.05$ level, although the NUTR main effect came close ($p = 0.081$). The PF and AL group means were significantly different ($p<0.05$ $z = 2.18$ with the PF mean being higher), while the ZD and AL and the ZD and PF group means did not differ significantly ($z = 0.59$ and $z = 1.59$ respectively). The AL and ZD YOKE, AL EXEC, and the ZD CONT female group means are considerably lower than the other female groups (Table 16). As can be seen in Figure 6, the above mentioned groups were significantly lower than their counterparts, indicating a degree of protection from upper stomach pathologies for these groups. Since the only overt variables being manipulated here are NUTR and STRESS, it indicates that the different NUTR groups are differentially affected by levels of STRESS.

Upper Stomach Pathology Number

The data are presented in Table 18. Again the data are seriously skewed and Hartley's F-max = 14.69 (k=18 and n=10) indicating a heterogeneity of within group variance problem. The log transformation, log (x+1), was executed on the data yielding a more homogeneous distribution (F-max = 3.55) of within group variances, but the data were still skewed (Table 19). The log transformed data were submitted to analysis of variance (Table 20). The NUTR main effect came closest to being significant ($p = 0.094$). The PF and AL group means did differ significantly ($p<0.05$, $z = 2.23$ with the PF mean being higher), while the other two

TABLE 16

Log of Upper Stomach Pathology Length*

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	0.682	0.629	0.153	0.49	mean
		0.706	0.807	0.451		var.
	Male	0.393	0.231	0.202	0.28	mean
		0.665	0.347	0.323		var.
	Totals	0.54	0.43	0.18	0.38	mean
Yoke	Female	0.228	0.665	0.056	0.32	mean
		0.484	0.852	0.135		var.
	Male	0.416	0.630	0.361	0.47	mean
		0.669	0.756	0.637		var.
	Totals	0.32	0.65	0.21	0.39	
Control (Cont)	Female	0.080	0.707	0.718	0.50	mean
		0.113	0.973	0.980		var.
	Male	0.636	0.765	0.556	0.65	mean
		0.665	0.699	0.658		var.
	Totals	0.36	0.74	0.64	0.58	mean
All	Female	0.33	0.67	0.31	0.44	mean
	Male	0.48	0.54	0.37	0.47	mean
	Totals	0.41	0.60	0.34	0.45	mean

*For n's see Table 9

TABLE 17

Unweighted Means Analysis of Variance Table for Log of Upper Stomach Pathology Length

Source	Df	MS	F-test
Sex	1	0.040	0.094
Nutr	2	1.096	2.565
Stress	2	0.700	1.638
Sex x Nutr	2	0.290	0.680
Sex x Stress	2	0.644	1.507
Nutr x Stress	4	0.658	1.540
Sex x Nutr x Stress	4	0.387	0.906
Error Term	157	0.427	

Fig. 6. Upper stomach pathology length

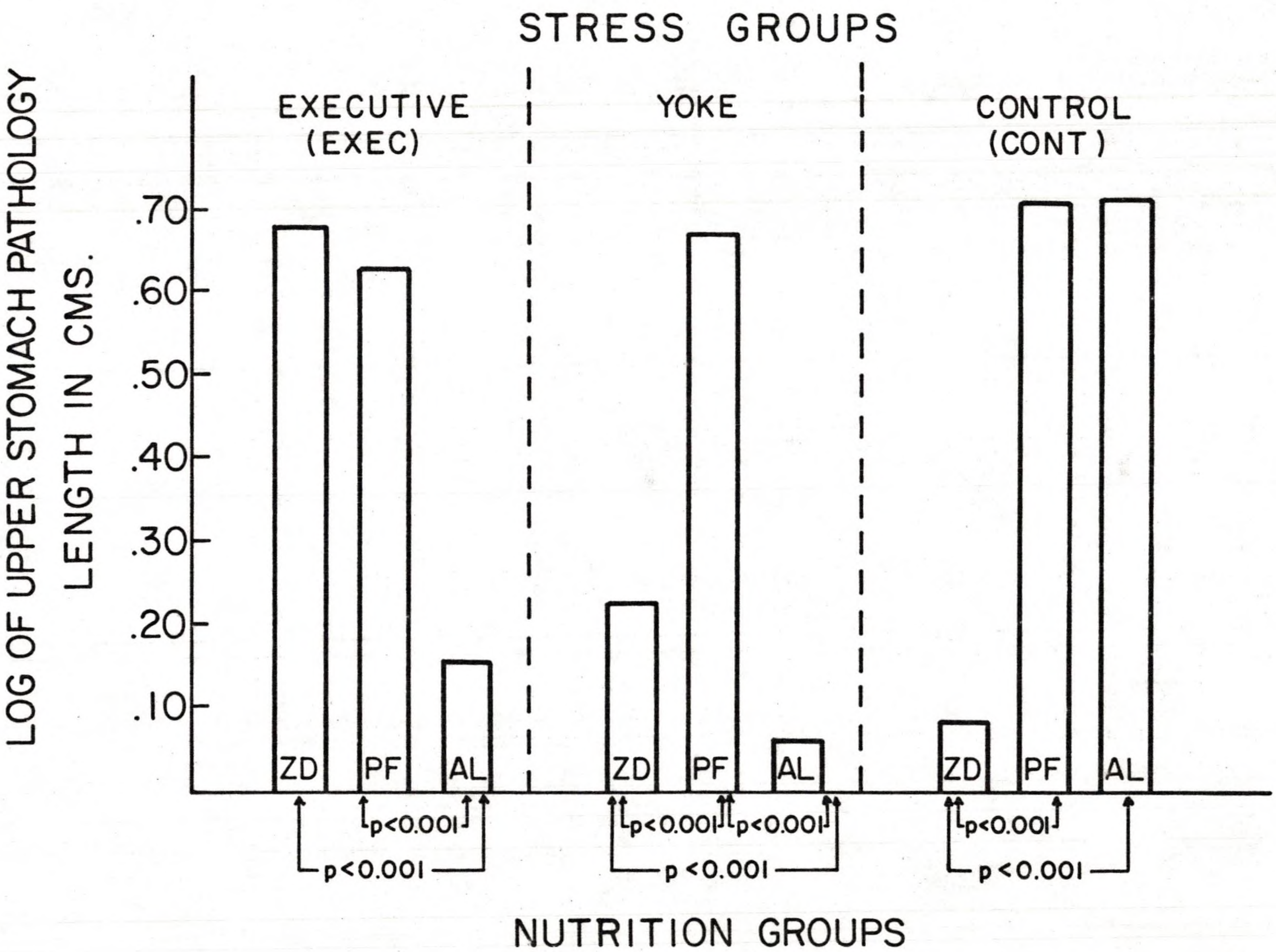

STRESS GROUPS
EXECUTIVE (EXEC)
YOKE
CONTROL (CONT)
LOG OF UPPER STOMACH PATHOLOGY LENGTH IN CMS.
.70
.60
.50
.40
.30
.20
.10
ZD
PF
AL
ZD
PF
AL
ZD
PF
AL
p<0.001
p<0.001
p<0.001
p<0.001
p<0.001
p<0.001
p<0.001
NUTRITION GROUPS

TABLE 18

Upper Stomach Pathology Number*

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	3.000	3.000	0.700	2.23	mean
		3.162	4.497	1.889		var.
	Male	1.889	1.100	1.000	1.33	mean
		3.655	1.524	1.633		var.
	Totals	2.44	2.05	0.85	1.78	
Yoke	Female	.800	3.700	0.222	1.57	mean
		1.687	4.322	0.441		var.
	Male	1.800	3.000	1.333	2.04	mean
		2.616	3.464	2.236		var.
	Totals	1.30	3.35	0.78	1.81	mean
Control (Cont)	Female	0.700	4.000	3.000	2.57	mean
		0.949	6.481	4.320		var.
	Male	4.500	3.100	2.700	3.43	mean
		6.502	4.202	2.946		var.
	Totals	2.60	3.52	2.85	2.99	mean
All	Female	1.50	3.57	1.31	2.13	mean
	Male	2.73	2.40	1.68	2.27	mean
	Totals	2.12	2.98	1.49	2.20	mean

*For n's see Table 9

TABLE 19

Log of Upper Stomach Pathology Number*

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	1.056	0.835	0.264	0.72	mean
		0.907	1.067	0.630		var.
	Male	0.609	0.519	0.468	0.53	mean
		0.894	0.686	0.659		var.
	Totals	0.83	0.68	0.37	0.63	mean
Yoke	Female	0.322	1.073	0.154	0.52	mean
		0.679	1.069	0.306		var.
	Male	0.698	0.988	0.584	0.76	mean
		0.827	0.972	0.698		var.
	Totals	0.51	1.03	0.37	0.64	mean
Control (Cont)	Female	0.399	0.997	0.886	0.76	mean
		0.528	1.099	1.036		var.
	Male	1.094	1.031	1.004	1.04	mean
		1.156	0.904	0.846		var.
	Totals	0.75	1.01	0.95	0.90	mean
All	Female	0.59	0.97	0.43	0.67	mean
	Male	0.80	0.85	0.69	0.78	mean
	Totals	0.70	0.91	0.56	0.72	mean

*For n's see Table 9

TABLE 20

Unweighted Means Analysis of Variance Table for Log of Upper Stomach Pathology Number

Source	Df	MS	F-test
Sex	1	0.550	0.742
Nutr	2	1.780	2.402
Stress	2	1.426	1.924
Sex x Nutr	2	0.608	0.820
Sex x Stress	2	0.978	1.320
Nutr x Stress	4	1.021	1.379
Sex x Nutr x Stress	4	0.492	0.665
Error Term	157	0.741	

contrasts, ZD vs. PF and PF vs. AL, were not significant ($z = 1.34$ and $z = 0.89$ respectively). These data are similar in trend to the upper stomach pathology length data above except that the number data are less powerful in discriminating differences among groups (Figure 7).

Wheel Turning Behavior

The wheel turning rates during the six twelve-hour blocks are given in Table 21. Responding rates for the EXEC Ss slowed rapidly until a steady rate of from 25 to 50 turns per hour was reached in the third block. The YOKE Ss showed a sudden rise in responding rate during the third period--this was the point where the number of shocks increased because of the EXEC Ss' slower responding and the addition of the conflictual shock. Generally CONT Ss exhibited steady responding rates from 1 to 39 turns per hour. The data are both seriously skewed and of heterogeneous within group variances (F-max = 469.76, k=108 and

Fig. 7. Log of upper stomach pathology number

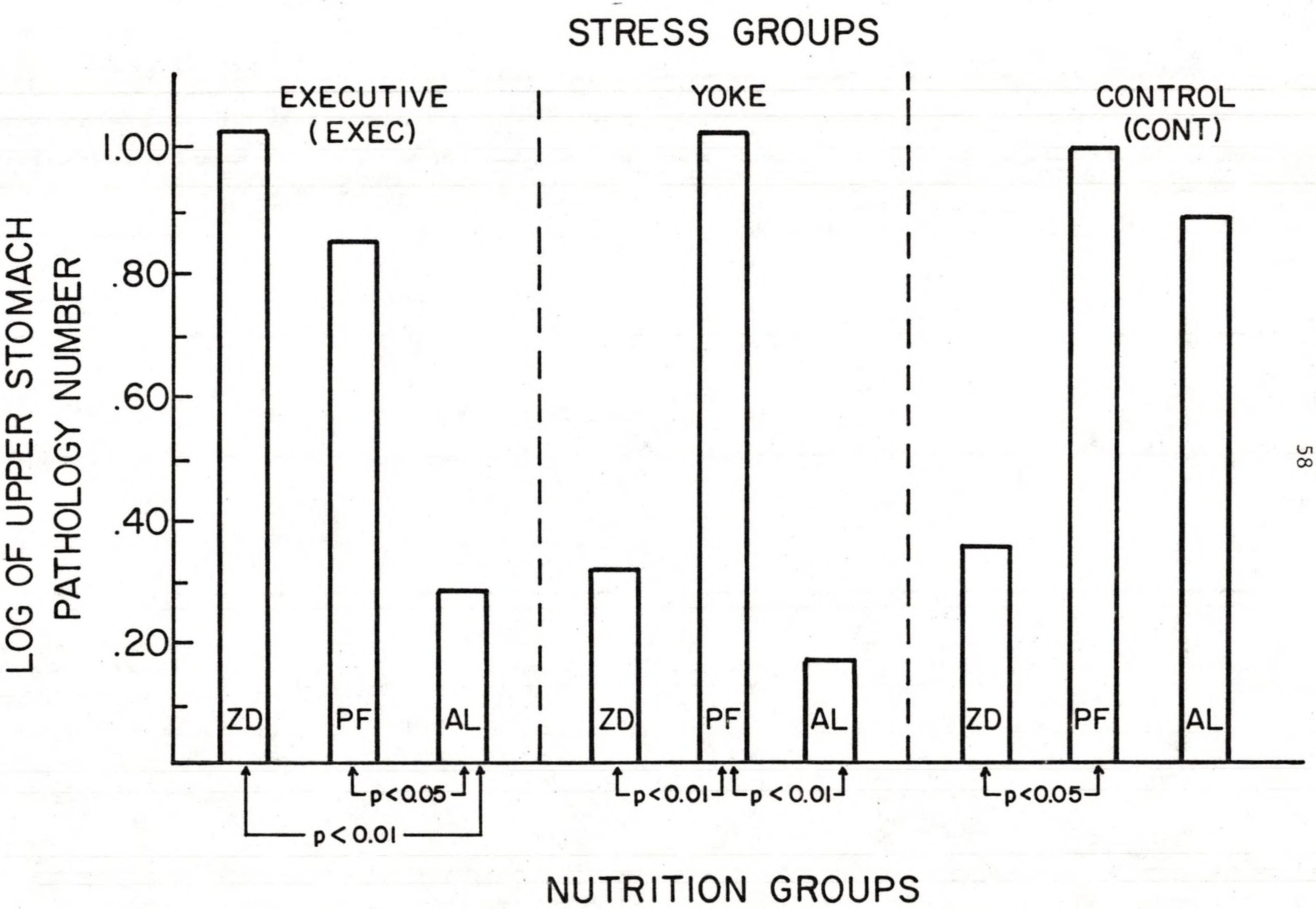

STRESS GROUPS
EXECUTIVE (EXEC)
YOKE
CONTROL (CONT)
LOG OF UPPER STOMACH PATHOLOGY NUMBER
1.00
.80
.60
.40
.20
ZD
PF
AL
ZD
PF
AL
ZD
PF
AL
p<0.05
p<0.01
p<0.01
p<0.01
p<0.05
NUTRITION GROUPS

TABLE 21

Wheel Turning Rates per Trial Block

EXECUTIVE STRESS*

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Nutrition: Pair Fed (PF)	Nutrition: Ad Libitum (AL)	Totals	Statistic
Trial 1	Female	1203.5	728.6	303.6	754.2	mean
		1010.8	642.5	240.3		var.
	Male	369.1	321.6	289.5	326.7	mean
		584.2	274.1	208.8		var.
	Totals	786.3	525.1	310.1	540.5	mean
Trial 2	Female	725.6	409.4	160.6	431.8	mean
		614.1	560.1	174.3		var.
	Male	159.5	203.0	176.3	179.4	mean
		235.1	238.8	173.9		var.
	Totals	442.6	306.2	168.5	305.7	mean
Trial 3	Female	39.61	44.70	42.98	42.41	mean
		10.37	32.23	8.10		var.
	Male	42.78	42.25	51.31	44.45	mean
		13.03	11.10	15.64		var.
	Totals	41.20	43.45	47.15	43.43	mean
Trial 4	Female	34.66	34.28	33.84	34.26	mean
		15.57	19.41	5.80		var.
	Male	34.42	32.32	41.15	35.96	mean
		11.88	11.88	14.92		var.
	Totals	34.54	33.30	37.50	35.11	mean
Trial 5	Female	34.07	31.42	32.15	32.55	mean
		13.07	20.98	9.16		var.
	Male	56.86	27.59	35.65	40.03	mean
		98.50	14.23	10.68		var.
	Totals	45.47	29.51	33.90	36.29	mean

*n=10 per cell

Table 21--Continued

EXECUTIVE STRESS*

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 6	Female	35.01	26.21	25.24	28.82	mean
		10.52	14.69	7.81		var.
	Male	28.29	20.04	60.98	36.44	mean
		12.66	13.58	89.05		var.
	Totals	31.65	23.13	43.11	32.63	mean

YOKE STRESS*

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 1	Female	115.32	525.57	138.86	259.9	mean
		200.29	706.89	148.96		var.
	Male	174.17	125.88	46.35	115.4	mean
		325.66	178.41	47.42		var.
	Totals	144.75	325.73	92.61	187.6	mean
Trial 2	Female	83.11	412.36	122.60	206.0	mean
		105.35	738.30	197.84		var.
	Male	93.61	279.24	40.29	137.7	mean
		217.15	437.07	63.03		var.
	Totals	88.36	345.80	81.45	171.9	mean
Trial 3	Female	209.77	619.89	348.44	392.7	mean
		231.68	618.81	708.31		var.
	Male	286.05	272.67	98.93	219.2	mean
		608.72	395.32	132.60		var.
	Totals	247.9	446.3	223.7	305.9	mean
Trial 4	Female	126.07	266.22	117.72	170.0	mean
		161.17	424.12	275.24		var.
	Male	49.78	89.95	86.92	75.6	mean
		61.09	87.93	146.20		var.
	Totals	87.9	178.1	102.3	122.7	mean

*n=10 per cell

Table 21--Continued

YOKE STRESS*

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 5	Female	58.88	137.16	32.62	76.22	mean
		97.24	201.98	40.31		var.
	Male	30.32	79.39	32.88	47.53	mean
		31.57	144.88	32.72		var.
	Totals	44.6	108.3	32.8	61.9	mean
Trial 6	Female	32.44	183.75	35.87	84.02	mean
		50.77	294.40	55.85		var.
	Male	30.72	57.66	21.80	36.73	mean
		34.55	73.89	25.06		var.
	Totals	31.58	120.71	28.84	60.37	mean

CONTROL STRESS*

Trial	Sex	Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 1	Female	14.79	29.53	13.71	19.34	mean
		23.41	66.32	19.69		var.
	Male	5.37	11.45	8.15	8.32	mean
		6.54	21.44	11.59		var.
	Totals	10.08	20.49	10.93	13.83	mean
Trial 2	Female	10.41	9.80	17.15	12.45	mean
		13.66	9.86	28.16		var.
	Male	9.16	18.26	4.36	10.60	mean
		16.63	53.19	6.25		var.
	Totals	9.79	14.03	10.76	11.52	mean
Trial 3	Female	4.11	16.43	10.72	10.42	mean
		5.46	35.40	16.42		var.
	Male	28.16	16.76	6.56	17.16	mean
		51.39	50.40	13.64		var.
	Totals	16.14	16.60	8.64	13.79	mean

*n=10 per cell

Table 21--Continued

CONTROL STRESS*

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 4	Female	20.17	35.70	12.76	22.89	mean
		28.07	55.22	23.29		var.
	Male	39.12	28.55	8.35	25.34	mean
		66.66	73.83	13.63		var.
	Totals	29.65	32.15	10.56	24.12	mean
Trial 5	Female	9.92	9.56	7.58	9.02	mean
		14.29	16.13	14.68		var.
	Male	16.34	26.91	1.87	15.04	mean
		40.98	75.13	2.15		var.
	Totals	13.13	18.24	4.73	12.03	mean
Trial 6	Female	20.79	31.79	18.32	23.63	mean
		18.02	46.40	30.49		var.
	Male	23.62	23.97	6.23	17.94	mean
		37.29	70.23	5.74		var.
	Totals	22.21	27.88	12.28	20.78	mean

*n=10 per cell

n=10; F-max = 15.0 is needed for k=108 and n=10--this value was derived by algebraic extrapolation of the function), therefore it was log transformed, log(x+1), with hopes of remedy (Table 22). This transformed data had been improved to an acceptable level (F-max = 13.13) and analysis of variance was computed (Table 23). It can be seen that a very large proportion of the variance is associated with the STRESS main effect and its interactions with TIME, the TIME main effect, and the error terms. The data were split along STRESS group lines and three new

TABLE 22

Log of Wheel Turning Rates per Trial Block

EXECUTIVE STRESS*

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 1	Female	6.624	6.075	5.466	6.05	mean
		1.218	1.160	0.951		var.
	Male	4.975	5.396	5.415	5.26	mean
		1.362	0.985	0.792		var.
	Totals	5.80	5.74	5.44	5.65	mean
Trial 2	Female	6.048	5.182	4.517	5.24	mean
		1.256	1.450	1.164		var.
	Male	4.381	4.848	4.830	4.67	mean
		1.133	0.959	0.872		var.
	Totals	5.20	5.00	4.06	4.96	mean
Trial 3	Female	3.673	3.544	3.768	3.63	mean
		0.267	0.943	0.192		var.
	Male	3.736	3.732	3.913	3.77	mean
		0.317	0.295	0.323		var.
	Totals	3.70	3.60	3.84	3.70	mean
Trial 4	Female	3.443	3.461	3.539	3.45	mean
		0.618	0.453	0.358		var.
	Male	3.513	3.442	3.671	3.51	mean
		0.355	0.394	0.421		var.
	Totals	3.49	3.45	3.58	3.48	mean
Trial 5	Female	3.494	3.192	3.467	3.33	mean
		0.380	0.969	0.276		var.
	Male	3.468	3.168	3.554	3.35	mean
		0.931	0.754	0.345		var.
	Totals	3.48	3.18	3.51	3.34	mean

*n=10 per cell

Table 22--Continued

EXECUTIVE STRESS*

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 6	Female	3.538	3.028	3.212	3.24	mean
		0.333	1.000	0.388		var.
	Male	3.268	2.758	3.715	3.18	mean
		0.528	0.930	0.763		var.
	Totals	3.40	2.89	3.50	3.21	mean

YOKE STRESS*

Trial	Sex	Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 1	Female	3.016	4.798	4.415	4.08	mean
		2.235	2.459	1.090		var.
	Male	3.671	3.764	3.345	3.59	mean
		2.079	1.887	1.157		var.
	Totals	3.31	4.28	3.88	3.84	
Trial 2	Female	2.938	4.593	3.868	3.58	mean
		2.190	2.128	1.576		var.
	Male	2.901	3.898	2.862	3.22	mean
		1.816	2.523	1.407		var.
	Totals	2.92	4.25	3.37	3.40	mean
Trial 3	Female	4.759	5.048	4.700	4.79	mean
		1.246	2.420	1.471		var.
	Male	4.386	4.582	4.098	4.31	mean
		1.678	1.896	0.988		var.
	Totals	4.57	4.83	4.40	4.55	mean

*n=10 per cell

Table 22--Continued

YOKE STRESS*

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 4	Female	3.682	4.528	3.673	3.98	mean
		1.814	1.462	1.211		var.
	Male	3.132	3.771	3.693	3.53	mean
		1.458	1.719	1.210		var.
	Totals	3.41	4.18	3.68	3.70	mean
Trial 5	Female	2.902	4.058	3.190	3.38	mean
		1.676	1.417	0.718		var.
	Male	2.892	3.310	3.150	3.12	mean
		1.161	1.654	0.901		var.
	Totals	2.90	3.65	3.17	3.25	mean
Trial 6	Female	2.454	3.882	3.117	3.15	mean
		1.578	1.774	0.867		var.
	Male	2.806	3.029	2.621	2.82	mean
		1.295	1.790	1.080		var.
	Totals	2.63	3.45	2.87	2.99	mean

CONTROL STRESS*

Trial	Sex	Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 1	Female	1.841	2.187	2.014	2.01	mean
		1.377	1.414	1.230		var.
	Male	1.319	1.692	1.587	1.53	mean
		1.105	1.231	1.172		var.
	Totals	1.53	1.94	1.79	1.77	mean
Trial 2	Female	1.882	1.902	1.847	1.88	mean
		1.106	1.111	1.487		var.
	Male	1.421	1.222	1.260	1.30	mean
		1.336	1.455	0.892		var.
	Totals	1.60	1.51	1.50	1.59	mean

*n=10 per cell

Table 22--Continued

CONTROL STRESS*

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 3	Female	1.235	1.900	1.524	1.55	mean
		0.892	1.265	1.458		var.
	Male	1.603	1.002	1.282	1.30	mean
		1.973	1.466	1.086		var.
	Totals	1.41	1.45	1.40	1.43	mean
Trial 4	Female	2.050	2.700	1.688	2.15	mean
		1.586	1.440	1.421		var.
	Male	1.983	1.759	1.460	1.73	mean
		2.074	1.614	1.22		var.
	Totals	2.03	2.23	1.57	1.94	mean
Trial 5	Female	1.588	1.629	1.415	1.54	mean
		1.363	1.201	1.164		var.
	Male	1.086	1.531	0.865	1.16	mean
		1.701	1.618	0.607		var.
	Totals	1.30	1.56	1.24	1.34	mean
Trial 6	Female	2.565	2.684	1.857	2.37	mean
		1.233	1.466	1.626		var.
	Male	1.618	1.290	1.624	1.51	mean
		1.938	1.572	0.939		var.
	Totals	2.09	1.99	1.74		mean

*n=10 per cell

TABLE 23

Unweighted Means Analysis of Variance Table for
Log of Wheel Turns

Source	Df	MS	F-test
Sex	1	28.570	3.282
Nutr	2	4.863	0.559
Stress	2	659.370	75.735***
Sex x Nutr	2	6.007	0.690
Sex x Stress	2	1.454	0.167
Nutr x Stress	4	14.578	1.674
Sex x Nutr x Stress	4	7.267	0.835
Error Term	154	8.706	
Time	5	28.090	22.925***
Sex x Time	5	1.791	1.461
Nutr x Time	10	1.079	0.880
Stress x Time	10	28.115	22.945***
Sex x Nutr x Time	10	0.571	0.466
Sex x Stress x Time	10	0.779	0.636
Nutr x Stress x Time	20	1.301	1.062
Sex x Nutr x Stress x Time	20	1.677	1.369
Error Term	770	1.225	

***$p<0.001$

analyses of variances were computed; the rationale behind this was to diminish the affects of the above-mentioned large variance contributers in order to look into their shadows for possible NUTR main effects and interactions.

The EXEC's data analysis is given in Table 24. The SEX by NUTR and the TIME by NUTR interactions approached the p = 0.05 level (p = 0.051 and p = 0.094 respectively), while the TIME main effect and its interactions with SEX and SEX by NUTR were significant ($p<0.001$, $p<0.001$, and $p<0.05$ respectively). The second degree interaction indicates that

TABLE 24

Analysis of Variance Table for Executive
(EXEC) Log of Wheel Turns

Source	Df	MS	F-test
Sex	1	3.373	2.411
Nutr	2	1.141	0.815
Sex x Nutr	2	4.426	3.164
Error	54	1.399	Not Tested
Time	5	58.631	118.430***
Sex x Time	5	2.222	4.489***
Nutr x Time	10	0.814	1.645
Sex x Nutr x Time	10	0.995	2.010
Time x Unit	270	0.495	Not Tested

***$p<0.001$

the lower order interactions and the main effects should be interpreted with caution because of possible inconsistencies in the trends among their means. The strong differences seem to appear during the first two trial blocks (Figures 8 and 9) for the females among NUTR groups and between SEX groups. ZD female Ss seem to be initially the most reactive to the stress followed in reactivity by the PF and the AL females. For male Ss, the ZD Ss were somewhat less reactive than the PF or AL males. The NUTR and SEX variables are interactive with TIME probably because of the different rates, in other words the slopes of the individual groups' curves are not the same.

The YOKE analysis is listed in Table 25. Other than the strong TIME main effect, only two other effects came near the $p = 0.05$ level, the NUTR and SEX main effects ($p = 0.122$ and $p = 0.189$ respectively).

Fig. 8. Executive (EXEC) Wheel Turning Rates

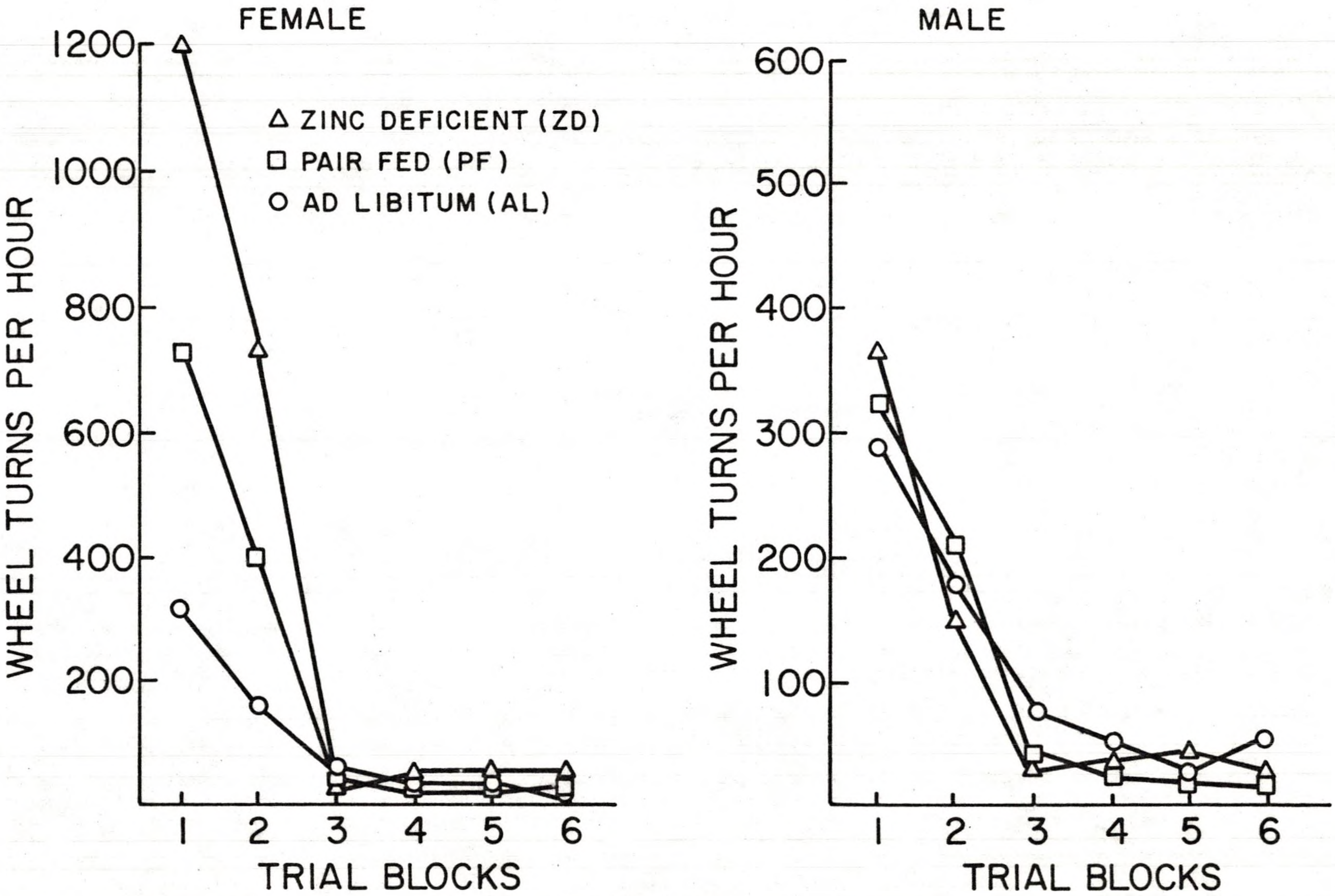

FEMALE
MALE
Δ ZINC DEFICIENT (ZD)
□ PAIR FED (PF)
○ AD LIBITUM (AL)
WHEEL TURNS PER HOUR
TRIAL BLOCKS
1200
1000
800
600
400
200
600
500
400
300
200
100
1
2
3
4
5
6

Fig. 9. Log of Executive (EXEC) Wheel Turning Rates

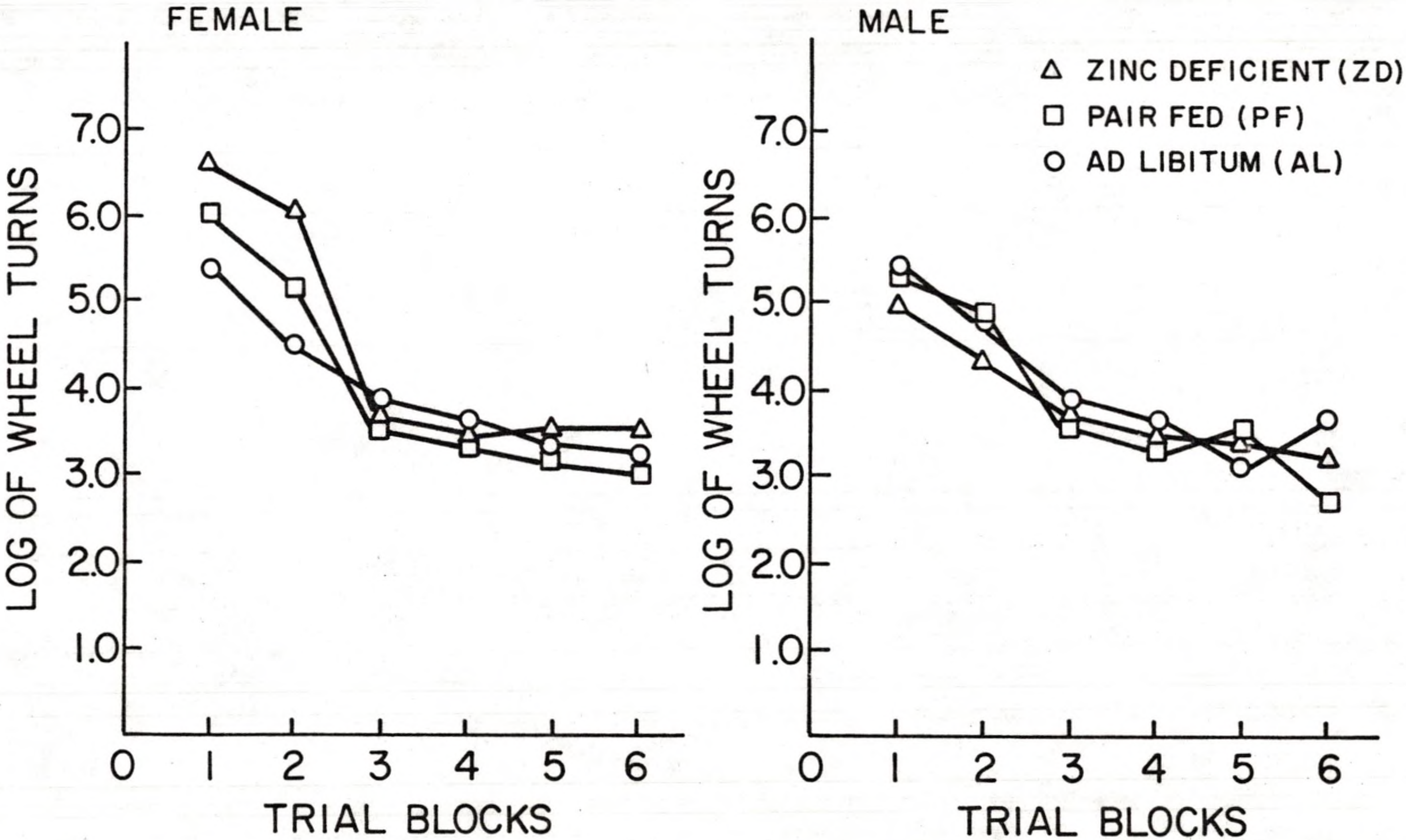

FEMALE
MALE
△ ZINC DEFICIENT (ZD)
□ PAIR FED (PF)
○ AD LIBITUM (AL)
LOG OF WHEEL TURNS
7.0
6.0
5.0
4.0
3.0
2.0
1.0
0
1
2
3
4
5
6
TRIAL BLOCKS

TABLE 25

Analysis of Variance Table for Yoke Log of Wheel Turns

Source	Df	MS	F-test
Sex	1	16.735	1.771
Nutr	2	20.713	2.192
Sex x Nutr	2	4.723	0.500
Error Term	54	9.448	
Time	5	18.729	13.615***
Sex x Time	5	0.193	0.140
Nutr x Time	10	0.708	0.514
Sex x Nutr x Time	10	1.023	0.744
Error Term	270	1.376	

***$p<0.001$

The groups means of 3.29, 4.11 and 3.56 (for ZD, PF and AL respectively) generated a significant contrast ($z = 2.21$ $p<0.05$) between the PF and ZD group means.

The CONT analysis is given in Table 26. Only the TIME main effect was significant, indicating that for the mere experience of being in the apparatus with CSs and food deprivation did not affect any group differentially.

Shock Rates

Shock rates in the six twelve-hour blocks are given in Table 27. Again the data were seriously skewed and of heterogeneous within group variances (F-max = 161.86 with k=108 and n=10). The data after the log transforms, log(x+1), log(1+log(x+1)), and log(1+log(1+log(x+1))), would not stabilize and by the time the data had reacted the third transformation

TABLE 26

Analysis of Variance Table for Control (CONT) Log of Wheel Turns

Source	Df	MS	F-test
Sex	1	22.021	2.588
Nutr	2	1.983	0.233
Sex x Nutr	2	1.482	0.174
Error Term	54	8.508	
Time	5	3.887	6.289***
Sex x Time	5	0.642	1.039
Nutr x Time	10	0.520	0.841
Sex x Nutr x Time	10	0.740	1.197
Error Term	270	0.618	

***$p<0.001$

TABLE 27

Shocks per Hour per Trial Block*

		Nutrition				
Trial	Sex	Zinc-Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 1	Female	5.58	3.53	3.98	4.36	mean
		10.32	1.85	3.24		var.
	Male	4.82	5.33	5.08	5.08	mean
		3.35	2.75	2.68		var.
	Totals	5.20	4.43	4.53	4.72	mean
Trial 2	Female	9.19	5.53	4.39	6.37	mean
		16.99	2.80	3.46		var.
	Male	5.56	5.93	5.68	5.72	mean
		3.23	2.75	3.03		var.
	Totals	7.38	5.76	5.04	6.04	mean

*n=10 per cell

Table 27--Continued

Trial	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Trial 3	Female	14.95	8.59	8.75	10.76	mean
		23.14	0.59	0.20		var.
	Male	8.43	8.39	8.34	8.39	mean
		0.714	0.55	0.82		var.
	Totals	11.69	8.49	8.55	9.57	mean
Trial 4	Female	16.67	8.74	8.85	11.42	mean
		25.11	0.38	0.15		var.
	Male	9.21	8.69	9.34	9.08	mean
		1.16	1.59	1.26		var.
	Totals	12.94	8.72	9.10	10.25	mean
Trial 5	Female	16.32	8.45	8.29	11.02	mean
		24.18	0.35	0.65		var.
	Male	8.57	8.10	8.56	8.41	mean
		0.33	1.02	0.50		var.
	Totals	12.45	8.28	8.43	9.72	mean
Trial 6	Female	14.01	8.45	8.35	10.27	mean
		16.90	0.49	0.80		var.
	Male	8.30	8.35	8.80	8.48	mean
		0.93	0.56	0.87		var.
	Totals	11.16	8.40	8.58	9.38	mean

*n=10 per cell

its accuracy had been destroyed. The reason for this problem with the data probably lies with the extreme differences in the variances complicated by a few of the variances being near zero. At this point the raw data was submitted to analysis of variance not to test for significant differences but to probe for possible trends (Table 28). The TIME and TIME by SEX effects were large. The TIME by SEX interaction is shown in Figure 10 and seems to be a function of the divergence of the two groups' curves over time; the females shock rate increased faster than did the makes shock rate.

Examination of the data of Table 27 shows a profound trend. The ZD females have without an exception the largest group means and largest variances. Group means and variances in groups other than the ZD females are generally very close, therefore, these groups were lumped together and an analysis of ZD females vs. non-ZD-females was done (Table 29 has the group means and Friedman's analysis of variance). Friedman's analysis of variance of ranks between the two groups and over the six trial blocks generated a significant ($p<0.001$, $X_r^2 = 42.0$ with df = 1) difference indicating that ranked data over all trial blocks was not random (the ZD females did in fact have higher group means than did the non-ZD female group).

Weight Data

At the beginning of the food deprivation period all Ss were weighed; the summary of these data appear in Table 30. The NUTR and SEX main effects were significant (Table 31). The AL group mean was significantly lower ($z = 4.11$ $p<0.001$ and $z = 3.05$ $p<0.01$) than the PF and ZD

TABLE 28

Analysis of Variance Table for Shock Rates

Source	Df	MS	F-test
Sex	1	818.716	0.537
Nutr	2	1236.252	0.811
Sex x Nutr	2	1298.236	0.851
Error Term	54	1524.693	
Time	5	1271.837	38.561***
Sex x Time	5	100.669	3.052*
Nutr x Time	10	47.318	1.435
Sex x Nutr x Time	10	30.915	0.937
Error Term	270	32.983	

*$p<0.05$
***$p<0.001$

TABLE 29

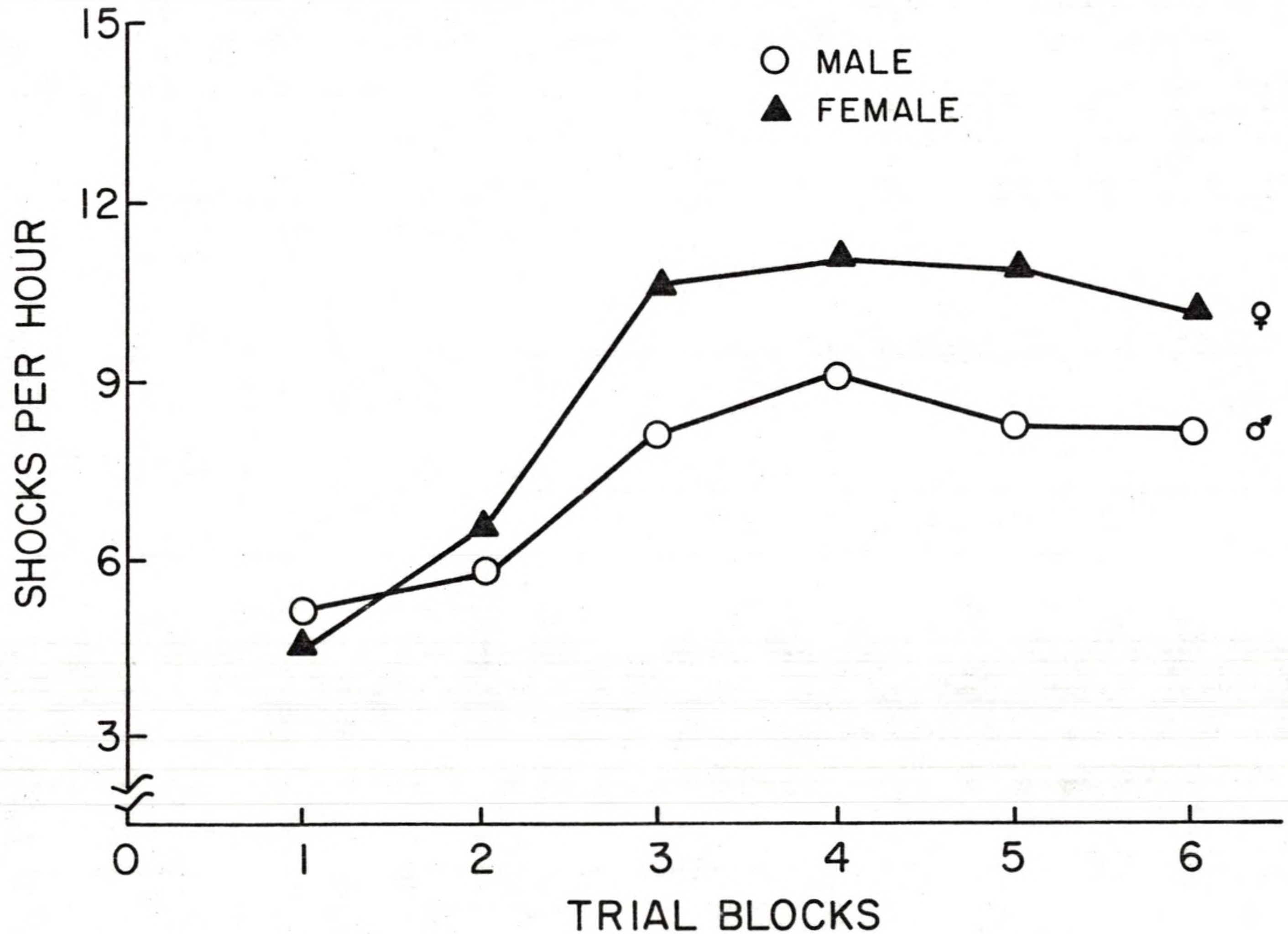
Friedman's Analysis of Variance of Shock Rates per Hour

	Zinc Deficient-Female		Non Zinc Deficient-Female	
Trial	$\bar{x}$	Rank	$\bar{x}$	Rank
1	5.58	1	4.55	2
2	9.19	1	5.42	2
3	14.96	1	8.50	2
4	16.67	1	8.97	2
5	16.31	1	8.39	2
6	14.00	1	8.45	2
		$\Sigma R = 6$		$\Sigma R = 12$

$X_R^2 = 42.00$

$p<0.001$

Fig. 10. Sex by trial interaction of shocks per hour



15
12
9
6
3
0
SHOCKS PER HOUR
O MALE
▲ FEMALE
♀
♂
1
2
3
4
5
6
TRIAL BLOCKS

TABLE 30

Pre-Food Deprivation Weights*

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	293.50	279.50	271.30	281.43	mean
		38.44	28.03	45.11		var.
	Male	451.60	470.30	421.00	447.63	mean
		56.74	45.08	48.36		var.
	Totals	372.55	374.90	346.15	364.53	mean
Yoke	Female	275.40	305.40	271.90	284.23	mean
		53.65	32.26	21.51		var.
	Male	475.30	464.90	431.30	457.16	mean
		57.56	34.50	48.40		var.
	Totals	375.35	385.15	351.60	370.70	mean
Control (Cont)	Female	299.00	290.80	264.30	284.73	mean
		43.46	32.63	21.81		var.
	Male	441.30	476.50	428.50	448.77	mean
		60.06	63.66	30.35		var.
	Totals	370.15	383.65	346.40	366.60	mean
All	Female	289.30	291.90	269.67	283.45	mean
	Male	456.07	470.57	426.93	451.19	mean
	Totals	372.68	381.23	348.05	367.32	mean

*weight in grams

TABLE 31

Analysis of Variance Table for Pre-Food Deprivation Weight

Source	Df	MS	F-test
Sex	1	1266051.000	645.404***
Nutr	2	17810.402	9.079***
Stress	2	586.033	0.299
Sex x Nutr	2	1648.537	0.840
Sex x Stress	2	321.257	0.164
Nutr x Stress	4	175.853	0.090
Sex x Nutr x Stress	4	2895.276	1.476
Unit	162	1961.640	

***$p<0.001$

group means respectively, while the PF and ZD group means were not different (z = 1.06). The male Ss' group mean was higher than that of the females' which of course is not surprising. In the following paragraphs where weight loss is considered, the above information should be kept in mind, as generally weight loss is a function of original weight of the Ss. The insignificance of other effects (STRESS and its interactions) supports the randomness of group assignments.

Weight loss during the food deprivation period is given in Table 32 and its analysis if given in Table 33. These data yielded a significant difference only in the Sex main effect, with the females losing less weight on the average than the males. The nonsignificant other effects supports the randomness of the STRESS assignments.

Weight loss during the wheel turning stress period data appears in Table 34 and its analysis appears in Table 35. The SEX and STRESS main effects as well as the STRESS and NUTR interactions with SEX

TABLE 32

Food Deprivation Weight* Loss**

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	26.500	24.200	26.500	25.73	mean
		5.017	6.941	7.735		var.
	Male	33.800	36.700	35.500	35.33	mean
		6.529	6.993	10.470		var.
	Totals	30.15	30.45	31.00	30.53	mean
Yoke	Female	25.500	27.300	27.100	26.63	mean
		6.570	5.618	6.367		var.
	Male	35.600	39.900	39.200	38.23	mean
		8.708	6.008	11.727		var.
	Totals	30.55	33.60	33.15	32.43	mean
Control (Cont)	Female	27.900	27.500	24.800	26.73	mean
		5.109	3.923	4.264		var.
	Male	35.800	37.500	32.500	35.27	mean
		6.460	7.778	8.209		var.
	Totals	31.85	32.50	28.65	31.00	mean
All	Female	26.63	26.33	26.13	26.37	mean
	Male	35.07	38.03	35.73	36.28	mean
	Totals	30.85	32.18	30.93	31.32	mean

*Grams
**n=10 per cell

TABLE 33

Analysis of Variance Table for Food Deprivation Weight Loss

Source	Df	MS	F-test
Sex	1	4420.352	85.711***
Nutr	2	33.472	0.649
Stress	2	58.822	1.141
Sex x Nutr	2	41.106	0.797
Sex x Stress	2	36.356	0.705
Nutr x Stress	4	54.706	1.061
Sex x Nutr x Stress	4	5.456	0.106
Unit	162	51.573	

***$p<0.001$

TABLE 34

Weight Loss* During Wheel Turning Stress**

Stress	Sex	Nutrition: Zinc Deficient (ZD)	Pair Fed (PF)	Ad Libitum (AL)	Totals	Statistic
Executive (EXEC)	Female	25.000	23.300	25.000	24.43	mean
		7.071	3.889	5.944		var.
	Male	47.100	47.000	41.500	45.20	mean
		9.666	11.314	9.095		var.
	Totals	36.05	35.15	33.25	34.82	mean
Yoke	Female	23.900	24.000	30.700	26.20	mean
		6.557	2.944	16.439		var.
	Male	46.300	45.800	37.500	43.20	mean
		4.809	6.215	8.436		var.
	Totals	35.10	34.90	34.10	34.70	mean
Control (Cont)	Female	21.000	20.900	22.700	21.53	mean
		3.091	3.542	3.434		var.
	Male	29.500	30.500	30.900	30.30	mean
		4.197	6.770	7.295		var.
	Totals	25.25	25.70	26.80	25.92	mean
All	Female	23.30	22.73	26.13	24.06	mean
	Male	40.97	41.10	36.63	39.57	mean
	Totals	32.13	31.92	31.38	31.81	mean

*Grams
**n=10 per cell

TABLE 35

Analysis of Variance Table for Weight Loss During Stress

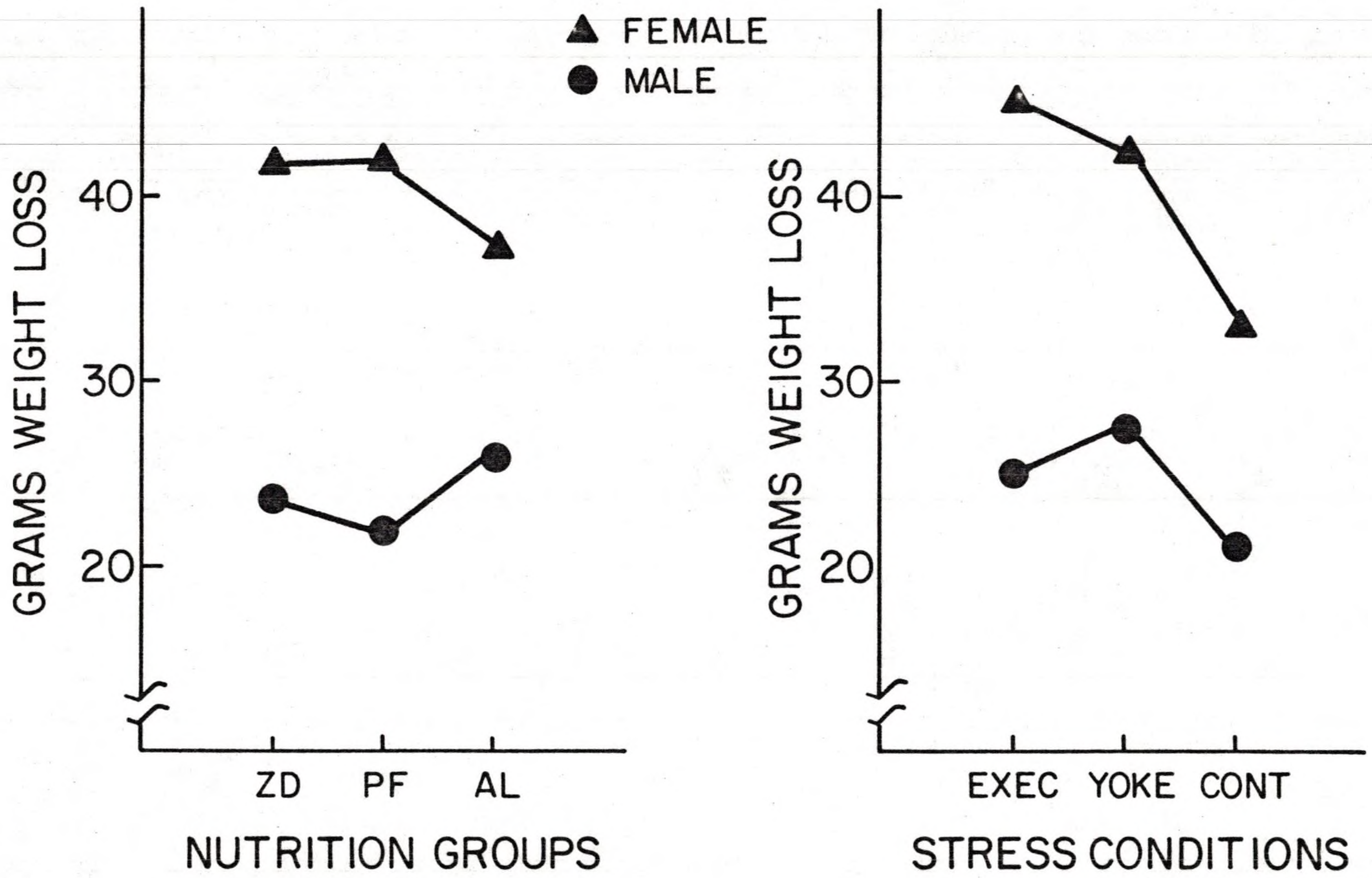
Source	Df	MS	F-test
Sex	1	10826.773	192.961***
Nutr	2	8.939	0.159
Stress	2	1563.706	27.869***
Sex x Nutr	2	284.340	5.068**
Sex x Stress	2	564.941	10.069***
Nutr x Stress	4	25.122	0.448
Sex x Nutr x Stress	4	90.222	1.608
Unit	162	56.109	

**$p<0.01$
***$p<0.001$

were significant. As would be expected the heavier male Ss lost more weight than did the female Ss. The EXEC and YOKE group means were not significantly different (z = 0.088), while both of these group means were significantly higher (they had larger weight losses) than that of the CONT group's (z = 6.50 and 6.41 respectively, $p<0.001$ for both).

The two interactions are graphed in Figure 11. The SEX by NUTR interaction seems to derive from a smaller difference between male and female AL Ss than is evident in the other NUTR groups. The SEX by STRESS interaction seems to be a function of a gradually decreasing SEX difference as one looks from EXEC to YOKE to CONT group means, possibly indicating that male Ss were more reactive to the STRESS than were the female Ss.

Fig. 11. Sex by nutrition and sex by stress interactions for weight loss during the stress treatments


FEMALE
MALE
GRAMS WEIGHT LOSS
40
30
20
ZD
PF
AL
NUTRITION GROUPS
GRAMS WEIGHT LOSS
40
30
20
EXEC
YOKE
CONT
STRESS CONDITIONS

CHAPTER IV

DISCUSSION

Before discussing the results of the previous chapter, Table 36 is offered as a concise summary. The boolean expressions in Table 36 seem to indicate few trends when all variables are considered at once. However, four different trends emerge when the following comparisons are made between: lower stomach pathology length and number; upper stomach pathology length and number; shock rate and wheel turning rate; and water consumption and weight loss. These four trends involve both biological and behavioral variables which are systematically influenced by the nutritional variables.

Lower Stomach Pathology

Although zinc deficient males were expected to have significantly more stomach pathology than normal males this hypothesis was not supported. This finding is inconsistent with the bulk of zinc literature; zinc deficient males have been shown to be more emotional in open field situations (Caldwell et al., 1970; Peterson, 1974), and slower maze learners (Halas et al., 1976). These findings would suggest that zinc deficient males should be more susceptible to pathology than normal males. On the other hand, zinc deficient females did not show similar behavioral deficits and therefore they were not expected to be pathology

TABLE 36

Summary of the Results

LOWER STOMACH PATHOLOGIES

A. Length

1. EXEC > YOKE > CONT
2. FEMALE > MALE
3. ZD-MALE < others

B. Number

1. EXEC > YOKE > CONT
2. FEMALE > MALE
3. others > ZD-MALE

UPPER STOMACH PATHOLOGIES

A. Length

1. CONT > YOKE ≃ EXEC
2. EXEC
    a. FEMALE [ZD > PF ≃ AL]
    b. MALE [ZD > PF ≃ AL]
3. YOKE
    a. FEMALE [PF > ZD > AL]
    b. MALE [PF > ZD ≃ AL]
4. CONT
    a. FEMALE [AL ≃ PF > ZD]
    b. MALE [PF > AL ; PF ≃ ZD ; AL ≃ ZD]
5. PF > ZD ≃ AL

B. Number

1. CONT > YOKE ≃ EXEC
2. EXEC
    a. FEMALE [ZD ≃ PF > AL]
    b. MALE [ZD > PF ≃ AL]
3. YOKE
    a. FEMALE [PF > ZD > AL]
    b. MALE [PF > ZD ≃ AL]
4. CONT
    a. FEMALE [AL ≃ PF > ZD]
    b. MALE [not consistent]

Table 36--Continued

SHOCK RATES

A. ZD-FEMALE > others

WHEEL TURNING RATES

A. EXEC

1. NUTR
   a. trials (1-2) ZD > PF > AL
   b. trials (3-6) not consistent
2. SEX
   a. trials (1-2) FEMALE > MALE
   b. trials (3-6) not consistent
3. SEX by NUTR
   a. trials (1-2) ZD-FEMALE > others
   b. trials (3-6) AL-MALE > others

B. YOKE

1. PF-FEMALE > others
2. PF > ZD ≃ AL
3. FEMALE > MALE

C. CONT not consistent

WATER CONSUMPTION

A. EXEC & YOKE

1. FEMALE [AL > PF > ZD]
2. MALE [AL > ZD > PF]

B. CONT

1. FEMALE [ZD > AL > PF]
2. MALE [PF > ZD > AL]

C. MALE > FEMALE

D. EXEC > YOKE > CONT

WEIGHT LOSS

A. EXEC

1. FEMALE not consistent
2. MALE [ZD ≃ PF > AL]

Table 36--Continued

WEIGHT LOSS

B. YOKE

1. FEMALE [ZD ≃ PF < AL]
2. MALE [ZD ≃ PF > AL]

C. CONT not consistent

D. MALE > FEMALE

E. EXEC ≃ YOKE > CONT

STRESS	NUTR=NUTRITION
EXEC=executive	ZD=zinc deficient
YOKE=yoked stress	PF=pair fed
CONT=controll stress	AL=ad libitum

prone. The behavioral deficits of zinc deficient females are different from those of zinc deficient males and will be discussed later. As an explanation for the negative results among the zinc deficient males, it is proposed that the zinc deficient males are a physically select group of animals, that is, a group that was culled by the zinc deficiency early in life. This hypothesis is supported by the mortality rate among the three nutritional groups. The zinc deficient litters suffered mortality rates three times greater than that of the normal litters (ZD = 27.95%; PF = 12.01%; AL = 9.3%). Variance terms of the mortality measure were even more divergent (ZD = 1753.68; PF = 130.18; AL = 108.13). These figures are well in line with other studies (Rowe, 1974). If mortality was lopsided in the zinc deficient group (i.e., higher mortality for males than females) then this could have accounted for the lower stomach pathology effect. Variance terms, particularly for pathology

number, were much smaller for the zinc deficient male group which adds further support to the suspected culling effects, in that, the zinc deficient males were a more homogeneous group than the other groups. Future studies might well look into the SEX by NUTR interactive effect on mortality.

Among females, zinc deficiency and undernourishment (pair feeding) did not increase the incidents of lower stomach pathology. These results support previously published data (Halas et al., 1976). Although there were no differences between nutritional groups, there was a difference between males and females. The males in all three nutritional groups had more pathology than their female littermates.

The lack of differences in lower stomach pathology among the three nutritional groups was not due to a failure of the stress variable. EXEC Ss in all three nutritional groups had more lower stomach pathology than YOKE and CONT Ss which is the classical observation in the stress literature and gives validity that the stress treatment was effective.

Upper Stomach Pathology

Since stress tends to produce pathology primarily in the lower stomach and duodenum, most investigators tend to ignore those few lesions that may occur in the upper stomach. However, in the present study, it was decided to measure and record the lesions that were found in the upper stomach. No predictions were made as to which nutritional or STRESS group would be more susceptible to pathology, but it was thought that the zinc deficient males would have more pathology than the

normal rats and executive Ss would have more pathology than Ss of less stressed groups.

Among EXEC and YOKE females, the AL Ss had less pathology than the ZD and PF Ss. Among males, the EXEC stress condition Ss broke along the zinc deficiency line, with ZD Ss having more pathology than either the undernourished (PF) or normal nourished (AL) Ss; whereas in the YOKE stress condition, the PF Ss had somewhat more pathology than either the ZD or AL Ss. The higher incidents of upper stomach pathology among malnourished rats is interesting because it suggests that normal rats, like normal humans, develop lesions in the lower stomach when they are subjected to stress. The data from this study suggest that malnutrition, especially zinc deficiency, may increase pathology incidence in the upper stomach over the incidence seen in normal nourished (AL) Ss when animals are subjected to stress. The obvious question is whether humans who suffered malnutrition have more upper stomach lesions than normal humans. As of now there is no answer to this question.

Wheel Turns and Shock Rates

During the first two trial blocks, the EXEC zinc deficient females had significantly more wheel turns than either the undernourished (PF) or normal (AL) females (Figure 8a, b). The undernourished females had more wheel turns than the normal females but the results did not reach statistical significance. These differences disappeared during the final four trial blocks. The EXEC male zinc deficient rats had more wheel turns than the undernourished males who in turn had more wheel turns than normal males but the results failed to reach the 0.05

significance level. Nevertheless the results of the zinc deficient and undernourished males paralleled those of their female littermates.

The wheel turns of the undernourished (PF) females in the YOKE stress group were significantly different from that of the zinc deficient or normal females. These results are intriguing in that undernourished males are significantly different in aggression than either the normal or zinc deficient males (Halas, Reynolds & Sandstead, 1977). What is not known is why rats who were undernourished behave differently from rats who were zinc deficient or were adequately fed. It is obvious that zinc deficiency and undernutrition (PF) produce different kinds of aberrant behavior in male and female rats.

When comparisons were made between male and female rats, it was found that females had more wheel turns than their male littermates during the first two trial blocks, but this difference disappeared in the last four trial blocks. This seems to indicate that females reacted more emotionally to stress than their male littermates in the avoidance situation but did not react differently under the conflictual shock avoidance phase.

Shock rates demonstrated a trend similar to that of wheel turns. The shock rates of zinc deficient females exceeded that of the undernourished and adequately fed females. Unlike wheel turns, group mean and variance differences continued throughout the entire six trial blocks. No such differences existed among the male groups. This behavioral data supports the upper stomach data in that females may survive prenatal malnutrition but cannot tolerate stress. On the other hand,

males do not survive prenatal malnutrition very well but those few males who do survive were able to tolerate stress reasonably well.

Overall, intergroup differences in shock rates and wheel turning rates were greatest during the first two trial blocks. This effect may be due to the nature of the task, straight avoidance, during the first two trial blocks as compared to conflictual shock avoidance during the remaining four trial blocks. The increased state of physical exhaustion may also play a role in the later trials. During the first trial block, zinc deficiency had a significant effect on female Ss as measured by both shock and wheel turning rates. This zinc deficient effect is in agreement with the research on emotionality in that others have found that zinc deficient rats were more emotional in settings such as the open field (Caldwell et al., 1970; Peterson, 1974).

These adverse effects were observed only among zinc deficient and undernourished rats who were subjected to stress. Those zinc deficient and undernourished rats who were not subjected to stress (CONT Ss) did not exhibit these aberrant behaviors nor did they have unusual lower stomach pathologies. Rehabilitated adult rats, who suffered prenatal malnutrition, usually appear and behave normally until some behavioral demand is made. Then the effects of the prenatal malnutrition will often times appear.

Water Consumption and Weight Loss

There was a marked similarity in trends between water consumption and weight loss at the STRESS and SEX main effect levels. In general, the more water consumed the greater the weight loss. The

nutrition variable had no effect on the water consumption or weight loss. The SEX effect, the lighter females drank less water and lost less weight than the heavier males. In the STRESS effect, the EXEC rats drank more water and lost more weight than YOKE rats who in turn drank more water and lost more weight than CONT rats. These two variables are responsible for most of the variance associated with weight loss and water consumption.

Conclusions

In future research the male-female dimension should be studied in relationship to both zinc deficiency and undernutrition. If it can be shown that the zinc deficient male is indeed culled during the first week of life, then this would have a profound influence on any experiment attempting to study the effects of zinc deficiency. Lower and upper stomach pathology in this study as well as the avoidance conditioning data presented elsewhere (Halas et al., 1976; Rowe, 1974) support this proposed culling. The actuarial study of pup mortality in relation to sex and nutrition is urged.

Other forms of stress should also be used to determine whether the present findings can be generalized to other forms of stress or whether the results presented here are unique to this particular mode of stress.

The wheel turning reaction to stress among the zinc deficient females was the most stunning result of this study. This finding and the results of other studies suggest that zinc deficiency has widespread adverse effects on the biological and behavioral systems of rats.

However, these adverse effects are not the same for male and female rats. Zinc deficient male rats do poorly in avoidance conditioning while zinc deficient females perform as well as normal females (Halas et al., 1976). Several aggression experiments (Halas et al., 1977) reported that zinc deficient female rats were more aggressive than normal rats while zinc deficient males were no more aggressive than normal male rats. Undernourished males were less aggressive than normal males but undernourished females were more aggressive than normal females. A recent study found that zinc deficient males were more aggressive than normal males (Peters, in press). These differences between males and females could be due to a higher mortality rate among male pups or due to differential effects of zinc deficiency and undernutrition on survivors in each sex. There is data to support both hypotheses and it is obvious that these issues are in an early stage of investigation.

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