

**THE EFFECT OF CHRONIC AND ACUTE
INSULIN-INDUCED HYPOGLYCEMIA ON SHOCK
ELICITED AGGRESSION**

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THE EFFECT OF
CHRONIC AND ACUTE INSULIN-INDUCED
HYPOGLYCEMIA ON SHOCK ELICITED AGGRESSION

An Abstract
Presented to
the Graduate Council of
Austin Peay State University

In Partial Fulfillment
of the Requirements for the Degree
Master of Arts

by
Elaine C. LaZizza Cronin

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ABSTRACT

Previous research has indicated that lowered blood-sugar levels (hypoglycemia) are associated with higher levels of aggression. The present experiment was designed to investigate this correlation in a shock-elicited aggression situation by replicating and extending an earlier study (Neideffer, Davis, & Travis, Note 1). Six groups of rats were randomly assigned to Chronic-Insulin, Acute-Insulin, Chronic-Phenol, Acute-Phenol, Chronic-Distilled Water, and Acute-Distilled Water treatments. Each subject received 200 1.5 mA shocks of 300 msec duration during aggression testing. The number of aggressive responses made by each subject was recorded automatically. The results lent further support to the correlation linking hypoglycemia with aggression.

THE EFFECT OF
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A Thesis

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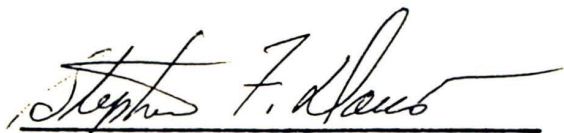
by

Elaine C. LaZizza Cronin

June 1978

To the Graduate Council:

I am submitting herewith a Thesis written by Elaine LaZizza Cronin entitled "The Effect of Chronic and Acute Insulin-Induced Hypoglycemia on Shock-Elicited Aggression." I recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Psychology.

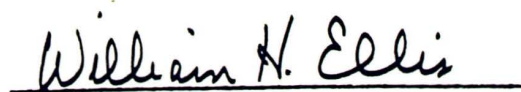

Major Professor

We have read this thesis and
recommend its acceptance:


Second Committee Member


Third Committee Member

Accepted for the Council:


Dean of the Graduate School

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CHAPTER I

INTRODUCTION

A possible correlation between blood-sugar level and aggression has been suggested by various subjective reports from a number of mental health workers. Specifically, the relationship suggests that lowered blood-sugar levels (hypoglycemia) accompany higher levels of aggression. Some support for this contention may be found in the literature. For example, Etheridge (1967) reported symptoms of anxiety, uncontrolled outbursts and aggression in his hypoglycemic patients. Similarly, Zivin (1970) reported symptoms such as restlessness, agitation, and irritability, in the emotional functioning of hypoglycemics. Based on similar observations, Moyer (1971) concluded:

Hypoglycemia, from whatever cause, is, in many cases, associated with tendencies to hostility and is another dysfunction in the blood chemistry which evidently sensitizes the neural substrates for aggression. (100)

Perhaps the most extensive human studies supportive of this contention are those dealing with the Quolla, a Latin American Indian tribe. In an observational study, Trotter (1973) reported that at least 40% of the tribal population could be classified as hypoglycemic.

This is particularly relevant when one considers that the Quolla are one of the most violently aggressive people in existence. In contrast to the tribe's behavior, Bolton (1973) reports that the ethical code of the Quolla stresses compassion and cooperation while disapproving of violence. Yet, available data showed that not a single country has a homicide rate equal to that of the Quolla. Moreover, the correlation between aggression and hypoglycemia is not limited to overt acts of aggression. Bolton (1976) further tested the Quolla for expression of fantasized hostility. Fantasized aggression expressed by hypoglycemic subjects was more than double that of subjects with normal blood-sugar levels.

A recent study conducted by Neideffer, Travis, Davis, Voorhees and Prytula (1977) experimentally tested the assumption that lower blood-sugar level is related to higher aggressiveness. Four groups of rats served as subjects. Three groups had their blood-sugar levels experimentally reduced by injections of regular U-40 zinc insulin. Dosages were single injections of 4-, 8-, and 12-units reflecting low, moderate, and high blood-sugar reductions, respectively. Subjects in the fourth group (control) were injected with Phenol,

which is used as the carrier in insulin preparations. Following injection, each subject was tested in a shock-elicited aggression situation similar to that used by Azrin, Rubin, and Hutchinson (1968). More specifically, the restrained rats were given unavoidable tail shocks of 1.50 mA intensity with a 30-msec duration every three seconds for 10 minutes. The results showed that aggression toward an inanimate target was inversely related to the size of the insulin injection, thus implying a causal relationship between hypoglycemia and aggression.

Neideffer, Davis and Travis (Note 1) have also recently reported data comparing the effects of chronic and acute hypoglycemia on aggressive behavior. In this study, 28 rat subjects were assigned to one of four groups: insulin-chronic (I-C), insulin-acute (I-A), Phenol-chronic (P-C), and Phenol-acute (P-A). Insulin-chronic subjects received two 6-unit injections, one every 12 hours, of regular zinc insulin daily for 29 days before aggression testing. Phenol-chronic subjects received two 6-unit Phenol injections, one every 12 hours, daily for the same period of time prior to testing. Groups I-A and P-A received two 6-unit insulin and two 6-unit Phenol injections, one every 12 hours, respectively, only on the day of testing. All subjects were tested in the

shock-elicited aggression situation. Each subject was given 200 1.5 mA shocks of 300 msec duration at three-second intervals. Aggression recordings were made at the end of the first and last five minutes of testing. Results showed that Group I-C was significantly more aggressive than the other three groups at the end of the first five minutes. By the end of the last five-minute period Groups I-C, I-A, and P-A were significantly more aggressive than Group P-C. Thus, the results of this experiment were generally supportive of the suggested relationship between blood-sugar level and aggression.

However, these data also raised several questions. For example, a substantial drop in performance was shown by Group P-C from the end of the first five-minute period until the end of the testing session. No such decrease was shown by Group P-A. Why a control group's performance would decline to this degree is certainly problematic and indicates the need for further investigation. Also, dosage-administration procedure may have affected the behavior of Group I-A, whose performance was similar to the P-A control group. As Group I-A received two 6-unit dosages separated by 12 hours, the effects of the first injection may have dissipated by the time the second injection was administered. Hence, the functional

dosage for this group would have been six units, not 12. This being the case, Group I-A would be expected, based on the Neideffer et al. (1977) data, to show aggressive behavior similar to that of control subjects rather than subjects with more substantial blood-sugar reduction.

The present study was designed to replicate the previous findings relating chronic insulin-induced hypoglycemia and shock-elicited aggression, and to address some questions raised by the Neideffer et al. (Note 1) study. To investigate the similar performance of Groups I-A and P-A, one daily 12-unit injection was administered to avoid dissipation. The substantial drop in performance by Group P-C was studied by the addition of a new control group to which injections of distilled water were administered.

CHAPTER II

METHOD

Subjects

Forty-two male albino rats purchased from the Holtzman Company, Madison, Wisconsin, served as subjects. All subjects were individually caged with food and water available on a free-feeding basis. The subjects were approximately 90 days old upon arrival at the laboratory, and 120 days old at the time of shock testing.

Apparatus

Testing was conducted in a shock-elicited aggression apparatus. The apparatus consisted of a white plastic tube with one end enclosed, measuring 21.5 cm in length and 7.5 cm in diameter. This device was mounted on a sheet of plexiglass which, in turn, was mounted on a wooden platform. Both the tube and plexiglass sheet were removeable to allow proper placement of the subject as well as cleaning of the tube between trials. At the enclosed end of the tube was a 1.5 cm hole through which the subject's tail was extended and fastened, with adhesive tape, to a wooden restraining rod 2.5 cm in diameter. Two pieces of copper wire permanently affixed to the

wooden rod 7.0 cm apart served as tail electrodes. When the rod was taped to the subject's tail it served as a restraining device as well as an electrode carrier. A 1.5 mA pulsating dc current was used to shock subjects during test sessions. A Jackson (Model 5-J-2) mA meter was used to monitor shock intensity.

The open end of the restraint tube faced a Lafayette omnidirectional lever (Model 80111) which was lightly taped with adhesive tape for easier subject grasp when the tube was in place. The lever was perpendicular to the open end, extending across mid-portion. The omnidirectional lever functioned as an aggression target for the test subject. When attacked the target activated a Lafayette (Model 5707 PS) impulse counter allowing the number of aggressive responses to be recorded. The apparatus was enclosed on top, end, and sides by a cardboard box to reduce external stimulation which might distract the subject.

Procedure

Twenty-nine days prior to aggression testing, six equal groups ($n=7$) were formed. On each of the 28 days preceeding and the day of testing itself, subjects in the Chronic Insulin (C-I), Chronic Phenol (C-P), and

Chronic Distilled Water (C-DW) groups received one daily 12-unit, subcutaneous injection of regular U-40 zinc insulin, .06% Phenol, and distilled water, respectively. Subjects in the Acute Insulin (A-I), Acute Phenol (A-P), and Acute Distilled Water (A-DW) groups received one 12-unit, subcutaneous injection of regular U-40 zinc insulin, .06% Phenol, and distilled water, respectively, only on the day of testing. Individual test sessions were conducted by removing the designated subject from its home cage one hour after receiving the appropriate injection. The order for running subjects was randomized. Injections were administered at staggered intervals so each subject would have an individual testing session. After recording its weight, the test subject was secured in the restraining tube and allowed five minutes to habituate to the apparatus. Following habituation, a 10-minute shock period began. During this period, each subject received 200 1.5 mA shocks of 300 msec duration, i.e., shocks were applied at three second intervals¹. The number of aggressive responses, attacks on the target rod, was recorded at two-minute intervals and at the end of the first five-minute interval.

CHAPTER III

RESULTS

Prior to analysis, all response scores were converted to $\log_{10} (X_i+1)$ scores in order to satisfy the mathematical assumption of normality of distribution. Group means for the entire ten-minute test session are shown in Figure 1. As visual inspection of the two-minute segment revealed several instances of no responding, analyses were performed only on the two five-minute segments and the entire ten-minute session. Results of these analyses will be presented separately.

First Five-Minute Segment

Analysis of variance yielded a significant type of injection effect, $F(2,36) = 9.21, p < .01$. Table 1 summarizes this analysis of variance. Subsequent evaluation of this effect (Neuman-Keuls procedure) indicated that the insulin subjects were significantly ($p < .01$) more aggressive than either the Phenol or Distilled Water subjects, which did not differ from each other.

Second Five-Minute Segment

As with the analysis of the first five-minute segment, a significant type of injection effect,

$F(2,36) = 7.15$, $p < .01$, was produced by the analysis of variance. This analysis of variance is summarized in table 2. Likewise, the Neuman-Keuls test indicated that the Phenol and Distilled Water subjects did not differ significantly from each other, but were significantly, ($p < .01$) less aggressive than the subjects tested under the insulin condition.

Complete Test Session

Paralleling the results of the two five-minute segments, analysis of variance performed on the data from the entire test session yielded a significant type of injection effect, $F(2,36) = 9.41$, $p < .01$. Table 3 summarizes this analysis. Similarly, the Neuman-Keuls tests indicated that the Phenol and Distilled Water treatments did not differ from each other, but were significantly ($p < .01$) less aggressive than the insulin subjects.

CHAPTER IV

DISCUSSION

The most striking results of the present experiment are the high levels of responding displayed by Groups C-I and A-I (see Figure 1). The high level of responding shown by Group C-I compliments that shown by the comparable group in the Neideffer et al. (Note 1) experiment and indicates that adaptation to the repeated lowering of blood-sugar level did not occur under the injection procedure used in the present experiment.

The high level of responding shown by Group A-I in the present study is certainly in agreement with the data reported by Neideffer et al. (1977), but does not corroborate the Neideffer et al. (Note 1) data. Differences in injection procedure might be responsible for the lack of aggressive responding shown by the Acute-Insulin subjects in the Neideffer et al. (Note 1) experiment. It will be recalled that the use of two 6-unit injections in that study raised the possibility of dissipation of the drug state prior to testing. The single 12-unit injection procedure was used in the present study to preclude such dissipation. The data indicate that it was successful.

Unlike the study by Neideffer et al. (Note 1), no drop in performance was manifested by the Chronic Phenol (Group C-P) subjects. This discrepancy may again be explained by appealing to the difference in injection administration procedure between the two studies. In the earlier study, the chronic subjects were injected twice a day for 29 days (i.e. 58 injections total). On the other hand, only 29 (total) injections were given in the present study. Thus, the concern over possible debilitation due to chronic Phenol injection raised by the Neideffer et al. (Note 1) experiment may have been due simply to chance or it may have been an artifact created by the particular injection procedure used.

As the Phenol and Distilled Water Groups did not differ from each other throughout the experiment, it would seem reasonable to suggest that Phenol may continue to be employed as a control injection. However, its use as a control would appear to be best exercised under the injection procedure employed in the present study.

It may be concluded that the present study has lent support to the negative correlation between lowered blood-sugar levels and increased aggression. However,

there is still much research to be done in this area. Although the present results indicate that the rat does not adapt to chronic lowering of blood-sugar level, these results may not adequately mirror the "real" state of affairs. An organism suffering from hypoglycemia may, indeed, experience numerous drops in blood-sugar level. However, it does not appear reasonable to suggest that each drop will be of exactly the same magnitude. A longitudinal study in which variable levels of insulin dosage are employed could yield information concerning responding under the non-equivalent decrease condition. Also, investigations employing another type of aggression task or situation, such as the paired-animal foot shock task, would be most beneficial in aiding further generalization.

REFERENCE NOTE

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FOOTNOTE

Intensity of shock has also been shown to be an important variable in eliciting aggressive response. Davis, Tramill, Voorhees, Mollenhour, and Prytula (1977) examined the relationship between shock modality and aggression. Rats were divided into three groups, each group receiving ac, half-wave (pulsating) dc, and full-wave (filtered) dc shock, respectively in the shock-elicited aggression situation. The results showed that half wave dc shock yielded significantly more aggressive responses than did ac or full-wave dc shock.

APPENDIX 1: TABLES

TABLE 1
 SUMMARY OF ANALYSIS OF VARIANCE
 FIRST FIVE-MINUTE RESPONSE PERIOD

Source	SS	df	MS	F
Acute vs. Chronic (A)	.01	1	.01	.04
Injection Type (B)	.68	2	2.34	9.21*
AxB	.52	2	.26	1.02
W. Cell	9.15	36	.25	
Total	14.34	41		

* $p < .01$

TABLE 2
SUMMARY OF ANALYSIS OF VARIANCE
SECOND FIVE-MINUTE RESPONSE PERIOD

Source	SS	df	MS	F
Acute vs. Chronic (A)	.12	1	.12	.46
Injection Type (B)	3.70	2	1.80	7.15*
AxB	.14	2	.07	2.77
W. Cell	9.31	36	.26	
Total	13.27	41		

* $p < .01$

TABLE 3
SUMMARY OF ANALYSIS OF VARIANCE
ENTIRE RESPONSE PERIOD

Source	SS	df	MS	F
Acute vs. Chronic (A)	.04	1	.04	.13
Injection Type (B)	5.33	2	2.67	9.41*
AxB	.23	2	.11	.40
W. Cell	10.21	36	.28	
Total	15.81	41		

* $p < .01$

APPENDIX 2: FIGURE

Figure 1: Group Mean Aggressive Responses

