Chapter 13

SOME EFFECTS OF CARISAPRODOL ON PAIN REACTIVITY

R. LYNN, Ph.D. and H. J. EYSENCK, Ph.D.*
(University of Exeter and University of London)

The experiment reported here was designed to test certain hypotheses relating to the effects of a new drug, Carisaprodol, on pain reactivity and various other psychological test responses; we were also interested in discovering how these responses would be related to personality. Our predictions were made on the assumption that Carisaprodol was acting as a C.N.S. depressant, an assumption made reasonable because of the close affinity of Carisaprodol to meprobamate (Berger *et al.*, 1959; Miller, 1959).

Subjects

Thirty subjects were tested in all; these were student volunteers at the University of Exeter. Each subject was tested three times: (a) under drug conditions, having been given a dose of 350 mg of Carisaprodol one hour before the beginning of the test; (b) under placebo conditions, having received a placebo indistinguishable from the drug; and (c) under "no drug" conditions. Five subjects were randomly selected to be tested under each of the six possible sequence conditions of a, b, and c.

Method

The subject was seated in an arm chair, his palms were cleaned with surgical spirit and the electrodes of the galvanometer were fixed to each palm with electrode jelly. Two minutes were allowed to elapse for the reading to become steady, and the resting level was then recorded (RL) (score in microamps). He was then told that his reaction to pain was going to be investigated and he was shown the thermal heat dolorimeter (Beecher, 1959) which stood immediately next to his chair. He was told that he would be asked to place the under side of his wrist on top of the aperture and the heat would be switched on. He was to keep his wrist on the aperture until he felt the prick of pain and then he should remove it. The intensity of the heat was set at 176 watts and pain was typically felt after about 6 sec. Two measures were taken from the PGR; the Anxiety Reaction

* We are indebted to the Wallace Laboratories for a grant which made this study possible, as well as for supplies of Carisaprodol ("Soma".)

(A) is the amount of increase in conductance as measured by the increase in microamps on the galvanometer scale, from the resting level to the reading when the subject places his wrist on the aperture of the thermal heat apparatus. The second measure was the Pain Reaction (P), the reading when pain was felt minus the resting level.

The subject was then instructed to lie back in the arm chair and close his eyes and his "orienting reflex" was extinguished. The stimulus was a mallet striking a block of wood; this stimulus was presented at 30 sec intervals until there was no PGR for three successive trials. The score recorded was the number of trials before the orienting reflex was extinguished (E).

He was then tested on the rotating spiral test (S) with 30 sec fixation (Eysenck, 1957). Finally he was tested for grip persistence (Costello and Eysenck, 1961). His maximum grip on the dynamometer was taken first (GM) and he was then asked to hold the pointer at half his maximum for as long as he could. His-persistence score was recorded in seconds (GT). In addition to these tests, each subject was given the Maudsley Personality Inventory (Eysenck, 1959).

Predictions

No prediction was made with respect to the resting level, as this measure was introduced merely in order to obtain a baseline from which to measure the changes in conductivity expected to result from the stimuli used to obtain scores A and P. We anticipated that the drug would *lower* the Anxiety and Pain responses, would *speed up* Extinction, *decrease* the duration of the spiral after-effect, and *increase* persistence.

Results

Mean scores are given in Table 1. It will be seen that with the exception of the persistence test all our expectations are, in fact, borne out. It is interesting to note that placebo reactions are intermediate between Drug and No Drug conditions; it is almost as if the placebo under these experimental conditions acquired some of the properties of the drug used. This is possibly due to the presence of certain features in the experiment which permit conditioning to take place, by pairing drug administration with depressant effects *before* the placebo is administered; such conditioning is, of course, only possible in certain groups of subjects, not in those in whom the placebo trial precedes the drug trial. An experimental investigation of this possibility would be of considerable interest.

Analyses of variance were carried out on all the data, and analyses of covariance on A and P, with Resting Level held constant. Furthermore, transformations of the skin resistance data were undertaken along the lines indicated by S.B.G. Eysenck (1956). These analyses will not be reported in detail as in none of them did the results achieve an acceptable degree of significance. It would appear reasonable to conclude that while Carisaprodol appears to have the predicted effects on all but one of the tests used, the dose chosen was too slight to make these effects sufficiently

TABLE 1
Scores

Test	*Drug*	*No Drug*	*Placebo*
Resting Level	32·77	29·73	31·33
Anxiety Reaction	4·63	5·27	4·50
Pain Reaction	9·37	11·27	10·20
Extinction	8·70	11·13	10·30
Spiral After-Effect	11·90	13·13	12·67
Grip; Maximum	65·63	66·03	65·27
Grip; Persistence	39·73	44·27	43·43

strong to overcome the very great variability in response to the tests which characterized our sample.

TABLE 2

N × RL_1	= —0·1799	E × RL_1	= 0·11998
RL_2	= 0·0587	RL_2	= 0·1308
(−) RL_3	= —0·1570	(+) RL_3	= 0·3677
N × A_1	= 0·2197	E × A_1	= 0·0013
A_2	= 0·2557	A_2	= —0·3070
(+) A_3	= 0·0702	(−) A_3	= —0·1230
N × P_1	= 0·0493	E × P_1	= —0·1387
P_2	= 0·3708	P_2	= —0·3372
(+) P_3	= 0·1365	(−) P_3	= —0·1757
N × Ex_1	= —0·1630	E × Ex_1	= 0·1971
Ex_2	= —0·3517	Ex_2	= 0·0808
(−) Ex_3	= —0·1388	(−) Ex_3	= —0·1014
N × S_1	= 0·0839	E × S_1	= —0·3373
S_2	= 0·2100	S_2	= —0·2994
(+) S_3	= 0·1863	(−) S_3	= —0·2453
N × GM_1	= —0·1770	E × GM_1	= —0·0222
GM_2	= —0·1065	GM_2	= 0·0087
(?) GM_3	= —0·1561	(?) GM_3	= —0·0007
N × GT_1	= —0·2152	E × GT_1	= 0·0631
GT_2	= —0·2616	GT_2	= 0·2817
(−) GT_3	= —0·2786	(+) GT_3	= 0·1732

It seemed relevant, therefore, to study the relationship between the experimental variables and personality dimensions N (neuroticism or emotionality) and E (extraversion–introversion), as determined by the M.P.I. (Eysenck, 1959), and as there were no significant drug effects, each person's questionnaire score was correlated with each test score three times, once for each successive administration of the test. (It is, of course, clear that to the degree to which the drug effects observed were *real* effects, this method would lower the observed correlations below the true correlations by confounding drug effects and personality.) The resulting correlations are given in Table 2, together with the direction of the relationship as it would be predicted from general theory; these predictions are given in brackets. They derive from the set of hypotheses developed in "*The Dynamics of Anxiety and Hysteria*", and extended in "*Experiments with Drugs*" (Eysenck, 1957, 1962). A (?) indicates that no prediction could be made.

Correlations of 0·362 and 0·464 are required to reach the 0·05 and the 0·01 level of P respectively, and relatively few of the observed values are as high as this. However, the three correlations in each set are nearly always identical with respect to sign, and may thus be taken as re-inforcing each other to some extent. In 11 cases out of 12 the observed correlations agree in sign with the predicted ones, taking the whole set of three into account; that is to say, either all three are in the predicted direction, or else two are in the right direction and the third one very slightly in the wrong direction. (Very slight, in this case, indicates values of 0·0013, 0·0087, and 0·0587.) In only one case (extraversion and extinction) is the direction of the forecast wrong; according to theory, extraverts should extinguish "orienting reflexes" more quickly, thus giving a negative correlation with number of trials fo extinction; the actual correlations are 0·1971, 0·0808, and —0·1014. All are insignificant, but only the last is negative, as required.* We may conclude from this survey of the observed correlations between personality and test scores that these are overwhelmingly in the predicted direction, but that the strength of the relationships indicated is below the level required for full statistical significance in most cases; if use had been made of the technique of "single-tail tests," then the number of significant correlations would have gone up decisively; Eysenck (1960) has given arguments against the use of this technique.

One prediction may require some discussion, viz. that relating to the PGR resting level. We have assumed that this is not, in fact, a true "resting level", as would be recorded, for instance, in sleep, but represents an autonomic "arousal" response to the situation of being in a strange room, full of apparatus, vaguely threatening and calling forth some degree of alertness. This "arousal" should adapt out in the course of several sessions

* It is possible that the positive correlation between extinction and extraversion, though not significant and contrary to theoretical expectation, deserves to be taken seriously. It is in line with a number of Russian studies showing that brain-injured subjects do not extinguish the orienting reflex as readily as do normals; also perhaps with the finding that animals higher in the phyletic scale extinguish these responses more readily (cf. Razran, 1961). It is to be regretted that so little work on this important response has been done in the West, and that there is almost none linking it with personality variables.

(and to some extent within each session), and when the figures are summed over subjects for the three successive sessions, we do, in fact, obtain an increase in resistance of some 15 per cent. (A similar adaptation is found with respect to the "orienting reflex", where there is a fall in number of responses of almost 50 per cent between first and third session.) From the fact that, theoretically, extraverts show quicker build-up of cortical inhibition (which is assumed to underlie adaptation, in so far as this is not due to peripheral factors), we would expect positive correlations between E and "resting level" resistance; this correlation should be highest on the third occasion, where subjects might be expected to have had enough time to adapt thoroughly. It will be seen that, indeed, the correlation for the third day is much the highest, as well as being the only significant one; this would seem to support the hypothesis. It is in line with this view thatin respect to extinction of the orienting reflex also, it is the correlation for the third day which is in line with prediction, while those for the preceding days are in the wrong direction. These points would probably repay more extended study.

SUMMARY

Thirty normal subjects have been given tests of pain reactivity, anxiety, "orienting reflex" extinction, spiral after-effect and persistence under conditions of "no drug", placebo, and 350 mg of Carisaprodol; they have also been administered the Maudsley Personality Inventory. Predictions were made with respect to (a) drug effects, and (b) personality correlates of test scores. Most of the predictions were, in fact, borne out by the results, but mostly at levels of statistical significance lower than 0·05. It is suggested that, in future work, larger doses of the drug may require to be used in order to obtain more clear-cut results.

Chapter 14

ETHYL ALCOHOL AND THE EFFECTS OF STRESS

J. EASTERBROOK*

Introduction

The physiological literature on ethanol can be described as confusing. The facts are distorted by errors, though there is enough redundancy among reports that some information comes through this noise (Easterbrook, 1961). Much the most important fault with the facts is the theory.

The psycho-pharmacology of ethanol (like that of other agents) has been influenced by the theoretical dichotomy of functions into depressant and stimulant. This has entailed difficulties because it has not been generally clear how sensitivity to discharge in the tissues of nervous assemblies may be related to activity by whole animals in psychological experiments. General theory has been inadequate as a source of standards of reference for psychopharmacology.

A point of general difficulty in the study of nervous efficiency which is reflected in the literature on ethanol is that the significance of rapid response to stimulation varies abruptly with the circumstances of test. When a single stimulus is effective and dominates an organism, it leads to intense experience and rapid impulsive action. Thus deprivations, assaults, intense stimulations and threats have been recognized to produce prevailing effects, known as "drive" (Hull, 1943) or general nervous arousal (Freeman, 1948), that have been described as activating or alerting because they facilitate such impulsive action. On the other hand, efficient and rapid adjustment to a multiplicity of disturbances can be achieved when the neural processes they initiate operate concurrently. To compound the confusion, it has become evident (Easterbrook, 1959) that rapidity of adjustment to stimulus complexes is impaired by the state of drive which produces rapidity in impulsive reaction.

Evidence to justify calling ethanol a CNS depressant refers primarily to rapidity of adjustment to stimulus complexes (Dale, Greenwood, Mellanby, Myers and Sherrington, 1938; Jellinek and McFarland, 1940). It seems to be true that subjects given relatively small doses of ethanol show poorer performance at complex tasks than do unemotional controls. At the same time, simple impulsive activity appears to be facilitated by ethanol. Indeed, it is known to be a stimulus to resting subjects. It causes positive response

*The writer is indebted to the committee of management of the Burden Neurological Institute, Bristol for the resources to carry out this investigation. The experiment described is part of a programme of work for the Ph. D degree in the University of London.

from chemo-receptors in the mouth and nose (Pfaffman, 1951; Diether, 1951; Kruger, Feldzamen and Miles, 1955; Lester and Greenberg, 1951), in blood vessels (Heymans, 1955; G. Liljestrand, 1953) and perhaps in the midbrain as well (von Euler and Soderberg, 1952; A. Liljestrand, 1953; Winterstein, 1961). It causes and facilitates depolarization of nerves directly (Gallego, 1948; Larabee and Pasternak, 1952; Grennell, 1957; Fischer, 1957; Ghosh and Quastel, 1954). It stimulates respiration (e.g. Dale *et al*, 1938; Gernandt, 1943; A. Liljestrand, 1953; G. Liljestrand, 1953; Holmberg and Martens, 1955; Loomis, 1952; Raffy, 1949) heart rate (e.g. Dodge and Benedict, 1915; Loomis, 1952; Holmberg and Martens, 1955) and endocrine activity (e.g. Eggleton, 1942, Kleeman, Rubin, Lamdin and Epstein, 1955; Perman, 1958; Kinzius, 1958; Santisteban, 1961). In its effects on both complex adjustment and impulse strength in quiescent subjects, ethanol operates like drive or general nervous arousal.

On the other hand, the emotional state of the subject apparently influences the effects of ethanol upon adjustment to stimulus complexes and also upon impulsive reactions. There is evidence to suggest that in emotion provoking conditions ethanol facilitates adjustment to complex situations. Such evidence comes primarily from experiments on the feeding behaviour of frightened animals (Dworkin, Raginsky and Bourne, 1937; Dworkin, Bourne and Raginsky, 1937; Masserman and Yum, 1946; Jacobsen and Skaarup, 1955; Conger, 1951), but its generality may be supported by studies on humans such as those of Ferrett, Barbut and Ducos (1951), and Vogel (1958). Under the influence of ethanol, ready responses to stimulus complexes are produced by subjects that otherwise behave inefficiently as a result of emotional states. In a similar way, according to evidence that is widely familiar (c.f. Easterbrook, 1961), ethanol reduces the intensity of impulsive reaction to intense drive-provoking stimuli so as, apparently, to reduce pain, fear and general stress. Here too, as in the case of adjustment to stimulus complexes, the effects of ethanol on subjects under weak stimulation are reversed in subjects under strong stimulation. Perhaps ethanol produces paradoxical effects of stimulus intensity (Pavlov, 1928).

The conception of ethanol as a stimulus contains hints for the explanation of these facts. It ought to act like other stimuli to precipitate the discharge and subsequent refractory phase of nerve cells it reaches. It ought, therefore, to facilitate impulsive response to all external stimulation and also to reduce the number of polarized cells available for subsequent external control and for recruitment by the more intense stimuli. It should act like noise. Finally, it should alter the normal relations of process intensity to stimulus intensity. It should do so in the fear–feeding conflict situation in such a way as to increase the intensity of the seeking processes and to reduce the intensity of the processes involved in fear.

The foregoing considerations have been formulated as hypotheses and put to test (Easterbrook, 1961). The hypotheses are:

(1) That increase in general nervous arousal due to increase in the intensity of prior stimulation will be associated with a reduction in the speed of response to adequate stimuli of low intensity in the presence of irrelevant stimulation.

(2) That small quantities of ethanol, internally, affect speed of response to adequate stimuli by facilitating response to weak stimuli and inhibiting response to strong stimuli.

(3) That small quantities of ethanol, internally, mimic the effects of irrelevant stimulation upon speed of response to relevant, adequate stimuli presented subsequently.

(4) That small quantities of ethanol, internally, affect the interaction between arousal and speed of response to adequate stimuli of low intensity in the presence of irrelevant stimulation, by : (a) increasing speed of response, in the case of subjects under the influence of strong drive-stimulation; and (b) reducing speed of response, in the case of subjects under the influence of weak drive-stimulation.

Method

The action selected for study was escape from electric shock. Differences in drive or arousal were produced in two ways: by preconditioning at different intensities of shock, and by varying the intensity of the irrelevant stimulus, white light. The use of aversive behaviour gives strong test to the hypothesis that the presence of the irrelevant light stimulus will alter the normal effects of drive.

Four intensities of relevant stimulus (SR) and four intensities of irrelevant stimulus (SI), in all their sixteen combinations, were presented to 16 subgroups from each of 3 major groups trained with different intensities of shock (ST) to produce three levels of relevant drive, arousal or stress. To observe the effect of a small quantity of ethanol on response in each condition doubled the required number of matched groups to a total of $(4\times4\times3\times2 =)$ 96 groups. Four animals were used in each group.

The intensities of shock for training were selected to produce relatively low emotion. The lowest intensity (60v) was 10 V higher than one which had been found inadequate to produce escape within 2 min. The highest intensity (100 V) was 50 V lower than one which had been found to make the escaping activity persist during inter-trial intervals.

The total of 4 rats was built up by running serially through the test conditions in four different sequences, duplicated daily. During sequences 1 and 3, ethanol was administered in morning tests, water in the afternoons, and the alternate order was followed for sequences 2 and 4. The four sequences were arranged in pairs to balance (as between extremes of stimulus intensity) any effects of order or time of testing, such as odours in the experimental chamber, prior disturbance in the stock room, weather and the condition of the apparatus. The four testing sequences are shown in Table 1.

The experimental chamber consisted of a space $6^1/_2$ in. $\times$ $6^1/_2$ in. $\times$ $5^1/_2$ in. (wide) in a box 13 in. $\times$ $6\ ^1/_2 \times 5^1/_2$ in. that had a metal cover for the opening at the top through which the rat was introduced. At one end of the box, a reflector carrying three light sources was separated from the experimental chamber by a frosted Perspex panel and a vacant space. The vacant portion of the box was divided from the experimental chamber by $^1/_{16}$ in vertical rods, set $^1/_2$ in. apart. Two side walls were covered by metal plates.

TABLE 1

Rata of test conditions showing the four sequences followed in serial testing

Number	*Training trials Relevant Stimulus Intensities (ST)*			*Testing trials Relevant Stimulus Intensities (SR)*				*Irrelevant Stimulus Intensities (SI)*			
	60 V	*70 V*	*100 V*	*60 V*	*70 V*	*100 V*	*150 V*	*0*	*2*	*10*	*312½**
1	1	2	3 (R)	1	2	3	4 (W)	1	2	3	4 (D)
2	3	2	1 (R)	4	3	2	1 (W)	4	3	2	1 (D)
3	3	2	1 (W)	4	3	2	1 (D)	4	3	2	1 (R)
4	1	2	3 (W)	1	2	3	4 (D)	1	2	3	4 (R)

(R) The parenthesized letters show the rate of rotation, by rat by day or by week.

* Light intensity in exposure meter units (Weston, Master III).

The chamber floor was a series of rods which could be electrified. When the shocking circuit was closed, these, the vertical rods and the side plates were each made electrically positive in random order for 25 msec once every 0·6 sec, the others serving as ground. Thus a rat in contact with only two electrodes had brief shocks during a twelfth of the time before it escaped. One with four points of contact was shocked for a sixth of the time.

At the end of the chamber opposite the light source was a movable plate of bright metal filling the available space and balanced against a switch to break electric circuits to grid and lamps. To escape the rat had to move this panel, as it easily did by standing with two paws on it or by pressure with its tail. This panel was never electrified. Above it, let into the lid of the box was a $^1/_{16}$ in. hole permitting entry of a small amount of room light.

On a shelf near the shock box, an electric fan produced a low ambient noise to mask the operator's movements.

The operator's control panel had means for selecting stimulus intensities, for initiating the stimuli and for resetting the entire switching and timing system. On each trial, stimulus levels having been selected, the operator had (a) to reset the system, (b) to press the light-starting switch, and (c) one second later to press the shock-starting switch. An electronic timer, zeroed at (a), began counting at (b) and stopped when the rat's switch was thrown, so that it recorded the time in seconds (to three decimal places) between pressure on the light-starting switch and completion of the rat's reaction. The tenth of seconds dial on this timer served to cue the operator in producing the one second inter-stimulus interval.

Three hundred and eighty-four rats served as subjects, being matched for strain (Wistar, albino, Glaxo) for sex (female) and for weight (140–160 g). They were randomly assigned to the conditions specified in the test rota.

A statistical procedure is mentioned below for supporting the assumption of the biological equivalence of the groups so composed.

All subjects were adapted to solitude in a darkened chamber by being kept for 18—23 hours just prior to testing in a cage that had been modified to resemble the experimental chamber in size and material. All were fed and watered *ad lib* up to the moment of testing.

Fifteen minutes before its test, each subject was given 1 ml of liquid by stomach tube and returned to its adaptation cage. For half the animals, this liquid was pure distilled water, but for half of them, twenty per cent of the water by volume had been replaced with ethyl alcohol, this dose being calculated at 1 mg of ethanol per gramme of average rat.

Observations were recorded by two different operators,* using the same equipment with common instructions. One made the morning observations while the other operated in the afternoon. Each was trained during pilot studies (a) to reproduce accurately the 1 sec interval between onset of stimuli, and (b) to administer the liquid by stomach tube smoothly (so that less than 20 sec was needed for the total operation beginning and ending with the rat in its adaptation cage).

The routine of testing days started with normal cleaning and feeding operations in the stock room which did not directly involve the experimental subjects. Half an hour after this work had ended, the morning experimenter injected his first rat, cleaned and polished the electrodes, started the masking noise and tested the equipment. The chamber was cleaned again after testing. An hour later the same procedure was repeated. The adaptation cages were then emptied and restocked for use on the following day.

Each rat was placed in the test chamber 15 min after entubation and allowed to explore it until she had pushed the movable panel once or until 2 min had elapsed. Six training trials were then given, with 2 min between the end of one and the beginning of the next. These were followed at the same rate by six test trials. Each rat, therefore, spent approximately 25 min in the test chamber.

For each rat, the operator recorded the conditions of each test and the time between light on and shock off, which was, of course, one second greater then the time the shock circuit was closed.

Analysis

Fourteen records in the ethanol sample and eighteen in the water sample were replacements for failure to meet fairly loose standards of consistency that are defined in detail in the original report (Easterbrook, 1961).

The raw times were transformed to remove the characteristic skew in distributions of reaction times. The transformation, selected by trial on the times for the first trial, was $100\sqrt{\frac{1}{t+1}}$ which of course is $100\sqrt{\frac{1}{RT+2}}$ because the measured time was 1 sec greater than the escape time (RT).

*The assistance of Mrs. Rita K. Carpenter is gratefully acknowledged.

The experiment yielded 4608 scores for speed of escape from shock. Thus, 4608 distinct factors might be required to account for the total variance in scores. It is the task of analysis to discover whether a more limited number of distinctions can serve this purpose to a useful degree, and if so to specify them.

The data fell conveniently into two sets of ($2\times6\times192$) 2304 scores, for both the training period (T 1—6) and the test period (T 7—12). Both sets of data were analysed in three stages:

(1) Simple analysis of variance, (a) to check that sample size was sufficient to produce the required compensation of random influences, and (b) to assess the importance of all the controlled variables, such as trials.

(2) Analysis of covariance and computation of least squares regression equations, (a) to smooth the differences between conditions in order further to support the assumption of biological equivalence of the test material, and (b) to distinguish significant from chance variations about the mean.

(3) Analysis of the integrated scores derived from the functions differentiated in (a), in order to assess the experimental hypotheses.

The details of the procedure used for smoothing and analysis of covariance are as follows:

(1) The voltage readings corresponding to the different values of shock stimuli were transformed into logarithms (base 10) for use in regression equations. The effectiveness of energy changes as stimuli is closely approximated by a log function of the physical values of these changes. The scale of irrelevant stimulus intensity is NOT a simple log. conversion of meter readings, however, but has an arbitrary component because there is no meaningful log. for zero energy change. The three positive values of light stimulus are indeed log. meter readings, but they were regarded as points on an arithmetic scale of stimulus intensity, which would have an arithmetic zero corresponding to the "no light" condition. This effectively codes the zero light stimulus as equivalent to an energy change of 0·1 units on the exposure meter scale. The empirical utility of this procedure was checked against the experimental data, first visually for simplicity of form of the relation between SI and speed of escape, and subsequently by an estimate of the error of fitting speed scores to these values. As will be seen below, a total sum of squares of 58,444 with 432 d.f. was attributable to the effects of SI. When the influence of SI was expressed in regression equations, however, a sum of squares of 4,163 was accounted for with eight degrees of freedom. The error of fit, then, is an MSV of 128·0, which is to be compared with the residual term of 132·9 in Table 3b. Evidently this somewhat arbitrary treatment of the transformation problem produced a scale which adequately approximated the effectiveness of the lights as stimuli. The values of the photic and the shock stimuli are shown in Table 2, which also displays the constants derived from them for use in regression analysis.

(2) The most significant independent variate was selected by reference to the variance analysis.

(3) Holding all other classifications constant, regression equations of the form $Y = a+b_1k_1(X)+b_2k_2(X)$ were then calculated for each cell. This form of equation was employed in place of the familiar $Y = a +$