EFFECTS OF TRANQUILLIZERS ON WEIGHT GAIN IN BROILERS

By

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THESIS

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CERTIFICATE

Certified that this thesis entitled "EFFECTS OF TRANQUILIZERS ON WEIGHT GAIN IN BROILERS" is a record of research work done independently by Smt. Santa K. George under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to her.

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SANTA B GEORGE.

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INTRODUCTION

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Commercial broiler production is relatively a new venture having developed mainly since the World War II and has made phenominal growth from 1935 onwards in advanced countries. Broilers are young chicken raised specifically for meat production. Since they are ready for market about two months of age, one can expect quicker returns on investment. With the establishment of commercial hatcheries and ready availability of superior broiler chicken in the recent days, broiler production is considered to be a lucrative industry. The broiler industry demands a fast growing chick capable of converting feed into meat with great efficiency. Feed continues to be the greatest single factor representing over 70% of the total cost of production. Higher weight gain, lesser mortality and better feed efficiency are factors that widen the difference between profit and loss in broiler farming. Successful poultry production depends on harmonious relationship with the environment to which the birds are subjected. Stress plays an important decisive role to make broiler production a thriving industry.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Stress in Poultry.

Stress has been defined as that within a living creature which result from inter-action of the organism with noxious stimuli (Pruthi, 1975). It may be physical, chemical or emotional factors along with management errors that cause physiological or mental tension. Under intensive system of poultry keeping, birds are invariably exposed to stress of one kind or other. Stress is a matter of degree and the maximum expression is the death of the bird from fright or shock even though no physical injury is involved. The cause and severity of stress to which the birds are exposed from day old to the point of disposal may be many and varying. Many organisms like bacteria and viruses with a potential for causing infectious diseases may remain dormant causing no apparent symptoms of disease in the birds. These infectious agents invade the tissues of the host when the resistance of the bird is lowered due to internal or external stress factors. Stress is not a single entity but an amalgamation of a number of factors which upset the well balanced physiological norm of a living being and may be very wide in its scope and manifestation. These factors put a strain on the production capacity of the birds and get expressed by lowering the vitality, performance, disease resistance and efficiency of feed utilisation.

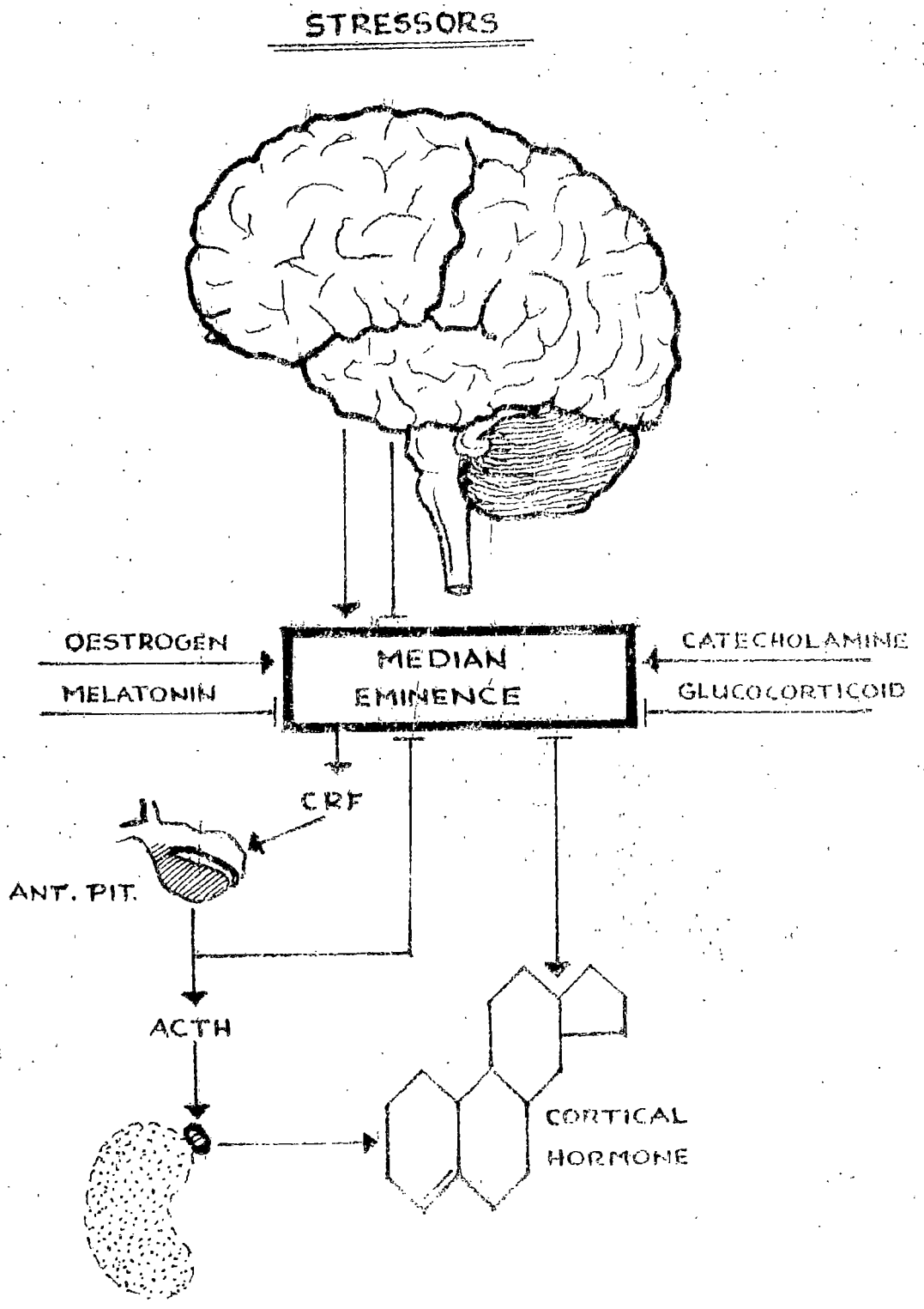
Factors associated with stress in rearing of poultry are i) transportation, ii) vaccination, iii) extremes in weather, iv) over-crowding, v) physical disturbance such as noise, strangers, frequent handling etc.

vi) poor feeding, vii) dehydration, viii) starvation, ix) debeaking, x) medication, xi) rapid growth etc. (Charles and Payne, 1966).

Chicken, like all animals are able to compensate for inadequate nutrition, faulty management or extremes in temperature by adjusting certain body function. But this ability is limited and when the limit is reached or surpassed they are affected by stress (Johnson and Ridden, 1974).

Stress factors stimulate hypothalamus of the brain and corticotrophin-release-factor is released into the blood stream. This acts on the anterior pituitary gland and adrenocorticotrophic hormone (ACTH) is produced which causes the release of cortico steroids and mainly corticosterone is released from the cortex of the adrenal gland. The cortico steroids limit the resistance of the body towards bacterial and viral infection and also antibody production. Growth is arrested since the secretion of the growth hormone is reduced. A change occurs in nutrient requirements under stress condition and the body demands more of vitamin A, B-complex, C and E and well balanced feed. Vitamin C is synthesised in the body but during summer the synthesis is disturbed. Vitamin C plays a role to decrease heat and cold stress on the birds (Gupta and Handa, 1976). Very high and low temperature inside the poultry house cause severe stress and it may even cause mortality besides low egg production.

A number of chemical substances are being added to broiler rations to obtain maximum weight gains. These chemical substances when employed as feed additives are believed to promote growth and improve feed conversion. Some of these are chiefly nutrients and some are medicaments like 'Cyproheptadine' (Sachidhanandan and Nair, 1971). Observations that certain feed additives especially antibiotics in animal feeds produce resistant organisms and some with transferable resistance promoted many countries to impose restrictions on their use. Additives when incorporated in broiler rations should bring about gains economically and cause no harm when meat or egg is consumed.



Source : Breazile, (1971).

Tranquillizers.

A quarter century ago, the tranquillizers or ataractics took their place as an important group of pharmacological agents in human and veterinary medicines and demanded by virtue of their unique pharmacologic action that a separate classification is necessary from the conventional depressants of the central nervous system.

The tranquillizing agents are to be distinguished from sedatives in the strict sense that one considers the sedative action of chloral hydrate, barbiturates and other central nervous system depressants. The tranquillizers are often referred to as 'ataractic agents'. Ataraxia by definition means 'not disturbed', perfect peace or calmness of mind (Booth, 1965).

Tranquillizers have brought about a great improvement in the management of severe emotional and mental disorders. One of the early developed tranquillizers reserpine was obtained from a plant (Rauwolfia serpentina) was known in India for centuries. Tranquillizer is a drug that will quieten a patient without notably impairing consciousness. The ideal tranquilliser would allay pathological anxiety and nervous tension without cerebral function; especially it would not cause sleepiness or even drowsiness (Lawrence, 1973).

Any substance which alters mental process or behaviour is generally termed 'psychotropic drugs'. Psycholeptic or anti-psychotic agents have

a depressing or inhibiting action. Thus, the sedative, tranquillising or ataractic drugs would be considered psycholeptic or antipsychotics. The term 'tranquillizer' has restricted meaning and should not be used to designate the entire group of psychopharmacologic agents. Thus in usual parlance, the psychotropics refer to therapeutically useful psychopharmacological drugs.

It is generally understood that a psycholeptic drug is one that elicits a calming effect reducing anxiety, tension, agitated or disturbed behaviour. The anti-anxiety drugs resemble the central nervous system sedatives in that they relax muscles, are mildly sedative and tend to produce drug dependency. However, a clean pharmacologic distinction cannot be made among these class of drugs.

Classification of psychotropic drugs:

Psychotropic drugs are classified in different ways. They may be classified depending on a) the chemical nature, b) pharmacological properties and c) clinical use. The classification based on clinical use include Major and Minor psycholeptic agents. The major psycholeptic agents are those with apparent or confirmed efficacy in the treatment of psychotic patients. The minor psycholeptic agents are those used in the treatment of neurotic and psychosomatic reactions.

Major neuroleptics:

i) Phenothiazine derivatives:

Chlorpromazine hydrochloride.

ii) Rauwolfia alkaloids:

Reserpine

iii) Thioxanthene derivatives:

Chlorprothixene

iv) Butyrophenone:

Haloperidol

Minor neuroleptics:

i) Phenothiazine derivatives:

Promethazine hydrochloride

ii) Benzodiazepine derivatives:

Chlordiazepoxide

Diazepam

Oxazepam

Nitrazepam

iii) Propanediol carbamates:

Meprobamate

iv) Compounds of miscellaneous structure:

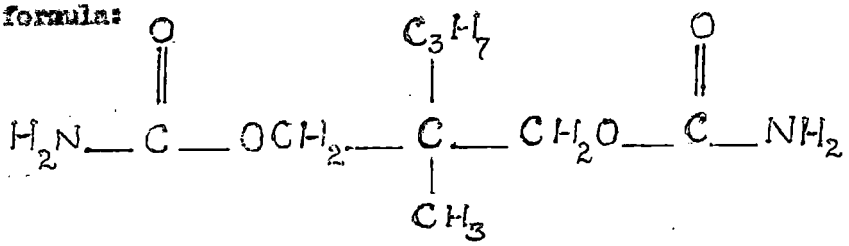
Chlormethazanone

MEPROBAMATE.

Meprobamate was originally synthesised in 1951 and developed as a potential muscle relaxant by Berger (1954).

Chemistry and structure-activity relationship:

Chemically it is 2-methyl, 2 propyl-1, 3 propanediol dicarbamate. It is a white crystalline powder and has a bitter taste. Meprobamate is soluble in water to the extend of only 0.79% at 37°C. The drug is stable in dilute alkali and acid solution and does not decompose in gastric and intestinal juices. It is a simple aliphatic compound with the following structural formula:



Meprobamate is the most potent compound in the series in paralysing action and in preventing convulsions and death caused by pentylene tetrazole. Stewart et al. (1959) prepared a series of 2 - methyl - 1,3 propanediol dicarbamates substituted in the 2 position, in place of the n-propyl group with 2-chloroethyl, 3 chloro propyl and 3 brom propyl group. The 2-chloro methyl 2-n propyl-1, 3 propanediol dicarbamate was also prepared and its action studied. Halogenation and minor alteration in the length of the substituted carbon chains resulted in significant decrease in potency from that of the parent compound. All the compounds had an action qualitatively similar to meprobamate. Meprobamate is a longer acting successor to mephenesin.

Pharmacological properties:

Meprobamate is commercially available under the popular brands of

Miltown and Equanil. It is also available in the market along with other sedatives, analgesics etc. under different trade names.

Action on central nervous system:

Mode of action of meprobamate on central nervous system is not well understood. It has anticonvulsant property, but it is of limited clinical use for this purpose. On withdrawal of large doses of meprobamate, convulsions are seen aggravated in epileptic patients. It has no specific depressant effect on reticular activating system. Pharmacological studies of Berger (1954) showed that meprobamate produced a reversible flaccid paralysis of skeletal muscles. Smaller doses elicited muscular relaxation and sedation. Berger (1954) observed that meprobamate antagonised the convulsive seizure evoked by either strychnine or pentylene tetrazol. Anaesthesia with barbiturates was found to be prolonged by meprobamate and the administration of the drug produced a taming effect on monkeys. Baird et al. (1957) demonstrated the effect of meprobamate on the electrical activity of the brain and was found to be not limited to the diencephalon but extends to the forebrain ganglia (amygdala, caudate nucleus, pallidum) in varying degree. These ganglia were found to exhibit different degrees of susceptibility to meprobamate. Lesions on the caudate nucleus may induce super sensitivity of the pallidum to the action of meprobamate.

EEG effects:

A characteristic action of meprobamate is its specificity for the

thalamus. It does not depress the cerebral cortical activity and the characteristic spindling of the EEG produced by barbiturate does not usually occur after normal doses. Sustained high dosage of meprobamate produce EEG pattern that are similar to those elicited by barbiturates. There are however, significant differences in the EEG observed after the two drugs (Berger, 1963).

Effects on sleep:

Meprobamate suppresses REM sleep as do the barbiturates. REM is resumed after withdrawal of the drug.

Autonomic nervous system:

No autonomic effects are seen with clinical doses.

Muscle:

Although skeletal muscle relaxation can presumably be measured objectively, quantitative data comprising the effect of meprobamate and other agents upon muscle spasm are difficult to obtain. There is evidence that sedation from meprobamate plays an important role in muscle relaxation (Domino, 1962).

Cardio-vascular and respiratory system:

In toxic doses meprobamate causes respiratory depression. Hypotension occurs occasionally with therapeutic doses.

Absorption, fate and excretion:

Meprobamate is well absorbed from the gastro-intestinal tract, reaches a peak plasma concentration and systemic effect within about 2-3 hours and has a half life of 10 hours. Meprobamate can induce microsomal enzyme systems in the liver and accelerated drug disposition. Pharmacodynamic tolerance and inter-action with other drugs thus occur. Meprobamate is quite uniformly distributed in the body and about 10% of the drug is excreted in an unchanged form in the urine within 24 hours. The rest is excreted as hydroxy meprobamate and glucuronide.

Toxic reaction and side effects:

The major side effects of meprobamate are sleepiness and ataxia. Hypotension may also occur. Allergic reactions have been reported in from 0.2 - 3.4% of different series of patients and appear most frequently in those with a history of dermatological or allergic conditions. Urticaria or erythematous rash is the most common manifestation. Acute non-thrombocytopenic purpura has also been reported. Angioedema and bronchospasm have also been reported. Meprobamate has been found to be associated with development of aplastic anaemia, thrombocytopenia, leukopenia, agranulocytosis and erythroid hypoplasia, but the number of reported cases has been very small.

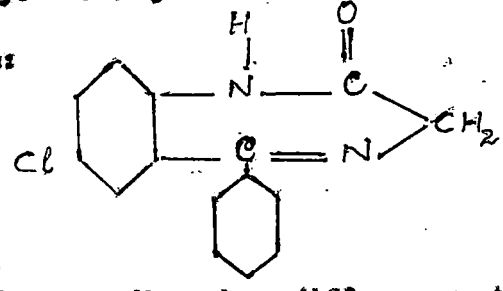
Clinical use of meprobamate:

There is no other drug introduced in the recent years which has

had a wide use as has meprobamate. Meprobamate has been used in anxiety states and many organic diseases with tension component. It is being used in the treatment of musculo-skeletal disorders, rheumatic conditions, alcoholism, psychomotor agitation and behavioural problems of children.

NITRAZEPAM.

The benzodiazepine derivatives presently available are chlordiazepoxide, diazepam, oxazepam, chlorazepate, flurazepam and nitrazepam. Nitrazepam is a new generation of benzodiazepine having remarkable hypnotic property. It is yellow in colour and tasteless. Its structure is as follows:



The benzodiazepines differ among themselves and it is difficult to characterise them as a class. Some of them appear to be more selective than the barbiturates in the suppression of anxiety. All benzodiazepines have hypnotic action, but the duration of action and side effects preclude the hypnotic use of some. They are considered to have a number of advantages over other hypnotic sedatives. The therapeutic index appears to be quite high. Benzodiazepines have a reputation for low incidence of 'hangover'; however, the hangover of nitrazepam is found to equal that from barbital (Walters & Lader, 1971) and amobarbital (Davies and Levine, 1967). The effects of benzodiazepines on the hepatic microsomal system

appear to be slight, although they stimulate the system in rats. However, stimulation of the microsomal system does not affect the metabolism of benzodiazepines.

Nitrazepam has been used extensively in Europe and in Common Wealth countries. Like the barbiturates it increases EEG activity but it decreases alpha and theta activity (Montague, 1971). As a hypnotic in man it is equiefficacious with short and intermediate acting barbiturates (Davies and Levine 1967; Haider, 1968; Matthew et al, 1969). After oral ingestion about 78% is absorbed. Redistribution takes place over a period of 8 - 12 hours. After this time the plasma concentration declines with a half life of 21 - 25 hours (Haider and Wendt, 1973) which may be the reason why hangover is about the same as that following amobarbital.

Pharmacological properties:

The effect of benzodiazepines in the relief of anxiety can readily be demonstrated in experimental animals. However, anxiety in experimental animals and man can hardly be equated. Since the neurophysiological or biochemical basis of anxiety is unknown, assessement of efficacy must be based on the general acceptance of benzodiazepines by the medical profession. The clinical popularity of these drugs apparently is the result of a mechanism of action that is yet undefinable. A study carried out by Boethens and Westerholm (1976) in Sweden revealed the wide use and acceptance of Nitrazepam in 1973 mostly at the expense of diazepam and combined products.

Benzodiazepines can be effectively used as hypnotics in conjunction with their use as anti-anxiety drugs. They do not suppress REM sleep in normal doses but do markedly diminish or eliminate stage-4 sleep. The significance of this is not known. Gaillo and Bassano (1975) observed in man that administration of flunitrazepam and nitrazepam produced a large decrease in rapid eye movement during first sleep cycle.

The benzodiazepines cause an increase in fast beta activity with an increase in amplitude of the EEG. All benzodiazepines increase seizure threshold and are anticonvulsant.

Skeletal muscle:

Benzodiazepines are widely used as muscle relaxant. Some muscle relaxation occurs after administration of any central nervous system depressant and there seems to be no particular advantage to any of them when given by oral route.

Absorption, fate and excretion:

Chlordiazepoxide is slowly absorbed and may take several hours to reach a peak plasma concentration and combined administration for several days is required for the plasma concentration to reach a plateau. Two active metabolites, a lactam and a demethylated derivative are formed. Diazepam in contrast is rapidly absorbed reaching a plasma concentration in one hour. The blood levels of rats orally administered with nitrazepam reaching a plateau which persisted for 90 minutes and then declined with

a half life of 90 minutes (Tanayama, et al. 1974). The limit of detection of nitrazepam in serum is found to be 5 $\mu g/ml$ (Moller, 1975). According to Yanagi et al.(1975) at least four kinds of reactions are involved in the bio-transformation of nimetazepam and nitrazepam and the former was found to be rapidly hydroxylated at C-3 while the C-3 hydroxylation of nitrazepam was very slow. The reduction of nitro group at C-7 and subsequent acetylation are important routes for the excretion of these drugs.

The tolerance and physical dependence occur with benzodiazepines as with all drugs of this class. Habituation to benzodiazepines is common, however, withdrawal symptoms after chronic use may not appear for a week after discontinuation of the drug. Yanagita et al. (1975) observed the development of physical dependence to repeated administration of nitrazepam in normal monkeys by withdrawal tests.

Toxic reaction and side effects:

The expected side effects of drowsiness and ataxia are extension of the pharmacological action of these drugs. In general, the clinical toxicity of the benzodiazepines is low. Weight gain which may be the result of a renewed appetite occure in some patients. Over-dosage with benzodiazepines is frequent but serious sequelae are rare. Over-dosage of nitrazepam up to 40 times the hypnotic dose produced no respiratory depression and loss of consciousness (Mathew et al. 1969). The striking advantage of benzodiazepines is the remarkable margin of safety.

Drug interaction:

It is infrequent with benzodiazepines. The relative lack of either side effects or drug interaction frequently makes these drugs the agents of choice in the treatment of anxiety states. Scaio et al. (1975) reported that nitrazepam in combination with alcohol was especially deleterious on psychomotor skills.

TRANQUILLIZERS IN CHICKEN.

The calm and serene atmosphere and a feeling of comfort will promote the normal physiological functions of the body. Tranquillizers are being prescribed by physcians to psychotic patients with good success. The use of tranquillizers as anti-anxiety drugs is not restricted to human patients. They are being employed in animals and veterinary practice in very many situations.

Reserpine and Chlorpromazine are two drugs widely used as tranquillising agents. Reserpine produces sedation in a wide variety of mammals and this action has been found to extend to wild turkeys (Earl, 1956), domestic turkeys (Carlson, 1956) and both domestic chicken and turkeys (Burger et al, 1959). A level of 500 mg reserpine per kg of diet produced 96% mortality while 5 mg per kg promoted growth in chicken (Burger, 1959). Feather pecking and cannibalism were found to be controlled on administration of reserpine in pheasants (Hewitt and Reynolds, 1957).

Burger et al, (1959) observed that chlorpromazine fed to White leghorn chicken at levels from 10 - 100 mg per kg diet produced a slight but significant increase in growth while at levels from 250 - 16000 mg per kg diet depressed growth and at higher levels there was 100% mortality.

The sedative effect of chlorpromazine in mammals was not seen extended to fowls (Burger et al, 1959) where as it was effective in increasing resistance to heat. The hypotensive activity of chlorpromazine though not tested in fowls is much weaker in mammals than that of reserpine. Chlorpromazine

had no effect on the plasma corticoid levels of chicken subjected to cold stress till 39 days of age (Buckland and Blagrave, 1973). Both chlorpromazine and reserpine were found to reduce the high ambient temperature stress in chicken (Van Matre and Burger, 1957).

Tranquillising agent meprobamate has been shown to have muscle relaxant action and taming effect on mammals as reported by Burger (1956). At relatively high levels of meprobamate in the diet inhibited growth in White leghorn chicken (Babcock and Taylor, 1957); at higher levels meprobamate had little effect to reduce the nervous and flighty characteristic (Garren and Hill, 1957) and it was suggested that the drug might be slightly toxic to the chicken (Garren and Hill, 1957).

The effects on administration of meprobamate on established breeds of chicken is although fairly known, its action on the broiler chicken which is endowed with rapid growth is not yet clear and much less the action of benzodiazepines in any type of chicken. In as much as cost of feed alone accounts over 70% in poultry production, savings on feed expenses will appreciably enhance the margin of profit. In the modern intensive system of rearing poultry in cages, factors responsible for stress in poultry must be kept minimum, if not completely avoided so as to achieve maximum productive efficiency. In the present investigation an attempt has been made to explore the possibility to reduce the feed cost and to improve the feed efficiency by incorporating tranquillizers such as meprobamate and nitrazepam in the rations so as to reduce stress factors and thereby to obtain maximum weight gains in broiler chicken.

MATERIALS AND METHODS

MATERIALS AND METHODS

A feeding trial of 30 days duration was carried out in the department of Pharmacology, College of Veterinary and Animal Sciences, Mannuthy to study the effect of two types of tranquillizers on weight gain when incorporated as feed additives in the rations for broiler chicken. One-day old commercial broiler chicken ('Starbro' brand) were procured from M/s. Indin Poultry Farm, Bangalore. All the chicken were of the same batch and were raised in the department in a battery brooder. They were wing banded on arrival and debeaked at 10 days of age. Starter and finisher basal rations for the chicken were compounded as per ICMR (1967) specifications. The ingredients and chemical composition of the basal diets are shown in table Ia and Ib.

At 30 days of age, the chicken were weighed individually and randomly assigned to eight groups of 21 each as uniformly as possible in respect of body weight and raised in weaner batteries with vertical tiers, each group having received a floor space of 2 m^2. The diets were also randomly allotted to the groups as detailed in table II. The group that received the basal diet alone formed as control while another four groups each had received basal diet incorporated with meprobamate* at levels of 0.2%, 0.4%, 0.6% and 1.2% of the diet and the remaining three groups were maintained on diets with the addition of nitrazepam * at levels of 0.005%, 0.01% and

* 1. Meprobamate used in the experiment was 'Equanil' a brand product of M/s. John Wyeth and Co., Bombay.
2. 'Hypnotex'brand of nitrazepam of M/s. Pharmaceutical and Chemical Industries, Bombay was used in the experiment.

0.015% respectively. Equanil and Hypnotex procured as tablets in strip packings with aluminium foils were finely powdered in a domestic type electric mixer. Calculated quantity of the drug was then thoroughly hand-mixed with the feed so as to have a uniform disperson of it in the feed, each time the feed so prepared for the respective groups was not more than 10 kg.

The chicken in all groups were maintained on starter diet till 38 days of age and switched on to finisher for the remaining period of the trial with and without the addition of respective drugs.

The experiment was conducted during peak summer. Feed and water were provided to the chicken ad libitum and were under identical conditions of management.

Prior to assigning to different groups, four chicken were randomly selected and subjected to hematological studies and histopathological examination of the liver. Similarly, one each at day 16 and four chicken on termination of the trial at day 30 from each group were sacrificed for hematological and histopathological studies. Birds died during the course of the trial were subjected to post-mortem examination and cause of death investigated.

Blood for haematological studies were drawn from the wing vein. Haemoglobin content was estimated by using a Sahli haemometer. Clotting time was determined by drawing blood into capillary tubes and breaking

the tube at intervals and the end point reached when a shred of clot was formed. The cell constituents of the blood was estimated as per the technique described by Nambiar, (1961). The histopathological examination of the liver was carried out with samples sectioned to 5 μ thickness and stained in haemotoxylin and eosin to be examined under light microscope.

The individual weights of the chicken at the initial, at day 8, at day 16 and final weight at day 30 were recorded correcting to the nearest 5 g. Feed consumed by the chicken during the trial was noted with due correction for spillage. Feed efficiency and cost per unit gain on different dietary treatments had been worked out. The data were statistically analysed as per methods described by Snedecor and Cochran (1967).

TABLE - I

Ingredients and chemical composition of the basal diet

a). Ingredient composition

Sl. No.	Ingredient, parts 100.	Starter	Finisher	Cost/100 kg Rs. ps.
1.	Ground nut cake	26	19	214.29
2.	Gingely oil cake	5	5	236.00
3.	Maize	32	43.5	143.31
4.	Wheat bran	10	5	145.00
5.	Rice bran	15	15	48.00
6.	Dried fish	10	10	155.00
7.	Starmin FS*	2	2	140.00
8.	Lard	—	0.5	400.00
		100	100	
9.	Rovimix @, g	25	25	117.14 per kg
10.	Bifuran £, g	50	—	110.24 ..
11.	Cost/kg of feed, Rs.	1.62	1.53	

1. Starmin FS* : (Shaw Wallace, Madras) containing: Calcium 28%; Phosphorus 7%; Iron 0.5%; Iodine 0.008%; Manganese 0.013%; Cobalt 0.005%; Sod-Chloride 17%.
2. Rovimix @ : Roche Products Ltd., Bombay, containing Vit.A-40,000. I.U; Vit B_2 - 20 mg; Vit.D_3 - 5000.I.U. per g.
3. Bifuran £ : Smith Kline and French (India) Ltd., Bangaloro containing Nitrofurazone 25% w/w; Furazolidon, 3.6% w/w.

b). Chemical composition of air dry feed.

S.No.	Nutrient %	Starter	Finisher
1.	Moisture	5.90	3.60
2.	Crude protein *	21.40	19.70
3.	Ether extractives	4.50	5.80
4.	Crude fibre	6.70	8.60
5.	Total ash	11.60	13.50
6.	NFE	49.90	48.60
7.	Insoluble ash	6.21	8.61
8.	Calcium	0.69	0.65
9.	Phosphorus	0.77	0.75

* N x 6.25

Table – II
Design of the experiment.

Dietary treatments	Group I	II	III	IV	V	VI	VII	VIII
No. of birds	21	21	21	21	21	21	21	21
Equanil % (Meprobamate)	Basal	Basal + 0.2%	Basal + 0.4%	Basal + 0.6%	Basal + 1.2%	-	-	-
Hypnotex % (Nit-rezepam)	-	-	-	-	-	Basal + 0.005%	Basal + 0.01%	Basal + 0.015%
Av. cost per kg feed, N.	1.57	2.68	3.79	4.90	8.23	3.26	4.95	6.64

Cost per tablet of 'Equanil' and 'Hypnotex' at the Department was N.0.22 and N.0.33 respectively.

RESULTS

RESULTS

Growth.

The growth rates of chicken in all the dietary treatments from 0 - 8, 0 - 16 and 0 - 30 days of trial are presented in tables III to X. Table XI shows the summarised values of growth, feed consumed by the chicken, feed efficiency (grams feed per gram gain), average daily gain in weight and feed cost per kg of gain. The data pertaining to the body weight gain were statistically analysed and the presented in table XII..

Mortality.

The chicken died during the course of trial from 0-8, 0-16 and 0-30 days in the respective treatment groups and the cause of death revealed on autopsy are set out in table XIII.

Birds died during the course of the trial in groups II, III, IV and V on autopsy exhibited extensive subcutaneous haemorrhage extending the entire length of the trachea. Patchy haemorrhagic area were seen at the region of the breast muscle, on the wings, legs and tarso-metatarsal regions. On the antero-ventral aspect of the lungs haemorrhages were seen. There was no evidence of meningeal haemorrhage. The leizons were almost identical in all the birds died, but occasionally slight variation could be noticed in individual cases. The birds died in group VII on autopsy revealed leizons suggestive of pericarditis and enteritis.

Haematology.

Data on the haematology of the chicken in the different dietary treat-

ments in respect of haemoglobin, cell constituents such as red cell count, total and differential leucocyte counts and clotting time of the blood at the commencement, at day 8, at day 16 and on termination of the trial at day 30 are depicted in tables XIV to XXIII. The summarised values on the haematology of the chicken on termination of the trial are presented in table XXIV.

Histopathological examination of liver.

Treatment groups	at day 0.	at day 16.	at day 30.
Gr. I	normal	normal	normal (Plate I Fig.I)
Gr. II	..	Disruption of hepatic cord.	In some areas disruption of hepatic cord and many of them with focal haemorrhages (Fig.2)
Gr. III	..	Focal haemorrhages in many areas	Diffuse haemorrhages scattered in some area of the liver (Fig. 3)
Gr. IV	..	Diffuse haemorrhages on the liver parenchyma	A few hepatic cells showed necrobiotic changes. Slight diffuse haemorrhages on liver parenchyma noticed. Disruption of hepatic cords. (Plate II Fig. 4).
Gr. V	..	Small focal area of haemorrhage and vacuoles with multiple well defined borders.	A few of the hepatic cells showed fatty changes. Vacuoles were seen as single one with multiple small vacuoles with well defined borders. Small focal area of haemorrhage (Fig. 5).
Gr. VI	..	normal	Few of the hepatic cells showed slight degenerative changes and occasional cell had become necrotic (Fig.6).

Gr. VII	.. ..	Enlargement of space disse
Gr.VIII	.. ..	Mild diffuse fatty degeneration

General behaviour of the birds.

There appeared to be no difference in the behaviour of the groups of birds when handled for weight recording or when observed in cages.

PLATE - I

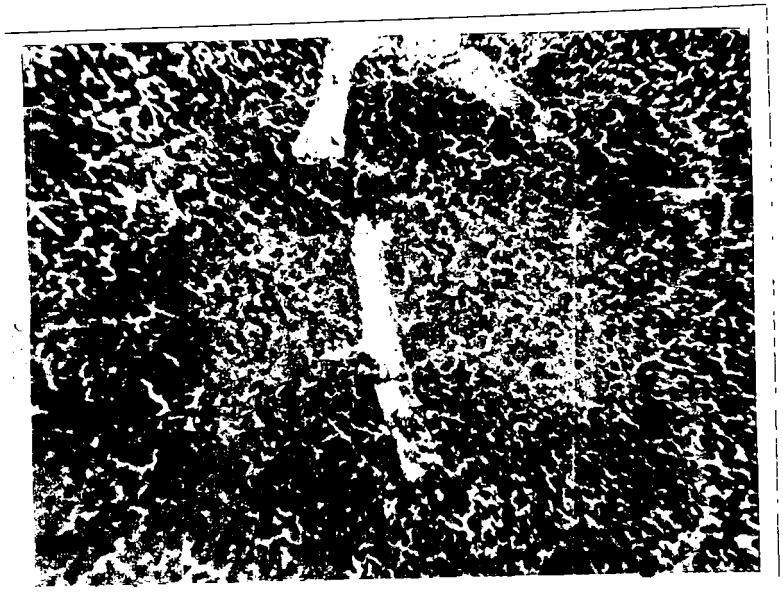


FIG. I

FIG. III

Table - III

Growth rate in chicken maintained on basal diet from 0 through 30 days, in kg.

S.No.	Chick No.	Initial wt.	at day 8	at day 16	at day 30
1.	64	0.310	0.500	0.610	1.000
2.	39338	0.315	0.520	0.670	Sacrificed at day 16
3.	39362	0.325	0.580	0.730	1.150
4.	38789	0.350	0.520	0.640	0.980
5.	38387	0.350	0.530	0.650	0.900
6.	38770	0.350	0.530	0.655	0.950
7.	39375	0.350	0.510	0.650	0.950
8.	39438	0.360	0.550	0.670	0.950
9.	39456	0.375	0.540	0.610	0.850
10.	38784	0.380	0.550	0.660	0.950
11.	39215	0.380	0.540	0.690	1.000
12.	38417	0.380	0.615	0.750	1.050
13.	38496	0.400	0.540	0.660	1.000
14.	39405	0.420	0.650	0.830	1.200
15.	39206	0.420	0.650	0.770	1.200
16.	38477	0.430	0.650	0.800	1.100
17.	38738	0.435	0.630	0.760	1.050
18.	38457	0.440	0.660	0.830	1.200
19.	39328	0.450	0.640	0.760	1.100
20.	39411	0.470	0.640	0.770	1.150
21.	38407	0.500	0.740	0.890	1.250
Mean		0.390	0.584	0.716	1.045

Table - V

Growth rate in chicken maintained on basal diet plus 0.4% "meprobamate" from 0 through 30 days, in kg.

S.No.	Chick No.	Initial wt.	at day 8	at day 16	at day 30
1.	39770	0.310	0.410	0.450	0.600
2.	39738	0.310	0.470	0.530	0.750
3.	38453	0.320	0.540	0.620	0.950
4.	38475	0.330	0.470	0.560	0.800
5.	38457	0.350	0.540	0.620	0.900
6.	39408	0.360	0.590	0.670	1.000
7.	39442	0.360	0.550	0.650	0.750
8.	38	0.365	0.570	0.660	Sacrificed at day 16
9.	38401	0.370	0.510	0.690	Died at day 28
10.	133	0.380	0.600	0.720	1.050
11.	38489	0.385	0.610	0.710	1.050
12.	38717	0.390	0.580	0.690	1.000
13.	38396	0.395	0.610	0.705	1.000
14.	39488	0.400	—	—	Died at day 8
15.	38442	0.410	0.605	0.730	1.000
16.	38454	0.420	0.610	0.730	0.950
17.	787	0.420	0.630	0.730	0.900
18.	39484	0.460	0.700	0.840	1.200
19.	38476	0.460	0.700	0.800	1.050
20.	39202	0.500	0.680	0.820	1.100
21.	38416	0.515	0.750	0.880	1.050
	Mean	0.390	0.586	0.687	0.950

Table - VI

Growth rate in chicken maintained basal diet plus 0.6% "meprobamate" from 0 through 30 days, in kg.

S.No.	Chick No.	Initial wt.	at day 8	at day 16	at day 30
1.	39498	0.310	0.510	0.630	0.950
2.	38740	0.310	0.500	0.605	0.900
3.	38771	0.310	0.500	0.600	0.900
4.	38389	0.340	0.510	0.605	0.850
5.	38448	0.350	0.530	—	Died at day 10
6.	39370	0.360	0.550	0.560	Died at day 24
7.	38777	0.370	0.560	0.650	Sacrificed at day 16
8.	39335	0.375	0.620	0.740	0.900
9.	38786	0.375	0.650	0.800	0.860
10.	29203	0.380	0.600	0.750	1.100
11.	39441	0.380	0.610	—	Died at day 10
12.	39417	0.390	0.600	0.700	1.000
13.	38451	0.400	0.640	0.830	1.050
14.	39355	0.405	0.610	0.655	Died at day 21
15.	39351	0.410	00650	—	Died at day 11
16.	39313	0.410	0.660	0.790	1.100
17.	39496	0.415	0.560	0.650	0.700
18.	579	0.430	0.610	0.700	0.850
19.	39372	0.460	0.750	0.890	1.250
20.	70	0.500	0.740	0.850	1.100
21.	39383	0.500	0.740	0.850	1.150
	Mean	0.389	0.604	0.714	0.977

Table - VII

Growth rate in chicken maintained on basal diet plus 1.2% meprobamate from 0 through 30 days, in kg.

S.No.	Chick No.	Initial wt.	at day 8	at day 16	at day 30
1.	39432	0.300	0.450	0.755	1.000
2.	39451	0.300	0.400	0.480	0.650
3.	38485	0.300	0.470	—	Died at day 13
4.	39402	0.350	0.470	—	Died at day 10
5.	38418	0.350	0.490	0.595	0.950
6.	39495	0.370	0.550	0.680	Died at day 22
7.	39375	0.370	0.500	0.620	0.850
8.	39376	0.375	0.500	0.610	1.000
9.	38430	0.380	0.500	0.600	0.850
10.	39430	0.380	0.565	0.660	0.950
11.	45	0.380	0.570	0.690	Sacrificed at day 16
12.	39350	0.380	0.600	0.715	0.975
13.	38435	0.410	0.600	0.660	Died at day 29
14.	38432	0.410	0.550	0.680	0.900
15.	39403	0.410	0.610	0.710	Died at day 24
16.	39395	0.430	0.550	0.640	0.850
17.	39485	0.435	0.570	0.690	Died at day 29
18.	38412	0.465	0.670	—	Died at day 11
19.	38468	0.470	0.635	—	Died at day 11
20.	38736	0.475	0.650	0.720	1.000
21.	84	0.490	0.740	0.850	1.000
	Mean	0.390	0.554	0.670	0.914

Table - VIII

Growth rate in chicken maintained on basal diet plus .005% nitrazepan from 0 through 30 days, in kg.

S.No.	Chick No.	Initial wt.	at day 8	at day 16	at day 30
1.	39715	0.320	0.530	0.660	1.050
2.	39201	0.350	0.500	0.590	0.900
3.	38500	0.350	0.510	0.630	0.950
4.	38491	0.355	0.550	0.660	1.100
5.	38441	0.360	0.440	0.570	0.900
6.	39490	0.360	0.560	0.710	1.200
7.	39440	0.360	0.500	0.610	0.900
8.	39463	0.370	0.500	0.590	0.950
9.	39406	0.375	0.560	0.680	0.950
10.	39471	0.375	0.610	0.710	1.100
11.	39379	0.380	0.560	0.660	Sacrificed at day 16
12.	39477	0.390	0.600	0.740	1.050
13.	38419	0.395	0.610	0.760	1.100
14.	39470	0.400	0.590	0.710	1.100
15.	38477	0.405	0.610	0.755	1.150
16.	38720	0.415	0.640	0.760	1.100
17.	38800	0.425	0.700	0.870	1.100
18.	39460	0.430	0.600	0.790	1.100
19.	39443	0.430	0.680	0.840	1.000
20.	38482	0.450	0.660	0.800	1.050
21.	39407	0.500	0.730	0.850	1.200
	Mean	0.390	0.582	0.711	1.040

Table - IX

Growth rate in chicken maintained in basal diet plus 0.01% nitrofurazone from 0 through 30 days, in kg.

S.No.	Chick No.	Initial wt.	at day 8	at day 16	at day 30
1.	38480	0.305	0.460	0.555	0.800
2.	38487	0.310	0.430	0.510	0.800
3.	39212	0.330	0.520	0.610	Died at day 27
4.	38718	0.355	0.530	0.730	1.150
5.	39366	0.360	0.540	0.630	0.900
6.	39421	0.360	0.540	0.640	1.000
7.	38450	0.365	0.630	0.740	1.000
8.	39359	0.370	0.610	0.660	1.000
9.	39214	0.370	0.450	0.700	0.950
10.	38452	0.375	0.580	0.710	1.100
11.	39473	0.380	0.520	0.560	0.650
12.	39357	0.385	0.600	0.650	Sacrificed at day 16
13.	38498	0.390	0.570	0.750	1.200
14.	39395	0.405	0.630	0.730	1.000
15.	39209	0.410	0.600	0.705	1.050
16.	39380	0.410	0.600	0.740	1.050
17.	17	0.415	0.610	0.730	1.050
18.	39425	0.450	0.690	0.810	1.050
19.	39385	0.460	0.700	0.860	1.200
20.	39207	0.490	0.710	0.860	1.150
21.	38438	0.510	0.750	0.800	1.050
	Mean	0.390	0.568	0.702	1.005

Table - X

Growth rate in chicken maintained on basal diet plus nitrogen 0.015% from 0 through 30 days, in kg.

S.No.	Chick No.	Initial wt.	at day 8	at day 16	at day 30
1.	39462	0.300	0.440	0.500	0.800
2.	38462	0.310	0.450	0.600	1.000
3.	39486	0.330	0.530	0.630	0.950
4.	39392	0.350	0.480	0.600	0.800
5.	39458	0.355	0.560	0.700	1.000
6.	38440	0.360	0.560	0.630	1.000
7.	38405	0.360	0.570	0.660	1.000
8.	38494	0.370	0.650	0.820	1.300
9.	90	0.380	0.640	0.720	0.950
10.	39439	0.380	0.560	0.660	Sacrificed at day 16
11.	38723	0.385	0.540	0.600	0.850
12.	39434	0.385	0.600	0.760	1.200
13.	39464	0.400	0.530	0.610	1.000
14.	35714	0.400	0.600	0.750	1.000
15.	61	0.405	0.640	0.730	1.000
16.	39399	0.410	0.650	0.750	0.850
17.	38399	0.420	0.700	0.850	1.200
18.	39468	0.440	0.740	0.750	0.950
19.	39465	0.450	0.710	0.840	1.200
20.	35796	0.465	0.750	0.900	1.300
21.	39403	0.500	0.770	0.940	1.200
	Mean	0.389	0.584	0.714	1.020

Table - XI

Summarised table of growth, feed consumption and Feed Efficiency in chicken reared from 0 through 30 days

S.No.	Item	Feed Gr.I Basal	Gr.II	Gr.III	Gr.IV.	Gr.V	Gr.VI	Gr.VII	Gr.VIII
1.	Feed consumed, in kg	41.090	39.190	39.090	38.000	33.640	40.340	37.715	36.465
2.	No. of chicken days	616	607	592	542	525	616	613	616
3.	Average feed consumed per chick per day, in g	66.7	64.5	66.0	70.0	64.0	65.4	60.6	59.1
4.	Average gain in weight per chick, in kg	0.665	0.641	0.560	0.588	0.524	0.650	0.615	0.631
5.	Average gain in weight per chick per day, in g	21.8	21.3	18.6	19.6	17.4	21.6	20.5	21.0
6.	Feed per unit gain	3.05	3.02	3.54	3.57	3.67	3.02	2.95	2.81
7.	Feed cost per kg gain a) Cost of feed, Rs.	4.80	4.76	5.58	5.60	5.78	4.76	4.65	4.43
	b) Cost of Drug, Rs.	—	4.32	7.78	11.78	24.22	4.98	9.74	13.91
	c) Total, Rs.	4.80	9.08	13.36	17.38	31.00	9.74	14.39	18.34

Table - XII

Analysis of Variance of body weights of chicken reared from 0 through 30 days

S.No.	Source	df	ss	mss	F
1.	Treatment	7	0.251	0.036	3.273 *
2.	Error	135	1.517	0.011	—
3.	Total	142	1.768	—	—

F at 5% = 2.08 * Significant at 5%

Critical values for the comparison of treatments.

Groups	I	II	III	IV	V	VI	VII	VII
I	-	0.065	0.068*	0.071	0.076*	0.065	0.065	0.065
II	-	—	0.068*	0.071	0.076*	0.065	0.068	0.065
III	-	—	—	0.071	0.059	0.068*	0.068	0.068*
IV	-	—	—	--	0.081	0.071*	0.071	0.071
V	-	—	—	—	—	0.076*	0.076*	0.076*
VI	-	—	—	—	—	—	0.065	0.065
VII	-	—	—	—	—	—	—	0.065
VIII	-	—	—	—	—	—	—	—

* Significant.

Table - XIII

Mortality pattern of the chicken under different dietary treatments from 0 through 30 days

S.No.	Treatment	Initial Number.	0-8 days	0-16 days	0-30 days	Chicken sacrificed	Mortality	Mortality %	Cause of Mortality.
1.	Group I	21	21	21	20	1	-	-	—
2.	Group II	21	21	20	19	1	1	4.76	Multiple haemorrhage
3.	Group III	21	21	20	18	1	2	9.52	Multiple haemorrhage
4.	Group IV	21	21	17	15	1	5	23.80	Multiple haemorrhage
5.	Group V	21	21	16	12	1	8	38.09	Multiple haemorrhage
6.	Group VI	21	21	20	20	1	-	-	—
7.	Group VII	21	21	20	19	1	1	4.76	Pericarditis and enteritis.
8.	Group VIII	21	21	20	20	1	-	-	—

PLATE - II

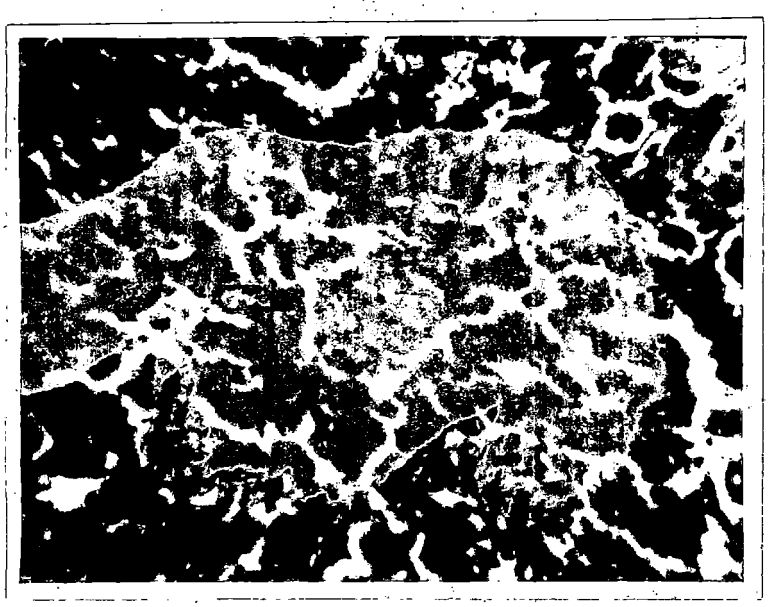


FIG. IV

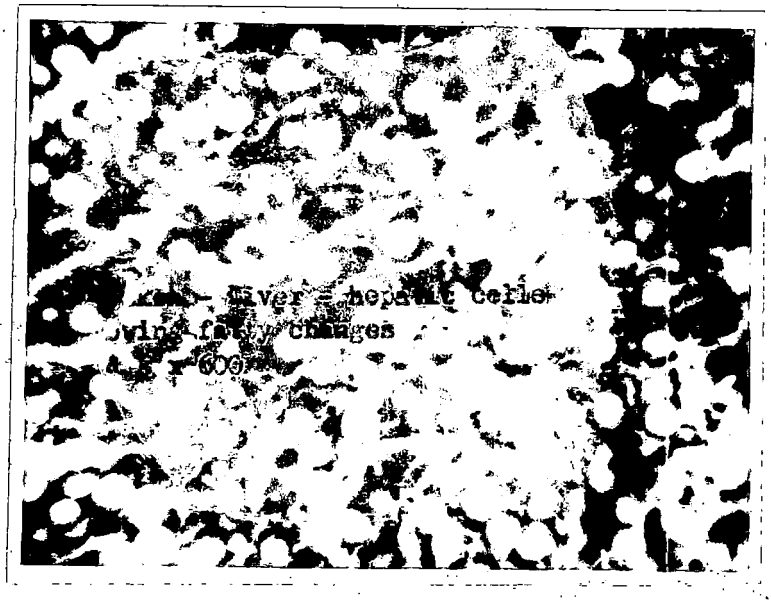


FIG. V

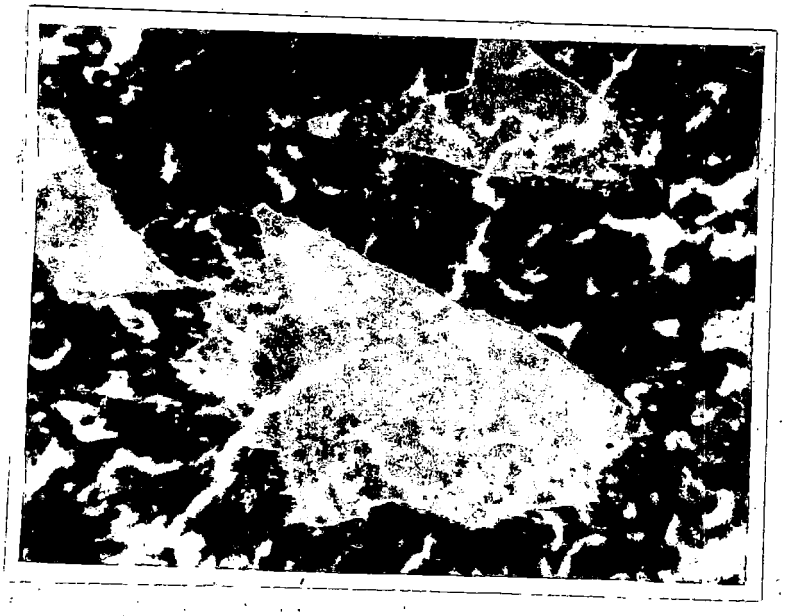


FIG. VI

Table - XIV

Haematology of the broiler chicken at 30 days of age (at day 0)

S.No.	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time mte., sec.
1.	39467	5.20	2.11	12.801	1	46	52	1	-	8 mt, 10 sec.
2.	38500	4.90	2.30	11.090	2	47	50	1	-	10 mt, 10 sec.
3.	39459	4.50	1.91	10.237	1	48	48	2	1	9 mt, 30 sec.
4.	39493	5.10	2.22	11.928	1	49	47	2	1	9 mt, 10 sec.
	Mean	4.92	2.06	11.514	1.25	47.5	29.25	1.5	0.5	9 mt, 15 sec.

Table - XV

Haematology of the chicken sacrificed at day 16.

Treatment	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time
Gr. I	39303	5.8	2.10	12.240	2	50	48	-	-	6 mt, 10 sec.
Gr. II	38778	6.1	2.20	14.510	3	49	46	-	1	10 mt, 20 sec.
Gr. III	38	5.9	2.3	11.210	2	48	48	1	1	19 mt, 50 sec.
Gr. IV	38777	6.05	1.98	10.670	3	47	48	2	-	19 mt, 15 sec.
Gr. V	45	5.70	1.90	9.812	4	50	45	1	-	24 mt, 30 sec.
Gr. VI	39379	5.7	2.10	12.170	2	49	49	1	1	6 mt, 40 sec.
Gr. VII	39337	6.0	2.10	11.220	2	49	46	1	-	9 mt, 10 sec.
Gr. VIII	39439	8.2	2.21	11.586	3	48	49	-	-	10 mt, 20 sec.

Table - XVI

Haematology of the broiler chicken maintained on basal diet at day 30.

Sl. No.	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time
1.	38477	7.20	2.90	9.008	3	47	48	1	1	7 mt, 50 sec.
2.	38457	6.20	1.80	8.095	2	46	49	2	1	8 mt, 10 sec.
3.	39395	6.20	3.10	8.127	4	46	46	2	2	6 mt, 10 sec.
4.	38770	6.40	2.60	10.140	3	47	48	1	1	10 mt, 30 sec.
	Mean	6.50	2.60	8.842	3	465	47.2	1.5	1.25	8 mt, 10 sec.

Table – XVII

Haematology of the broiler chicken maintained on basal diet plus meprobamate 0.2% at day 30.

S. No.	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time
1.	39423	6.20	2.00	10.400	3	46	50	–	1	8 mt, 20 sec.
2.	39415	5.50	2.10	11.201	2	48	49	1	–	10 mt, 10 sec.
3.	39377	6.80	2.40	10.240	4	46	48	2	–	7 mt, 50 sec.
4.	39791	7.00	2.60	9.878	3	45	50	1	1	12 mt, 15 sec.
	Mean	6.37	2.47	10.429	3.0	46.25	49.25	1	0.5	9 mt, 39 sec.

Table - XVIII

Haematology of Broiler chicken maintained on basal diet plus meprobamate 0.4% at day 30.

S.No.	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time
1.	38453	7.20	2.08	12.016	2	48	46	1	1	18 mt, 10 sec.
2.	133	8.00	2.31	10.103	4	46	46	2	2	15 mt, 40 sec.
3.	39342	7.80	2.13	9.878	3	46	49	1	1	16 mt, 10 sec.
4.	38476	6.80	2.02	10.121	2	48	48	2	-	18 mt, 10 sec.
	Mean	7.45	2.13	10.530	2.75	47	47.25	1.5	1.0	16 mt, 23 sec.

Table - XIX

Haematology of broiler chicken maintained on basal diet plus meprobamate 0.6% at day 30.

S.No.	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time
1.	39498	6.80	2.02	10.148	2	49	49	-	-	14 mt, 10 sec.
2.	70	7.20	1.92	8.617	3	47	48	1	1	14 mt, 20 sec.
3.	39496	7.80	2.10	9.148	2	46	48	2	2	10 mt, 10 sec.
4.	38451	7.20	1.89	10.231	3	48	47	2	-	16 mt, 30 sec.
	Mean	7.25	1.98	9.536	2.5	47.5	48.0	1.0	0.75	14 mt, 47 sec.

Table - XX

Haematology of the broiler chicken maintained on basal diet plus meprobamate 1.2%, at day 30.

S.No.	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time.
1.	38485	5.20	1.90	9.064	2	50	48	-	-	22 mts, 20 sec.
2.	38435	5.40	2.00	10.141	-	52	46	-	2	15 mts, 30 sec.
3.	39485	4.80	1.80	8.674	1	50	49	-	-	18 mts, 20 sec.
4.	39376	6.20	1.80	10.156	1	49	48	2	-	16 mts, 30 sec.
	Mean	5.40	1.87	9.508	1.0	50.2	47.7	5	5	18 mts, 25 sec.

Table - XXI

Haematology of the broiler chicken maintained on basal diet plus nitrazepam 0.005%, at day 30.

S.No.	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time
1.	38715	5.80	2.60	14.080	2	45	52	-	1	8 mte, 15 sec.
2.	39443	6.20	2.10	16.178	3	48	49	-	-	9 mte, 50 sec.
3.	39406	6.80	1.94	12.016	-	46	53	-	1	8 mte, 15 sec.
4.	38500	5.80	2.01	8.814	1	50	47	2	-	10 mte, 10 sec.
	Mean	6.15	2.21	12.772	1.5	47.2	50.2	0.5	0.5	9 mte, 07 sec.

Table - XXII

Haematology of the broiler chicken maintained on basal diet plus nitrazepam 0.01%, at day 30.

S.No.	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time
1.	38480	5.55	2.64	16.435	4	46	46	4	0	5 mts, 10 sec.
2.	39385	6.80	1.86	12.330	4	40	52	3	1	4 mts, 50 sec.
3.	39421	6.20	2.32	10.280	3	46	50	1	-	7 mts, 20 sec.
4.	39380	6.20	2.04	11.140	3	46	49	2	-	6 mts, 40 sec.
	Mean	6.18	2.21	12.546	3.5	44.5	49.25	2.5	0.25	6 mts, 10 sec.

Table - XXIII

Haematology of the broiler chicken maintained on basal diet plus nitrazepam 0.015%, at day 30.

S.No.	Chick No.	Hb g/100 ml	RBC $10^6/m^3$	WBC $10^3/m^3$	E%	H%	L%	M%	B%	Clotting time.
1.	38486	5.80	2.34	12.240	2	50	46	2	-	7 mte, 5 sec.
2.	61	6.00	2.50	10.180	3	46	45	4	2	6 mte, 30 sec.
3.	38399	6.20	1.96	9.871	4	45	49	1	1	8 mte, 10 sec.
4.	90	4.20	1.34	11.241	3	48	47	2	1	7 mte, 20 sec.
	Mean	5.55	2.03	10.883	3	47.2	46.7	2.25	0.75	7 mte, 16 sec.

Table - XXIV

Summarised table for the haematology of broiler chicken under different dietary treatments at 30 days.

S. No.		Initial value at day 0	Gr.I	Gr.II	Gr.III	Gr.IV	Gr.V	Gr.VI	Gr.VII	Gr.VIII	No. of observations
1.	Hb gm/100 ml	4.92	6.50	6.37	7.45	7.25	5.40	6.15	6.18	5.55	4
2.	RBC/10^6cm	2.06	2.60	2.47	2.13	1.90	1.87	2.21	2.21	2.03	4
3.	WBC/10^3cm	11.514	8.842	10.429	10.530	9.536	9.508	12.772	12.546	10.883	4
Differential count.											
4.	Eosinophil %	1.25	3	3.0	2.75	2.5	1.0	1.5	3.5	3	4
5.	Heterophil %	47.5	46.5	46.25	47.0	47.5	50.2	47.2	44.5	47.2	4
6.	Lymphocyte %	49.25	47.2	49.25	47.25	48	47.7	50.2	49.25	46.7	4
7.	Monocyte %	1.5	1.5	1	1.5	1.0	0.5	0.5	2.5	2.25	4
8.	Basophil %	0.5	1.25	0.5	1.0	0.75	0.5	0.5	2.5	0.75	4
9.	Clotting time (mts. sec.)	9,15	8,10	9,39	16,23	14,47	18,25	9,07	6, 0	7,16	4

DISCUSSION

DISCUSSION

Growth.

It will be seen from the summarised table XI that the chicken maintained on the basal diet had the maximum gain in weight during the trial period from 0 - 30 days and that they had the highest average daily gain in weight. Incorporation of tranquillisers in broiler ration in the present study did not promote growth rate. Addition of nitrazepam in the diet in general, had manifested better growth rate than the addition of meprobamate. Incorporation of nitrazepam in the diet at a level of 0.005% did not inhibit growth in chicken. The chicken that were receiving 0.01% nitrazepam in the diet in Gr.VII had the lowest average daily gain in weight amongst the three levels involved in the present study. The apparent variation between treatments noticed in growth rate was not statistically significant.

The chicken maintained on diets incorporated with meprobamate had retarded growth rate and lower average daily gain as the dose levels were increased from 0.2% to 0.4% and from 0.6% to 1.2%. An apparent but non-significant higher average daily gain was observed in chicken in Gr.IV than in Gr.III. Addition of meprobamate at the lowest level studied (0.2%) in Gr.II had an almost equal rate of growth when compared to Gr.I chicken on basal diet. Significantly low growth rate was observed in chicken, Gr.III (0.4%) and Gr.V (1.2%). The observation that meprobamate inhibited growth in chicken at all levels involved in the present study was in full agr[illegible] with that of Babcock and Taylor (1957) and Carren and Hill (1957).

Feed consumption and feed efficiency.

The chicken maintained on the basal diet in Gr.I had consumed maximum feed (41.09 kg) during the course of the present study (Table XI) than any of the groups, the average feed consumption per day had been 66.7 g. It can be seen that as the dose level of nitrazepam in the diets was increased, a progressive decrease in feed consumption was evident in the respective dietary treatments. The trend in feed consumption had reflected on the growth rate of the chicken. The feed required per unit gain in weight in chicken maintained on basal diet was 3.05. An increase in feed efficiency was observed from 3.02 to 2.95 and 2.81 as the level of nitrazepam in the diet was increased from 0.005% in Gr.VI to 0.01% in Gr.VII and 0.015% in Gr.VIII respectively. The difference in feed efficiency was not appreciable when compared between treatments. The observations of Nair (1976) in broiler chicken in regard to feed efficiency viz., 2.8; 2.9 and 3.5 when rations supplemented with 'Neftin-50','TM-5' and '3-Nitro Hoechst' respectively were favourably comparable with the addition of nitrazepam in the diets for broiler chicken.

Although addition of meprobamate in the diets had slightly improved the feed efficiency at the lowest level studied (3.02 in Gr.II), higher dose levels viz, 0.4%, 0.6% and 1.2% brought about a progressive decrease. The feed efficiency was 3.54 in Gr.III, 3.57 in Gr.IV and 3.67 in Gr.V. Similar observations were reported by Babcock and Taylor (1957) in cockrels maintained on rations supplemented with meprobamate at levels ranging from 0.2% to 2.2%.

The feed required per unit gain remained unaltered in treatment groups III and IV (3.54 and 3.57 respectively) was in agreement with the observations made by Babcock and Taylor (1957) on similar levels of meprobamate in poultry rations.

Mortality.

As the trial advanced from 0 to 30 days, meprobamate not only inhibited growth rate but, it drastically reduced the availability of chicken in the respective groups depending on the dosage level of the drug. The availability of chicken in terms of 'chicken days' was reduced from a normal figure of 616 at day 30 in Gr.I to 607, 592, 542 and 525 in groups II, III, IV and V respectively, the reduction had been due to mortality (Table XI). Mortality was consistent with increasing levels of meprobamate in the diet. The per centage of mortality was lowest 4.76 in 0.2% of meprobamate in the diet (Table XIII), 9.52 in 0.4%, 23.8 in 0.6% and 38.09 in 1.2%. The mortality in chicken maintained on diets supplemented with meprobamate had thus reached the extreme condition of death than the suggestion made by Garren and Hill (1957) that the drug might be slightly toxic to chicken. In as much as the leisons observed on autopsy of the birds died during the course of the trial were almost similar such as multiple haemorrhage and that there was no such mortality in any other groups, the toxicity of meprobamate on continued feeding was almost certain. Leisons observed on autopsy of the bird died in Gr.VI were suggestive of pericarditis and enteritis and could not be attributed

to the effects of nitrazepam. It is interesting to note that multiple haemorrhage was consistantly seen in all the toxic cases of meprobamate. Such a multiple haemorrhage and purpurea has been reported as a side effect for meprobamate by other workers. But the occurrence of similar side effect has been very few in human practice. The general incidence of such a toxicity in the experiment suggests that the birds are more sensitive to this 'side effect' than other animals. The exact mechanism of causing the multiple haemorrhage is not yet fully understood. This may be either affecting the clotting mechanism or increasing the cappillary permeability.

Haematology.

The summarised values on haematology of chicken (Table XXIV) revealed that the drugs had not influenced the nature and content of the blood constituents studied. However, the R.B.C. count, total and differential leucocyte count while remaining almost similar in birds maintained on diets supplemented with nitrazepam and on basal diet, there was reduction of such values in birds receiving meprobamate. There appeared to have a tendency for longer clotting time for the chicken that had received meprobamate, reaching at the highest in birds receiving 1.2% in the diet. This increased clotting time might be attributed to the hepatotoxicity of the drug at the dose levels given.

Histopathological studies.

On histopathological examination of the liver, diffuse haemorrhage

scattered all over the liver parenchyma, disruption of hepatic cord, multiple vacuoles and necrotic changes observed in chicken maintained on diets incorporated with meprobamate were suggestive of toxicity of the drug at any of the dose levels involved in the present study. The histopathological lesions in the liver of chicken receiving nitrazepam were very mild and insignificant.

General behaviour of the birds.

Tranquillisers involved in the present study had little effect on the general behaviour of the birds either when handled or observed in cages. Although Garren and Hill (1957) had made a similar observation, it was at variance with the reports of Selling (1955) and Borrus (1955) in regard to the calming effect of meprobamate in man and experimental animals. It could be inferred that the drugs were not effective in reducing anxiety and stress in chicken.

Economics.

Feed cost per kg of gain in broiler chicken fed with and without the addition of tranquillisers in diets, revealed that incorporation of meprobamate at 1.2% in Gr.V was toxic to the birds and least economic (Rs.31.00) followed by Rs.17.33 in Gr.III and Rs.9.08 in Gr.II. Similarly, a higher feed cost was observed in diets supplemented with nitrazepam viz., Rs.18.34 in Gr.VIII, Rs.14.39 in Gr.VII and Rs.9.74 in Gr.VI. On the other hand, chicken maintained on the basal diet without the addition of tranquillisers, the feed cost had been Rs.4.80 per kg of gain (Table XI).

It may be noted that the tranquillizers incorporated in the present study were purchased in the tablet form intended for human patients from the retail market. Several factors influence the market price of drugs. Generally, there would be several fold increase in the cost of raw drugs at the point of manufacture and the finished product at the actual consumer and the difference would be still wider between certain preparations intended for human and veterinary practice. To cite an example, it has been seen that there is about twelve fold increase in the price of tetracyclines in human therapeutic dosage form than that are generally used as animal feed supplements. Hence the cost of feed worked out on addition of tranquillizers in the present study has been exaggerated to this extend and cannot be relied. However, it imparts ample opportunity to assess the relative merit of the different dietary treatments.

Meprobamate had not promoted higher growth rate in chicken and it was toxic on continued feeding at any of the levels studied and hence not advisable to incorporate in practical broiler rations. Since there had not been an appreciable higher growth rate and any added advantage in supplementing broiler rations with nitrazepam and all the more the prohibitively high cost of the drug were sufficient grounds to refrain from making positive recommendations at the prevailing prices of nitrazepam.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

A feeding trial of 30 days duration was instituted in the Department of Pharmacology, College of Veterinary and Animal Sciences, Mannuthy to determine the effect of two tranquillizers such as meprobamate and nitrazepam on weight gains when incorporated as feed additives in the rations for broiler chicken. At 30 days of age, the chicken were weighed and randomly assigned to eight groups of 21 each as uniformly as possible in respect of body weight and raised in weaner batteries with vertical tiers each group having received a floor space of 2 m^2. The group that had received basal diet alone formed as control while another four groups each had received basal diet incorporated with meprobamate ('Equanil') at levels of 0.2%, 0.4%, 0.6% and 1.2% of the diet and the remaining three groups received diets supplemented with nitrazepam ('Hypnoter') at levels of 0.005%, 0.01% and 0.015% respectively. The chicken in all groups were maintained on starter diet till 39 days of age and switched on to finisher for the remaining period of the trial with and without the addition of respective drugs. Feed and water were provided to the chicken ad libitum and all the chicken were under identical conditions of management. The individual weights of the chicken at the initial, at day 8, 16 and at day 30 were recorded. Haematological studies and histopathological examinations of the liver of the chicken prior to the commencement of the experiment, at day 16 and on termination of the trial were carried out. Feed consumed by the chicken during the trial period was recorded. Feed efficiency and

feed cost per kg gain in weight on different dietary treatments were worked out.

The following observations were made:

1. The chicken maintained on the basal diet had the maximum gain in weight during the trial period from 0 - 30 days.

2. Incorporation of tranquillizers in the broiler rations did not promote growth rate faster than the chicken on the basal diet.

3. Addition of nitrazepam in feed had manifested better growth rate than the addition of meprobamate.

4. Chicken maintained on basal diet had consumed maximum feed than any other groups, the average daily feed consumption in them had been 66.7 g.

5. As the dose level of nitrazepam in the diets was increased, a progressive decrease in feed consumption was noticed.

6. Feed required per unit gain in weight in chicken maintained on basal diet was 3.05. An increase in feed efficiency was observed commensurate with the increasing level of nitrazepan in the diet, while meprobamate at all levels studied were found to be toxic to the chicken.

7. Addition of meprobamate in the diet caused mortality in chicken, the maximum being in groups that had received the highest dose level.

8. There appeared to have a tendency for longer clotting time for

the blood in chicken that had received meprobamate in the diet.

9. Histopathological examination of the liver showed diffuse haemorrhage scattered all over the liver parenchyma, disruption of hepatic cord and multiple vacuoles and necrotic changes in chicken maintained on diets incorporated with meprobamate.

10. Neither meprobamate nor nitrazepam had any effect on the general behaviour of the birds either when handled or observed in cages.

11. Addition of tranquillizers in the diet enhanced the cost per kg gain in weight of the chicken several folds than the chicken on the basal diet.

From the above observations it was concluded that:

1. Addition of tranquillizers in the diets for broiler chicken had not produced any beneficial effect in regard to weight gain, feed efficiency and economics of weight gain.

2. There was no added advantage on adding tranquillizers involved in the present study in the basal diet for that chicken.

3. Meprobamate was found to be toxic to broiler chicken.

4. Addition of tranquillizers in the diets for broiler chicken was uneconomic.

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ABSTRACT

EFFECTS OF TRANQUILLIZERS ON WEIGHT GAIN IN BROILERS

BY
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ABSTRACT OF A THESIS

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ABSTRACT

A feeding trial of 30 days duration was carried out in 30 days-old commercial broiler chicken to study the effects of two tranquillizers such as meprobamate and nitrazepam incorporated at varying levels in the rations for chicken, on weight gains. There were eight dietary treatments each consisted of 21 birds. The group that had received basal diet without the addition of tranquillizers formed as control while four groups each received basal diet incorporated with meprobamate (Equanil) at levels of 0.2%, 0.4%, 0.6% and 1.2% of the diet and the remaining three groups received diets added with nitrazepam (Hypnotex) at levels of 0.005%, 0.01% and 0.015% respectively.

The gain in body weight, feed consumption and haematology of the chicken were recorded and economics of weight gain in the respective treatments worked out. The results of the study indicated that incorporation of tranquillizers in the diet did not promote growth in chicken at a faster rate than the basal diet. Addition of meprobamate in the diet caused mortality in chicken, the maximum had been in groups that received the highest dose levels. There appeared to have a tendency for longer clotting time for the blood in chicken that had received meprobamate and that their liver showed varying degrees of degenerative changes. None of the tranquillizers at any of the levels studied had any effect on the general behaviour of the birds.

Based on the results it was concluded that addition of tranquillizers had not produced any beneficial effect in broiler chicken either in promoting a faster growth rate or n savings in feed consumption.