GENETIC BASIS OF EGG YOLK CHOLESTEROL IN MEYER STRAIN OF WHITE LEGHORNS

THESIS

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CERTIFICATE

This is to certify that the thesis entitled "GENETIC BASIS OF EGG YOLK CHOLESTEROL IN MEYER STRAIN OF WHITE LEGHORNS" submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Animal Genetics and Breeding to the Tamil Nadu Agricultural Univeristy, Coimbatore is a record of bonafide research work carried out by Thiru K.S. RAMAN under my supervision and guidance and that no part of this thesis has been submitted for the award of any other degree, diploma, fellowship or other similar titles or prizes and that the work has not been published in part or full in any scientific or popular journal or magazine.

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C O N T E N T S

INTRODUCTION

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One of the highly significant medical problems of the day concerns the various relation of dietary fats, including unsaturated fatty acids to ischaemic heart diseases and atherosclerosis. The medical war against heart attacks and strokes is primarily a battle against arteriosclerosis - the build up of cholesterol and other fatty substances in the walls of arteries. The fatty substances accumulate slowly in the form of patches known as plaques. Gradually, these plaques enlarge and become scarred in a painless but relentless process that leads to many mortal dangers. One such danger is the blockage of blood flow by large plaques, depriving the heart, brain and other organs of oxygen and other vital nutrients. Researches have shown that process of arteriosclerosis cannot be reversed or stopped, only slowed down at best.

There is extensive evidence that the level of cholesterol in the plasma of most people can be lowered by appropriate dietary modification. Generally such lowering can be achieved most practicably by partial replacement of the dietary sources of saturated fats with sources of polyunsaturated fatty acids and by a reduction in the consumption of foods rich in cholesterol.

Faithful and continued consumption of a cholesterol lowering diet over a period of years can reduce the coronary attack rate in middle aged men.

Fat controlled diets developed by the American Heart Association provided a maximum of 300 mg cholesterol daily, 35 percent of the kilocalories in the form of saturated fats and 10 percent of the kilocalories in the form of polyunsaturated fats (Zukel, 1969).

As per 1972 data on cholesterol content of foods published by the U.S. Department of Agriculture as quoted by Seeley et al. (1972) beef, lamb and pork were restricted to three ounces per week and only skim milk and lean meats may be used. No more than three egg yolks per week were permitted; liver may be used, but only as a substitute for eggs. Shellfish, except shrimp were allowed.

The American Heart Association fat controlled diets were designed to lower elevated levels of serum cholesterol and other lipids in an effort to reduce the risk of heart diseases (White and Havenstein, 1976). Significant and sustained reduction in serum cholesterol and plasma lipids have been demonstrated in persons adhering

to the diet. Low density lipoprotein (LDL) serum cholesterol levels were decreased and high density lipoprotein (HDL) serum cholesterol levels were increased (Hulley et al., 1977). Dietary intervention produced encouraging results of young survivors of acute myocardial infarctions (Loren, 1975). However Laird (1975) has stated that five to seven percent of the young children in the United States were genetically predisposed to atherosclerosis.

Due to vast development of poultry industry, the rate of consumption of eggs has been increased and the cholesterol content of egg has a bearing on the atherosclerotic heart disease in human. Avian eggs contain large amounts of cholesterol located exclusively in the yolk. Most of the portion of cholesterol exists as free cholesterol, a minor proportion is present as cholesteryl ester. Cholesterol when ingested in the form of egg yolk increases the circulating levels of lipid.

Egg yolk lipid differs from most other fat containing food stuffs in that it contains not only triglycerides but also phospholipids and large quantities of cholesterol.

Seventeen grams of egg yolk contains approximately 280 milligrams of cholesterol. Most of the fat is in the form of lipoprotein. Since the dietary cholesterol forms one of the sources which manipulates the levels of lipid in circulating blood.

The cholesterol content of egg yolk may be affected by genetic and a number of environmental factors. It can be lowered to 35 percent by feeding a plant sterol, sitosterol (Clarenburg et al., 1971). However, the genetic influence on the deposition of cholesterol in the yolk may set the limits within which environmental manipulations can change its level. One of the first studies to suggest a genetic basis for yolk cholesterol was that of Edwards et al. (1960) who found differences in yolk cholesterol levels between various breeds and strains. A number of studies have established that genetics may play a role in controlling cholesterol levels in many species. Wilcox et al. (1965) in serum cholesterol level in chickens; Harris and Wilcox (1963) in yolk cholesterol level in chickens; Lepore and Marks (1965) in cholesterol level in the eggs of Coturnix quail and Clarkson and Lofland (1970) in plasma cholesterol in Squirrel monkeys.

The present study aims to determine the genetic parameters of egg yolk cholesterol and plasma cholesterol

and their relationship with other economic traits in chickens. The ultimate objective is to explore the possibility of development of lines of chicken with low egg yolk cholesterol level.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

I Cholesterol:

As early as 1815, Chebreul gave the name cholesterine to a white waxy material which was isolated from gall stones. Later on the white waxy material was given the name cholesterol.

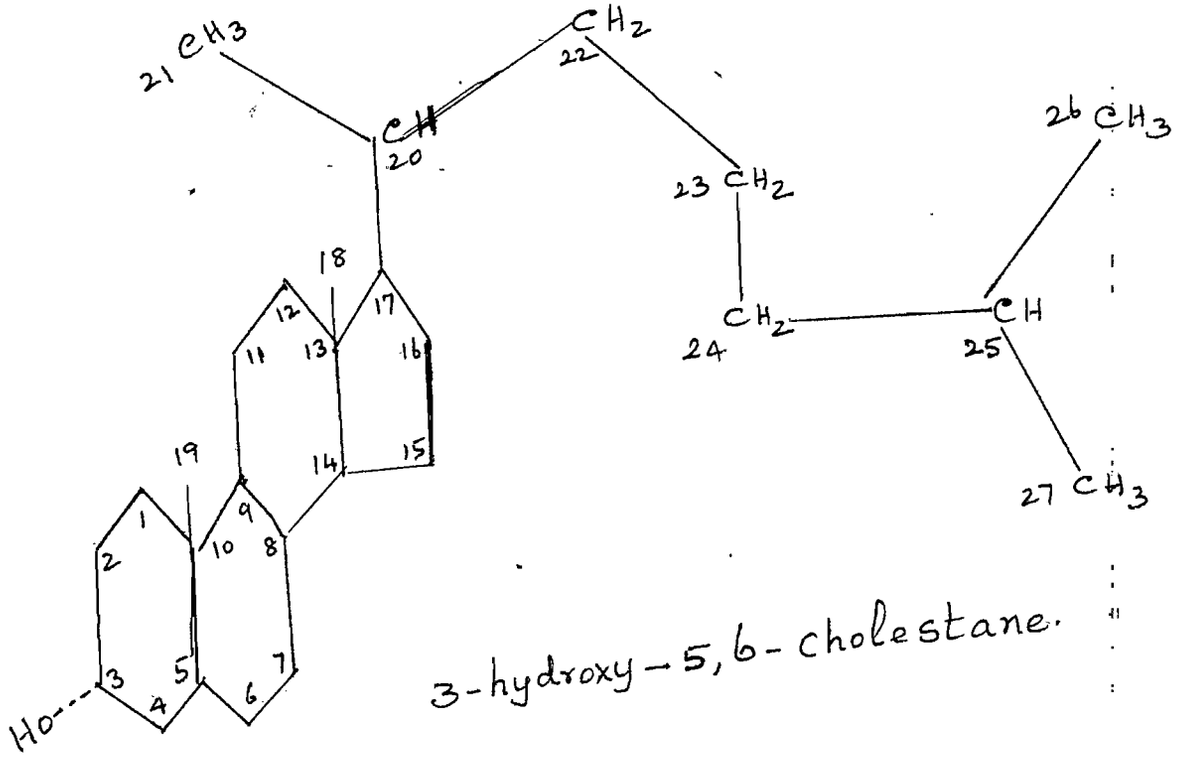
i) Biochemistry of cholesterol:

Ramakrishnan et al. (1980) has dealt with biochemistry, regulation and control of biosynthesis of cholesterol in detail.

Cholesterol comes under the main classification of non phosphorylated lipids and belongs to the group of steroids. The group derived lipid is a 'catch all' group in Bloor's classification. It includes the hydrolysis products of simple and compound lipids and also various other compounds such as steroids, terpenes, fatty acids, alcohols, fatty aldehydes, ketones etc. Cholesterol is the steroid alcohol (Steros^{G} = solid). All steroids may be considered as derivatives of a fused and fully saturated ring system called cyclopentanoperhydrophenanthrene or sterane.

Cholesterol (chole^{G} = bile) is the principal sterol of vertebrates and is especially abundant in nervous tissues,

brain, gall stones and bile. It is an essential constituent of animal cells. Its parent hydrocarbon is cholestane, $C_{27}H_{48}$. In addition to an OH group at C_3 there is a double bond between C_5 and C_6. Chemically it is hence named as 3 - hydroxy - 5, 6 - cholestane.



3-hydroxy-5,6-cholestane.

ii) Metabolism of cholesterol:

Cholesterol is essential to life and is present in all animal cells. Brain, egg yolk, liver, meat are good sources of cholesterol in the diet. Each person gets all his cholesterol in the body by biosynthesis from acetyl Co A from fatty acids, glucose and to a limited extent amino acids through the metabolites. An average non vegetarian diet provides only 0.3 g of cholesterol per day although the turnover of cholesterol in the body is about 1.3 g per day.

iii) Biosynthesis of cholesterol:

Cholesterol is synthesised from simple precursors like acetate and water. It can be synthesised from many of the intermediates from carbohydrates, protein and fatty acid metabolism, pyruvic acid, acetoacetic acid, alcohol, leucine, alanine and octanoic acid are found to act as precursors for cholesterol.

It is also found that all these compounds at some stages pass through acetyl Co A as well as HMG Co A (Hydroxy Methyl Glutaryl). Tissues known to synthesise cholesterol efficiently in the body include liver, intestines, adrenal cortex, skin and aorta. The enzymes concerned with the synthesis of cholesterol are present in microsomal fraction.

Pasternak (1979) stated that the cholesterol absorbed largely from the diet but synthesized by tissues such as liver and gastrointestinal tract, is metabolised in a variety of ways. Even metabolism to cholic acid and other bile acids (which are excreted) is not a fully degradative pathway, in that further oxidation of bile acids does not occur. Moreover bile acids are very effectively reabsorbed in the gut and are eliminated only slowly from the circulation. Cholesterol is the only food stuff and one of the few cell constituents, that is not capable of complete oxidation to Co_2 and H_2O by animals. Once absorbed into the circulation it cannot easily be eliminated; metabolism to the bile acid 'pool' soon becomes saturated and what cholesterol is excreted unchanged (in the bile) is largely reabsorbed in the lower intestine.

Regulation and control of cholesterol biosynthesis:

Feed back control:

Cholesterol inhibits its own synthesis in liver at the HMG Co A reductase enzyme stage and thus is involved in feed back control. This is effective only in the liver.

Fasting and diabetes:

HMG Co A reductase in liver is reduced during fasting, but not in diabetes mellitus. This explains the decreased

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synthesis of cholesterol during fasting. Its increased formation in diabets is due to diversion of acetyl Co A for cholesterogenesis. When dietary cholesterol is increased it is observed that the cholesterol biosynthesis is reduced proportionately to a certain limit but at the same time it could not be completely suppressed.

Species variation:

It is now believed that extra hepatic synthesis of cholesterol mainly in intestine, is also proceeding in certain species especially in man, which may be regulated by other factors than cholesterol in diet alone. There is a species variation regarding the assimilation of exogenous cholesterol. Cholesterol feeding causes hypercholesterolemia and onset of atherosclerotic lesions in rabbit, pig, monkey and man but not in dog, rat and cat which are resistant to it. In dogs and cats thyroidectomy or thiouracil treatment is necessary to produce hypercholesterolemia and increased deposition of cholesterol on aortic walls.

Hypophysectomy:

Cholesterol biosynthesis is depressed in hypophysectomised animals.

Hyper and hypothyroidism:

In hypothyroidism, basal metabolic rate is decreased, the oxidative process in the body get, depressed and the intermediary metabolites and diverted towards cholesterogenesis resulting in hypercholesterolemia. In hyperthyroidism, on the other hand, although the rate of cholesterol synthesis is increased its rate of turnover is very much higher, resulting in a lowering of the cholesterol to normal levels. Thyroid hormones facilitate the synthesis as well as the degradation of cholesterol, the latter effect being more predominant.

Insulin:

Insulin increases the overall oxidation of glucose and the peripheral oxidation of ketone bodies and other intermediates, more acetyl Co A is driven through citric acid cycle rather than the cholesterol pathway.

Androgens and estrogens:

Androgens increase cholesterol synthesis and in their absence cholesterol level is reduced. Estrogens not only decrease cholesterol synthesis but protect pre-menopausal woman from hypercholesterolemia.

Polyunsaturated fatty acids:

Polyunsaturated fatty acids bring down cholesterol in blood by esterfying, solubilising and mobilising the

cholesterol to liver. The oxidation of cholesterol and conversion to bile acids in liver is thus enhanced. Vegetable oils like corn oil and cotton seed oil are very good sources for polyunsaturated fatty acids, while animal fats like butter fat and beef fat are very poor sources.

Drugs and other agents:

Choloxin, atromids, and neomycin have a beneficial effect in decreasing cholesterol levels in blood by increasing the faecal excretion of coprosterol and bile acids which arise from the body's cholesterol reserves. Triparanol blocks the biosynthetic pathway of cholesterol at the desmosterol stage. Nicotinic acid, vanadium salts and ethyl-p-cholorophenoxy-isobutyrate are also cholesterol-lowering substances.

Catabolism of cholesterol and faecal excretion:

The liver is the main site of cholesterol catabolism. It is found that 60 percent of the total cholesterol of liver is converted to bile acids and excreted in bile. The rest (40%) is excreted as a solution into bile into intestine from where a part is reabsorbed, and another part is reduced by intestinal bacteria to coprosterol. The coprosterol is excreted in faeces along with bile salts

as neutral sterols. Some of the bile salts may be reabsorbed but then it will depress the further catabolism of cholesterol in liver and formation of more bile acids by a feed back control.

In the gonads, adrenal cortex, corpusluteum and placenta cholesterol is the precursor of biosynthesis of various steroid hormones. These hormones are released into circulating blood and are modified or destroyed in the liver and excreted through urine mostly with the steroid ring intact. Compared to the amount of cholesterol catabolised by liver, this amount converted to steroid hormone is quantitatively negligible but physiologically very important.

A small quantity of cholesterol is oxidised to seven dehydrocholesterol and finally converted into cholecalciferol (Vit.D_3) by irradiation in the skin. Vit.D_3 finally excreted in faeces mostly or through the skin in small amounts, along with a little cholesterol or bile.

iv) Cholesterol and atherosclerosis:

Atherosclerosis is a condition associated with hardening of aorta and arterial walls (sclerosis) due to variable combinations of changes in the intima of arteries

consisting of local accumulation of lipids, complex carbohydrates, blood and blood products, fibrous tissues and calcium deposits are associated with medical changes. Deposition of cholesterol esters needs special mention.

Atherosclerosis occurs in diseases with elevated levels of cholesterol and low density lipoproteins in blood as detailed below:

Elevated concentration of very low density lipoprotein (mainly triglycerides) (Pre-beta fraction) with normal concentration of low density lipoprotein (density 1.006 to 1.063).

Elevated levels of low density lipoprotein containing chiefly cholesterol (beta fraction) and normal very low density lipoprotein.

Elevated levels of both very low density lipoprotein and low density lipoprotein (Pre-beta and beta fractions).

The ratio of phospholipid to cholesterol in beta lipoprotein which is normally one tends to decrease in atherosclerosis as also the ratio between alpha to beta lipoprotein decreasing from about 0.9 to 0.5. There is also change in muco-polysaccharide pattern. There is an

increase in chondroitin sulphate-A and hyaluronic acid compared to heparitin sulphate which is potent activator of lipoprotein lipase and also an anticoagulant. Recently, importance is given to scavenging role of high density lipoprotein in removal of deposited cholesterol and preventing atherosclerosis.

The various diseases in which there is increased serum lipids in blood are diabetes mellitus, nephrotic syndrome, hypothroidism and familial hyperlipoproteinemias. Investigation of total cholestorol, triglycerides and lipoprotein electrophoresis is very helpful in diagnostic biochemistry of atherosclerosis.

Atherosclerosis has been successfully induced in herbivorus animals like rabbit, pig and monkey by feeding cholesterol. However, other animals like dog, rat and cat are resistant. If atherosclerosis is to be experimentally induced in the latter species, thiouracil and other hypothyroid drugs are to be fed along with cholesterol.

Process of ageing brings about significant changes in blood vessel wall as the metabolism of cholesterol slows down as age advances. The enzymes electroproteinase and elastomucase required for solubilising and lipolysis of fat are decreased with age. Again as age advances the

elasticity of vessel wall decreases thereby decreasing the lateral pressure and there is a turbulent flow of blood due to greater axial pressure. For these reasons ageing invites atherosclerosis.

In man, many factors are believed to play role in the incidence of atherosclerosis. Obesity, lack of exercise, high fat diet (containing saturated fat), emotional stress, excessive cigaratte smoking, alcoholism, high blood pressure etc. are considered as risk factors that upset the pattern of serum lipid levels leading to the incidence of atherosclerosis in the long run. A strong correlation exists between coronary heart disease, hypertension hypercholesterolemia, serum free fatty acid levels, serum very low density lipoprotein and low density lipoprotein and atherosclerosis.

Among dietary methods, a diet low in fat and sugar is found helpful. Substitution of animal fat by vegetable oils in the diet which is increasingly being adopted in recent years to prevent atherosclerosis is believed to depend upon the beneficial value of polyunsaturated fatty acids. These polyunsaturated acids solubilise the deposited cholesterol by a process of esterification and mobilise the cholesterol to liver for catabolism.

The study supported by the National Institute of Health found that the rate of progression of arteriosclerosis affecting the coronary arteries was proportional to the amount of lipids in the blood (American Dietetic Association, 1981).

Cholesterol is one of the many contributory factors for the formation of atheromatous lesions. Neverthless atherosclerotic complications are more prevalent in hypercholesteremic than in normocholesteremic individuals (Searcy, 1969).

Tandon (1983) had shown that gall stone disease occured frequently in the northern India and that 80 to 90 percent of the stones were of the cholesterol type i.e. more than 70 percent of their dry weight was constituted by cholesterol. The cholesterol stones were associated with bile supersaturated with cholesterol and the occurence was three times more frequently in women than in men. Parous women were affected more often than nulliparous women. Cholesterol monohydrate crystals formed the bulk of cholesterol gall stones besides the varying amounts of calcium carbonate, phosphate and pigment in different layers.

II Egg yolk cholesterol:

1) Biochemistry:

Egg yolk has been implicated as having cholesterogenic properties (Messinger et al., 1950). According to Cook (1958) cholesterol has been definitely established as a precursor of the steroid hormones. Yolk has been indicated as one of the excretory pathways for metabolic cholesterol (Harris and Wilcox, 1963).

Egg yolk cholesterol was exclusively present in the low density fraction and lipovitellins as an integral part of lipoprotein structure (Martin et al., 1963; Cook, 1968).

El-Maguid and Quisenberry (1968) stated that there was an influence of egg consumption on performance, blood cholesterol and livability. Hen day egg production was increased, feed efficiency was improved and mortality was reduced. Yolk cholesterol was not noticeably altered.

Connor et al. (1969) showed that 90 percent of the cholesterol in the brain of the chicken embryo was synthesized, but the cholesterol in the remainder of the body came from the yolk of the egg, suggesting the possibility that the cholesterol level in the yolk might be related to embryonic development and the hatching of chicks.

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Sim et al. (1980) studied the metabolic fate of ^{14}C-ovo cholesterol in the egg yolk fed rat. The rat groups fed egg yolk powder excreted more than 95 percent ingested ovo-cholesterol. An unknown lipid factor present in egg yolk accelerated cholesterol turnout rate and excretion via faeces.

ii) Mean values of egg yolk cholesterol:

Achard et al. (1934) found 17.5 mg/g in chicken eggs, and 26.5 mg/g in the duck egg. Harris and Wilcox (1963), Miller and Denton (1962), Fisher and Leveille (1957) have reported 22 to 26 mg, 15.5 to 17.5 mg and 17 to 22.5 mg cholesterol per gram of yolk respectively.

Much higher levels of 29, 21, 20 and 27 and 21 to 24 mg/g. yolk have been reported by Combas and Helbacka (1960), Daghir et al. (1960), Chand et al. (1972) and Chand and Sapra (1973) respectively.

Miller and Denton (1962), Chung et al. (1965), Weiss and Fisher et al. (1967) and Lall and Slinger (1973) reported values from 11.5 to 15.5 mg/g for White Leghorn eggs. Rangachar et al. (1970) studied the cholesterol content of six breeds of chickens and reported values from 10.1 to 15.0 mg/g. yolk and he has also reported much lower level of 6.5 mg cholesterol/g. yolk for Indian ducks.

Lepore and Marks (1965) reported an average content of 72.7 mg cholesterol per gram of fat or 26.0 mg/g. yolk.

Bartov et al. (1971) reported a range of egg yolk cholesterol of 11 to 15 mg per gram yolk. Turk and Barnett (1971) reported a range in yolk cholesterol values of 11 to 20 mg/g. yolk.

Turk and Barnett (1971) reported 8.4 mg of cholesterol per gram of egg. Seeley et al. (1972), in a compilation of the cholesterol content of foods, gave a level of 14.8 mg/g. yolk.

Cunningham et al. (1973) measured the egg yolk cholesterol level in a closed population of White Leghorns. Egg yolk cholesterol level ranged from 921.4 mg% to 1755.6 mg% with population average being 1267.8 mg%.

Washburn and Nix (1974a) stated that the genetic stocks namely the Athens Randombred population (ARB) and the Athens Canadian Randombred population (AC) differed in the mean yolk cholesterol content and also blood cholesterol. The mean yolk cholesterol content, for the AC (meat strain) population was 22.8 mg (range 19-27 mg) cholesterol per gram of yolk with a standard deviation of 2.8 mg. The mean yolk cholesterol content for the ARB (crosses of meat and egg strain) population was 19.2 mg

cholesterol per gram of yolk with a standard deviation of 1.2 mg. A wide distribution in AC population indicated that genetic selection to change yolk cholesterol was possible.

Tomita et al. (1975) also studied the cholesterol content of seven breeds of chickens and found 10.6 to 15.7 mg per gram of yolk and he also reported a lower cholesterol content of 17.3 mg/g in eggs from Osaka ducks.

Rotenberg and Christensen (1976) found a mean of 13.9 mg cholesterol/g. egg yolk. Godfrey et al. (1976) found 10.9 mg/g. yolk in eggs from Webster Red strain hens. Kudchodkar et al. (1976) found 16.4 mg cholesterol/g. yolk in White Leghorn eggs.

Becker et al. (1977) estimated the analysis of chicken egg yolk cholesterol as 15.64 mg cholesterol/g wet yolk.

Cunningham (1977) stated the cholesterol values of Araucana eggs ranged from a 1094 mg. cholesterol/100 g. yolk to 1684 mg/100 g.yolk with a mean of 1315 mg% and also found 1163 and 1255 mg% for White Leghorns and plymouth Rock respectively.

Somes et al. (1977), Cunningham (1977) and Peterson et al. (1978) reported 21, 13.2 and 12 to 16 mg/g.yolk

respectively for Araucana eggs, levels not significantly different from white eggs from White Leghorns and brown eggs from Plymouth Rock hens.

Bair and Marion (1978) found a much lower concentration 16.0 mg/g.yolk and a mean value of 458 mg per egg in turkeys, and he also reported an average of 14.3 mg cholesterol per gram of yolk in chickens. Chand et al. (1978) reported that the yolk cholesterol concentration (mg/g.yolk) ranging from 18.93 ± 0.47 mg to 31.74 ± 1.38 mg.

The yolk cholesterol content was not significantly different among the pure bred birds, the means being 34.14 ± 5.08 mg per gram, 33.66 ± 5.92 mg/g and 34.69 ± 4.97 mg/g for Rhode Island Red, White Leghorns and Australorp respectively. The Australorp x Rhode Island Red cross bred birds showed a significantly lower level of yolk cholesterol content with a mean of 28.35 ± 4.14 mg/g (Anglin and Briles, 1980).

Bitman and Wood (1980) reported that dove eggs had a relatively high cholesterol concentration of 22.5 mg/g. yolk. Hollands et al. (1980) found the mean yolk cholesterol level (mg%) as 1387 ± 12, 1279 ± 9, 1373 ± mg% for the unselected, low and high line birds. Bitman and Wood (1980) found duck eggs, from Mallard and Black ducks contained 21.1 and 25.0 mg cholesterol/g. yolk.

Chand (1980) indicated the different avian species, in the increasing order of egg yolk cholesterol concentration (mg/g.yolk) were, White Leghorn chicken (20.61 mg) Japanese quail (21.78 mg) Turkey (22.84 mg), duck (26.23 mg) and pigeon (34.28 mg). The differences in concentration of egg yolk cholesterol of chicken, quail and turkey were not significant and so was the case of duck and turkey. Pigeon egg yolk cholesterol, was significantly higher than that of all other species studied.

Bair and Marion (1978) found a much lower concentration (16.2 mg/g) in eggs from three breeds of ducks (Mallard, Muscovy and Pekin-Rouen) and reported 22.0 mg/g.yolk as cholesterol concentration for the dove.

The cholesterol concentration (mg/g.wet yolk) recorded as 23.9 ± 1.24 mg in fertile eggs was slightly but not significantly, higher than that recorded in infertile eggs as 20.8 ± 0.80 mg (Chand, 1979).

Total egg yolk cholesterol:

Turk and Barnett (1971) found 678 mg per turkey egg. Becker et al. (1977) estimated total yolk cholesterol as 225.73 ± 0.66 mg in chickens. The yolk cholesterol content was lower (312.27 mg per yolk) in Australorp with a range of 239.81 ± 14.25 to 512.36 ± 27.99 mg (Chand et al., 1978).

Bitman and Wood (1980) reported that the domestic turkey had a total cholesterol of 681 mg.

iii) Genetic basis of egg yolk cholesterol:

Gaujoux and Krijanowsky (1932) and Achard et al. (1934) indicated that the duck's egg was markedly richer in yolk cholesterol than the hen's egg.

According to Chavous et al. (1965), Collins et al. (1966) and Chand et al. (1972) significant breed differences existed with respect to yolk cholesterol.

Hickman (1974) stated that eggs produced by Araucana hens were lower in cholesterol content than other eggs.

Gissel et al. (1976) reported within breed and between breed variations in cholesterol content of eggs. Within breed variation was higher than that between breeds.

Seven inbred lines of chickens showed differences in yolk cholesterol (Bair and Marion, 1978).

Bair and Marion (1978) studied the yolk cholesterol in eggs from various avian species and species listed in increasing concentrations of cholesterol per gram of yolk were guinea fowl, chicken, pheasant, quail, turkeys, duck, goose and dove with an overall range of 12.77 to 21.99 mg of cholesterol per gram of yolk and he also found significant

differences in cholesterol concentrations between domestic and wild genetic groups of turkeys and ducks.

iv) Heritability of egg yolk cholesterol:

Scheinberg et al. (1953) found large difference between the percent of the sire component to total variance of wet yolk weight (0.34%) and the dam component (6.14%). Yao and Skinner (1959) had reported the heritability through sire component as 0.388 and through dam component as 0.333 while Jaffee (1964) estimated 0.428 and 0.435 through sire and dam component respectively.

Harris and Wilcox (1963), using the mean of two consequetively laid eggs per hen for analysis found that yolk cholesterol expressed, as mg.cholesterol/g. wet yolk differed between dam families within sires in a random bred White Leghorn strain.

Turk and Barnett (1971) reported that egg production strains have lower cholesterol values than broiler strains.

Cunningham et al. (1974) analysed the mean of the three eggs per hen as mg. percent of yolk cholesterol in a White Leghorn population. One generation of bidirectional selection was initiated. The intra-sire daughter dam regression provided a heritability of 0.24.

Washburn and Nix (1974a) studied the heritabilities of two random bred population (Crosses of meat type and egg production - ARB population and meat stock AC population) viz., ARB and AC population by half sib procedure as 0.23 and 0.36 respectively.

Ali et al. (1974) reported that the heritability of egg yolk cholesterol was 0.24 in a closed population of White Leghorns. Becker et al. (1975) in a random bred White Leghorns estimated the heritabilities of mg. cholesterol and total cholesterol as 0.14 and 0.31 respectively.

Becker et al. (1977) estimated the heritability of mg.cholesterol/g. dry yolk was 0.15. He also suggested that the mg.cholesterol in total yolk should be reduced to be meaningful to the consumer because the whole egg was eaten.

Becker et al. (1977) reported the heritability of total yolk cholesterol as 0.27 $\pm$ 0.07. The heritability of mg.cholesterol in total yolk was larger than the heritability of mg.cholesterol/g. dry yolk probably reflecting the higher heritability of yolk weight which was component of mg.cholesterol in total yolk.

v) Correlation of egg yolk cholesterol with egg production traits:

a) Age and egg yolk cholesterol:

Significantly higher values for yolk cholesterol were observed when the birds were in moult (March and Bailey, 1959; Rao et al., 1964).

Palafox (1968) studied the effects of age, energy source and concentration on yolk lipids and cholesterol in White Leghorns. Yolk lipids and cholesterol concentration of 30 to 50 week old pullets were not significantly different.

Turk and Barnett (1971) found that cholesterol concentration in the egg was not affected by the hen's age. Chand et al. (1978) reported that the yolk lipids did not change significantly upto 12 months of age in Australorp.

The egg, yolk, shell weight as well as yolk cholesterol content did not increase significantly during the first five months of production (Chand et al., 1978).

Yolk cholesterol tended to decrease as age of hen increased (Bair and Marion, 1978). Egg, albumen, yolk and shell weight, yolk lipid and yolk cholesterol contents were recorded from hens of Australor from 6 to 18 months of age. Highly significant effect due to advancing age was recorded, for all the traits studied (Chand et al., 1978).

Bair et al. (1980) indicated that cholesterol concentration in chicken egg yolk decreased significantly from an average of 18.9 to 15.8 mg per gram of yolk from the first to fifty eggs after the onset of production.

b) Yolk cholesterol and egg production:

A negative correlation between egg production and yolk cholesterol level was found; no association was found between yolk cholesterol and egg size (Cunningham et al., 1973).

Ali et al. (1974) stated that there was a negative relationship between egg production and the amount of yolk cholesterol. Washburn and Nix (1974) stated that a significant negative phenotypic correlation was found between yolk cholesterol and egg production.

Washburn and Marks (1977) showed that there was a direct relationship between clutch length and yolk cholesterol concentration. The mg.cholesterol/g.yolk was 17.2, 17.3, 16.0 and 15.5 for the clutch size of three, four, five and six eggs respectively. Selection for increased clutch length decreased the cholesterol concentration in egg yolk.

c) Yolk cholesterol and egg weight:

Cunningham et al. (1974) reported that the egg weight and yolk cholesterol was not correlated.

Because of the great variation in egg and body size, total cholesterol per egg varied from 43 mg in Japanese quail to 845 mg in the great black-backed gull. When total cholesterol was plotted against egg weight, the plot indicated that cholesterol was a relatively constant proportion of the egg, irrespective of species (Bitman and Wood, 1980).

d) Yolk cholesterol and yolk weight:

Harris and Wilcox (1963) estimated that the phenotypic correlation between cholesterol and yolk weight was negative (-0.09 to -0.17). Nicholas et al. (1963) indicated a negative correlation between yolk weight (g) and yolk cholesterol concentration (mg/g.yolk) and between yolk size and cholesterol concentration.

Forsythe (1963) pointed out that the proportion of the yolk tends to be higher in smaller eggs than in larger ones. Chand et al. (1972) reported that the correlation was not high between yolk weight and yolk cholesterol in White Leghorn birds.

The total cholesterol concentration of egg yolk and cholesterol concentration in the lipid (mg. per gram of lipid) were not significant and this was due to the significant differences in the percent yolk weight and lipid concentration (Chand and Sapra, et al., 1973).

The genetic correlation between mg.cholesterol and yolk weight were negative indicating that selection for low cholesterol might increase yolk weight (Becker et al., 1975).

Becker et al. (1977) reported that the genetic correlation between mg.cholesterol/g.dry yolk and each of the three yolk weight traits varied from 0.28 to 0.35 with the environmental correlation of 0.07 and phenotypic correlation of 0.05. A genetic change in mg.cholesterol/g. dry yolk would result in a corresponding positive change in yolk and moisture weight. An environmental shift in mg.cholesterol/g.dry yolk would probably result in little change in yolk and moisture weights.

The correlation coefficient values between egg and yolk weight were highly significant and positive, which reflected that larger eggs had havier yolks both in terms of absolute weight and relative values. The correlation coefficient values of eggs weight and yolk weight, yolk weight and cholesterol concentration (mg/g.yolk), and between yolk weight and cholesterol content were 0.83, -0.37, 1.0 respectively (Chand et al., 1978).

e) mg.cholesterol/g. yolk with total yolk cholesterol:

Becker et al. (1977) reported that a high genetic

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correlation of 0.71 to 0.74 between mg.cholesterol/g. dry yolk and cholesterol in total yolk.

vi) Egg yolk cholesterol and blood cholesterol:

Human studies have indicated minimal or even no changes in serum cholesterol at different levels of eggs in the diet (Mayer et al., 1954; Keys et al., 1956; Slater et al., 1976 and Porter et al., 1977).

Marion et al. (1960) reported a significant (-0.29) inverse phenotypic relationship between serum cholesterol and yolk cholesterol.

There was influence of feeding on yolk cholesterol as well as serum cholesterol level which was suggestive of a relationship between yolk and serum cholesterol level (Harris and Wilcox, 1963).

Due to close relationship between heart diseases and cholesterol and lipid intake in general (American Heart Association, 1968) a comprehensive study of cholesterol of eggs and plasma cholesterol of hens was made and reported that a specific weight of egg yolk lipid caused a higher and more rapid increase in serum cholesterol level than did the same weight of any other dietary fat (Well and Bronte-Stewart, 1963).

The concentration of cholesterol in yolk and plasma showed a reverse relationship (Chand and Sapra, 1973).

The phenotypic and genetic correlations for yolk cholesterol and blood cholesterol for the AC (meat strain) population were 0.14 and 0.03 and for ARB (crosses of meat and egg strain) population were 0.0 and 0.59 respectively (Washburn and Nix, 1974).

The phenotypic correlations between yolk and plasma cholesterol were small (0.04, -0.04 and -0.08 for unselected, low and high lines respectively) as reported by Hollands et al. (1980).

Sim et al. (1980) studied the effect of dietary egg yolk on serum cholesterol levels of White Leghorn cockrels and found that high dietary levels of crystalline cholesterol had a greater influence on raising the serum cholesterol concentration than an equal amount of dietary cholesterol fed in the yolk form. No significant effect was observed at the dietary cholesterol level less than 0.2 percent, irrespective of cholesterol source. The correlation coefficients between dietary and serum cholesterol levels were 0.97 and 0.77 for the crystalline and egg yolk powder cholesterol source respectively.

vii) Influence of drugs and diet on egg yolk cholesterol:

Kurnic et al. (1958) did not observe appreciable changes in cholesterol content of the yolk of the eggs from hens fed one percent cholesterol in the diet. Further eggs from hens maintained for four and half years on the diet supplemented with five percent dried egg yolk did not show altered yolk cholesterol concentration when samples following the dietary regime.

Leveille and Fisher (1959) and Combs and Halbacka (1960) demonstrated that diets containing 10 percent corn oil increased yolk cholesterol whereas 10 percent animal fat did not effect yolk cholesterol level.

Wood et al. (1961) have shown a marked elevation in yolk cholesterol with the addition of one percent cholesterol to the diet and noted that the presence of 10 percent corn oil enhanced the increase.

In contrast to body depot lipids, egg lipids changed rapidly in response to changes in dietary fat (Hilditch and William, 1964; Bitman, 1976).

Bartov et al. (1971) studied effects of different vegetable oils on yolk cholesterol levels; supplementing laying diet with 20 percent safflower oil or coconut oil produce a significant increase in yolk cholesterol.

Dietary lipids in the unsaturated form potentiated the absorption of free cholesterol (Sim and Bragg, 1977).

Hood et al. (1978) found the effect of dietary monoterpenses on the cholesterol level of eggs. Feeding five monoterpenes phorone or 200 mg cholesterol per day to hens did not significantly changed the level of cholesterol in the egg yolk.

Diazocholesterol significantly decreased concentration of cholesterol in yolk and increased desmosterol. Diazocholesterol with B-sitosterol decreased cholesterol and increased desmosterol and total sterols in yolk (Dam et al., 1979).

Sharma et al. (1979) found that the compounds like garlic sarpagandha and nicotinic acid reduced egg cholesterol concentrations significantly irrespective of the dose added, in White Leghorns. After withdrawal the cholesterol content of eggs from birds given nicotinic acid and garlic powder tended to increase.

No difference was exhibited in egg yolk cholesterol among any of the groups of Coturnix quail which was given various fibre sources like alfalfa, wheatbran, dried brewer's grain, cellulose and pectin (Sutton et al., 1980).

Sutton et al. (1980) indicated in Coturnix quail that quantity of cholesterol deposited in the egg on which fibre intake, energy consumed or egg production had very little effect and that there was an inverse relationship between serum and tissue cholesterol levels and total quantity of cholesterol excreted via the egg.

There was no significant difference of treatments of the effect of dietary alfalfa of varying saponin content in yolk cholesterol level, hen day egg production and Haugh units (Nakaue et al., 1981).

Egg quality and yolk cholesterol were not significantly affected by energy level of the diet (Campos and Ferreira, 1981).

Environmental factors with yolk cholesterol:

The cholesterol content of egg yolk might be affected by a number of environmental factors (Clarenburg et al., 1971). Moudgal (1978) indicated that housing systems did not have a significant effect on cholesterol content but increase in dietary protein level from 17 to 25 percent resulted in lowering the cholesterol content significantly in White Pekin Ducks.

viii) Yolk cholesterol and atherosclerosis:

Chicken was one of the most sensitive animal models for cholesterol metabolism and experimental atherosclerosis studies (Katz and Stamler, 1953; Linsey et al., 1955).

Diets high in cholesterol and fat have produced a form of atherosclerosis in many species (Clarkson, 1971; McGill, 1979; Vesselinovitch, 1979). Shih (1979) found the atherogenicity effect of USP and purified cholesterol in Japanese quail.

ix) Selection for yolk cholesterol:

Ali et al. (1975) reported that a closed population of White Leghorns was divided into 'low' and 'high' egg yolk cholesterol sublines. One generation of bidirectional selection for low and high egg yolk cholesterol resulted in 71.45 mg% difference between sublines.

Two hundred and ninty seven mg of egg yolk cholesterol separated the 'low' and 'high' subline breeders in generation two. The low subline has averaged 93.5 ± 4.6 eggs per breeder during the last five months of laying year while the high subline has averaged 74.9 ± 6.4 eggs during the same period (Ali et al., 1975).

Marks and Washburn (1977) reported that they were not able to decrease yolk cholesterol but were able to increase it by direct selection.

III Blood cholesterol:

The action of dominant gene has been suggested as operating in humans (Wilkinson et al., 1948; Schaefer et al., 1953; Pipe and Orrild, 1956; Harris Jones et al., 1957).

Wilcox et al. (1963) concluded that serum cholesterol in chickens exhibited additive genetic variation.

Andrews et al. (1968) concluded that egg cholesterol originated from serum cholesterol indicating a relationship between yolk and serum cholesterol.

i) Mean values:

Average serum cholesterol in the young pullet (six weeks age) as well as adult hen (eight months) were 146 and 228 mg per 100 ml respectively (Wilcox et al., 1963).

The mean plasma cholesterol was 169 mg% with a standard deviation of 58 mg% in AC population. The serum cholesterol level of the ARB population was 230 mg% with a standard deviation of 63 mg% (Washburn and Nix, 1974).

Kaminski et al. (1979) found the concentration of cholesterol were 159.2 and 131.1 mg% in male and female blood serum respectively.

Kaminski et al. (1979) reported the serum cholesterol value as 145.7 mg, 157.8 mg, 226.3 mg and 184 mg/100 ml

at four, 20, 28 and 32 weeks respectively for female. The male cholesterol values were 239 mg, 155 mg, 175 mg, 194 mg and 168 mg/100 ml at four, eight, 20, 28 and 32 weeks of age respectively.

The mean plasma cholesterol (mg%) in females of fifth generation was 97 ± 1.2 mg%, 69 ± 8 mg% and 135 ± 1.5 mg% for the unselected, low line and high line respectively (Hollands et al., 1980).

Mean values for plasma cholesterol level ranged from 235 ± 49 to 361 ± 86 mg% for high line and 205 ± 41 to 281 ± 74 mg% for low line across eight generations (Marks and Siegel, 1980).

ii) Heritability:

Bumgardner (1955) obtained an estimate of 0.42 as heritability for serum cholesterol in a New Hampshire line. Quantitative inheritance has been postulated in rats by Kohn (1950) and in mice by Weibust (1969) who obtained realized heritabilities of 0.48 for males and 0.59 for females, in two generations of crosses of unrelated inbred strains.

Cherms et al. (1960) reported heritability estimates as 0.19, 0.41 and 0.30 by sire, dam and combined component respectively in a randombred population of White Leghorns.

Serum cholesterol levels were measured at six to nine weeks of age in random bred White Leghorns as well as lines selected for differentiation in cholesterol. Significant differences between dam families were noted in two successive years and heritability was estimated to be 0.30. After three generations of selection two lines have been developed which differ markedly and significantly in serum cholesterol level (Cherms et al., 1960).

Wilcox et al. (1963) reported heritability estimates for plasma cholesterol of 0.34 for sire and 0.17 for dam component in a population of random bred White Leghorns.

Average heritability of serum cholesterol was 0.25 (through sire and dam component) (Wilcox et al., 1963) which was close to that observed by Cherms et al. (1960).

The heritability estimate for serum cholesterol level in ARB population was 0.56. There was no significant sire effect on plasma cholesterol in the AC population and the heritability estimate indicated no genetic variance for plasma cholesterol in the population (Washburn and Nix, 1974).

The heritability for plasma cholesterol by regression of progeny on mid parent was 0.25 in the high line and 0.16 in the low line (Marks and Siegel, 1980).

Hollands et al. (1980) estimated the heritability of the plasma cholesterol of the unselected line (White Leghorns) as 0.2 to 0.3. The estimates of the two sexes agreed reasonably well thus showing little evidence of either dominance or maternal effects. The realised heritabilities in the selected lines were somewhat lower than the heritability from the unselected line.

iii) Correlations:

a) Age and blood cholesterol:

Serum cholesterol levels were decreased from six weeks through 12th week and then increased upto 20 weeks in chicken (Estep et al., 1969)

Kaminski et al. (1979) reported the effect of age on lipoproteins and cholesterol in blood serum of White Rock hens and the cholesterol values were 145.7 mg, 157.8 mg, 226.3 mg and 184 mg/100 ml at four, 20, 28 and 32 weeks respectively for females; the male cholesterol values were 239, 155, 175, 195 and 168 mg/100 ml at four, eight, 20, 28 and 32 weeks of age respectively.

b) Sex and blood cholesterol:

In human, the levels of high density lipoprotein (HDL) cholesterol in females were higher than in males (approximately ten mg/100 ml plasma) and females have a

lower incidence of coronary heart disease (CHD) (Castelli et al., 1977).

Kaminski et al. (1979) studied the relationship between the total cholesterol in blood serum and cholesterol deposition in certain tissues of broiler chickens at nine weeks and found concentrations of cholesterol were 159.2 mg/100 ml and 131.1 mg/100 ml for males and females in blood serum.

In single comb White Leghorns males at nine to 10 weeks of age had higher plasma cholesterol (113 mg%) levels than females (100 mg%) of the same age (Hollands et al., 1930).

c) Blood cholesterol and body weight:

Bumgardner (1955) observed a significant positive genetic correlation between cholesterol level and body weight at four weeks of age.

Weiss and Fisher (1957) found no relationship between adult body weight and adult cholesterol level.

There was a statistically significant positive correlation between cholesterol level of the young pullet and adult cholesterol level and a negative correlation between cholesterol level and body weight at six weeks of age (Wilcox et al., 1963) and he found the correlation of 0.13 between cholesterol level in the young chicken and

adult body weight (eight months) was quite low and was identical to the repeatability value observed by Hardy et al. (1962) for cholesterol level in serum collected at six and seven weeks from the same birds.

Swierczewska et al. (1981) estimated the cholesterol in blood serum of Baile-Brwinoskie (meat type) and Rhode Island Red (egg type) cocks and hens. There was a close correlation between serum cholesterol and body weight at five weeks of age than at eight weeks of age. The correlation being positive in the meat type cocks and negative in the meat type hens, egg type, cocks and hens.

d) Blood cholesterol and egg production traits:

No significant correlation between egg production and adult cholesterol level was noticed (Lorenz et al., 1938). Weing andFisher (1957) reported a non significant positive correlation between the rate of egg production and serum cholesterol, while Johnson et al. (1959) reported a non-significant negative correlation.

Leveille and Fisher (1959), Svacha (1959) and Smith (1969) reported a significant negative correlation between rate of egg production and serum cholesterol.

Substantialy positive genetic correlation were observed between cholesterol level and body weight at eight weeks,

age at first egg, egg production and albumen quality. A substantial negative correlation was observed for adult body weight (Wilcox et al., 1963).

Andrews et al. (1968) reported that in the laying fowl the specific activity of plasma cholesterol rose during the first 32 hours of production.

The estimated genetic correlation for plasma cholesterol and egg production was negative (Hollands et al., 1980).

Hollands et al. (1980) reported the genetic correlation of plasma cholesterol with age to first egg, and egg weight as -0.047, 0.191 respectively in White Leghorns and phenotypic correlations of plasma cholesterol with the same traits in order as -0.140, 0.002 respectively.

iv) Influence of diet on blood cholesterol:

Goldsmith et al. (1960) stated that excretion of bile acids in human increased 20 to 25 percent concomitantly with a decrease in serum cholesterol when polyunsaturated fatty acids were fed.

The apparent association between cholesterol levels and livability might have been caused by starvation just before death, because fasting could cause elevated cholesterol level (Hardy et al., 1962).

Edwards and Jones (1964) reported both yolk and blood cholesterol of hens have increased following consumption of high levels of cholesterol in the diet.

Cholesterol supplementation of the fat diet showed a significant increase in the faecal bile acid excretion (Portman and Stare, 1959; Wilson, 1964).

An increase in the faecal output of total neutral sterols was also demonstrated in human studied when unsaturated fatty acids were included in the diet (Grundy and Ahrens, 1966; Wood et al., 1966; Moore et al., 1968; Connor et al., 1969).

Daghir and Porooshani (1968) showed that dietary cholesterol significantly increased serum and liver cholesterol. Commercial egg strains were grown on diets containing different levels of protein. The protein level influenced significantly in serum cholesterol levels (Estep et al., 1968).

Cholesterol in the mammalian system was eliminated entirely by excretion into the faces as neutral steroid and bile acids (Danielson and Tchen, 1969).

Addition of every additional 100 mg of dietary cholesterol would cause elevation of five mg serum cholesterol (Halloway, 1969).

Bartov et al. (1971) studied effects of different vegetable oils in plasma cholesterol levels. Changes in plasma cholesterol levels due to dietary treatment were not uniform for the hens of each treatment, except in case of sterol unsupplemented coconut oil wherein 90 percent of the hens fed this oil exhibited higher plasma cholesterol levels.

Kruski and Narayan (1972) pointed out that liver exhibited the greatest response to a cholesterol diet. Liver and serum were considered as a pool of cholesterol and the concentration of cholesterol in the liver was the principal factor controlling cholesterol metabolism in chickens.

Among the plasma (or serum) lipoproteins induced by cholesterol feeding, the very low density lipoprotein cholesterol was markedly increased in pigeons, quail and chickens (Jones and Dobrilovic, 1969; Day et al., 1974; Kruski and Narayanan, 1972; Narayan and Calhoun, 1975) and in rabbits (Shore et al., 1974; Day et al., 1974).

Cholesterol feeding resulted in a hypercholesteremia with a distinctive hyperlipoproteinemia in different animals (Mahley, 1978).

Serum cholesterol level was elevated in the pectin and wheat bran diets. There was an inverse relationship between both serum and tissue cholesterol level versus the total quantity of cholesterol excreted via egg (Sutton et al., 1980).

Comparative hypercholesterolemic properties of two dietary cholesterol sources viz. crystalline and egg yolk powder were investigated in White Leghorn male chicks. High dietary levels of crystalline cholesterol had a significantly greater influence on raising the serum cholesterol concentration than an equal amount of dietary yolk cholesterol. No significant effect was observed at the dietary cholesterol level less than 0.2 percent irrespective of cholesterol source. The correlation coefficients between dietary and serum cholesterol levels were 0.97 and 0.77 for the crystalline and egg yolk powder cholesterol source respectively (Sim et al., 1980).

Serum cholesterol level was elevated in the birds (coturnix quail) fed the pectin and wheat bran diets (Sutton et al., 1980).

In White Leghorn cockrels, the serum cholesterol value with the cholesterol-free diet was lowest (113.0 mg%) and the highest was with 1.0 percent crystalline cholesterol diet (384.8 mg%) (Sim et al., 1980).

The serum cholesterol levels with egg yolk diets at graded levels (from 0.25 to 1.5%) were significantly lower than that of one percent crystalline cholesterol level (0.25%). However, egg yolk cholesterol diet produced slightly but significantly higher serum cholesterol level than the crystalline form (Sim et al., 1980).

In comparison to recrystallized cholesterol treated quail, oxidised cholesterol treated quail exhibited significantly increased serum and liver cholesterol concentrations and increased severity of atherosclerotic lesions (Donaldson, 1982).

Wu and Donaldson (1982) stated that serum cholesterol had a positive association with liver cholesterol in cholesterol-fed quail.

Panda et al. (1983) found that tannic acid at one percent and fat or oil at two percent had a synergistic effect on increase in plasma cholesterol. The fat and oil increased plasma cholesterol but the increase was greater with fat. The results were of significance in human nutrition in view of the tannic acid content of tea and coffee.

Mol et al. (1983) studied the effect of dietary protein and cholesterol on cholesterol concentration and lipoprotein

pattern in the serum of chickens found that in the groups on cholesterol containing diets there was a shift in the lipoprotein pattern from the low density lipoprotein to the intermediate density lipoprotein and very low density lipoprotein.

Normal rabbits fed with dietary cholesterol developed cholesterol deposits in all of their blood vessels, not just the coronary arteries. In human cholesterol accumulated preferentially in the coronary arteries. Watanabe rabbits developed a heart disease that closely resembled what occured in human (Anonymous, 1983).

v) Blood cholesterol and atherosclerosis:

Many investigators have demonstrated a correlation between raised serum lipid levels and the incidence of coronary heart disease and atherosclerosis. Havel and Carlson (1962) stated that the serum cholesterol has been one of the most chief factor in causing the incidence of coronary heart disease and atherosclerosis besides other factors such as phosph lipid ratio, lipoprotein concentration, serum triglycerol concentration etc., causing the heart disease.

Andrew Gement (1966) found absorption of cholesterol was one of the various facets of complicated mechanism of

atheroma formation, the major part of the mechanism was metabolic enzyme and hormone controlled. Cholesterol found in atheromas was not synthesized locally but was derived from blood.

Elevated serum cholesterol level played a central role in the genesis of atherosclerosis and coronary heart disease (Connor, 1968).

High density lipoprotein (HDL) was considered as a protective factor against chronic heart disease (CHD) and very low density lipoprotein (VLDL) and low density lipoprotein (LDL) were considered to promote the disease process. Recent reports indicated that the ratio of high density lipoprotein (HDL) to low density lipoprotein (LDL) cholesterol or high density lipoprotein (HDL) to low density lipoprotein (LDL) and very low density lipoprotein (VLDL) cholesterol may be a measure of resistance to Atherosclerosis in human (Gluek, 1976; Kannel et al., 1979; Nakai et al., 1981).

The plasma high density lipoprotein (HDL) cholesterol level was negatively correlated to the risk of coronary heart disease (CHD), whereas low density lipoprotein (LDL) and very low density lipoprotein (VLDL) cholesterol had a positive association in human (Gordon et al., 1977; Castelli et al., 1977; Anonymous, 1978; Kannel et al., 1979).

The degree of diet aggravated atherosclerosis was studied by Bitman and Wood (1980) in Japanese quail. No atherosclerosis was seen in birds not fed with cholesterol. With cholesterol feeding, male SUS (Susceptible) birds exhibited the greatest incidence of atherosclerosis whereas the female RES (Resistant) birds exhibited the least. Aortic cholesterol concentration was considerably higher in males as compared to females. The high concentration of cholesterol circulating in the blood was the only possible explanation of higher incidence of atherosclerosis.

vi) Selection for blood cholesterol:

Serum cholesterol levels were measured at six to nine weeks of age in randombred White Leghorns as well as lines selected for differentiation in cholesterol. Significant differences between dam families were noted in two successive years and heritability was estimated to be 0.30. After three generations of selection two lines have been developed which differed significantly in serum cholesterol level (Cherms et al., 1960).

Genetic correlations would be of use in predicting the performance of lines selected for high and low cholesterol level. Their magnitude suggested that there was a genetic

relationship between cholesterol level of the young chicken and subsequent body weight, age at first egg, egg production and albumen quality (Wilcox et al., 1963).

Hollands et al. (1980) studied the response to five generations of selection for blood cholesterol levels in White Leghorns. The respons to five generations of selection for high and low plasma cholesterol levels was examined in two lines of single comb White Leghorn chickens derived from the same population.

Differences between the two selected lines in the fifth generation was 37 mg percent for males and 33 mg percent for females. High cholesterol levels were associated with high mortality. After five generations of selection for plasma cholesterol, yolk cholesterol was 109 mg% lower in the low selected line than in the controls (Hollands et al., 1980).

MATERIALS AND METHODS

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I Population under study:

This experiment was carried out at the poultry section of the Livestock Research Station, Kattupakkam during the year 1980 to 1982. Meyer strain single comb White Leghorns maintained under index selection programme were used for the study.

II Collection of data:

The pedigreed birds belonged to fifth generation under index selection programme were taken for collection of data for egg yolk cholesterol, plasma cholesterol and other economic traits. A total of 696 pullets belonging to fifth generation under 28 sire families were included in the study. The data was collected at 32, 40 and 72 weeks ages of the birds.

III Management of birds:

Pedigreed chicks were raised in a single hatch and kept in brooder houses upto ten weeks of age, sexed and then put into grower house. At 20 weeks of age all the birds were weighed, wing badged and housed in the layer house with a floor space of three square feet per pullet. They were reared under intensive deep litter system in a

semi-monitor type aluminium roofed houses. Paddy husk was used as litter material.

In each pen, one aluminium two way trapnest of 3' x 1.5' with three holes was used for trapnesting. The birds were trapnested from 7.00 A.M. to 5.00 P.M. daily. Egg weights were recorded upto 40 weeks of age.

Feeding:

The chicks were provided with adlibitum feed by hanging metal feeders and running water channels were used for watering. Separate grit hoppers were provided with shell grit which was made available as free choice. The feeding and other management practices were kept constant throughout the experimental period. The chicks were fed adlibitum from 0 to 8 weeks of age, grower mash from 8 to 20 weeks of age and layer mash thereafter. The composition of mashes are given in the Appendix I.

Collection of eggs:

Three consequative eggs per hen were collected and analysed for egg yolk cholesterol. The eggs were stored at a temperature of 21°C before the analysis of egg yolk cholesterol.

IV Experimental methods:

i) Determination of egg yolk cholesterol:

Egg yolk cholesterol determination involved extraction of cholesterol from the egg and measurement of isolated cholesterol. Yolk cholesterol was determined by a modification of Folch et al. (1956) extraction method by Washburn and Nix (1974). Eggs were hard cooked as per the method of Baker et al. (1930). In the modified extraction procedure yolks were separated from the remainder of the egg. A sample of one gram yolk was mixed with 15 ml of 2 : 1 chloroform - methanol and shaken 12 times by hand, five ml of distilled water added and the sample again was shaken 12 times by hand. After centrifugation at 2500 rpm for ten minutes, the aqueous methanol layer was removed by suction and discarded. The chloroform layer was filtered through fibre glass filter paper into a test tube, stoppered and stored at 5°C for assay of mg cholesterol/g. yolk by the standard Zlatkis method (Zlatkis et al., 1953).

colourimetric assay:

To 0.1 ml of egg yolk extract was added three ml of aldehyde-free glacial acetic acid followed by two ml of ferric chloride reagent.

This ferric chloride reagent was made up by dissolving 10 g $FeCl_3$ in 100 ml of aldehyde-free glacial acetic acid

and then diluting two ml to 200 ml with concentrated sulphuric acid. After mixing the intensity of the purple colour was measured at 560 mu in a Erma photoelectric colourimeter. The amount of cholesterol was then estimated by reference to a standard curve. This method was rapid and did not involve saponification since equimolar concentration of cholesterol ester and cholesterol gave colours of equal intensity.

ii) Determination of total plasma cholesterol:

Principle:

When a solution of ferric chloride in acetic acid was added to the plasma, the plasma proteins were rapidly precipitated leaving the liberated cholesterol in the supernatent solution. A diluted aliquot of the supernatent fluid from the plasma when treated with a specific amount of concentrated sulphuric acid, yielded a colour that was stable, sensitive and reproducible, reacting according to Beer's law. The absorbance of coloured solution was measured in Erma Photoelectric colourimeter and the total cholesterol level was read against the standard graph prepared previously.

Collection of plasma:

A two ml blood was collected from the brachial vein of the bird. Individual blood samples were placed in

heparinised test tubes and centrifuged. Aliquots of plasma were frozen for later analysis of cholesterol via a colourimetric assay.

Method:

The standard method of Zlatkis (Zlatkis et al., 1953) was used in the estimation of plasma cholesterol. This procedure was adopted because of its suitability for its short working time and necessity of collecting only a small amount of blood for the experiment.

Reagents used in the estimation of total plasma cholesterol:

Ferric chloride stock reagent:

This reagent was prepared by dissolving 840 mg of $FeCl_3\ 6H_2O$ in 100 ml of aldehyde-free glacial acetic acid.

Ferric chloride precipitating reagent:

This was prepared by diluting the stock reagent 1 : 10 with aldehyde-free glacial acetic acid.

Ferric chloride blank and diluting reagent:

This reagent was prepared by diluting 8.5 ml of the stock reagent to 100 ml with aldehyde-free glacial acetic acid.

Cholesterol stock standard:

The cholesterol stock standard was prepared by dissolving 100 mg of pure, dry cholesterol in 100 ml of

aldehyde-free glacial acetic acid.

Cholesterol working standard:

This was prepared by adding one ml of stock standard to 0.85 ml of the stock reagent of ferric chloride and made upto ten ml with aldehyde-free glacial acetic acid.

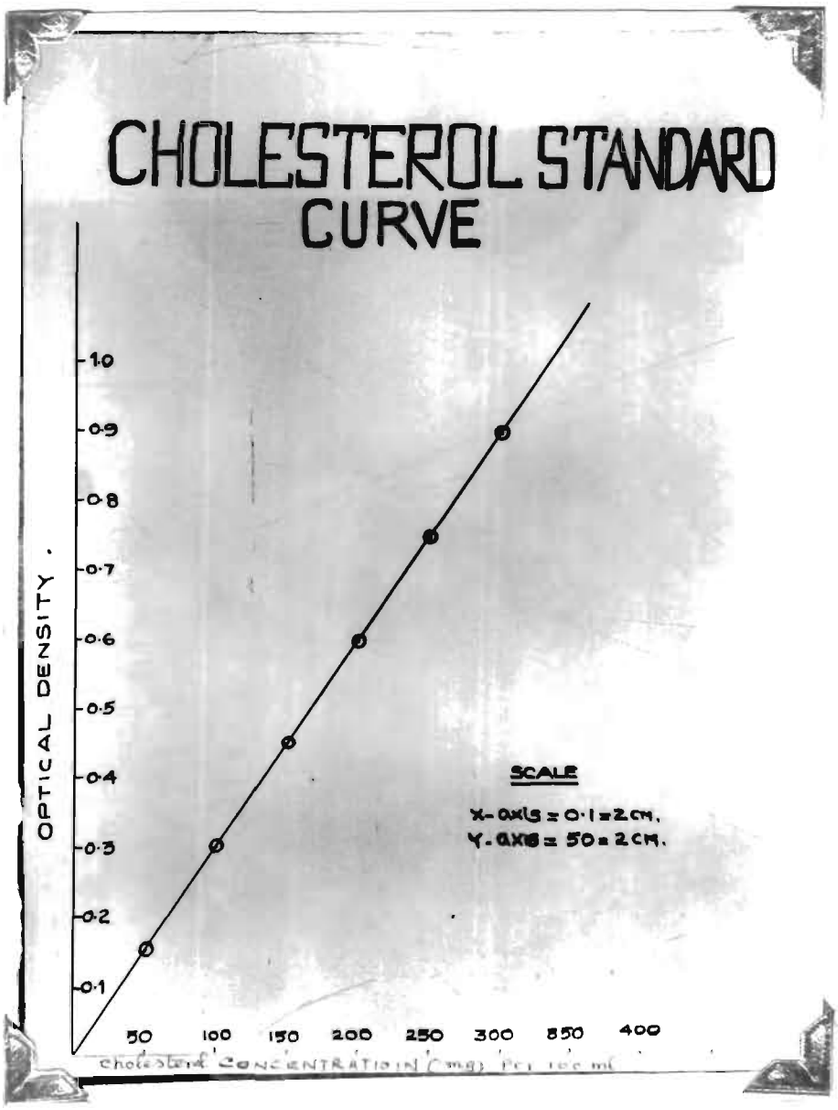
Concentrated sulphuric acid.

Preparation of the standard graph for plasma cholesterol:

A fresh working standard which contained 0.1 mg in one ml was prepared. Zero, one, two and three ml of this freshly prepared working standard were diluted to four ml with the diluting solution of ferric chloride; 0.1 ml of physiological saline was added to each tube. Three ml aliquot of this solution were withdrawn from each tube and two ml of concentrated sulphuric acid were added to each. The solutions were mixed thoroughly by means of tapping the tubes with the fingers and the solutions were allowed to cool to the room temperature. The absorbances of the three standards in comparison with that of the blank were measured in Erma Photoelectric colourimeter set at 560 mu. From the figures obtained the standard graph was constructed with the concentration along the abscissa and the optical density along the ordinate.

Estimation of total cholesterol in plasma:

0.1 ml of plasma was pipetted into four ml ferric


CHOLESTEROL STANDARD CURVE
OPTICAL DENSITY.
1·0
0·9
0·8
0·7
0·6
0·5
0·4
0·3
0·2
0·1
50
100
150
200
250
300
350
400
SCALE
X-axis = 0·1 = 2 cm.
Y-axis = 50 = 2 cm.

chloride precipitating reagent and this was mixed well by tapping with fingers. After two or three minutes, the mixture was filtered into a small test tube. To a three ml aliquot of the clear yellow filtrate two ml of concentrated sulphuric acid was added. The reagents were mixed well and the resulting solution was permitted to cool down to room temperature. Then the absorbance of the solution in comparison with that of the blank was measured in Erma Photoelectric colourimeter set at 560 mu. Benenson (1955) reported the permitted variation for blood constituents. The control of accuracy by photoelectric colourimeter was as checked by Benenson (1955), for cholesterol and cholesterol ester to the extent of $\pm$ 0.9 percent. For each sample of plasma, a control test was also done and the average reading was taken. If the absorbance value exceeded 1.0, the final colour was diluted 1 : 1 with blank reagent in order to extent the range of analysis. The optical density was read against the concentration from the standard graph.

Calculation:

$$\text{mg\% cholesterol} = \frac{\text{Optical density of the unknown sample}}{\text{Optical density of the standard}} \times 100$$

V Definition of characters under study:

mg.cholesterol/g. Yolk:

Among avian species, strain or line differences existed primarily with respect to the amount of cholesterol per unit weight of yolk, hence mg.cholesterol/g.yolk was taken as a measure of egg yolk cholesterol and defined as the egg yolk cholesterol concentration expressed in milligram per gram of yolk.

Total yolk cholesterol:

Total yolk cholesterol was taken as a trait since the total yolk cholesterol is the amount of cholesterol a person would ingest by way of one egg and it is obtained as the egg yolk cholesterol concentration per gram of yolk multiplied by the total yolk weight and expressed in milligram.

Plasma cholesterol:

The plasma cholesterol has a correlation with egg yolk cholesterol. Hence it was taken as a indirect measure for easy selection for low cholesterol and it is defined as the concentration of cholesterol per 100 ml of plasma and is expressed as milligrams percent.

Hatch weight:

Hatched out chicks were weighed in a single pan electrical balance and expressed as gram.

Body weight at 20 weeks:

The pullets/cockrels were weighed at the age of 20 weeks in an electric Keroy balance of 0.1 g accuracy and expressed as gram.

Age at sexual maturity:

The age of the bird in days when it laid its first egg.

Body weight at sexual maturity:

Weight of the bird in gram on the day of first egg measured with an electric Keroy balance of 0.1 g accuracy.

Body weight at 32 weeks:

The pullets/cockrels were weighed at the age of 32 weeks in an electric Keroy balance of 0.1 g accuracy and expressed as gram.

Body weight at 40 weeks:

The pullets/cockrels were weighed at the age of 40 weeks in an electric Keroy balance of 0.1 g accuracy and expressed as gram.

Egg number:

Number of eggs laid by a pullet upto completion of 40 weeks of age.

Egg mass:

Calculated as the total weight of egg (gram) laid by a pullet upto completion of 40 weeks of age.

Egg weight:

Average weight of eggs taken for analysis at appropriate weeks, weighed in single pan electrical balance of 0.0001 g accuracy, and expressed as gram.

Hard cooked egg weight:

The eggs and tap water sufficient to cover the eggs were brought to a boil on 'high' of the heater in a 200 ml covered saucepan. The pan was removed from the heating surface and left to stand for 25 minutes. The hard cooked egg were weighed in a single pan electrical balance of 0.0001 g accuracy and expressed as gram.

Yolk weight:

Yolks were separated after the eggs were hard cooked. Individual yolk was weighed in a single pan electrical balance of 0.0001 g accuracy and expressed as gram.

Shape index:

The length and width of the egg were measured upto 1/10th of a mm with a Vernier calipers. The length was measured as the distance between the poles and the width

as the distance at the maximum circumference of egg.

Shape index was calculated by the formula:

$$\text{Shape index} = \frac{\text{Greatest width}}{\text{Greatest length}} \times 100$$

VI Method of statistical analysis:

i) Estimates of heritability:

The heritability was calculated by half sib analysis with unequal number of progeny as per King and Henderson (1954). The statistical model was,

$$Yij = \mu + S_i + eij$$

where Yij = the observations on the j^{th} individual of the i^{th} sire

μ = general mean

Si = effect due to i^{th} sire with $E(Si) = 0$ and $V(Si) = \sigma S^2$

eij = random effect due to error with $E(eij) = 0$ and $V(eij) = \sigma e^2$

Analysis of variance:

Source of variation	df	S.S.	M.S.	E.M.S.
Between sires	(S-1)	SS_s	MS_s	$\sigma e^2 + K\sigma S^2$
Within sires	$\sum_{i=1}^{S} (ni-1)$	SS_e	MS_e	σe^2

S = Number of sires

$$K = \frac{1}{S-1}\left[\sum_{i-1}^{S} ni - \frac{\sum ni^2}{\sum ni}\right]$$

where ni = number of progenies for i^{th} sire

$$\sum_{i=1}^{S} ni = \text{total number of progenies} = N$$

The variance components σ_S^2 estimates $1/4^{th}$ of the additive genetic variance, $1/16^{th}$ of the additive x additive genetic variance and the various amounts of the sex - linked variance depending upon the species.

The variance components σ_e^2 estimates the remainder of the genetic variance plus all the environmental variance.

$$\text{Heritability} \quad h_S^2 = \frac{4\,\sigma_S^2}{\sigma_S^2 + \sigma_e^2}$$

The standard error of the heritability was calculated as per Swiger et al. (1964).

$$SE\,(h^2S) \cong 4\sqrt{\frac{2(N-1)\,(1-t)^2\,\{1+(k-1)\,t\}^2}{K^2\,(N-S)\,(S-1)}}$$

where t = intra class correlation

$$= \frac{\sigma_S^2}{\sigma_S^2 + \sigma_e^2}$$

A weighted average of the heritability estimates for the data observed at three age groups (32, 40 and 72 weeks) was taken, the weight being the reciprocals of the

variance of each estimate. The variance of this average estimate was computed as a reciprocal of the sum of the weights. The computational equations were:

$$h^2_A = \frac{\frac{1}{V(h^2_{32})} h^2_{32} + \frac{1}{V(h^2_{40})} h^2_{40} + \frac{1}{V(h^2_{72})} h^2_{72}}{\frac{1}{V(h^2_{32})} + \frac{1}{V(h^2_{40})} + \frac{1}{V(h^2_{72})}}$$

$$V(h^2_A) = 1 \Big/ \left(\frac{1}{V(h^2_{32})} + \frac{1}{V(h^2_{40})} + \frac{1}{V(h^2_{72})} \right)$$

$$S.E.(h^2_A) = \sqrt{V(h^2_A)}$$

h^2_A = Weighted average of the heritability

h^2_{32} = Heritability at 32 weeks

h^2_{40} = Heritability at 40 weeks

h^2_{72} = Heritability at 72 weeks

ii) Estimation of genetic correlation:

Genetic correlations were calculated using sire component of variance and covariance (Becker, 1968).

Analysis of variance and covariance:

Source of variation	df	MS_x	MSP_{xy}	MS_y	EMSP
Between sires	(S-1)	$MS_{s(x)}$	$MSP_{s(xy)}$	$MS_{s(y)}$	$\sigma_{exy}+K\sigma_{gxy}$
Within sires	$\sum_{i=1}^{S}(ni-1)$	$MS_{e(x)}$	$MSP_{e(xy)}$	$MS_{e(y)}$	σ_{exy}

Covariance $COV_{s(xy)}$ estimates 1/4th of additive genetic covariance, 1/16th of additive x additive genetic covariance and other non-additive covariances. The genetic correlation between two traits x and y was calculated as given below:

$$r_G(xy) = \frac{COV_{s(xy)}}{\sqrt{\sigma^2_{s(x)} \cdot \sigma^2_{s(y)}}}$$

where $r_G(xy)$ = genetic correlation of the traits x and y

$COV_s(xy)$ = sire component of covariance of traits x and y

$\sigma^2_{s(x)}$ = sire component of variance of trait x

$\sigma^2_{s(y)}$ = sire component of variance of trait y

The standard error of genetic correlation was calculated as per Robertson (1959).

$$SE\ r_G = \frac{1 - r_G^2}{\sqrt{2}} \sqrt{\frac{SE\ (h^2 x)}{2} \cdot \frac{SE\ (h^2 y)}{2}}$$
$$h\ x \qquad\qquad h\ y$$

where SE r_G = standard error of the genetic correlation

SE (h^2x) = standard error of heritability of trait x

SE (h^2y) = standard error of heritability of trait y

h^2x, h^2y = heritabilities of traits x and y

$r_{(G)}$ = genetic correlation between the traits

Phenotypic correlations:

Calculated as simple product moment correlations

$$\gamma P(xy) = \frac{\xi xy - \frac{\xi x \cdot \xi y}{N}}{\sqrt{\left(\xi x^2 - \frac{(\xi x)^2}{N}\right)\left(\xi y^2 - \frac{(\xi y)^2}{N}\right)}}$$

The standard error of phenotypic correlations were calculated by the formula

$$SE\ (rP(xy) = \sqrt{\frac{1 - r^2 P\ (xy)}{N - 2}}$$

where N is the total number of pairs of observations.

Environmental correlations:

The environmental correlations were calculated as per Pillai and Sinha, (1968).

$$r_E = \frac{E \ \mathrm{COV}\ (xy)}{\sqrt{(E \ \mathrm{var}\ x)\ (E \ \mathrm{var}\ y)}}$$

The analysis of the data was made with uptron computer of the reliance computer centre, Bangalore.

RESULTS

RESULTS

I Egg yolk cholesterol:

The cholesterol levels in eggs were measured as

a) mg.cholesterol/g.yolk

b) Total yolk cholesterol (mg)

The above measurements were made at 32, 40 and 72 weeks of ages of the birds belonging to index selection programme.

i) Mean values:

The mean values with standard error and coefficient of variation are presented in Table I.

a) mg.cholesterol/g.yolk:

The mean values ranged from 16.16 mg at 32 weeks to 17.84 mg at 72 weeks of age with gradual increase as the age of bird advanced. The pooled mean over the ages was 16.87 mg. The coefficient of variation of this character was between 14.97 to 16.85 percent.

The differences in the mean values at different ages were highly significant ($P < 0.01$).

b) Total yolk cholesterol:

The mean values ranged from 218.18 mg at 32 weeks to 334.54 mg at 72 weeks with an over all mean over ages was 246.81 mg. The coefficient of variation was between 16.97

TABLE I

MEAN VALUES OF MEASUREMENTS OF EGG YOLK CHOLESTEROL

Age of the bird		Measurement of egg yolk cholesterol: mg.cholesterol/ g. yolk	Measurement of egg yolk cholesterol: Total yolk cholesterol (mg)
32	Number	696	696
	Mean	16.16	218.18
	S.E.	0.0917	1.4037
	C.O.V.(%)	14.9736	16.9754
40	Number	696	696
	Mean	17.35	258.00
	S.E.	0.1109	3.2174
	C.O.V.(%)	16.8504	32.8978
72	Number	163	163
	Mean	17.84	334.54
	S.E.	0.2123	4.9338
	C.O.V.(%)	15.1918	18.8293
Pooled	Number	1555	1555
	Mean	16.87	246.81
	S.E.	0.0782	1.4410
	C.O.V.(%)	16.3897	23.0079

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to 32.90 percent showing existence of high variability for this character. The differences in the mean values at the three age groups were highly significant ($P < 0.01$).

ii) Heritability:

Heritability was worked out for the measurements of egg yolk cholesterol using 696 progenies belonging to 28 sires. The mean squares and estimates of heritability with the standard error are presented in Table II.

a) mg.cholesterol/g.yolk:

The heritability estimates were 0.2415, 0.2377 and 0.2955 at 32, 40 and 72 weeks of age respectively indicating more or less the same heritability at all ages as may be judged from the magnitude of their standard errors. But the pooled estimate over all ages was 0.2886 with a standard error of 0.0137 indicating the heritability of the character was low in this population. The sire differences for this character were significant at all the ages.

b) Total yolk cholesterol:

The heritability estimates were 0.2556, 0.2210 and 0.2239 at 32, 40 and 72 weeks of age respectively indicating more or less similar heritability at these age of the birds.

TABLE II

ESTIMATES OF HERITABILITY OF EGG YOLK CHOLESTEROL

Age of the bird (weeks)	Source of variation	df	Mean squares	
			mg.cholesterol/ g. yolk	Total yolk cholesterol
32	Between sires	27	20.5480**	14456.7**
	Within sires	668	7.8956	5349.5
	Heritability		0.2415	0.2556
	Standard error		0.1408	0.1260
40	Between sires	27	22.4051**	17740.3**
	Within sires	668	8.5385	7216.7
	Heritability		0.2377	0.2210
	Standard error		0.0412	0.1104
72	Between sires	27	11.4041*	5069.6^{NS}
	Within sires	135	7.4047	3778.9
	Heritability		0.2955	0.2239
	Standard error		0.0146	0.3794
Pooled	Heritability		0.2886	0.2355
	Standard error		0.0137	0.0811

** Highly significant ($P < 0.01$)
* Significant ($P < 0.05$)
NS Not significant. ($P > 0.05$)

But the pooled estimate over all ages was 0.2355 with a standard error of 0.0811 indicating a low value of heritability for this character also.

Sire effects were highly significant ($P < 0.01$) at all ages except at 72 weeks of age.

iii) Correlations:

Correlations of the measures of egg yolk cholesterol with the following production traits were computed to study the association of cholesterol level with production traits.

hatch weight,
body weight (20 weeks),
age at maturity,
weight at maturity,
body weight (32 weeks),
body weight (40 weeks),
egg weight,
yolk weight,
plasma cholesterol and
shape index.

a) mg.cholesterol/g.yolk with egg production traits:

1. Correlations at 32 weeks of age:

The estimates of genetic, phenotypic and environmental

correlations of mg.cholesterol/g.yolk at 32 weeks of age of the bird with production traits are presented in Table III.

Correlations with body weight traits:

Correlations with hatch weight, body weight (20 weeks), weight at maturity, body weight (32 weeks) were estimated. The genetic correlations with body weights were positive and ranged from 0.1259 at 20 weeks of age to 0.8036 at hatch. They were moderate to high except for the correlation between mg.cholesterol/g.yolk and body weight at 20 weeks of age. Highest correlation was encountered with hatch weight. The phenotypic and environmental correlations were very low and were negative in most instances.

Correlation with age at maturity:

The genetic correlation was negative and fairly high (-0.4291), while phenotypic and environmental correlations were low.

Correlation with egg weight:

The genetic correlation was positive and medium while phenotypic and environmental correlations were low.

Correlation with yolk weight:

The genetic correlation was negative and fairly high while phenotypic and environmental correlations were low.

TABLE III

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATIONS OF Mg.CHOLESTEROL/G.YOLK WITH OTHER TRAITS AT 32 WEEKS OF AGE

S.No.	Traits correlated with mg.cholesterol/g.yolk	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_P)	S.E.	Environmental (r_E)
1.	Hatch weight	0.8036	0.0259	-0.0295	0.0379	-0.0809
2.	Body weight (20 weeks)	0.1259	0.3860	-0.0503	0.0379	0.0119
3.	Age at maturity	-0.4291	0.1037	0.0548	0.0379	0.0700
4.	Weight at maturity	0.3525	0.6845	0.0044	0.0380	-0.0058
5.	Body weight (32 weeks)	0.3023	0.3564	-0.0644	0.0379	-0.0104
6.	Egg weight	0.3095	0.3382	0.0636	0.0379	0.1435
7.	Yolk weight	-0.5354	0.1849	0.0867	0.0378	0.1969
8.	Total yolk cholesterol	0.1107	0.3985	0.8490	0.0201	0.9224
9.	Plasma cholesterol	0.9512	0.0351	0.7942	0.0230	0.8785
10.	Shape index	0.1028	0.3384	0.1701	0.0374	0.1459

Correlation with total yolk cholesterol:

The genetic correlation was low with a large standard error while the phenotypic and environmental correlations were very high with small standard error. However, the reliability of genetic correlation obtained is questionable due to a very high standard error.

Correlation with plasma cholesterol:

The genetic, phenotypic and environmental correlations were very high between these two characters indicating very close genetic, phenotypic and environmental relationship between the characters.

Shape index:

The genetic, phenotypic and environmental correlations were uniformly low indicating that the shape index was independent of the cholesterol concentration in egg yolk.

2. Correlations at 40 weeks of age:

The estimates of genetic, phenotypic and environmental correlations of mg.cholesterol/g.yolk at 40 weeks of the age of the bird with production traits are presented in Table IV.

Correlation with body weight traits:

Correlations with hatch weight, body weight (20 weeks),

TABLE IV

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATIONS OF Mg. CHOLESTEROL/G. YOLK WITH OTHER TRAITS AT 40 WEEKS OF AGE

S.No.	Traits correlated with mg.cholesterol/g.yolk	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_P)	S.E.	Environmental (r_E)
1.	Hatch weight	0.1184	0.0393	-0.0213	0.0379	-0.0054
2.	Body weight (20 weeks)	-0.1566	0.3635	-0.0318	0.0379	-0.0197
3.	Age at maturity	0.1165	0.0684	0.0486	0.0379	0.0107
4.	Weight at maturity	0.1218	0.4198	-0.0392	0.0379	0.0406
5.	Body weight (40 weeks)	0.2292	0.3529	0.0025	0.0380	0.1517
6.	Egg weight	0.2123	0.1849	0.0301	0.0379	0.2105
7.	Yolk weight	0.1716	0.1437	0.0093	0.0380	0.4580
8.	Total yolk cholesterol	0.6181	0.1286	0.6702	0.0282	0.4050
9.	Plasma cholesterol	0.1582	0.1847	0.6550	0.0287	0.2056
10.	Shape index	-1.0345	0.1113	0.4685	0.0335	0.4730
11.	Egg mass	0.2762	0.1766	0.0712	0.0379	0.2096
12.	Egg number	-0.2464	0.1833	0.0364	0.0379	0.1298

weight at maturity, weight (40 weeks) were estimated. The genetic correlation of this character with body weight traits were generally positive and low except it was negative with body weight at 20 weeks. The phenotypic and environmental correlations were low.

Correlation with age at maturity:

The genetic correlation was low, while the phenotypic and environmental correlations were very low.

Correlation with egg weight:

The genetic and environmental correlations were low, while the phenotypic correlation was very low.

Correlation with yolk weight:

The genetic correlation was low and phenotypic correlation was very low, while the environmental correlation was high.

Correlation with total yolk cholesterol:

The genetic, phenotypic and environmental correlations were uniformly high indicating the close relationship between the characters.

Correlation with plasma cholesterol:

The genetic, environmental correlations were low while the phenotypic correlation was very high.

Correlation with shape index:

The phenotypic and environmental correlations were medium while the genetic correlation was unreliable.

Correlation with egg mass:

The genetic and environmental correlations were low, while phenotypic correlation was very low.

Correlation with egg number:

The genetic correlation was negative and low and phenotypic correlation was very low, while environmental correlation was positive and low.

3. Correlations at 72 weeks of age:

The estimates of genetic, phenotypic and environmental correlations of mg.cholesterol/g.yolk at 72 weeks of the age of the bird with other traits are presented in Table V.

Correlation with egg weight:

The genetic correlation was low and negative, while the phenotypic and environmental correlations were very low.

Correlation with yolk weight:

The genetic correlation was negative and very high while the phenotypic and environmental correlations were very low.

TABLE V

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATION OF Mg.CHOLESTEROL/G. YOLK WITH OTHER TRAITS AT 72 WEEKS OF AGE

S.No.	Traits correlated with mg.cholesterol/g.yolk	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_P)	S.E.	Environmental (r_E)
1.	Egg weight	-0.2759	0.0795	0.0299	0.0788	0.0349
2.	Yolk weight	-0.9403	0.0073	0.0517	0.0787	0.0583
3.	Total yolk cholesterol	0.1093	0.2022	0.8418	0.0425	0.8404
4.	Plasma cholesterol	0.2531	0.0935	0.8448	0.0422	0.8539
5.	Shape index	0.5993	0.0733	0.0635	0.0787	0.0296

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Correlation with total yolk cholesterol:

The genetic correlation was low while the phenotypic and environmental correlations were very high.

Correlation with plasma cholesterol:

The genetic correlation was low while the phenotypic and environmental correlations were very high.

Correlation ~~of~~ with shape index:

The genetic correlation was positive and high, while the phenotypic and environmental correlations were very high.

b) Total yolk cholesterol with egg production traits:

1. Correlations at 32 weeks of age:

The estimates of genetic, phenotypic and environmental correlations of total yolk cholesterol at 32 weeks of the age of the bird with production traits are presented in Table VI.

Correlation with body weight traits:

Correlations with hatch weight, body weight (20 weeks) weight at maturity, body weight (32 weeks) were estimated. The genetic correlations with body weights ranged from 0.0343 at 20 weeks to 0.7251 at weight at maturity showing the magnitude of genetic relationship was variable at

TABLE VI

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATIONS OF TOTAL YOLK CHOLESTEROL WITH OTHER TRAITS AT 32 WEEKS OF AGE

S.No.	Traits correlated with total yolk cholesterol	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_p)	S.E.	Environmental (r_E)
1.	Hatch weight	0.1726	0.0652	-0.0736	0.0379	-0.0678
2.	Body weight (20 weeks)	0.0343	0.3482	-0.0896	0.0378	-0.0599
3.	Age at maturity	-1.1153	0.0125	0.0334	0.0379	0.0722
4.	Weight at maturity	0.7251	0.3408	-0.0929	0.0378	-0.0549
5.	Body weight (32 weeks)	0.4922	0.1464	0.0349	0.0379	-0.0373
6.	Egg weight	0.2561	0.3213	0.4112	0.0346	0.3312
7.	Yolk weight	-1.0348	0.7650	0.5509	0.0317	0.4675
8.	Plasma cholesterol	0.1121	0.3352	0.8710	0.0186	0.8266
9.	Shape index	0.1790	0.3044	0.1425	0.0376	0.1190

different ages of the bird. Body weight (32 weeks), weight at maturity had high and very high genetic relation with the character. The phenotypic correlations were very low and negative to hatch weight, body weight (20 weeks) and weight at maturity. The environmental correlations were very low and negative to body weights.

Correlation with age at maturity:

The genetic correlation was spurious. The phenotypic and environmental correlations were very low.

Correlation with egg weight:

The genetic correlation was medium, while phenotypic and environmental correlations were high and low respectively.

Correlation with yolk weight:

The genetic correlation was spurious, while phenotypic and environmental correlations were high.

Correlation with plasma cholesterol:

The genetic correlation was low whereas the phenotypic and environmental correlations were very high.

Correlation with shape index:

The genetic, phenotypic and environmental correlations were low.

2. Correlations at 40 weeks of age:

The estimates of genetic, phenotypic and environmental correlations of total yolk cholesterol at 40 weeks of the age of the bird with production traits are presented in Table VII.

Correlations with body weight traits:

Correlations with hatch weight, body weight (20 weeks), weight at maturity, body weight (40 weeks) were estimated. The genetic correlations were very low and negative except weight at maturity wherein it was very low. The phenotypic correlations were very low and negative. The environmental correlations were low with all characters except with body weight wherein it was medium.

Correlation with age at maturity:

The genetic, phenotypic and environmental correlations were very low.

Correlation with egg weight:

The genetic correlation was negative and very low, while phenotypic and environmental correlations were positive and medium.

TABLE VII

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATIONS OF TOTAL YOLK CHOLESTEROL WITH OTHER TRAITS AT 40 WEEKS OF AGE

S.No.	Traits correlated with total yolk cholesterol	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_p)	S.E.	Environmental (r_E)
1.	Hatch weight	-0.2223	0.0643	-0.0358	0.0379	-0.0037
2.	Body weight (20 weeks)	-0.2153	0.6032	-0.0119	0.0379	0.0334
3.	Age at maturity	0.0155	0.1171	0.0047	0.0380	0.0044
4.	Weight at maturity	0.0520	0.7215	-0.0158	0.0380	0.0714
5.	Body weight (40 weeks)	-0.0844	0.6279	-0.0544	0.0379	0.3195
6.	Egg weight	-0.0062	0.3288	0.3717	0.0352	0.3798
7.	Yolk weight	0.1071	0.2485	0.5563	0.0315	0.8190
8.	Plasma cholesterol	0.1606	0.3133	0.4739	0.0334	0.4686
9.	Shape index	0.6218	0.2046	0.7908	0.0232	0.7805
10.	Egg mass	0.2160	0.3094	0.1282	0.0376	0.3864
11.	Egg number	0.7332	0.1532	0.1132	0.0377	0.2462

Correlation with yolk weight:

The genetic correlation was low, while phenotypic and environmental correlations were high and very high respectively.

Correlation with plasma cholesterol:

The genetic correlation was low, while phenotypic and environmental correlations were high.

Correlation with shape index:

The genetic correlation was very high. The phenotypic and environmental correlations also were very high indicating close relationship between the characters.

Correlation with egg mass:

The genetic, phenotypic correlations were low while environmental correlation was medium.

Correlation with egg number:

The genetic correlation was high while phenotypic and environmental correlations were ~~small~~ low indicating a high genetic relationship.

3. Correlations at 72 weeks of age:

The estimates of genetic, phenotypic and environmental correlations of total yolk cholesterol at 72 weeks of the age of the bird with other production traits are presented in table VIII.

TABLE VIII

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATION OF TOTAL YOLK CHOLESTEROL WITH OTHER TRAITS AT 72 WEEKS OF AGE

S.No.	Traits correlated with total yolk cholesterol	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_P)	S.E.	Environmental (r_E)
1.	Egg weight	-0.2161	0.4804	0.4663	0.0697	0.4818
2.	Yolk weight	0.1686	0.3587	0.5283	0.0669	0.5357
3.	Plasma cholesterol	0.1199	0.5778	0.9452	0.0257	0.9423
4.	Shape index	0.4103	0.5571	0.0457	0.0787	0.0028

Correlation with egg weight:

The genetic correlation was low and negative. The phenotypic and environmental correlations were high.

Correlation with yolk weight:

The genetic correlation was low while phenotypic and environmental correlations were high.

Correlation with plasma cholesterol:

The genetic correlation was low while the phenotypic and environmental correlations were very high.

Correlation with shape index:

The genetic correlation was high while phenotypic and environmental correlations were very low.

II Plasma cholesterol:

This character had close relationship with egg yolk cholesterol and its study would be useful in early selection of birds for low egg yolk cholesterol. This character was studied in both sexes at 32, 40 and 72 weeks of age.

i) Mean values:

Mean values with standard error and coefficient of variation are presented in Table IX.

TABLE IX

MEAN VALUES OF PLASMA CHOLESTEROL

Age of the bird (weeks)		Plasma cholesterol (mg%)	
		Male	Female
32	Number	173	696
	Mean	146.24	157.69
	S.E.	2.1259	0.9161
	C.O.V.(%)	19.1216	15.3246
40	Number	173	696
	Mean	170.10	189.97
	S.E.	2.2756	1.2846
	C.O.V.(%)	27.5957	17.8417
72	Number	173	163
	Mean	224.50	216.13
	S.E.	0.9943	2.8371
	C.O.V.(%)	5.8100	16.7594
Pooled	Number	519	1555
	Mean	180.1937	178.2600
	S.E.	1.8066	0.9200
	C.O.V.(%)	22.8200	20.3400
Pooled over sex	Number	2074	
	Mean	178.74	
	S.E.	0.8242	
	C.O.V.(%)	20.99	

Plasma cholesterol (mg%):

a) Male:

The mean values ranged from 146.24 mg percent at 32 weeks to 224.50 mg percent at 72 weeks of age with gradual increase as the age of the bird advanced. The coefficient of variation of the plasma cholesterol was between 10.27 to 27.60 percent. The pooled mean over all ages was 180.19 mg percent.

The differences in the mean values at different ages were highly significant ($P < 0.01$).

b) Female:

The mean plasma cholesterol values ranged from 157.69 at 32 weeks to 216.13 mg percent at 72 weeks. The increase in values of the plasma cholesterol was noticed as the age of the bird advanced. The pooled mean over the ages was 178.26 mg percent. The coefficient of variation of the character was between 15.32 to 17.84 percent. The differences in the mean values at different ages were highly significant ($P < 0.01$).

Effect of sex:

The differences in the mean values of males and females were highly significant ($P < 0.01$) at each age group as well as for pooled means over the ages. Females had larger values than males.

ii) Heritability:

Heritability was worked out for the plasma cholesterol using 173 and 696 progenies of males and females respectively belonging to 28 sires. The mean squares and estimates of heritability with the standard error are presented in Table X.

a) Male:

The heritability estimates were 0.7129, 0.7168 and 0.7487 at 32, 40 and 72 weeks of age respectively, indicating more or less similar high heritability at these ages. Significant sire differences at all ages indicated sires as a large source of variation for plasma cholesterol. But the pooled estimate over all ages was 0.7542 with a standard error of 0.0865 indicating the heritability of the character was high.

b) Female:

The heritability estimates were 0.3187, 0.2970 and 0.3054 at 32, 40 and 72 weeks of age respectively indicating similar heritability. The heritability estimates were medium. But the pooled estimate over all ages was 0.3057 with a standard error of 0.0867 indicating the heritability of the character was medium.

TABLE X

ESTIMATES OF HERITABILITY OF PLASMA CHOLESTEROL

Age of the bird (weeks)	Source of variation	Mean squares			
		df	Male	df	Female
32	Between sires	27	1455.80**	27	3072.0**
	Within sires	145	650.70	668	972.4
	Heritability		0.7129		0.3187
	Standard error		0.1483		0.1490
40	Between sires	27	1672.20**	27	3427.0**
	Within sires	145	744.66	668	1142.0
	Heritability		0.7168		0.2970
	Standard error		0.1587		0.1230
72	Between sires	27	1753.68**	27	2080.1*
	Within sires	145	689.00	135	1348.1
	Heritability		0.7487		0.3054
	Standard error		0.1437		0.1214
Pooled	Heritability		0.7542		0.3057
	Standard error		0.0865		0.0867

** Highly significant ($p < 0.01$)
* Significant ($p < 0.05$)

The sire differences for this character also were present at all the ages.

iii) Correlations with egg production traits:

a) Male:

1. Correlations at 32 weeks of age:

The estimates of genetic, phenotypic and environmental correlations of plasma cholesterol (32 weeks) with body weights and plasma cholesterol level (40 weeks) are presented in Table XI.

Correlation with body weight (20 weeks):

The genetic correlation was high while phenotypic and environmental correlations were low.

Correlation with body weight (32 weeks):

The genetic correlation was low while phenotypic and environmental correlations were high.

Correlation with plasma cholesterol (40 weeks):

The genetic correlation was very high. The phenotypic and environmental correlations were high indicating a close relationship with these two characters.

2. Correlations at 40 weeks of age:

The genetic, phenotypic and environmental correlations of plasma cholesterol (40 weeks) with body weights are presented in the Table XII.

TABLE XI

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATIONS OF PLASMA CHOLESTEROL WITH OTHER TRAITS AT 32 WEEKS OF AGE (MALE)

S.No.	Traits correlated with plasma cholesterol	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_P)	S.E.	Environmental (r_E)
1.	Body weight (20 weeks)	0.4710	0.2753	0.2475	0.0660	0.2409
2.	Body weight (32 weeks)	0.1039	0.1127	0.4443	0.0565	0.4252
3.	Plasma cholesterol (40 weeks)	0.9567	0.0738	0.8853	0.0152	0.8704

TABLE XII

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATIONS OF PLASMA CHOLESTEROL WITH OTHER TRAITS AT 40 WEEKS OF AGE (MALE)

S.No.	Traits correlated with plasma cholesterol	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_p)	S.E.	Environmental (r_E)
1.	Body weight (20 weeks)	0.4146	0.2809	0.3258	0.0629	0.3323
2.	Body weight (32 weeks)	0.1006	0.1150	0.4728	0.0546	0.4583
3.	Body weight (40 weeks)	0.4804	0.1018	0.4113	0.0585	0.3917

Correlation with body weight (20 weeks):

The genetic correlation was high while phenotypic and environmental correlations were medium.

Correlation with body weight (32 weeks):

The genetic correlation was low while the phenotypic and environmental correlations were high.

Correlation with body weight (40 weeks):

The genetic and phenotypic correlations were high, while the environmental correlation was medium.

b) Female:

The plasma cholesterol was correlated with the following production traits: hatch weight, body weight (20 weeks), age at maturity, weight at maturity, body weight (32 weeks), body weight (40 weeks), egg weight, yolk weight, mg.cholesterol/g.yolk, total yolk cholesterol and shape index, to study the relationship between the plasma cholesterol and the measurements of egg yolk cholesterol as well as production traits in relation to selection of birds for low cholesterol level in the population.

1. Correlation at 32 weeks of age:

The estimates of genetic, phenotypic and environmental

correlations of plasma cholesterol at 32 weeks of the age of the bird with production traits are presented in Table XIII.

Correlations with body weight traits:

Correlations with hatch weight, body weight (20 weeks), weight at maturity, body weight (32 weeks) were estimated. The genetic correlations with body weights varied from 0.1387 at 20 weeks of age to 0.7511 at hatch showing the magnitude of genetic relationship was variable at different ages of the bird. Hatch weight and weight at maturity had a very high genetic correlation. The phenotypic and environmental correlations were very low in general.

Correlation with age at maturity:

The genetic correlation was negative and fairly high while phenotypic and environmental correlations were low indicating a high negative genetic relationship with age at maturity.

Correlation with egg weight:

The genetic correlation was very low and the phenotypic correlation was medium, while the environmental correlation was low.

TABLE XIII

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATIONS OF PLASMA CHOLESTEROL WITH OTHER TRAITS AT 32 WEEKS OF AGE

S.No.	Traits correlated with plasma cholesterol	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_P)	S.E.	Environmental (r_E)
1.	Hatch weight	0.7511	0.0285	-0.0082	0.0380	-0.0726
2.	Body weight (20 weeks)	0.1387	0.3445	0.0214	0.0380	0.0077
3.	Age at maturity	-0.4857	0.0870	0.0356	0.0379	0.0600
4.	Weight at maturity	0.6599	0.3952	-0.0434	0.0379	0.0156
5.	Body weight (32 weeks)	0.4917	0.2664	0.0046	0.0380	-0.0250
6.	Egg weight	0.0791	0.3328	0.3498	0.0356	0.1488
7.	Yolk weight	-0.4110	0.2913	0.5027	0.0328	0.2102
8.	mg.cholesterol/g.yolk	0.9512	0.0351	0.7942	0.0230	0.8785
9.	Total yolk cholesterol	0.1121	0.3352	0.8710	0.0186	0.8265
10.	Shape index	0.1079	0.2988	0.1901	0.0373	0.1655

Correlation with yolk weight:

The genetic correlation was medium and negative. Phenotypic correlation was high and environmental correlation was low.

Correlation with mg.cholesterol/g.yolk:

The genetic, phenotypic and environmental correlations were positive and very high between these two characters, indicating the very close relationship between the characters.

Correlation with total yolk cholesterol:

The genetic correlation was low while the phenotypic and environmental correlations were high.

Correlation with shape index:

The genetic, phenotypic and environmental correlations were low.

2. Correlation at 40 weeks of age:

The estimates of genetic, phenotypic and environmental correlations of plasma cholesterol at 40 weeks of the age of the bird with production traits are presented in Table XIV.

Correlations with body weight traits:

Correlations with hatch weight, body weight (20 weeks) weight at maturity, body weight (40 weeks) were estimated.

TABLE XIV.

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATIONS OF PLASMA CHOLESTEROL WITH OTHER TRAITS AT 40 WEEKS OF AGE

S.No.	Traits correlated with plasma cholesterol	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_P)	S.E.	Environmental (r_E)
1.	Hatch weight	-1.2440	0.1309	0.0018	0.0380	-0.0143
2.	Body weight (20 weeks)	0.0353	0.5753	-0.0246	0.0379	0.0572
3.	Age at maturity	0.1934	0.1032	0.0247	0.0379	-0.0008
4.	Weight at maturity	-0.4575	0.5208	-0.0108	0.0380	0.0194
5.	Body weight (40 weeks)	-0.6995	0.6279	-0.0807	0.0379	0.4985
6.	Egg weight	0.1297	0.2943	0.3369	0.0357	0.6887
7.	Yolk weight	0.1267	0.2257	0.4904	0.0331	0.3930
8.	mg.cholesterol/g.yolk	0.1582	0.1847	0.6550	0.0287	0.2056
9.	Total yolk cholesterol	0.1606	0.3133	0.4738	0.0334	0.4686
10.	Shape index	-1.0568	0.0158	0.4240	0.0342	0.4263
11.	Egg mass	-0.4407	0.2381	0.1174	0.0377	0.7130
12.	Egg number	0.7397	0.1395	0.3971	0.0348	0.3971

The genetic correlations with body weights varied from -0.4575 at weight at maturity to -1.2440 at hatch, showing the magnitude of genetic relationship was variable at different ages of the bird. Hatch weight had a spurious genetic correlation. The correlation with weight at maturity was highly negative and with body weight at 40 weeks was very highly negative. The phenotypic correlations were very low. Environmental correlations were also low with all traits except with body weight (40 weeks) wherein it was high.

Correlation with egg weight:

The genetic correlation was low and positive. The phenotypic correlation was medium, while the environmental correlation was very high.

Correlation with yolk weight:

The genetic correlation was low and positive. The phenotypic correlation was high while the environmental correlation was medium.

Correlation with mg.cholesterol/g.yolk:

The genetic correlation was low while the phenotypic correlation was very high. The environmental correlation was low.

Correlation with total yolk cholesterol:

The genetic correlation was low, while the phenotypic and environmental correlations were high.

Correlation with shape index:

The genetic correlation was spurious, while the phenotypic correlation and environmental correlation were high.

Correlation with egg mass:

The genetic correlation was high and negative. Phenotypic correlation was low. Environmental correlation was very high.

Correlation with egg number:

The genetic correlation was very high and phenotypic and environmental correlations were medium.

3. Correlation at 72 weeks of age:

The estimates of genetic, phenotypic and environmental correlations of plasma cholesterol with other traits at 72 weeks of age of the bird are presented in Table XV.

Correlation with egg weight:

The genetic correlation was low and negative while the phenotypic and environmental correlations were high.

TABLE XV

GENETIC, PHENOTYPIC AND ENVIRONMENTAL CORRELATIONS OF PLASMA CHOLESTEROL WITH OTHER TRAITS AT 72 WEEKS OF AGE

S.No.	Traits correlated with plasma cholesterol	Correlations				
		Genetic (r_G)	S.E.	Phenotypic (r_p)	S.E.	Environmental (r_E)
1.	Egg weight	-0.0696	0.2428	0.4284	0.0712	0.4298
2.	Yolk weight	0.1424	0.1676	0.4959	0.0684	0.4831
3.	mg.cholesterol/g.yolk	0.2531	0.0935	0.8448	0.0422	0.8539
4.	Total yolk cholesterol	0.1119	0.5778	0.9452	0.0257	0.9423
5.	Shape index	0.6289	0.1961	0.0213	0.0788	-0.0255

Correlation with yolk weight:

The genetic correlation was low and positive while phenotypic and environmental correlations were high.

Correlation with mg.cholesterol/g.yolk:

The genetic correlation was medium, while the phenotypic and environmental correlations were very high.

Correlation with total yolk cholesterol:

The genetic correlation was low, while phenotypic and environmental correlations were high.

Correlation with shape index:

The genetic correlation was very high and positive while the phenotypic and environmental correlations were very low.

III Egg production traits:

The study aims to study the inheritance of egg yolk cholesterol and plasma cholesterol and their genetic relationship with other important egg production traits so that selection for low egg cholesterol can be practised without affecting the economic traits in poultry. Since the production traits were correlated with cholesterol measurements the information on the average values and the heritability of the production traits were studied.

The mean values and heritability estimates of the production traits are presented in Table XVI.

i) Mean values:

In the population studied the mean values of hatch weight was 36.52 g and age at sexual maturity 165.69 days. The body weight increased from 1061.19 g at 20 weeks to 1398.42 g at 40 weeks of age with a gradual increase as the age advanced. The egg weight ranged from 45.18 g at 32 weeks to 57.00 g at 72 weeks with a gradual increase as the age advanced. Similarly hard cooked egg weight increased from 44.41 g at 32 weeks to 56.17 g at 72 weeks. There was increase in yolk weight also from 13.47 g at 32 weeks to 18.72 g at 72 weeks. However, shape index remained the same through ages. The egg number was 64.55 eggs and egg mass was 3135.88 g at 40 weeks of age.

The variability in all the traits ranged from 4.64 to 15.14 percent.

ii) Estimates of heritability:

The estimates of heritability for all the production traits under study were low in general in the population. Only in case of body weight traits they were medium.

TABLE XVI

MEAN VALUES AND ESTIMATES OF HERITABILITY OF PRODUCTION TRAITS

S.No.	Traits	Age (weeks)	n	Mean ± S.E.	C.O.V.	h^2 ± S.E.
1.	Hatch weight(g)		696	36.52±0.11	7.94	0.2238±0.00
2.	Body weight(g)	20	696	1061.19±5.08	12.63	0.1194±0.19
3.	Age at maturity (days)		696	165.69±0.29	4.64	0.1965±0.01
4.	Weight at maturity (g)		696	1192.46±5.62	12.44	0.1012±0.21
5.	Body weight(g)	32	696	1325.18±4.40	8.75	0.3058±0.16
		40	696	1398.42±6.49	12.24	0.1519±0.24
6.	Egg weight (g)	32	696	45.18±0.13	7.55	0.2552±0.12
		40	696	48.37±0.19	8.62	0.3281±0.14
		72	163	57.00±0.39	8.76	0.2389±0.07
7.	Hard cooked egg weight (g)	32	696	44.41±0.13	7.91	-
		40	696	47.49±0.19	10.77	-
		72	163	56.17±0.39	8.90	-
8.	Yolk weight(g)	32	696	13.47±0.04	7.99	0.1566±0.08
		40	696	14.67±0.07	14.19	0.1621±0.04
		72	163	18.72±0.13	8.84	0.3852±0.05
9.	Shape index	32	696	72.85±0.16	5.85	0.1525±0.06
		40	696	72.66±0.30	11.06	0.1998±0.09
		72	163	72.82±0.24	4.19	0.1386±0.07
10.	Egg number	40	696	64.55±0.36	14.80	0.1620±0.07
11.	Egg mass (g)	40	696	3135.88±17.99	15.14	0.2927±0.12

DISCUSSION

DISCUSSION

The study on genetic basis of egg yolk cholesterol was aimed to determine the inheritance of egg yolk cholesterol as well as the plasma cholesterol and to estimate the genetic relationship of these two characters with other economically important egg production traits in poultry. It is the objective to explore the possibility of development of lines of poultry with low egg yolk cholesterol without deterioration in the other economic traits in the lines to be developed.

The age of the birds was also considered since the egg yolk cholesterol content changes with age. mg.cholesterol per gram yolk and total yolk cholesterol were taken at the ages of 32, 40 and 72 weeks. The plasma cholesterol was also included in the study for the possibility of earlier selection since this character was highly related to egg yolk cholesterol. The plasma cholesterol was studied in both sexes.

I Egg yolk cholesterol:

i) Mean values of egg yolk cholesterol:

a) mg.cholesterol/g.yolk:

The population when pooled over ages had a mean value of 16.87 mg with a coefficient of variation of 16.30 percent. Considerable variation was found among various species and

inbred lines in the concentration of cholesterol as well as the total amount of cholesterol per egg (Nicholas et al., 1963; Chavous et al., 1965 and Bair and Marion, 1978). The value obtained in the present study was slightly higher than that reported by Chung et al. (1965), Weiss et al. (1967), Rangachar et al. (1970), Bartov et al. (1971), Lall and Slinger (1973), Bair and Marion (1979) and Hollands et al. (1930), but lowerer than that reported by Fisher and Loveille (1957), Combs and Halbacka (1960), Daghir et al. (1960), Harris and Wilcox (1963), Lepore and Marks (1965), Chand and Sapra (1973), Washburn and Nix (1974a), Chand et al (1979) and Anglin and Briles (1980). This value concurred with Miller and Denton (1962), Kudchodkar et al. (1976) and Becker et al. (1977).

The differences in the strain, age structure of the population as well as the method of estimation might have contributed to the discrepancy noticed.

The value of the character increased with advancing age. This concurred with March and Bailey (1959) and Rao et al. (1964), but Turk and Barnett (1971) found that cholesterol concentration was not affected by hen's age. Chand et al. (1978) stated that yolk lipids did not change significantly upto 12 months of age in Australorp.

b) Total yolk cholesterol:

The overall mean value was 246.81 mg with a coefficient of variation of 23.01 percent. The variability of this measurement was more when compared with mg.cholesterol/g.yolk.

The results of the present study were in agreement with that obtained by Becker et al. (1977). Similar to mg. cholesterol/g.yolk, the values of total yolk cholesterol increased with advancing age.

ii) Heritability:

The heritability estimates of mg.cholesterol/g.yolk were found to be 0.2415 ± 0.1408, 0.2377 ± 0.0412 and 0.2955 ± 0.0146 taken at 32, 40 and 72 weeks of age with an overall pooled estimate of 0.2866 ± 0.0137. All the estimates were moderate and of similar magnitude with estimates at higher ages being more precise in view of smaller standard errors.

The heritability estimates of total egg yolk cholesterol recorded at 32, 40 and 72 weeks of age were 0.2556 ± 0.1260, 0.2210 ± 0.1104, 0.2239 ± 0.3794. The estimate when pooled over all the ages was found to be 0.2355 ± 0.0311. The standard error of the estimates especially when the measures were taken in older hens were high compared to that of mg.cholesterol/g.yolk. Although the estimates of heritability

for both measures of cholesterol were the same in all the age groups it appears that mg.cholesterol/g.yolk would be a better measure in view of the smaller sampling errors of their heritability estimates.

Becker et al. (1975) contrary to the present findings have observed a higher heritability 0.31 for total yolk cholesterol compared to 0.14 for mg.cholesterol/g.yolk. Becker et al. (1977) have concluded that the higher heritability for the total yolk cholesterol than the mg. cholesterol/g.yolk might be due to higher heritability of egg yolk which is a component of mg.cholesterol in total yolk. The disagreement of the present study with that of Becker et al. (1975) might be related to low heritability of yolk weight in the strain used for the study.

The range of heritability estimates reported in the literature was from 0.14 (Becker et al., 1975) to 0.43 (Jaffe, 1964) for mg.cholesterol per gram yolk. The values obtained in the present study were within this range and was in consistent with those reported by Ali et al. (1974) Cunningham et al. (1974) and Becker et al. (1977).

There were only two estimates (Becker et al. 1975, 1977) for total yolk cholesterol and were 0.31 and 0.27 respectively. The present estimates were not much different from those reported earlier.

Sire component of variances for mg.cholesterol/g.yolk were significant in all age groups while it was significant at 32 and 40 weeks age groups in the case of total yolk cholesterol.

This indicates that the genetic variability for mg. cholesterol/g.yolk is more consistent with advancing age than that of total egg yolk cholesterol possibly due to the confounding effect of the yolk weight. Genetic variability for mg.cholesterol/g.yolk was also reported by Harris and Wilcox (1963) and Washburn and Nix (1974). Harris and Wilcox (1963) demonstrated a significant dam family difference while Washburn and Nix (1974) demonstrated a significant sire family difference. The moderate heritability for the traits obtained in the study suggested that family selection could be of use in altering the level of cholesterol in the egg yolk in the desired direction. mg.cholesterol/g.yolk would be a better of the two measures in view of less variability and bias due to confounding effect of yolk weight.

iii) Correlations with egg production traits:

a) Correlation with body weight traits:

The genetic, phenotypic and environmental correlations of mg.cholesterol/g.yolk and total yolk cholesterol with hatch weight and mature weights taken at 20, at first egg, 32 and 40 weeks of age were computed. A high genetic correlation

of mg.cholesterol/g.yolk was observed with hatch weight. All the rest of the correlation were either low or moderate invariably with large standard errors indicating that the cholesterol measures will have no genetic association with mature body weight. The phenotypic, environmental correlations between the body weight traits and the cholesterol measures were nearer to zero indicating of no relationship between these traits possibly the physiological basis for cholesterol and the body weight traits are not the same and it could be possible to select for low cholesterol content without affecting the body weight traits. There are no comparable studies available in the literature wherein the association of cholesterol contents with body weights were investigated in poultry.

b) Correlation with age at maturity:

A high negative genetic correlation of -0.4291 ± 0.1037 was observed between mg.cholesterol/g.yolk at 32 weeks and age at maturity. The total yolk cholesterol taken at 32 weeks of age had a genetic correlation of more than one indicating the lack of association between the two traits. The genetic correlations at 40 weeks with age at maturity was low. The phenotypic and environmental correlations between cholesterol content and age at maturity were uniformly low. The results indicate that selection for low mg.cholesterol/g.yolk at 32 weeks might increase the age at maturity while

the cholesterol measures taken at 40 weeks would have no influence on the age at maturity.

c) Correlation with egg number and egg mass:

Cholesterol contents of egg produced by birds at 40 weeks of age were investigated for their genetic, phenotypic and environmental correlations with egg number and egg mass upto 40 weeks of age. A medium and negative genetic correlation of egg number was found with mg.cholesterol/g. yolk while the genetic correlation with total yolk Cholesterol was high and positive. It indicates that selection for low mg. cholesterol/g.yolk is unlikely to reduce the egg number while it may be much less a fact when the selection is for low total egg yolk cholesterol. The genetic correlation of egg mass with cholesterol measures were positive and moderate. It may indicate that there may be a marginal decrease in egg mass for selection against cholesterol content. The phenotypic and environmental correlations of egg yolk cholesterol with egg number and egg mass were positive and ranged from low to moderate. These findings were in contrast with the findings of Cunningham et al (1973), Ali et al. (1974), Washburn and Nix (1974) who have reported a negative phenotypic correlation between egg production and egg yolk cholesterol.

d) Correlation with egg weight:

The measure of mg.cholesterol/g.yolk was found to be

positively correlated with egg weight when the egg yolk cholesterol measures were taken at 32, 40 weeks of age and negatively correlated when it was taken at 72 weeks of age. A similar change from positive to negative genetic correlation was also observed with advancing age when the egg weight was correlated with total yolk cholesterol.

It was also observed that the genetic correlations of total yolk cholesterol with egg weight had large standard errors while those with mg.cholesterol/g.yolk had small standard errors when correlated with cholesterol measures taken at 40 and 72 weeks of age.

The results indicate a possible reduction in egg weight when selection is to be practiced against mg.cholesterol/g. yolk measure on eggs laid at 32 or 40 weeks of age while the reverse could be a factor when the cholesterol measure is taken on eggs laid by older birds.

The phenotypic and environmental correlations were all positive and ranged from low to moderate. The results of the studies on phenotypic correlation of egg weight with egg yolk cholesterol content appeared in the literature were variable. Cunningham (1974) obtained no correlation between the traits. Becker et al. (1975) obtained a negative phenotypic correlation, while Chand et al. (1978) found a positive phenotypic correlation.

e) Correlation with yolk weight:

The genetic correlation of mg.cholesterol/g.yolk were negative and very high at 32 and 72 weeks of age while it was low at 40 weeks of age indicating that high negative genetic relationship between these two traits. Hence, selection for low egg yolk cholesterol will be followed with increase in yolk weight. However, the correlation of total yolk cholesterol with yolk weight was low. Thus the two measurements of the same trait viz. egg yolk cholesterol differed in their relationship with yolk weight. If the selection is based on total yolk cholesterol there will not be much effect on the yolk size. The genetic correlation obtained with mg.cholesterol/g.yolk and yolk weight was in agreement with the results obtained by Harris and Wilcox (1963), Nicholas et al. (1963) and Becker et al (1975). The results indicated that selection for mg. cholesterol/g.yolk taken on eggs laid at 40 weeks of age will not significantly alter the yolk weight. Further a high environmental correlation between the traits also indicates that it may be possible to alter these two traits by attending to nutritional and other environmental factors.

f) Correlations with shape index:

The genetic correlations ~~were~~ was low at 32 weeks while it was very high at 72 weeks. It indicated that selection for

low egg yolk cholesterol would affect shape index if selection was made at 72 weeks. With the cholesterol content of eggs laid at 40 weeks of age the shape index seems to be positively correlated very high phenotypically and environmentally. The genetic correlation with mg. cholesterol/g.yolk was unreliable while that with total yolk cholesterol was high and positive. The prediction of alteration in the shape index when selection is based on mg.cholesterol/g.yolk is not conclusive.

iv) Correlations among mg.cholesterol/g.yolk, total yolk cholesterol and plasma cholesterol:

Correlation between mg.cholesterol/g.yolk and total yolk cholesterol:

The genetic correlation of mg.cholesterol per gram yolk with total yolk cholesterol was variable between age groups while the phenotypic and environmental correlations between these two traits remained high throughout. All three correlations between these two traits were high when the cholesterol contents were measured from eggs laid at 40 weeks of age which may indicate that the selection against either trait will lower the value of the trait.

Correlation between mg.cholesterol/g.yolk and plasma cholesterol:

The genetic phenotypic and environmental correlations between mg.cholesterol/g.yolk taken on eggs laid at 32 weeks

of age and plasma cholesterol were uniformly high indicating a possibility of indirect selection for low content of cholesterol/g.yolk by selecting against plasma cholesterol content at early ages. Andrews et al. (1968) observed that the plasma cholesterol contributed the egg yolk cholesterol and the present study gives a corroborative evidence of the same.

The genetic relationship of the plasma cholesterol content and egg yolk cholesterol content at 40 weeks of age were variable from low to medium for 40 and 72 weeks observation. The phenotypic and environmental correlations were high with the possible exception of environmental correlation at 40th week. A Negative phenotypic correlations were obtained by Marion et al. (1960) and Chand et al. (1973) and supra. Low to high genetic correlation was reported by Washburn and Nix (1974). The age structure of the population, the level of production and the genetic differences between the populations might be the reasons for the difference.

Correlation of total yolk cholesterol and plasma cholesterol:

Total yolk cholesterol and plasma cholesterol were lowly correlated genetically but their phenotypic and environmental correlations were high for all the age groups studied.

II Plasma cholesterol:

i) Mean values:

In males the mean values of plasma cholesterol was 180.19 mg percent ranging from 146.24 mg percent at 32 weeks to 224.50 mg percent at 72 weeks. In females the average values were 178.26 mg percent with range of 157.69 mg percent at 32 weeks to 216.13 mg percent at 72 weeks. In both sexes the mean values increased with advancing age. The sex effect was also very high, the males having lower values than females. The present mean values in the population were low when compared to the values reported by Wilcox et al. (1963), Washburn and Nix (1974), Kaminski et al. (1979) and Marks and Siegel (1980).

ii) Heritability:

Heritability values were very high (0.7129 to 0.7487) in males while the heritability estimates were medium (0.2970 to 0.3187) in females. The pooled estimates were 0.7542 $\pm$ 0.0365 and 0.3057 $\pm$ 0.0367 for males and females respectively.

The heritability estimates indicate that individual selection will be effective in lowering the plasma cholesterol level. The significant sire differences in both males and females also attributed the presence of high genetic variation through sire families. The heritability estimates

were in agreement with that obtained by Bumgardner (1955), Cherms et al. (1960), Wilcox et al. (1963), Hollands et al. (1980) and Marks and Siegel (1980). The heritability results were less than those of Washburn and Nix (1974).

iii) Correlations with egg production traits:

Correlation with body weight traits:

In females the genetic correlations were low to very high. Hatch weight and weight at maturity were having high genetic correlation showing that the selection for hatch weight or weight at maturity would increase the plasma cholesterol levels in females. In males the genetic correlation were high with body weight at 20 and 40 weeks while it was low at 32 weeks. Therefore, the selection for body weight at 20 or 40 weeks would increase the plasma cholesterol levels in males. The phenotypic correlations were low at all ages and negative at 40 weeks. However, Wilcox et al. (1963) reported low negative phenotypic correlation between blood cholesterol and body weight at 32 weeks.

Correlation with age at maturity:

The genetic correlation was negative and fairly high at 32 weeks indicating that selection for low plasma cholesterol would increase the age at sexual maturity similar to the findings for mg.cholesterol/g.yolk with age at maturity.

Phenotypic correlation was very low but positive. Wilcox et al. (1963) obtained a low negative phenotypic correlation.

Plasma cholesterol taken at 40 weeks of age had low genetic relationship and very low phenotypic and environmental relationship with age at maturity. Similar associations were also found between mg.cholesterol/g.yolk of eggs laid by 40 weeks old birds and their age at maturity. The results indicate that selection for low plasma cholesterol will have very little effect on age at maturity.

Correlations with egg number and egg mass:

The plasma cholesterol taken at 40 weeks of age was found to be positively correlated genetically with egg number and negatively correlated with egg mass. The results indicated that plasma cholesterol will adversely affect the egg number but improve the egg mass. Wilcox et al. (1963) reported positive genetic correlation between blood cholesterol and egg production and it is in agreement with present study.

Correlation with egg weight:

The genetic correlation was low in general between the two traits. Selection for low plasma cholesterol may not affect the egg weight. The phenotypic correlation was medium and increased with age. Wilcox et al. (1963) obtained a low and negative phenotypic correlation between adult blood cholesterol level and egg weight.

Correlation with yolk weight:

Genetic correlation was high and negative at 32 weeks while they were low at 40 and 72 weeks. Therefore, the early selection at 32 weeks for low plasma cholesterol would increase the yolk weight and at the other ages it may not affect the yolk weight.

Correlation with shape index:

The genetic correlation was low at 32 weeks and high at 72 weeks of age. It indicated that selection for low plasma cholesterol at 32 weeks of age would not affect the shape index.

SUMMARY

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The present study was undertaken to determine the genetic parameters of egg yolk cholesterol and plasma cholesterol and their relationship with other economic traits *in chickens*.

The ultimate objective is to explore the possibility of development of strains/lines of chickens with low egg yolk cholesterol level.

The experiment was carried out at Poultry section of Livestock Research Station, Kattupakkam during the period from 1980 to 1982. Meyer strain single comb White Leghorns raised in a single hatch and maintained under index selection programme were used as experimental materials. A total of 696 pullets belonging to fifth generation under 28 sire families were included in the study.

The egg yolk cholesterol was measured as mg.cholesterol/g.yolk and total yolk cholesterol. The plasma cholesterol was also studied.

The cholesterol measures were taken at 32, 40 and 72 weeks of age of the birds. Plasma cholesterol measure was also taken in males at respective ages. Heritability

of cholesterol measures, body weight at hatch, 20 weeks maturity, 32 and 40 weeks of age, age at maturity, egg number, egg mass (40 wks), egg weight, yolk weight, shape index were computed.

The genetic, phenotypic and environmental association of cholesterol measures with production traits were investigated. Hatch weight, body weight at 20 weeks, maturity, 32 and 40 weeks of age were the body weight traits whose association with cholesterol measures were investigated. Among the reproductive traits, age at maturity, egg number and egg mass upto 40 weeks, egg weight, yolk weight and shape index were considered to investigate their genetic, phenotypic and environmental associations with cholesterol measures. The genetic, phenotypic and environmental correlations among cholesterol measures were also investigated.

mg.cholesterol/g.yolk increased from 16.16 $\pm$ 0.09 mg at 32 weeks to 17.84 $\pm$ 0.21 mg at 72 weeks with an overall pooled average of 16.87 $\pm$ 0.08 mg. The level of total yolk cholesterol and plasma cholesterol also showed an increasing trend with advancing age. The total yolk cholesterol ranged from 219.18 $\pm$ 1.40 mg at 32 weeks to 334.54 $\pm$ 4.93 mg at 72 weeks with an overall average of 246.81 $\pm$ 1.44 mg.

The plasma cholesterol ranged from 146.24 ± 2.13 mg% (32 weeks) to 224.50 ± 0.99 mg% (72 weeks) in males while in females it ranged from 157.69 ± 0.92 mg% (32 weeks) to 216.13 ± 2.84 mg% (72 weeks).

Sire family differences for the three cholesterol measures were significant at all age groups except for total egg yolk cholesterol measured at 72 weeks of age. The heritability estimates of egg yolk cholesterol measures were moderate at all ages with the mg.cholesterol/g.yolk giving a more precise estimate especially at 40 and 72 weeks of age with small standard errors compared to total yolk cholesterol. The estimates pooled over ages for mg. cholesterol/g.yolk and total yolk cholesterol were 0.2866 ± 0.0137 and 0.2355 ± 0.0811 respectively. The range of heritability estimates indicate that family selection would be effective in changing the level of egg yolk cholesterol to the desired direction.

The heritability estimates of plasma cholesterol were uniformly very high in males at all ages with a pooled estimate of 0.7542 ± 0.0865. In females the estimates were moderate in all the age groups and pooled over estimate was 0.3057 ± 0.0867.

mg.cholesterol/g.yolk had a high genetic correlation with hatch weight, while it had a low to moderate correlation with mature weights indicating that for low egg yolk cholesterol is unlikely to affect the mature body weight. Total yolk cholesterol had a low to moderate genetic correlation with body weight measures.

mg.cholesterol/g.yolk measured from eggs laid at 32 weeks of age had a high positive genetic correlation with age at maturity while the same measure of cholesterol taken on eggs laid at 40 weeks of age had no genetic influence on age at maturity.

A low non significant genetic correlation was encountered between mg. cholesterol per gm of yolk and the egg number indicating the efforts to reduce the cholesterol content in yolk may possibly not affect the egg number very seriously.

The genetic correlation of egg mass with both egg yolk cholesterol measures were positive and moderate and there may be a marginal decrease in egg mass if selection is directed to lower the egg yolk cholesterol content.

Egg weight was found to be positively correlated with cholesterol measures at 32 and 40 weeks of age and

negatively correlated when the measures were taken on eggs laid at 72 weeks of age. A possible reduction in egg size cannot be ruled out when selection is to practiced against egg yolk cholesterol content on eggs laid by younger hens.

The genetic correlation of yolk weight with mg. cholesterol/g.yolk was high and negative while it was low with total yolk cholesterol.

The egg yolk cholesterol content in older hens were highly correlated with shape index.

The phenotypic and environmental correlations between cholesterol measures and production traits were mostly low.

The genetic correlation of total yolk cholesterol with production traits had a very high standard error and inconclusive in few instances. It appeared that mg. cholesterol per gram yolk taken on eggs laid by hens around 40 weeks of age was more reliable in view of small standard errors of the measurement and heritability of the trait. The genetic, phenotypic and environmental correlations with production traits had also low standard errors.

The genetic, phenotypic and environmental correlations between the two egg yolk cholesterol measures were variable depending upon the age at which they were measured. However

when the measures were taken at 40 weeks of age they were highly correlated.

mg.cholesterol/g.yolk present in eggs laid at 32 weeks of age had high genetic, phenotypic and environmental correlation with plasma cholesterol indicating a possibility of indirect selection for low egg yolk cholesterol by basing the selection on plasma cholesterol. The relationship of plasma cholesterol and mg.cholesterol/g.yolk at later ages were variable. Total yolk cholesterol and plasma cholesterol was lowly correlated genetically.

Plasma cholesterol was found to be highly correlated with body weight positively and negatively correlated with age at maturity.

The genetic correlation of egg weight with plasma cholesterol was low while that of yolk weight with plasma cholesterol was high and negative. Plasma cholesterol at 40 weeks of age was found to be positively correlated genetically with egg number and negatively with egg mass. The shape index was lowly correlated with plasma cholesterol.

The results indicated that selection for low egg yolk cholesterol content could be directed by inclusion of mg. cholesterol/g.yolk in the selection index without seriously affecting the production traits.

In males the plasma cholesterol taken at 32 weeks of age if included in the index would help the male line selection to produce low egg yolk cholesterol lines.

BIBLIOGRAPHY

BIBLIOGRAPHY

Achard, C., Levy, J., Georgia, N., and Kakis. 1934. Lecholesterol des ailments. Arch. Mal Appar. Dig. Mal. Nutr. 24: 785 - 793. cited by Bair and Marion, 1978.

Ali, M.N., Krueger, W.F. and Bradley. 1975. Selection for low and high egg yolk cholesterol. Poultry Sci. 54:1726.

Ali, M.N., Krueger, W.F., Fanguy, R.C. and Bradley, J.W. 1974. Poultry Sci. 53: 384.

American Dietetic Association. 1981. Hand book of clinical Dietetics, New Haven and London, Yale University Press.

American Heart Association. 1968. The National Diet. heart study. Final report. Circulation 37: suppl.1.

Andrew Gement. 1966. Adsorption of cholesterol by components of the intima in atherosclerosis. Am. Jl. of Medl. Sci. 252: 92/192, 96/196.

Andrews, J.W.Jr., Wagstaff, R.K. and Edwards, Jr., H.M. 1968. Cholesterol metabolism in the laying Fowl Amer. J. Physiol. 214: 1078 - 1083.

Anglin, H.A. and Briles, C.O. 1980. The genetic association of shell colour with yolk cholesterol content in three breeds. Poultry Sci. 59: 7: 1578.

Anonymous, 1978. Blood lipids and coronary heart disease. Nut. Rev. 36: 239-241.

Anonymous, 1983. Cholesterol - Heart Disease Link - Illuminated. 221: 4615.

Bair, C.W. and Marion, W.W. 1978. Yolk cholesterol in eggs from various avian species. Poultry Sci. 57: 5: 1260-1265.

Bair, C.W., Marion, W.W. and Hotchkiss, D.K. 1980. Relationship of yolk cholesterol and onset of egg production Poultry Sci. 59: 666-668.

Baker, R.C., Darfler, J.M., Rehkugler, G.E. 1980. Poultry Sci. 59: 545-549.

Bartov, I., Bornstein, S. and Budowski, P. 1971. Variability of cholesterol concentration in plasma and egg yolks on hens and evaluation of the effect of some dietary oils. Poultry Sci. 50: 1357-1364.

Becker, W.F. 1968. Manual of procedures in quantitative genetics Washington, State University Press, Washington.

Becker, W.A., Spencer, J.V., Verstrate, J.A. and Mirosh, L.W. 1975. Genetic analysis of yolk cholesterol of chicken eggs. Poultry Sci. 54: 1731.

Becker, W.A., Spencer, J.V., Verstrate, J.A. and Mirosh, L.W. 1977. Genetic analysis of chicken egg yolk cholesterol Poultry Sci. 56: 895-901.

Benenson, B. 1955. Permitted variation for different blood constituents practical clinical Biochemistry Harold Varley (1963) ed. third edn. William Heinmann, Medical Books Ltd., London.

Bitman, J. 1976. Status report on the alteration of fatty acid and sterol composition in lipids, in meat, milk and eggs. pp. 200-237 in Fat content and compositionof animal products. Nat. Acad. Sci. Washington, D.C. cited by Bitman and Wood (1980).

Bitman, J., and Wood, D.L. 1980. Cholesterol and cholesteryl esters of eggs from various avian species. Poultry Sci. 59: 2014-2023.

Bumgardner, H.L. 1955. Studies on the serum cholesterol levels of the chicken. Ph.D. thesis. Univ. of Maryland cited by Hollands et al. (1980).

Campos, E.J. and Ferriera, M.O.O. 1981. Effects of strain dietary energy level and storage time on egg quality and yolk cholesterol Nut. Abst. 52: 8: 4091.

Castelli, W.P., Doyle, J.T., Gordon, T., Hames, C.G., HJortland, M.C., Hulley, S.B., Kagan, A., and Zukel, W.J. 1977. HDL cholesterol and other lipids in coronary Heart Disease. The cooperative lipoprotein phenotyping study. Ciruclation 55; 767-772. cited by Wu and Donaldson (1982).

Chand, D. 1979. A note on the effect of fertilisation on egg yolk cholesterol content in White Leghorns. Indian Poultry Gaz. 63: 4: 169-170.

Chand, D. 1980. A note on yolk cholesterol content in various avian species. Indian. Poultry Gaz: 64: 3: 97-100.

Chand, D., Arora, K.L. and Arneja, D.V. 1972. Egg size and yolk cholesterol content in White Leghorn and four indigenous breeds of Indian Fowl. Indian. J. Poult. Sci. 7: 13-16.

Chand, D., Georgia, G.C. and Razdan, M.N. 1978. Variation in gross components, yolk lipid and yolk cholesterol content of eggs during pullet year production in Australorp. Ind. J. Poult. Sci. XIII. 73-77.

Chand, D. and Sapra, K.L. 1973. Egg yolk lipid and cholesterol concentration in Australorp, White Cornish, New Hampshire and their reciprocal crosses. Indian. J. Poult. Sci. 8: 124-130.

Chavous, L.G., McClung, H.R. and Gardiner, E.E. 1965. Genetic variations among breeding combinations in egg yolk cholesterol Poultry Sci. 44: 184-186.

Cherms, F.L.Jr., Wilcox, F.H. and Shaffner, C.S. 1960. Genetic studies of serum cholesterol level in the chicken. Poultry Sci. 39: 889-892.

Chung, R.A., Rogler, J.C. and Stadelman, J. 1965. The effect of dietary cholesterol and different dietary fats on cholesterol content and lipid composition of egg yolk and various body tissues. Poultry Sci. 44: 221-229.

Clarengburg, R., Kimchung, I.A. and Wakefield, I.M. 1971. Reducing the egg cholesterol level by including emulsified sito sterol in standard chicken diet. J. Nutrition. 101: 289-297.

Clarkson, T.B. 1971. Animal models for atherosclerosis. North Carolina Med. J. 32: 88-98. cited by Wu and Donaldson (1982).

Clarkson, T.B. and Lofland, H.B. 1970. Genetic control of plasma cholesterol level in squirrel monkeys. circulation: 43: 4-8.

Collins, W.M., Terril, A.E., Zervas, N.P. and Constantine, R.F. 1966. A study of the effect of sex-linked carrying and rate upon egg yolk cholesterol Poultry Sci. 45: 1077.

Combs, G.F. and Helbacka, N.V. 1960. Studies with laying hens. Effect of dietary fat protein levels and other variables in practical rations. Poultry Sci. 39: 271-279.

Connor, W.E. 1968. Dietary sterols, their relationship in atherosclerosis. J.Amer.Diet.Ass. 52: 202-208.

Connor, W.E., Johnston, R., and Lin, D.S. 1969. Metabolism of cholesterol in the tissues and blood of the chick embryo. J.Lipid Res.10: 388-394.

Connor, W.E., Stone, D.B. and Armstrong, M.L. 1969. Cholesterol balance and faecal neutral sterols and bile acid excretion in normal manfed dietary fats of different fatty acid composition. J.Clin. Invest. 48: 1363-1375.

Cook, R.P. 1958. Cholesterol. Academic Press Inc., New York.

Cook, W.H. 1968. A study of the hen's eggs pp 109-132. in egg quality. T.C.Carter, ed. oliver and Boyd, Edinburgh.

Cunningham, F.E. 1977. Composition of Araucana eggs. Poultry Sci. 56: 463-467.

Cunningham, D.L., Kruegar, W.F. and Fanguy. 1973. Poultry Sci. 52: 4: 2017.

Cunningham, D.L., Krueger, W.F., Fanguy, R.C. and Bradley, J.W. 1974. Preliminary results of bidirectional selection level in laying hens. Poultry Sci. 53: 384-391.

Daghir, N.J., Marion, W.W. and Balloun, S.L. 1960. Influence of dietary fat and choline on serum and egg yolk cholesterol in the laying chicken. Poultry Sci. 39: 1459-1466.

Daghir, N.J. and Porooshani, J.M. 1968. Effect of pyridoxine deficiency on lipid metabolism in the chick. Poultry Sci. 47: 1094-1097.

Dam, R., Labate, M.E., Tam, S.W. and Cuervotorres, C. 1979. Effects of diazocholesterol and B - sitosterol on egg cholesterol deposition in coturnix quail. Poultry Sci. 58: 4: 985-987.

Danielson, H. and Tchen, T.T. 1969. Steroid metabolism in metabolic pathways. Vol.II.pp. 117-168. D.M. Greenberg, ed. 3rd ed. Academic Press, New York.

Day, C.E., Barker, B. and Stafford, W.W. 1974. 1974. Composition of very low density lipoproteins from cholesterol fed animals. Comp.Biochem. Physiol. 49 B 501-505.

Donaldson, W.E. 1982. Atherosclerosis in cholesterol fed Japanese quail; Evidence for amelioration by dietary Vit.'E'. Poultry Sci. 61: 2097-2102.

Edwards, H.M., Jr., Diggers, J.C., Dean, R. and Carmon, J.I. 1960. Studies on the cholesterol content of eggs from various breeds and/or strains of chickens. Poultry Sci. 39: 487-489.

Edwards, H.M.Jr. and Jones, V. 1964. Effect of dietary cholesterol on serum and egg cholesterol levels over a period of time.

El. Maguid, F.S. ABD. and Quisenberry, J.H. 1968. Influence of egg consumption on performance, blood cholesterol levels and livability. Poultry Sci. 47: 1668.

Estep, G.D., Fanguy, R.C. and Ferguson, T.M. 1969. The effect of age and heredity upon serum cholesterol levels in chickens. Poultry Sci. 48: 1908-1911.

Estep, G.D., Fanguy, R.C. and Quisenberry. 1968. Factors affecting serum cholesterol levels in chickens. Poultry Sci. 47: 1667.

Fisher, H. and Leveille. 1957. Observations on the cholesterol linoleic and linolenic acid content of eggs influenced by dietary fats. J.Nutr. 63: 119-129.

Folch, J., Lees, M. and Sloane Stanley, G.H. 1956. A simple method for the isolation and purification of total lipids from animal tissues. J.Biol.Chem. 226: 497-509.

Forsythe, R.H. 1963. Chemical and physical properties of eggs and egg products. Cereal Sci. To day: 8: 309-312 & 328. cited by Chand et al. (1978).

Gaujoux, E., Krijanowsky, A. 1932. Teneur Comaree en cholesterol du jaune d' eouf des oiseaux de basse - cour. Soc. Biol. 110: 1083-1084. cited by Bair and Marion, (1978).

Gissel, C., Lindfeld, A., Atthelmig, K.H. 1976. Investigations on egg yolk cholesterol content in 10 breeds. Archir fur Geflii gelkundi. 40: 177-181. cited in Poultry Abstr: 3:8.

Gitte, D.S., Khan, A.G. 1981. Genetic and environmental factors affecting serum and egg yolk cholesterol in chicken. Indian Poultry review. XII: 11: 23-29.

Glueck, C.J. 1976. Alpha - lipoprotein cholesterol, beta-lipoprotein cholesterol and longevity. Artery. 2: 196-198.

Godfrey, J.C., Luttinger, R., Taylor, H.D., San Hueza, G.M. 1976. Dietary plant sterol induced reduction of egg yolk cholesterol in the chicken. Nutr. Rep. int. 13: 263-271.

Goldsmith, G.A., Hamilton, J.G., and Miller, O.N. 1960. Lowering of serum lipid concentrations. Mechanism used by unsaturated fats, nicotinic acid and neomycin excretion of sterols and bile acids. Arch. Int. Med. 105: 512-517.

Gordon, T., Castelli, W.P., HJortland, M.C., Kannel, W.B. and Dawber, T.R. 1977. Diabets, blood lipids and the role of obesity in coronary heart disease for women. Ann. Intern. Med. 87: 393-397. cited by Wu and Donaldson (1982).

Grundy, S.M. and Ahrens, E.H. 1966. An evaluation of the relative methods for measuring the balance of stcrols in man. Isotopic balance versus chromatographic analysis. J.Clin.Invest.45: 1503-1515. cited by SIM et al. (1980).

Halloway, P.W., Peluffo, R., and Wakil, S.J. 1962. Biochem: Biophys: Res: Commun: 6: 270.

Harris Jones, J.N., Jones, E.G. and Wells, P.G. 1957. Xanthamatosis and essential hypercholesterolemia. The Lancet, 272: 855-857.

Harris, P.C., Wilcox, F.H. 1963. Studies on egg yolk cholesterol 1. Genetic variation and some phenotypic correlations in a randombred population, Poultry Sci. 42: 178-182.

Hardy, L.B., Auger, H.V. and Wilcox, F.H. 1962. Genetic difference in serum cholesterol in chickens. Am. J.Physiol. 202: 997-1001.

Havel, R.J. and Carison, C. 1962. Lipid pharmacology pp.357. R. Paoletti (1964) Academic Press.

Hickman, M. 1974. The amazing Araucanas. org. Garden. Farm. 21: 58-61. cited by Bitman and wood (1980).

Hilditch, T.P. and Williams, P.N. 1964. The chemical constitution of neutral fats. 4th ed. John Wiley and Sons, Inc. New York, NY.

Hollands, K.G., Grunder, A.A., and Williams, C.J. 1980. Response to five generations of selection for blood cholesterol levels in White Leghorns. Poultry Sci. 59: 1316-1323.

Hood, R.L., McC.Bailey, W. and Svoronos, D. 1978. The effect of dietary monoterpenes on the cholesterol level of eggs. Poultry Sci. 57: 1: 304-306.

Hulley, S., Cohen, R. and Widdowson, G. 1977. Plasma high density lipoprotein cholesterol level. Influence of risk factor intervention. JAMA: 238: 2269.

Jaffe, W.P. 1964. The relationship between egg weight and yolk weight. Brit. Poultry Sci. 5: 295-298.

Johnson, (Jr.) D., Mehring (Jr.)A.L., and Titus, H.W. 1959. Variability of the blood plasma cholesterol of laying chickens. Poultry Sci. 38: 1109-1113.

Jones, R.J. and Dobrilovic. 1969. Aortic cholesterol and the plasma lipoproteins of the cholesterol fed cockrel. Proc. Soc. Exp. Biol. Med.130: 163-167.

Kaminski, J.E., Labe-Drohobycka and Skrzynska, J. 1979. Effect of age on lipoproteins and cholesterol in blood serum of White Rock hens. Nut.Abst. 50: 6: 2659.

Kaminski, J.E., Labe-Drohobycka and Skrzynska, J. 1979. Relationship between total cholesterol in blood serum and cholesterol deposition in certain tissues of broiler chickens Nut. Abst. 50: 6: 2737.

Kannel, W.B., Castelli, W.P. and Gordon, T. 1979. Cholesterol in the prediction of atherosclerotic disease. New perpectives based on the Framingham study. Ann. Intern. Med. 9: 85-91.

Katz, L.N. and Stamler, J. 1953. Experimental atherosclerosis, charles C.Thomas Springfield, IL. cited by Sim et al. (1980).

Keys, A., Anderson, J.T., Adelson, O. and Fidanza, F. 1956. Diet and serum cholesterol in man. Lack of effect of dietary cholesterol. J.Nutr. 59: 39-56.

King, S.C. and Henderson, C.R. 1954. Heritability studies of egg production in the domestic fowl. Poultry Sci. 33: 155.

Kohn, H.I. 1950. Changes in plasma of the rat during fasting and influence of genetic factors upon sugar and cholesterol levels Amer. J.Physiol. 163: 410-417.

Kruski, A.W. and Narayan, K.A. 1972. The effect of dietary supplementation of cholesterol and its subsequent withdrawls on the liver lipids and serum lipoproteins of chickens. Lipids 7: 742-749.

Kudchodkar, B.J., Horlick, L. and O'Neil, J.B. 1976. Absorption of dietary B-sitosterol in laying hens and its incorporation into the egg. J.Nutr.106: 1629-1636.

Kurnick, A.A., Sutton, J.B., Pasvogel, M.V. and Kemmerer, A.R. 1958. Effect of betaine, choline and methionine on the concentration of serum, tissue and egg yolk cholesterol. Poultry Sci. 37: 1218.

Laird, W.P. 1975. Childhood and diet as related to atherosclerosis Clin.Pediatr. 14: 485.

Lall, S.P. and Slinger, S.J. 1973. Nutritional evaluation of rapeseed oils and rapeseed soap stocks for laying hens. Poultry Sci. 52: 1729-1740.

Lepore, P.D. and Marks, H.L. 1965. Genetic variation of some chemical components of Coturnix quail egg yolk. Poultry Sci. 44: 184-186.

Leren, P. 1975. Prevention of coronary Heart Disease by diet. Postgrad.Med.J. 51: 44.

Leveille, G.A. and Fisher, H. 1959. Observation on lipid utilisation in hens fed vegetable and animal fat supplemented diets. Poultry Sci. 37: 658-664.

Linsey, S., Nicholas, C.W. and Chaikoff, I.L. 1955. Aortic lesion induced in the bird by diethyl-stilbestrol injection and cholesterol feeding. Arch. Pathol. 59: 173-184.

Lorenz, F.W., Enteman, C. and Chaikoff, I.L. 1938. The influence of age, sex and ovarian activity on the blood lipids of the domestic fowl. J.Biol. Chem. 122: 619-633.

Mahley, R.W. 1978. Alterations in plasma lipoproteins induced by cholesterol feeding in animals including man p. 181-197 in Disturbances in lipid and lipoprotein metabolism. J.M. Dietachy, A.M. Gotto.Jr. and J.A.Ontko ed. Am.Physiol. Soc. Bethesda, M.D. cited by Wu and Donaldson (1982).

Mareh, B.E. and Bailey, J.M. 1959. Dietary modification of serum cholesterol in the chick. J.Nutr. 69:105-110.

Marion, W.W., Daghir, N.J., Balloún, S.L. and Forsythe, R.H. 1960. Egg yolk and serum cholesterol values as influenced by dietary fatty acids. Poultry Sci. 39: 1271-1272.

Marks, H.L. and Siegel, H.S. 1980. Divergent selection in Japanese quail for the plasma cholesterol response to ACTH. Poultry Sci. 59: 1700-1705.

Marks, H.L. and Washburn, K.W. 1977. Divergent selection for yolk cholesterol in laying hens. Brit. Poultry Sci. 18: 179-188.

Martin, W.G., Tattrie, N.H. and Cook, W.H. 1963. Lipid extraction and distribution studies of egg yolk lipoproteins Can. J. Biochem. Physiol. 41: 657-666.

Mayer, G.A., Connel, W.F., Dewolfe, M.S., Beveridge, J.M.R. 1954. Diet and plasma cholesterol levels. Amer. J. Clin. Nutr. 2: 316-322.

Mc.Gill, Jr., H.C. 1979. Appraisal of cholesterol as a causative factor in atherogenesis. Am. J. Clin. Nutr. 32: 2632-2636.

Messinger, W.J., Porosowska and Steele, J.M. 1950. Effect of feeding egg yolk and cholestrol on cholesterol on cholesterol level. Arch. Int. Med. 86: 189-194. cited by Sim et al. (1980).

Miller, E.C. and Denton, C.A. 1962. Serum and egg yolk cholesterol of hens fed dried egg yolk. Poultry Sci. 41: 335-337.

Mol, M.A.E., DE.Smet, R.C., Terpstra, A.H.M., West, C.E. 1983. Effect of dietary protein and cholesterol on cholesterol concentration and lipoprotein pattern in the serum of chickens. Poultry Abstr. 9: 3: 610.

Moore, R.B., Anderson, J.T., Taylor, H.L., Keys, A. and Frantz, (Jr.) I.D. 1968. Effect of dietary fat on the fecal excretion of cholesterol and its degradation products in man J.Clin. invest.47: 1517-1534.

Moudgal, R.P. 1978. The influence of housing system and dietary protein level on carcass cholesterol content of White Pekin Ducks. Indian Poultry Gaz. 62.2:61-63.

Nakai, T., Tamai, T., Yamada, S., Kobayashi, T., Hayashi, T., Kutsumi, Y. and Oida, K. 1981. Plasma lipids and lipoproteins of Japanese blood and umbolical cord of blood. Artery. 9: 2: 132-150.

Nakaue, H.S., Lowry, R.R., Cheeke, P.R., and Arscott, G.H. 1981. The effect of dietary alfalfa of varying saponin content on yolk cholesterol level and layer performance Nut. Abst. 52:1: 384.

Narayan, K.A. and Calhoun, W.K. 1975. The influence of some dietary factors and/or tread mill exercise on rat and chicken tissue lipids and serum lipoproteins pp. 383-401 in Atherosclerosis Discovery C.E.Day.ed. plenumpress New York. NY.

Nicholas, E.L., Marion, W.W., Balloun, S.L. 1963. Effect of egg yolk size on yolk cholesterol concentration. Proc. Soc. Exptl. Biol. Med. 112: 378-380. cited by Chand et al. (1978).

Palafox, A.L. 1968. Effect of age, energy source and concentration on yolk lipids and cholesterol. Poultry Sci. 47: 1705.

Panda, N.C., Sahu, B.K. and Dehuri, P.K. 1983. Effect of tannic acid with dietary fat oroil on growth, plasma cholesterol and other blood parameters of chicks. Poultry Abstr. 9:5: 992.

Pasternak, C.A. 1979. An introduction to human biochemistry, Oxford University Press. New York. Toronto.

Peterson, D.W., Lilyblade, A., Clifford, C.K., Ernest, R., Clifford, A.J. and Dunn, P. 1978. Composition of cholesterol content of Araucana eggs and commercial White eggs. J.Amer.Diet.Ass. 72: 45-47.

Pillai, S.K. and Sinha, N.C. 1968. Statistical methods for biological workers Ramprasad and sons. Agra-3.

Piper, J. and Orrild, L. 1956. Essential famitial hypercholesterolemia and xanthamatosis Am.J.Med.21: 34-46.

Porter, M.W., Yamanaka, W., Carlson, S.D. and Flynn, M.A. 1977. Effect of dietary egg on serum cholesterol and triglyceride of human males. Amer.J.Clin.Nutr.30:490-495.

Portman, O.W. and Stare, F.J. 1959. Dietary regulation of serum cholesterol levels. Physio.Revs. 39: 407.

Ramakrishnan, S., Prasannan, K.G., Rajan, R. 1980. Text of Medical biochemistry orient Longman, Madras-2.

Rangachar, T.R.S., Setty, S.V.S. and Hegde, V.R. 1970. Cholesterol content in eggs of chicken and duck, Mysore, J.Agri.Sci.4: 146-151.

Rao, M.R., Pal, A.K. and Razdan, M.N. 1964. Studies on some of the lipid constituents in the blood of chicken under different physiological conditions.Part II. lipid concentration in pre-laying just started laying, at the height of laying, moulting and non-laying Rhode Island Red Chicken. Indian. Vet.J.41: 260-263.

Robertson, A. 1959. The sampling variance of the genetic correlation coefficient. Biometrics. 15: 469-485.

Rotenberg, S. and Christensen, K. 1976. Spectrophotometric determination of total and free cholesterol in egg yolk and Animal tissues. Acta. Agr. Scandanavia 26: 94-98. cited by Bitman and Wood (1980).

Schaefer, L.E., Drachman, S.R., Steinberg, A.G. and Aldersberg, D. 1953. Genetic studies on hyper-cholesterolemia, frequency in a hospital population and in families of hyper-cholesteremic index patients. Am.Heart. J.46: 99-116.

Scheinberg, S.L., Ward, H. and Nordskog, A.W. 1953. Breeding for egg quality 1. Heritability and repeatability of egg weight and its components Poultry Sci. 32: 504-510.

Searcy, R.L. 1969. Cholesterol and atherosclerosis. Diagnostic Biochemistry. 174-175.

Seeley, R.M., Crinear, P.E. and Watt, B.K. 1972. Cholesterol content of foods J.Am. Diet Assoc.61:134-149.

Sharma, R.K., Singh, R.A., Pal, R.N. and Aggarval, C.K. 1979. Cholesterol content of chicken egg asaffected by feeding garlic, sarpagandha and nicotinic acid. Nut.Abst.50: 12: 6520.

Shih, J.C.H. 1979. Atherogenicity of USP and purified cholesterol, Poultry Sci. 58: 4: 1107.

Shore, V.G., Shore, B. and Hart, R.G. 1974. Changes in apolipoproteins and properties of rabbit very low density lipoproteins on induction of cholesteremia. Biochemistry. 13: 1579-1585.

Sim, J.S., Kitts, W.D. and Bragg, D.B. 1980. Metabolic fate of 14C - OVO cholesterol in the egg egg yolk fed rat. Poultry Sci. 59: 7: 1662.

Sim, J.S., Kitts, W.D. and Bragg, D.B. 1980. Effect of dietary egg yolk on serum cholesterol levels of White Leghorn cockrels. Poultry Sci. 59: 1812-1817.

Slater, G., Mead, J., Dhopeshworkar, G., Robinson, S. and Alfin-Slater, R.B. 1976. Plasma cholesterol and tryglycerides in men with added eggs in the diet Nutr. Rep. Int. 142: 249-260.cited by Sim *et al.*(1980).

Smith, Jr., D.N. 1969. The effect of high levels of fat, choline, Vit B.12 and Vit.E on the occurence of fatty livers in caged layers. Ph.D. dissertation: Texas A & M University. cited by Gitte, D.S. and Khan, A.G. (1981).

Somes, Jr., R.G., Francis, P.V. and Tlustohowicz, J.J. 1977. Protein and cholesterol content of Araucana chicken eggs. Poultry Sci 56: 1636-1640.

Sutton, C.D., Muir, W.M., Begin, J.J. and Johnson, T.H. 1980. Effect on fibre on cholesterol metabolism in the coturnix quail Poultry Sci. 59: 7: 1665.

Svacha, R.L. 1959. Effect of cholesterol type of fat, energy level and lipotropic agents in the diet of the domestic fowl. Ph.D. Dissertation Texas A & M University. cited by Gitte and Khan (1981).

Swierczewska, E., Szymkiewicz, M., Rzeszewska, Z. and Murkocinska, N. 1981. Relation between cholesterol in blood serum of hens and chickens of meat and general purpose types. Nut. Abst. 52: 5: 2175.

Tandon, R. 1983. Medical times XIII: 11: 4-5.

Tomita, Y., Yamada, A., Hatashi, K., and Honbo, T. 1975. Cholesterol content and fatty acid composition in egg yolks and blood plasma of poultry. Bull. Fac. Agri.Kagashima Univ. 25: 89-93. cited by Bitman & Wood (1980).

Turk, D.E., and Barnett, B.D. 1971. Cholesterol content of market eggs. Poultry Sci. 50: 1303-1306.

Vesselinovitch, D. 1979. Animal models of atherosclerosis their contributions and pitfals. Artery 5:3: 193-206.

Washburn, K.W., and Nix, D.F. 1974. A rapid technique for extraction of yolk cholesterol. Poultry Sci. 53: 1108-1122.

Washburn, K.W. and Nix, D.F. 1974a. Genetic basis of yolk cholesterol content, Poultry Sci. 53: 109-115.

Washburn, K.W. and Marks, H.L. 1977. Relationship of yolk and plasma cholesterol levels to position of eggs in clutch. Poultry Sci. 56: 1676-1678.

Weibust, R.S. 1969. Two way selection for plasma cholesterol levels in mice. Genetics (suppl.No.2/ part 2) 61: 562. cited by Hollands et al. (1980).

Weiss, H.S. and Fisher, H. 1957. Plasma lipid and organ changes associated with the feeding of animal fat to laying chickens. J.Nutr. 61: 267-280.

Well, V.M. and Bronte-Stewart, B. 1963. Egg yolk and serum cholesterol levels. Importance of dietary cholesterol intake. Brit. Med.J. 1: 577-581.

Whyte, H.M. and Haventein, N. 1976. A perspective view of dieting to lower the blood cholesterol. Am.J. Clin.Nutr. 29: 784.

Wilcox, F.H., Cherms, Jr., I.D., Vanvleck, I.D., Harvey, W.R. and Shaffner, C.S. 1963. Estimates of genetic parameters of serum cholesterol level, Poultry Sci. 42: 37-42.

Wilkinson, C.F., Hand, E.A. and Fliegelman, M.T. 1948. Essential familial hypercholesterolemia. Ann.Int. Med. 29: 671-686. cited by Wilcox et al. (1963).

Wilson, J.D. 1964. The quantification of cholesterol excretion and degeneration in the isotopic study in the rat; the influence of dietary cholesterol J.lipid Res. 5: 409-417.

Wood, J.D., Biely, J. and Topliff, J.E. 1961. The effect of diet, age and sex on cholesterol metabolism in White Leghorn chickens canadian. J.Biochem. Physiol. 39: 1705-1715.

Wood, P.D., Shioda, R. and Kinsell, L.W. 1966. Dietary regulation of cholesterol metabolism Lancet 2:604-607.

Wu, T.C. and Donaldson, W.E. 1982. Effect of cholesterol feeding on serum lipoproteins and atherosclerosis in atherosclerosis-susceptible and atherosclerosis-resistant Japanese quail. Poultry Sci.61: 2407-2414.

Yao, K.T.S., Skinner, J.I. 1959. Heritability and genetic correlations of albumen weight and yolk size in chicken eggs. Poultry Sci. 38: 1262.

Zlatkis, A., Zak, B., and Boyle, A.J. 1953. A new method for the direct determination of serum cholesterol J. lab. clin. Med. 41: 486-492.

Zukel, M.C. 1969. Revising booklets on fat - controlled meals. J. Am. Diet. Assoc. 54: 20.

APPENDIX

APPENDIX - I

Composition of chick mash:

Ingredients	Parts per 100
Yellow maize	50
Groundnut oil cake	28
Gingelly oil cake	2
Wheatbran	10
Fish meal	10

Eggomine 200 g per 100 kg of feed

Amprolsol 50 g per 100 kg of feed

Neftin 100 g per 100 kg of feed

Rovimix 20 g per 100 kg of feed

Composition of grower mash:

Ingredients	Parts per 100
Yellow maize	60
Groundnut oil cake	15
Gingelly oil cake	5
Wheatbran	10
Fish meal	10

Eggomine 200 g per 100 kg of feed

Neftin 100 g per 100 kg of feed

Rovimix 20 g per 100 kg of feed

Composition of layer mash:

Ingredients	Parts per 100
Yellow maize	65
Groundnut oil cake	10
Gingelly oil cake	5
Wheat bran	10
Fish meal	10

Eggomine 200 g per 100 kg feed

Neftin 100 g per 100 kg feed

*Rovimix 20 g per 100 kg feed

* Rovimix brand mixture contains stable vitamins, A + B_2 + D_3 having a guaranteed potency of 40,000 I.u. vitamin A, 25 mg of vit. B_2 and 5,000 I.u. of vitamin D_3 per gram.

APPENDIX II (Continued)

ANALYSIS OF COVARIANCE FOR THE TRAITS UNDER STUDY AT 32 WEEKS OF AGE

Source of variation	df	Mean sum of products: Mg.cholesterol/g.yolk and Hatch weight	Body weight (20 weeks)	Age at maturity	Weight at maturity	Body weight (32 weeks)	Egg weight
Between sires	27	4.3530	4481.8000	-5.0093	91.4070	104.7800	2.4884
Within sires	668	-0.5484	133.2000	1.2644	-2.0400	-2.7635	1.1778

		Mean sum of products: Mg.cholesterol/g.yolk and Yolk weight	Total yolk cholesterol	Plasma cholesterol	Shape index
Between sires	27	0.0486	118.9000	89.8330	8.9514
Within sires	668	0.5082	81.2610	50.0020	1.4643

APPENDIX II (Continued)

ANALYSIS OF COVARIANCE FOR THE TRAITS UNDER STUDY AT 40 WEEKS OF AGE

Source of variation	df	Mean sum of products: Mg.cholesterol/g.yolk and Hatch weight	Body weight (20 weeks)	Age at maturity	Weight at maturity	Body weight (40 weeks)	Egg weight
Between sires	27	93.0370	-47.5930	29.1110	4721.8000	13799.0000	226.0000
Within sires	668	-1.3084	- 6.2006	6.8593	503.5200	2149.7000	90.8980

		Mean sum of products: Mg.cholesterol/g.yolk and Yolk weight	Total yolk cholesterol	Plasma cholesterol	Shape index
Between sires	27	158.7100	1527.6000	10.8800	30.7550
Within sires	668	113.3300	1154.1000	6.8851	38.2410

APPENDIX II (Continued)

ANALYSIS OF COVARIANCE FOR THE TRAITS UNDER STUDY AT 72 WEEKS OF AGE

Source of variation	df	Mean sum of products				
		Mg.cholesterol/g.yolk and				
		Egg weight	Yolk weight	Total Cholesterol	Plasma Cholesterol	Shape index
Between sires	27	0.0573	0.0961	167.3300	84.9210	1.9977
Within sires	135	0.4799	0.2603	131.2500	82.5760	0.2459

APPENDIX II (Continued)

ANALYSIS OF COVARIANCE FOR THE TRAITS UNDER STUDY AT 32 WEEKS OF AGE

Source of variation	df	Mean sum of products: Total yolk cholesterol and Hatch weight	Body weight (20 weeks)	Age at maturity	Weight at maturity	Body weight (32 weeks)
Between sires	27	76.4810	-219.2600	-0.0109	1230.2000	1237.9000
Within sires	668	-7.110	-291.2800	20.1620	-296.1200	-152.7700

Source of variation	df	Mean sum of products: Total yolk cholesterol and Egg weight	Yolk weight	Plasma cholesterol	Shape index
Between sires	27	50.6480	11.6060	1100.1000	122.0400
Within sires	668	42.0400	18.6590	727.5600	18.4750

APPENDIX II (Continued)

ANALYSIS OF COVARIANCE FOR THE TRAITS UNDER STUDY AT 40 WEEKS OF AGE

Source of variation	df	Mean sum of products: Total yolk cholesterol and Hatch weight	Body weight (20 weeks)	Age at maturity	Weight at maturity	Body weight (40 weeks)
Between sires	27	-15.0560	-0.0466	3.5556	499.5600	1441.2000
Within sires	668	- 0.3600	147.9800	1.1108	350.8300	1795.5000

Source of variation	df	Mean sum of products: Total yolk cholesterol and Egg weight	Yolk weight	Plasma Cholesterol	Shape index
Between sires	27	64.7220	103.6900	9.5741	31.9140
Within sires	668	65.0520	80.2940	6.2253	25.3200

APPENDIX II (Continued)

ANALYSIS OF COVARIANCE FOR THE TRAITS UNDER STUDY AT 72 WEEKS OF AGE

Source of variation	df	Mean sum of products			
		Egg weight	Yolk weight	Total yolk cholesterol and Plasma cholesterol	Shape index
Between sires	27	134.6800	55.6920	2568.3000	57.2130
Within sires	135	150.6100	54.2740	2069.1000	-0.5336

APPENDIX II (Continued)

ANALYSIS OF COVARIANCE FOR THE TRAITS UNDER STUDY AT 32 WEEKS OF AGE

Source of variation	df	Mean sum of products: Plasma cholesterol and Hatch weight	Body weight (20 weeks)	Age at maturity	Weight at maturity	Body weight (32 weeks)
Between sires	27	39.8610	2.0556	-58.5930	1764.7000	1643.9000
Within sires	668	-4.9173	0.4020	10.8260	54.3710	-66.2510

Source of variation	df	Mean sum of products: Plasma cholesterol and Egg weight	Yolk weight	Shape index
Between sires	27	15.4810	1.9722	93.4630
Within sires	668	12.2080	5.4208	16.5970

APPENDIX II (Continued)

ANALYSIS OF COVARIANCE FOR THE TRAITS UNDER STUDY AT 40 WEEKS OF AGE

Source of variation	df	Mean sum of products: Plasma cholesterol and Hatch weight	Body weight (20 weeks)	Age at maturity	Weight at maturity	Body weight (40 weeks)
Between sires	27	-63.3680	-88.2960	1.2731	-4.3426	20.6570
Within sires	668	- 0.0161	-179.5700	-0.0024	1.1228	32.9280

Source of variation	df	Mean sum of products: Plasma cholesterol and Egg weight	Yolk weight	Shape index
Between sires	27	1.4149	0.5690	0.1426
Within sires	668	1.3864	0.4534	0.1607

APPENDIX II (Continued)

ANALYSIS OF COVARIANCE FOR THE TRAITS UNDER STUDY AT 72 WEEKS OF AGE

Source of variation	df	Mean sum of products		
		Plasma cholesterol and		
		Egg weight	Yolk weight	Shape index
Between sires	27	76.5650	32.8870	29.1300
Within sires	135	78.4180	28.5670	-2.8041