Proceedings

ROCKINGHAM POULTRY SERVICEMAN'S ORGANIZATION

POULTRY HEALTH SEMINAR

Roanoke — Salem, Virginia
Sheraton Motor Inn

September 12-13, 1973

Sponsored by

Cooperative Extension Service
Virginia Polytechnic Institute
and State University

in cooperation with the

Rockingham Poultry
Serviceman's Organization

and

Virginia Department
of
Agriculture and Commerce

Issued in furtherance of Cooperative Extension work, Acts of May 8 and June 30, 1914, in cooperation with the U. S. Department of Agriculture. W. E. Skelton, Dean, Extension Division, Cooperative Extension Service, Virginia Polytechnic Institute and State University, Blacksburg, Virginia 24061.

PROGRAM

Sheraton Motor Inn

WEDNESDAY, September 12, 1973

7:30 - 8:15 Registration

Morning Session - Chairman, Jonn Martin

8:25 Welcome - Richard Eaton

8:30 What Management Expects from The Serviceman - Chip Strickler

9:15 Virginia Poultry Laboratory Services - Fred Rea

10:00 Break

10:30 Broiler Breeder Flock Management - Bill Little

11:15 Turkey Breeder Flock Management - Sass Saffores

12:00 Lunch

Afternoon Session - Chairman, Suresh Singh

1:30 Hatchery Management - Milt Hendrixson

2:15 Poultry Nutrition - Hal Yacowitz

3:00 Break

3:30 Poultry Mycoplasmas - Rudy Killingsworth

4:15 E. Coli Infections - Burnie Gross

5:00 Break

Evening Program - Chairman, Richard Eaton

6:00	Speaker: Professor George W. Litton, Emeritus Professof of Animal Science, V.P.I. & S.U.

THURSDAY, September 13, 1973

Morning Session - Chairman, Dick Boyd

9:00	Gangrenous Dermatitis - Stan Moum
9:45	Viral Diseases of Chickens and Turkeys - Rob DuBose
10:30	Break
11:00	Molds and Their Toxins - Ray Harris
11:45	Panel Discussion - Chairman, Merlin Fahrney - All Speakers A time for discussion of topics that were not completed during the program
12:30	Adjourn

This program has been planned by the Rockingham Poultry Serviceman's Organization.

Richard Eaton	President
Merlin Fahrney	Vice President
Bruce Grover	Secretary - Treasurer

PROGRAM PARTICIPANTS

Dr. Dick Boyd	Rocco Turkeys Inc., Harrisonburg, Va.
Dr. R. T. DuBose	V.P.I. & S.U., Blacksburg, Va.
Mr. Richard Eaton	Long Foods, New Market, Va.
Mr. Merlin Fahrney	Rockingham Poultry Marketing Cooperative, Broadway, Va.
Dr. W. B. Gross	V.P.I. & S.U., Blacksburg, Va.
Dr. J. R. Harris	University of North Carolina, Raleigh, North Carolina
Mr. Milton Hendrixson	Goldsboro Milling Company, Goldsboro, North Carolina
Dr. R. D. Killingsworth	Eli Lilly and Company, Apex, North Carolina
Mr. Bill Little	Arbor Acres Farms, Concord, North Carolina
Professor George W. Litton	Emeritus Professor of Animal Science, V.P.I. & S.U., Blacksburg, Va.
Dr. Stanley Moum	Merck and Company, Salisbury, Md.
Dr. Fred Rea	Virginia Department of Agriculture and Commerce, Richmond, Va.
Mr. O. P. Saffores	The Amburgo Company, Philadelphia, Pa.
Dr. Suresh Singh	Rockingham Poultry Marketing Cooperative, Broadway, Va.
Mr. Charles O. Strickler	Rocco Feeds, Harrisonburg, Va.
Dr. Hal Yacowitz	The Amburgo Company, Philadelphia, Pa.

CONTENTS

PAGE	TITLE	AUTHOR
1	Virginia Poultry Laboratory Services	Fred. W. Rea
8	Broiler Breeder Flock Management	Bill Little
20	Turkey Breeder Flock Management	O. P. Saffores
26	Poult Quality and Hatchery Management	Milton Hendrixson
29	Poultry Nutrition	Hal Yacowitz
39	Mycoplasma	R. D. Killingsworth
44	E. Coli Infection	W. B. Gross
46	Gangrenous Dermatitis	Stanley G. Moum
49	Viral Diseases of Chickens and Turkeys	R. T. DuBose
53	Molds and Their Toxins	J. R. Harris
58	Enrollment	

VIRGINIA POULTRY LABORATORY SERVICES

Fred W. Rea
Coordinator of Laboratories

A. HISTORY OF THE LABORATORIES

How it all began.

Prior to 1930, diagnostic services only were available from V.P.I. By 1930, poultry diagnosis was started in a small laboratory used for blood testing. This served the needs of the poultry industry until things really got tight during the depression. The poultry people organized and went to the State Legislature for help. From this request for help, the Legislature put into law the State Code of Virginia requiring the State Board of Agriculture to maintain and operate, at some suitable location in the county of Rockingham, a laboratory for the diagnosis of poultry diseases. The city of Harrisonburg donated the lot and the present laboratory was built and opened in 1942.

Expansion of the diagnostic laboratory system.

The State Code was changed to read, "in the county of Rockingham and in such other places within the State as the Board may determine." A laboratory was set up in Richmond shortly after the opening of the Harrisonburg Laboratory. In 1948 laboratory buildings were built in Ivor, Wytheville, Warrenton and Accomac. In 1960, the Lynchburg Laboratory was opened. The Central Laboratory at Richmond moved into the new consolidated building in 1970. (See Appendix I for locations.)

B. OPERATIONAL SET-UP OF LABORATORIES

All laboratories are in the Department of Agriculture and Commerce, Division of Animal Health and Dairies. The State Veterinarian is the director of this division and the laboratory section is headed by the veterinary laboratory coordinator. Each of the regional laboratories is under the direct supervision of a veterinary bacteriologist supervisor. The technical staff includes a veterinary bacteriologist and technicians. Each laboratory also has laboratory aides, custodial, and secretarial personnel.

The requirements for a veterinary bacteriologist are a four-year degree in science with one year experience in a veterinary laboratory for a veterinary bacteriologist supervisor, a four-year degree in science, and three years of experience. Requirements for the coordinator are a degree in veterinary medicine and five years experience in laboratory disciplines. Technician positions require a two-year college science course and/or equivalent experience in laboratory work.

C. POLICY OF THE LABORATORIES

It may be well to note, at this time, that the laboratories are general laboratories intended to serve all segments of agriculture in the diagnosis of diseases. Not only are they equipped and staffed for poultry, but these facilities are available to the beef cattle, horse, sheep, swine, and dairy industries. Our staff and equipment is geared to multi-species diagnostic tests.

Regarding poultry services, it is the policy of the laboratory section to accept all types of poultry. However, our monthly reports indicate that it is primarily involved with chickens and turkeys. The laboratories

will accept both young and adult birds for laboratory diagnosis of disease problems. Anyone associated with the brood poultry industry may submit birds. This incldes the owner of a backyard flock of only a few birds up to and including management of the larger integrated poultry operations. As many of you know, the majority of our submissions are brought to the laboratories by the poultry servicemen.

Now, let us see what the laboratories need from you, the serviceman. First, we need the birds which most likely represent the problem on the farm as you see it. Second, and this is of utmost importance, we need a complete history on the flock from which you are submitting the birds. I know you have heard this many times and yet there are some who neglect to tell all they know about the flock. You get rushed and seem to forget that the laboratory personnel was not at the farm when the birds were picked up. Then there is the old hang-up that a lot of us have at times - we just hate to write it down. Well, it is just about a must if we are to get the job done which we work at day in and day out - the solving of problems which plague our poultry flocks. A good history will first tell the laboratory who the flock belongs to and where it is located. It will tell the size, the age and the species. We in the laboratory like to know vaccination records and treatments. This helps a great deal in the interpretation of what is seen when examining and culturing the birds. Other things of importance are listed on the regular laboratory history forms which are supplied by each laboratory for your use.

D. DIAGNOSTIC SERVICES

I would like to go over the various services you can expect to receive when submitting poultry to our laboratories.

1. Anti-mortem Examination

The live bird is observed for any abnormalities that would produce disease problems. It is at this time that the veterinary bacteriologist looks over the bird to determine if there are symptoms of central nervous disease, paralysis, external parasites, and may take blood samples for further laboratory tests.

2. Gross Pathology

Necropsy observation is made of all body systems to detect any abnormal findings. Birds that are a few days old are cultured to detect any bacterial infection. If birds show evidence of a virus condition, the needed specimens are collected. Routine microscopic examinations are made on intestinal scrapings to detect the presence of coccidia.

3. Laboratory Tests

a. Culture

All of the regional laboratories are staffed and equipped to do culture work for the presence of bacterial and fungi pathogens. This enables the laboratories to confirm suspected diseases which are caused by common bacterial and mycotic pathogens. Advance bacterial and fungi culture work is also performed in the central laboratory at Richmond. Mycoplasma culture is usually performed in the Richmond Laboratory; however, this can be done in the regional laboratories.

b. Virus Isolation

Specimens collected in the regional laboratories from birds suspected of having virus diseases are sent to the central laboratory. Our Richmond virus laboratory is well equipped

and staffed to carry out the isolation of the viruses normally found in poultry.

c. Histopathological Studies

There are many times when the regional laboratories need further microscopic studies of tissues to confirm a suspected poultry disease. This may involve bacterial, virus, toxin, or nutritional diseases. The proper specimen is fixed in Formalin solution and sent to the Richmond Laboratory for sectioning, staining and study by the veterinary pathologist. Again, this laboratory, like the virus laboratory, is especially well equipped to handle these tissues. It may be noted here that both the histopath and the virus laboratories are set up in the central laboratory so as to serve all the various livestock and poultry industries.

d. Serology

The majority of the blood serology testing for poultry is done in our Harrisonburg Laboratory. The tube agglutination test is performed on chicken and turkey and turkey serum for Mycoplasma, pullorum, and typhimurium. Test for Mycoplasma synoviae is done upon special request. The confirmatory test on positive samples found on the tube test for M. gallisepticum is run in the Richmond Laboratory using the H. I. method. This serology test is also used to detect Newcastle Disease and one of the pneumonia virus diseases of cattle.

e. Antigen for Serology Tests

(1) Pullorum is produced in the Harrisonburg Laboratory, and is evaluated each year by the antigen committee of the Northeastern Conference on Avian Diseases. The Mycoplasma

antigen is produced in our Richmond Laboratory. We buy the antigen used in typhimurium testing and the antigen used for M. synoviae is bought by those in the industry who request the testing. We have been able to obtain antigen for Mycoplasma H. I. testing from the Federal government.

E. REPORTING LABORATORY FINDINGS

It is the policy of our laboratories to make reports on findings as soon as they are noted. Preliminary reports are given at the time the birds are submitted to the laboratory for necropsy. Many cases can be diagnosed following the post-mortem and microscopic check for coccidia. Recommendation for treatment and also control methods are given at this time. One or two days are needed to make a report of bacterial cultural findings. Mycotic culture may take up to one week for reporting. Virus isolations are very time consuming, depending on the cell line needed for growth and the number of transfers it takes before the virus can be identified. For histopathological studies it usually takes two to three days to prepare the slide after the tissue reaches Richmond, and the time elapse before the slide is read depends upon the availability of the pathologist. A final written report giving both the primary diagnosis and any other conditions noted is made on all laboratory cases. Recommendations for treatment are given when indicated.

F. SUMMARY

There are other services which the laboratories give to the poultry industry that have not been listed, and I am sure there will be more added as the years go on. As the title of my talk would indicate, we are a service group, and this is what we in the laboratory section continue to work at -- giving an acceptable service to the industry.

APPENDIX I

LABORATORY DIRECTORY

COORDINATOR OF LABORATORIES - FRED W. REA, D.V.M.
1 North 14th Street
Richmond, Virginia 23219

LABORATORY	ADDRESS	TELEPHONE
Accomac	Accomac, Virginia 23201	787-2585
Harrisonburg	116 Reservoir Street Harrisonburg, Virginia 22801	434-3897
Ivor	Ivor, Virginia 23866	859-2771
Lynchburg	Box 4191 Lynchburg, Virginia 24502 (3/4 mile East on Route 460)	846-8860
Richmond	Consolidated Laboratories Building, Room 162 1 North 14th Street Richmond, Virginia 23219	770-2446
Warrenton	234 West Shirley Avenue Warrenton, Virginia 22186	347-3131
Wytheville	Box 436 Wytheville, Virginia 24382	228-5501

All laboratories are onen a five-day week, Monday through Friday. The hours are from 8:15 A.M. until 5:00 P.M., with the exception of Harrisonburg which is open from 7:45 A.M. until 4:30 P.M.

BROILER BREEDER FLOCK MANAGEMENT

Bill Little

This paper is only a guide and thought provoker and is not to be considered as a specific program to be followed on all flocks. I cannot mark a calendar as to the changes that need to be made, because of environmental differences, feed differences, and breed differences. The Management Program that I will outline will be generalized, with several rules that may help the breeder serviceman make decisions from flock to flock.

1. Before the Chicks Arrive:

Chicks should be brooded as far as possible by older birds. Brooder house security is important. Keep visitors out. A separate caretaker is essential.

Be sure you are ready. Start Clean. Brooder houses and all equipment should be thoroughly washed and disinfected. Litter should be dry and free from mold. Check all equipment carefully to make certain it is in good working condition. Brooders should be operating 24 hours before chicks arrive. Waterers should be filled several hours before chicks arrive. Put feeder lids on chick-size feeders in place, but do not put the feed out until two or three hours after the chicks arrive. We will need to provide one inch of feeding space per bird when chick feeders are used, or one feeder lid per 100 chicks for the first three to five days and two one gallon chick waterers per 100 chicks.

2. Brooding Management:

Brooding Temperature:

Thermometers are useful before the chicks arrive in adjusting the brooding equipment to attain a floor temperature of 90 to 95 degrees F. Afterwards, however, chick comfort should be the criterion for the correct brooding temperature. Watch the behavior of the chicks, particularly at night, to determine if the brooder heat is correct. Comfortable chicks are quiet and uncomplaining. As they grow, lower the temperature about five degrees per week to approximately sixty-five degrees.

Be particularly observant of the chicks after vaccinating, debeaking, and other stresses. A temporary increase in brooding temperature and medication through the drinking water or feed may be required.

After the first two days, gradually expand the brooder guards as the chicks grow and as weather permits. With each expansion, move the waterers and feeders out away from the hover. After seven to ten days, the use of the brooder guards should be discontinued.

GOOD VENTILATION A MUST:

In conventional brooder houses where there are openings and windows, adjust the ventilation according to the outside temperature and existing weather conditions just before chicks arrive. Watch the chicks carefully and begin acclimating them to reduced brooder house temperatures after the first two days. After three to four weeks, chicks should be accustomed to much lower temperatures. The ideal room temperature for brooding is 65 degrees F. On most days, climate and weather permitting, windows or curtains should be kept open during the middle of the day. During early morning and late afternoon, compensation should be made for cooler outside temperatures. Any fan thermostats should be adjusted gradually to maintain the optimum house temperature.

In controlled-environment houses, proper ventilation also must be maintained. Here, too, ideal room temperature is 65 degrees F.

The slight extra cost of maintaining adequate brooder heat in a well-ventilated, draft-free house will be more than compensated for by the development of a healthy and vigorous flock.

CLEAN, FRESH WATER IS ESSENTIAL:

Allow chicks to have water two to three hours prior to giving them feed. Use two one-gallon chick waterers per 100 chicks for the first two weeks. Be sure that the drinking water is warm for the first two days. Wash and sanitize the waterers daily. When changing to automatic waterers, or when making any equipment change, do so gradually. Leave chick waterers in and operating for a few days until you are sure that all birds have found the new ones. Then promptly remove the chick waterers from the pens. A good water sanitizer can be used to help control disease-producing organisms, algae, and fungi in the drinking water.

WATCH THE CHICKS:

Frequent visits to the brooder house, particularly during the first week or ten days, will definitely pay dividends. Be sure the chicks are eating and drinking. If they tend to huddle or congregate in certain spots, check the brooding equipment and check for drafts along the floor.

FEEDING DURING BROODING PERIOD:

Feed small quantities at a time, but often, during the first two to three days. This stimulates chick activity and helps avoid feed spoilage and waste. Feed a well-fortified, properly balanced Chick

Starter mash. It should contain 20-21% protein and 920-960 calories of productive energy per pound, and should be self fed for the first four to six weeks.

TROUGH FEEDERS:

If feeder lids are used for the first three to six days, supplement them with chick size troughs beginning the second day. Continue to use them for three weeks; then, use larger troughs for the remainder of the brooding period. Adjust the feeder height so that the bottom of the feeder is one inch above the backs of the birds (providing excessively deep feeder troughs are not being used.)

AUTOMATIC FEEDERS:

In houses equipped with automatic feeders, the chicks should have access to the feeders as soon as possible. Gradually move the trough feeders toward the automatic feeder. Once the chicks are eating well from the automatic feeder adjusted so that the bottom of the trough is one inch higher than the backs of the birds to help keep the feed clean and to avoid waste.

LITTER

Litter management is most important. Remove caked or moldy material to minimize disease risks and be sure to remove excessively wet or dusty litter.

MEDICATION AND VACCINATION:

Vaccinating procedures vary considerably from one area to another. If there is a question about your vaccination program, check carefully with a qualified pathologist and your vaccine supplier. Before you medicate, be sure you know what you are medicating for. If you have

trouble, check your management procedures. The cause of the problem still may exist and, unless corrected, treatment may be of little or no value.

PRECISION DEBEAKING:

In order to help reduce picking when the birds are older, your chicks should be debeaked. Debeak at six to nine days of age, using the precision debeaking technique. In precision debeaking, about one half of both the upper and lower beak is removed. If the chick is held properly against the cauterizing blade, the lower beak will be slightly longer than the upper. This method of debeaking is permanent if done properly. There is less stress on the birds; it is performed at an ideal time; it helps prevent feed waste; it is easily done with a minimum of labor.

GROWING MANAGEMENT (6 THROUGH 18 WEEKS OF AGE)

REARING:

Unless the flock will be grown in the same pens where brooded, the growing house and equipment should be thoroughly cleaned, disinfected and fumigated before the growing birds arrive. Be sure to clean and fumigate the bulk feed bins. New litter, free of mold and excessive dust, should be used. Avoid moving birds during extremes of weather. Wait until it moderates.

Waterers should be cleaned and sanitized daily. Feeders should be kept clean and free of debris. Maintain a good litter condition. Provide plenty of draft-free ventilation. Keep feeders and waterers adjusted so that the bottom of the trough is one inch higher than the backs of the birds. Equipment should be located so that the birds never have to go more than ten feet for feed or water.

Whenever detected, remove obvious culls and sexing errors. Do not remove otherwise healthy birds because of feather or shank color. Such variations are meaningless, and these birds should perform as well as any in the flock.

Take all necessary precautions to control external and internal parasites. Periodic worming of the flock may be necessary. Use 3/4-inch poultry netting on windows and doors to keep out wild birds and predatory animals.

FEEDING GROWING PULLETS:

Feed intake must be limited to avoid the development of fat, early-maturing pullets. A lean healthy pullet grown according to the recommendation of the Primary Breeder is the goal of a controlled feeding program during the growing period. Body weight, development, and the onset of sexual maturity are closely related to a controlled lighting and feeding program. By increasing or decreasing the feed intake, one can regulate a bird's development and body weight. The amount of feed to be fed during the growing period will be determined by the weight of the pullets. Approximate amounts can be found in all breeder Feeding and Management Booklets, but remember these are only guides.

CONTROLLED FEEDING PROGRAMS:

Three controlled feeding programs are recommended:

1. "Skip Every Other Day"
2. "Limited Every Day
3. "Skip Two Days Per Week"

When managed properly, each program gives excellent results. Each requires that careful attention be given to the birds, weather, equipment,

and housing ventilation. Change from the Starter to the Developer feed at the beginning of the sixth week. At this time, if the flock is in good physical condition, begin to limit the daily nutritional intake.

NOTES ON CONTROLLED GROWING FEED PROGRAMS:

1. The Starter and Developer rations fed for the first 14 weeks (98 days) should contain a level of coccidiostat that will allow the flock to develop an immunity against coccidiosis. If a commercial coccidiosis vaccine is used, follow the manufacturer's directions explicitly.

2. Adequate feeder space is ABSOLUTELY NECESSARY TO ENABLE ALL BIRDS TO EAT AT ONE TIME. A minimum of four inches of feeder space per bird is required.

3. IT IS IMPORTANT THAT FEED BE MADE AVAILABLE TO THE BIRDS BEFORE THE LIGHT DAY BEGINS. FEEDERS SHOULD BE FILLED DURING THE DARK HOURS. AUTOMATIC FEEDERS SHOULD RUN CONTINUOUSLY UNTIL THE DAY'S FEED ALLOTMENT IS COMPLETELY CONSUMED.

4. Each day's feed allowance must be ACCURATELY WEIGHED.

5. Birds grown on either of the Skip-Day feeding programs may be given cracked or small grain to keep them active. Feed grain in the litter (one pound per 100 birds) on "no feed" days only. Divide the grain into three equal feedings. Do not feed on excessively hot days.

6. During periods of stress, disease, and medication, place the birds on full feed. Return to controlled feeding as soon as the birds have recovered.

7. Feed one pound of hen-size grit per 100 birds per week beginning the 7th week (43 days). Feed the grit on a "feed day."

8. On the "Skip 2 Days per Week" feeding program do not skip feeding on consecutive days. Suggestion: Skip feeding on Sundays and Wednesdays.

DETERMINING AVERAGE BODY WEIGHTS:

During the 8th week (50-56 days), sample-weigh approximately 5% of birds in each pen. To be sure the sample is representative, make a random selection of birds from different areas of each pen. Take sample weights in a similar manner each week; thereafter, throughout the growing period. Birds should be weighed on the same day of each week, and at approximately the same time of day, preferably in the afternoon. Birds on "Skip Day" feeding programs should be weighed at approximately the same time of day on "no-feed" days.

LIGHTING THE GROWING BIRDS:

It is advisable to delay the onset of egg production of broiler breeder pullets in order to have satisfactory early egg size. Two programs are effective to accomplish this: (1) a feed-control program and (2) a light-control program. In most instances, the two are used simultaneously.

Long hours of daily light hasten the bird's sexual development. Growing programs to control sexual maturity must limit light stimulation. Several methods can be used. Each depends a great deal on the varying length of the natural light day throughout the year, or the use of light-proof houses. These programs are discussed under two broad types of housing; (a) conventional buildings with windows which permit daylight to enter, or (2) totally dark houses (environmentally controlled) with zero light leakage. The most important factor in all lighting programs for growing birds is that the length of the light day should

never be allowed to increase.

THE CONSTANT LENGTH OF LIGHT DAY PROGRAM:

Determine the hours of natural daylight the birds will receive when they are 18 weeks of age (126 days) supplementing natural daylight with artificial light. EXAMPLE: If the length of natural daylight will be 12 hours when the birds become 18 weeks (126 days) of age, then, the amount of natural daylight plus artificial light should be 12 hours throughout the growing period. Provide this constant light-day length from one-day of age through the 18th week.

WINDOWLESS HOUSES:

For birds grown in totally dark houses, provide from 8 to 10 hours of artifical light daily. Do not vary or increase the number of light hours; maintain it constantly each day from one day of age through the 18th week.

MANAGEMENT: CHANGE OVER PERIOD FROM GROWING TO PRODUCTION (18TH WEEK THROUGH 24TH WEEK)

Beginning with the 18th week, pullets should be fed 3 pounds of oyster shells per 100 birds one day each week.

LIGHTS:

If birds have been on a constant light program for 18 weeks, daylight hours should equal the amount of light the birds have had up to that point. For those birds that reach 18 weeks on decreasing light, stop the decrease by use of artificial lights at 18 weeks. Hold this amount of light constant for one week and then plan to have the birds on 14 hours of light by the time they are 24 weeks of age. Additional light should

be added one hour at a time if possible; addition of less than one-half hour seems to have no stimulation on heavy breed birds.

I would suggest that you get a copy of Sunrise and Sunset times for the year from your nearest weather station for use in planning. Dusk and dawn have some effect which will vary from farm to farm; because a breeder house in a valley will not be affected by these as much as one located on top a hill.

The following table will show how the various hatch dates are affected by length of day at Salisbury, Maryland, which is almost the same latitude as Harrisonburg, Virginia.

HATCH DATE	DATE AT 18 weeks	DATE AT 24 weeks	DAYLIGHT (SUN. TO SUN.) at 24 Weeks of Age
1-1	5-7	6-18	14:50
2-1	6-7	7-19	14:34
3-1	7-5	8-16	13:36
4-1	8-4	9-15	12:20
5-1	9-3	10-15	11:05
6-1	10-4	11-15	9:55
7-1	11-3	12-15	9:19
8-1	12-5	1-16	9:36
9-1	1-5	2-16	10:36
10-1	2-4	3-18	11:49
11-1	3-7	4-18	13:10
12-1	4-6	5-18	14:15

Never let light increase on birds up to 19 weeks of age; never let light decrease from 19 weeks on.

FEEDING DURING THE CHANGEOVER:

At 21 to 22 weeks of age, change from a developer feed to breeder feed but stay on restricted feed until birds hit 5% production and at this time go to 32 to 34 pounds of feed per 100 birds per day.

When we combine the lighting program with the feeding program, we should reach 5% production between 24 and 25 weeks of age; if birds do

not mature sexually fast enough to bring on production at this time, re-evaluate the changeover program, and make slight adjustments if required - do not try to bring the birds into production by increasing the feed enough to put on fat.

MANAGEMENT DURING LAY:

When the birds hit 50% production if they are not on 15 hours of light, go to 15 hours and increase feed by 1 to 2 pounds per 100 birds per day. It is advisable to control the feed intake of the breeder flock once it has passed the peak of egg production in order to regulate the body weight of the hen. But the program needs close supervision. During cold weather, the birds may require less feed. Regulating the body weight of the bird MUST be associated with the weight of the bird at the time it lays its first eggs, ENVIRONMENTAL TEMPERATURE, EGG PRODUCTION, AND CALORIC CONTENT of the ration. Any table showing feed consumption on a controlled feeding program for layers can only be a guide. Depending on the conditon of the flock, the birds may require more or less feed. Important points for controlled feeding are:

a) After production has peaked and then declines four percent, reduce the daily feed allotment by ½ pound per 100 birds. If this causes an abnormal drop in egg production, return the flock to the amount of feed consumed prior to this first feed reduction.

b) As egg production continues its normal decline, reduce the daily feed allotment by ½ pound per 100 birds EACH WEEK until 30 pounds of feed are fed per day. Then, make no further reductions if egg production is not declining.

c) Do not reduce the feed intake during periods of stress or disease.

d) Increase feed allocation to the previous level if daily production per hen decreases more rapidly than normal.

A good group of pullets will make a good group of hens; a poor group of pullets seldom pay their way. Keep them lean and mean and they will reward you with additional broiler chicks. Do not allow the pullets to put on fat as they are coming into production. Remember, for each ¼ pound of excess weight these birds have at the time of peak production will take two pounds of feed per 100 birds per day for the rest of their lives.

TURKEY BREEDER FLOCK MANAGEMENT

O. P. Saffores

Breeder Flock management starts with the breeder hen. In adopting an old adage, for illustration, "which came first, the turkey or the egg" we will assume, for our purposes the turkey came first. Management must take into consideration the breeder hen and start with a breeder that has the potential to produce a large number of poults that are disease free and have the capacity for maximum growth.

Feeding the Breeder Hen Candidate

We, as breedermen, have in the past fed a breeder hen much like we would a meat bird, just pour the coal to her and let her get big and fat to selection age, approximately 17-18 weeks; we then select our breeders, and in trying to keep internal body fat off, we will put the selected breeders on a holding ration, a high-fiber feed, trying to save dollars in a less expensive feed and keep the weight of the breeder down. This, as far as I'm concerned, has not been successful. First, the turkeys eat that much more to satisfy their energy needs and it ends up costing you as much or more than a normal finisher feed would. The birds with the increased consumption end up too heavy at lighting time, so neither goal is achieved.

I believe there are two approaches that are much better. One is physically restricting feed and the other is to feed a low protein ration. Both, in my opinion, should be started by 12 weeks of age and

continued until lighting time. When considering this approach, be sure to consult your nutritionist to get a balanced diet. If fed indiscriminately, you could get into trouble. If one is going to use either of the two methods, restriction, or a low protein feed, it would be well to select your flock prior to starting such a program so your reject birds do not suffer. Even though this is a small number, probably 5-10% of a flock, it would be an economical loss. The other alternative would be to select at 21 weeks.

Facilities

The present and future need for product year round requires special facilities. Dark-out buildings - birds that are maturing on a lengthening day will mature too early, resulting in poor egg production. In order to control the maturity, one must use dark-out buildings, so any hen hatched between August 1 and April should go through a dark-out period. I would strongly advise against any short cuts from this proven method; they are just too risky. I went through a costly lesson last year trying to bypass this procedure. The dark-out phase should be started just as the birds start to show sexual activity (a few hens starting to squat). I believe that it is possible to start darkening-out hens too early; you should wait until they have reached sexual maturity.

The type of building is not all that important, nor is complete darkness. There can be dim light as long as it is diffused by the time it reaches the bird. I have found that using black curtains with a curtain release works extremely well and can be adapted to any curtain house. The principle is to plan on how much light you want and have this from daylight until noon. Roll the curtains up at noon and at ½ hour past dark. A time clock activates the curtain minder and the

curtains drop and the fans go off. This eliminates our problem of power failure at night, which has resulted in disaster.

It is important in a dark-out facility to have adequate air movement. I plan on a complete change of air ever 1½ minutes. I recommend a minimum of 3 sq. ft. of air per hen.

Breeder Hen Building

Set up with semi-trap nests arranged so the hen can be locked out of the nests and penned up away from the immediate nest area.

Allowing 5 hens per nest and 5 sq. ft. per hen is adequate, including the nest area.

Tom Building

A tom building should be separate from the hen buildings, not a part of it - the light should be controlled and fan ventilated. It should be constructed so the amount of light the tom will receive can be controlled.

After selection, the next step is:

Vaccination

Breeders should be vaccinated for Pox, Newcastle, Avian Encephalomyelitis, Cholera, and Erysipelas. You should check with your local State Veterinarian to get an approved vaccination program.

Lighting

There are three considerations in determining a lighting program:

1. The amount of light
2. Age to light breeders, hens and toms
3. Type of lights

First, the amount of light. There have been many studies about the amount of light, and the general feeling is that you should give a mini-

mum of 5 candle power for hens for 14-16 hours. Anything over 16 is wasted, and anything under 14 is inadequate.

Age at lighting is another highly controversial subject. I believe that the work that has been done will show that the optimum time for lighting should be 31 weeks of age.

The type of lighting. I would recommend regular incandescent light bulbs. Quartz lights work very well if there are no objects to cause shadows. Remember, in lighting toms they should be prelit 3-4 weeks prior to the hens and with a fraction of the intensity -- ½ to 1 foot candle power is sufficient for toms, and with the lower intensity light, they will be less likely to go into a molt.

AI and scheduling

The usual period of time after lighting to begin AI is 2 weeks. This will vary a couple of days either way. The best method to determine the time is to go in to a flock and pen up 100 turkeys and try to open the hens. If 80% open, go ahead and IA, then come back within 5 days with a second AI and 5 days with a third AI. This will give you three inseminations within 10 days; then, go on a 2-week schedule for ten weeks; and go either a 10-day or 7-day schedule. Another important step is to keep on a schedule until your hens go to market. Don't skip the last insemination; this can cause a very sharp drop in fertility for the last week's eggs.

Egg Handling

Management can do more in upgrading a breeder-flock's product by proper egg handling than any one single phase of management.

Egg handling has to start with egg collection. Eggs have to be collected regularly and often; during the peak lay period this will be

constantly. This not only improves your quality of egg but also brings you an economic gain by having less culls, primarily in egg breakage.

As soon as the eggs are gathered they should be cleaned and sanitized. Don't wait until the next morning or that evening; do it after every gathering. There are two methods of egg sanitizing. (1) Fumigation. (This if done properly with the proper humidity and heat is an excellent method). The problem arising here in having a proper cabinet and getting the job done properly. (2) Eggwashing (this, as far as I am concerned, is the most efficient way to handle hatching eggs). Most eggs are dipped at the hatchery so it is necessary to have as clean an egg as possible before dipping. This can be done at the farm level with proper egg washing equipment, properly supervised. The other benefit of on the farm washing is the labor factor. Equipment that will clean, sanitize, and dry in one operation has a tremendous amount of appeal in the labor saving feature alone.

Broody Control

There are many ways to handle broodys. As is often said, "Do something, even if it's wrong," could apply in this, the roughest part of breeder-hen management. It starts with the recognition of a broody, and once recognized what is to be done with her. It is important to change her environment, get her off the nest for 2-3 days to break her broody cycle and get back into production. This can be done by switching pens or by placing broodys in broody pens, rotating these. Like I've said, this is the toughest part of breeder hen management, and one where the Breeder Man must work out his own system. One fact to remember. There is a strong correlation between high egg-production and broody control.

Record Keeping

It is important for the farm manager to have accurate records, so that he may stay on top of and know how the flock is doing - these should contain:

1. The vaccine schedule, days and type of vaccine used
2. Date of lighting
3. AI dates
4. Egg records, recording culls and type of culls. If your cull eggs are running too high, you should know what kind of culls they are so you may be able to do something about it
5. Graph the projected egg production and post in egg house so the people on the farm can see how they are doing
6. Fertility and hatchability should be maintained on a graph as well

I have hit the high spots and will be glad to answer any questions I can in a bull session. I would like to stress the Man in management. You can lay out the finest breeder program in the world, but if the Man isn't there to carry out the functions, the program will fail.

Today we are in an ever-increasing cost spiral with a huge demand for quality products, faced with competition for getting labor, a situation that is ever getting worse. We either attract the too-young or the too-old to do a job that takes dedication and a concentrated effort. This doesn't make the Breeder Hen Managers job any easier, and there are no easy solutions to this problem.

POULT QUALITY AND HATCHERY MANAGEMENT

Milton Hendrixson

How well a turkey does during its life is to a large extent, determined during the first two weeks of its life. Legs, livability, condemns, and dollar reture are for a large part established during the beginning two weeks of life. Feed or nutrition, grower husbandry, and poult quality are the main factors which determine the future of a turkey during the critical first two weeks of life.

The production of a quality poult as a responsibility of a hatchery starts with the egg being produced on the farm and ends with the poult placed on the growing farm. The production of a quality poult is the result of many operational procedures being properly carried out. The failure to properly perform any one step will depress poult quality.

Some of the procedures that must be carried out in a precise manner are listed for your information.

Disease-free breeders maintained on a clean farm with clean nests are a must. Furthermore, eggs must be sanitized as soon after lay as possible, preferably within an hour. Eggs must be stored in clean egg rooms, in clean egg cases and transported in clean trucks.

At the farm and at the hatcher, eggs should be held at 65°F and 80 to 85% humidity. While an egg is being held in storage it is constantly losing moisture. By keeping the humidity in the egg rooms high this moisture

loss is reduced. Once an egg has been sanitized, avoid any procedure such as handling where the egg can be contaminated.

Turkey eggs should be dipped by some method that draws an antibiotic solution into the egg prior to setting. Turkey eggs should be four days old before setting.

Turkey eggs should be set in clean machines having a clean source of intake air to prevent contamination of the egg while in the setter or hatcher. It is desirable to treat the machines on a continuous or periodic basis with a disinfectant to insure that the eggs stay free of bacteria and molds while in the machines.

Poults should be taken from the hatchers before they dry-down too mech, and be held in poult rooms with controlled humidity at 80% to prevent dehydration.

Sexing of poults should be done as soon as possible after the poult comes from the hatcher. Poults will live better if they are sexed before drying out too much.

All equipment, floors, wall, and air in a hatchery should be kept extra clean at all times by constant cleaning and filtering. No unnecessary personnel, equipment or materials should be permitted to be in a hatchery for anyone may harbor or carry disease. All personnel should be required to change into clean work clothes when entering a hatchery.

Poults should be delivered in new boxes in freshly cleaned trucks. Never reuse a paper box. Keep the poult comfortable while delivering and plan to arrive at the farm 24 hours after the poult has been pulled from the hatcher. For best results, never place poults in a house

that is not ready with feed, water, and heat. As soon as poults are placed inside the brooder rings, get all noise and personnel away and let poults adjust to their new situation.

POULTRY NUTRITION

Hal Yacowitz

This year has been very chaotic for both nutritionists and purchasing agents. Due to high priced ingredients, rapidly shifting prices, and shortages of some ingredients, nutritionists have had to reformulate feeds more often than in the past. Constant reformulation and shortage of ingredients have resulted in some nutritional problems in the field. Before discussing some field problems seen this past year, data will be presented on broiler feed conversions. The purpose of these data is to provide some goals which servicemen can work toward in improving broiler performance.

Table I shows the performance of the three best and three poorest broiler flocks in an operation which produces approximately 400,000 broilers per week. The average performance of all flocks is also shown. All data were converted to a broiler weight of 3.75 pounds, using the figures of four points of growth equals one point of conversion. This was done to facilitate the comparison of flocks of different ages and weights. The figures representing the three best and three poorest flocks each represent an average of 50 - 60,000 broilers.

During four weeks in July 1973, the average difference in conversion between the best and poorest flocks was 0.21 pounds of feed per pound of weight. Thus the poor flocks consumed about 10% more feed per pound of body weight than the best flocks. Since all flocks were on exactly the same rations, the differences in performance are due to

all the management factors that are involved in broiler production. The ultimate goal of the serviceman is to have all flocks perform as well as the best flocks.

Table II shows a comparison of field performance of broilers with the performance of broilers grown on the AMBURGO research farm. The rations were produced at the feed manufacturer's mill and a portion shipped to the AMBURGO farm so that field rations and research farm rations were identical. The source of chicks used in the field and on the research farm was also the same. The data were converted to a 3.75 pound body weight.

It can be noted that the broilers on the research farm, in small floor pens, under ideal management conditions, performed much better than the best three flocks grown under field conditions. This means that the true performance potential of a given ration is not reached under field conditions.

The higher stress level under field conditions is apparently depressing broiler performance. We obtain little or no growth response from antibiotics and many feed additives under our research farm conditions, while we see marked improvements in performance under field stress conditions. When we learn all the reasons for this marked difference in field and test farm performance, we can look forward to significant improvements in feed conversions.

Let us now turn to some problems seen under practical conditions.

Weak bones in broilers - During the past few years we have been seeing a weak bone condition in broilers in the various broiler producing areas. The condition in young chicks is characterized by abnormal, irregular, spongy cartilage on the ends of the tibia. Birds

having this condition show leg weakness. This condition was described in 1965 by Drs. Leach and Nesheim of Cornell (1). Dr. Ray Harris of North Carolina State University is now seeing this condition in many field flocks of broilers. The condition is worse in flocks from breeders that have had problems with staphylococci or coliform infections.

Bone ash assays on birds affected with this condition are normal. In my laboratory, normal bone ash values ranging from 54% - 55% were recently obtained in affected broilers at 10 and 23 days of age. Bone ash from chicks showing vitamin D deficiency will show values of 30% or lower.

Increased fortification of rations with calcium, phosphorus, vitamin D and trace minerals has had little or no effect on this abnormal cartilage condition. Dr. Leach, now at Penn State, finds that he can influence the incidence of this condition by selective breeding but has not been able to overcome the problem by nutritional means.

Faulty cartilage development is a problem in fast growing individuals of all species, including humans, and requires additional research to find a solution.

Water Belly (Ascites) in Turkey Poults - We recently encountered an interesting case of ascites in broiler strain turkey poults. These poults were started in batteries and shipped to the grower at 10 days of age. Water belly started a few days after the poults were put on the floor. Mortality ranged from 2-8% and lasted for a 7-10 day period. There was no problem in a large white strain of turkeys hatched locally and started in floor pens, at day old. The large strain turkeys were fed the same ration as the broiler strain turkeys.

The starter ration assayed 1.13-1.56% salt but was calculated to contain only 0.47% salt. Though the salt level of 1.13-1.56% caused ascites in the shipped poults, it caused no problems in those poults which were not exposed to the stress and dehydration of shipping. As pointed out by Mr. Milt Hendrixson of Goldsboro Milling Co., dehydration is a serious problem in poults. Dehydration during shipment followed by a higher than normal salt level in the ration, apparently triggered the ascites problem.

In a paper presented at the recent Poultry Science meeting by Morrison et al. (2) of the Ontario Agricultural College, they studied ascited in battery reared poults fed various salt levels. It is interesting that Morrison et al. found no differences in water intake in young poults fed 0.7, 1.2, 1.7 and 2.7% salt in the ration during the period from 0-14 days of age. We know that most animal species show increased water intake and increased water excretion as the level of salt in the ration is increased. This is due to the action of salt in the blood acting on the thirst regulating center in the brain (hypothalamus) and causing the animal to have a sensation of thirst. The young turkey poult, according to the data of Morrison et al., does not show increased thirst due to high salt intake. Apparently their thirst regulating mechanism is not fully developed at that age. If the poults increased their water intake, they would have excreted the salt and it would not have resulted in ascites. This would explain the high susceptibility of young poults to water belly, while older turkeys are relatively free of this problem.

Litter eating in poults - Another field problem is the tendency of young poults to eat litter.

Dr. Charles, at the University of Georgia, found that very high levels of folic acid corrected this problem in one trial but were not effective when the test was repeated.

In some field cases of litter eating, reported by Dr. Poss of the Earl B. Olson Farms, Inc., in Minnesota, the poults had a previous history of enteritis during the first few weeks of life. This was followed by a tendency to eat litter. Consumption of litter is usually followed by multiple nutritional deficiency symptoms, such as rickets, white muscle syndrome (due to lack of vitamin E and selenium), folic acid deficiency, etc. Since the poults are not eating, their consumption of coccidiostat is reduced and they often show coccidiosis outbreaks. Mycosis problems will also develop since the litter is high in fungi.

In many cases, the problem of litter eating can be avoided by suppressing the initial enteritis condition with antibacterial antibiotics fed at high levels, followed by use of an antifungal agend to prevent mycosis.

Dr. Al Kurnick, of Hoffman La Roche, has reported a malabsorption syndrome in chicks and poults. This syndrome results in multiple deficiency symptoms and responds to vitamins A, D, and E in the drinking water. Assay of the feed shows that these nutrients are present at adequate levels. It is possible that the malabsorption syndrome is due, in part, to litter eating and failure to consume sufficient feed.

Reduced growth and poor feed conversions in turkeys due to inadequate lysine levels - The essential amino acid, lysine, has been in very short supply. In addition, the high cost of proteins makes natural lysine expensive to use. As a result, many feed companies have had to reduce the levels of lysine in their rations.

In order to determine the effects of using suboptimal lysine levels, Dr. Leo Jensen, now at the University of Georgia, reported the effects of feeding 0.62% and 0.87% lysine in 17% protein rations to large white tom turkeys from 16 to 20 weeks of age (3). The 0.87% level is close to that recommended by the National Research Council.

Feeding the suboptimal lysine level (0.62%) resulted in one pound less gain and increased feed consumption by 11.5% per pound of gain, during the four-week period from 16 to 20 weeks.

Dr. Jensen concluded that excessive reduction of protein or lysine levels below the requirements for optimum performance is false economy.

Vitamin D_3 deficiency and weak egg shell problems

In layers raised in confinement, in windowless houses, the only source of vitamin D_3 is that present in the vitamin premix. None of the natural feed ingredients supply any appreciable amount of vitamin D_3. Failure to include the premix in a ration, or any loss of vitamin D_3 in the premix due to instability, can result in deficiency symptoms.

A number of D_3 deficiency problems in layers occurred during the past year. Decreased egg production, very week egg shells, and increased mortality in the hens due to D_3 deficiency can be rapidly alleviated by administering D_3 in water. The problem can be avoided by using properly stabilized D_3 at adequate levels in the vitamin premix and not including trace minerals in the premix package. Trace minerals will accelerate the destruction of vitamin D_3, particularly in a hot, humid environment.

High vitamin E levels and disease resistance

In human nutrition, the use of megavitamin dosage levels is expounded by some scientists and condemned by others. Megavitamin dosages deal with the use of massive levels of vitamins, far above

the normal levels required for growth or maintenance. One example is the use of massive levels of vitamin C for prevention of colds in humans. The effectiveness of these massive dosages of vitamin C is still undergoing extensive research.

At the recent Poultry Science meeting, Heinzerling, et al. (4) of Colorado State University reported that the use of very high levels of added vitamin E (136,000 I.U. per ton of feed) significantly reduced mortality and improved growth in E. Coli infected chicks. Hemagglutination titers also increased with added vitamin E resulted in increased antibody production.

These findings suggest that we may be able to improve disease resistance in chicks using the megavitamin concept.

Summary of information that can be of practical application

1. In a typical integrated broiler operation, feed conversions could be improved by 10% if all growers could attain conversions equal to the top three growers.
2. Excessive reduction of lysine levels in turkey rations from 16 - 20 weeks of age can result in reduced performance and reduced profits.
3. Recent research using very high vitamin E levels showed that vitamin E reduced mortality and improved growth in E. coli infected chicks.

TABLE I

PERFORMANCE OF

3 BEST AND 3 POOREST BROILER FLOCKS*

Week Sold	Feed Conversion Before Condemnations		Difference In Conversion	Average Conversion of All Flocks**
7/1/73	Poorest 3 flocks	2.23		
	Best 3 flocks	2.03	0.20	2.13
7/8/73	Poorest 3 flocks	2.21		
	Best 3 flocks	2.01	0.20	2.12
7/15/73	Poorest 3 flocks	2.25		
	Best 3 flocks	1.99	0.26	2.11
7/22/73	Poorest 3 flocks	2.19		
	Best 3 flocks	2.02	0.17	2.09
		Avg.	0.21	

*All data calculated to 3.75 lb. average body weight

**Average of approximately 400,000 broilers sold per week

TABLE II

COMPARISON OF FIELD PERFORMANCE

AND RESEARCH FARM PERFORMANCE OF BROILERS*

	Source of Data**	Weight	Feed Conversion Before Condemnations	Difference
		lb.		%
1)	Field (Approximately 400,000 broilers)	3.75	2.13	
2)	Field (3 best flocks- approximately 50,000 birds)	3.75	2.00	- 6.
3)	AMBURGO Research Farm (6 pens of 50 broilers each)	3.75	1.75	- 18

*AMBURGO Research Farm test conducted by C. Knestrick

**All birds fed the same rations, mixed by feed manufacturer

References

1. Leach, R. and M. C. Nesheim, J. Nutr. 86, 236, 1965.

2. Morrison, W. D., A. E. Ferguson, J. R. Pettit and D. C. Cunningham. Abstracts, 62nd Annual Meeting Poultry Science Assoc. 1973.

3. Jensen, L. S., University of Georgia Extension Service, Publication PH 3-3, August 1973.

4. Heinzerling, R. H., C. F. Nockels and C. L. Quarles. Abstracts, 62nd Annual Meeting Poultry Science Assoc. 1973.

MYCOPLASMA

R. D. Killingsworth

I have been asked to speak to you on Mycoplasma in poultry, but since I profess to be no expert in this field, I would like to concentrate on my presentation on only two Mycoplasmae. Both are found in chickens. You are familiar with both and have dealt with them in your operations. I speak of M. gallisepticum and M. synoviae. Gentlemen, I have no startling new facts about these organisms to offer you, only a review of things we have learned in the past.

I spoke a moment ago about these organisms being found mainly in chickens, but as we talk about these two organisms and the potential problems they present this winter, I feel we must also consider the turkeys in the area, especially since we can get cross infections with turkeys. If there are skeptics here who say that as sophisticated as our industry is, cross-infection couldn't occur, let me tell them about the company in mid-1973 that had a flock of M.g. positive turkey hens that they thought were too young to sell and so decided to keep and treat the poults because they were not egg dipping. Do you suppose this was a possible source of cross-infection to chickens and a potential reservoir of infection for clean turkeys? Well, as you might suspect, both happened in 1973! Some of the broiler flocks were infected, as well as some of the clean turkeys.

Another example in 1973 is the company that did not want to believe that M.s. could be transferred to turkeys from chickens. Turkey poults

were placed on a farm where some broilers that were M.s. positive had been left in the house. These broilers were not destroyed but left to roam as they would, even though the house was well cleaned out for the turkeys. When the turkeys were ranged, the chickens ate with them. The turkeys developed respiratory problems and leg problems diagnosed by culture as M.s. -- not M. meleagridis as the company wanted to believe.

This problem I have just mentioned could have been averted if the serviceman involved had been "on the ball." What if this had not been turkeys, but another broiler flock. Would you, as a conscientious serviceman, let a situation like this happen? THINK! Not only would your company be losing money because of drug costs, but the performance of the birds involved would be reduced, which in turn would cause the grower to gripe about YOUR bad birds. Whom are we kidding -- him or ourselves? Think back. I'm sure you remember that old Chinese proverb: "He who keep yard chickens to keep his house chickens company has M.s.-essed in his nest!"

Seriously, remember--Mycoplasma gallisepticum causes air sacculitis in chickens and turkeys as well as sinusitis in turkeys.

Mycoplasma synoviae causes synovitis and air sac problems in chickens and turkeys.

Let's turn our attention to only M.g. for a moment. There are probably very few M.g. positive birds around right now. Nevertheless, we do not want to forget about M.g. Let's take a little problem that does occur in the field. What if, suddenly, a few positive M.g. plate tests show up, even though these birds have been testing negative. What do we do and what do we look for?

First, Do Not Panic! Second, resample. Then, two weeks later, resample again. If additional positives to plate tests turn up and borderline or rising HI titers of above 1:80 are encountered, consider these birds positive and either decide to sell them or resample and get some culture work done, even if you must go out of state to do it.

If the samples only turn up positive to plate test and negative to HI, then pull out your vaccination records. It is reported in the literature that following IB and ND vaccination, and IB, ND, and AE vaccination sometimes a transient positive M.g. reaction will occur. This may occur if the samples were pulled within one to two weeks after these combination vaccinations were given. This is a transient reaction, and it usually disappears in 10 to 14 days after the vaccine is given.

It is also reported that these "odd" reactions will sometimes occur in M.s. positive birds. In this case, the birds will usually be showing positive, or weakly positive, M.s. tests at the time of the positive M.g. plate test. Sometimes the M.s. will not show up as positive until after the transient M.g. positive test has again become negative.

Let's turn our attention to Mycoplasma synovia. As most of us remember, researchers in the past have considered M.s. to cause joint infections primarily. Now, we consider M.s. to be a source of respiratory infections as well as joint infections. So let's review some of the things we should start thinking about, in case some problem flock happens to come along this fall and winter.

It has been reported by Drs. Klevin, King, Anderson, and Yoder of Georgia and other workers that the severity of M.s. infections is greatly enhanced by vaccination reactions associated with viral

respiratory vaccines. With this fact in mind, shouldn't we use as mild a vaccination program as possible on these positive chicks, as well as place these chicks in houses in areas away from other birds and traffic?

Ventilation - Poor ventilation is one of the major contributing factors to air sacculitis. We all know that this is one reason why we always see more air sac condemnation during the cooler months. We know how to correct improper ventilation, but it is sometimes one of the hardest jobs to get accomplished.

If we have to go on fuel allocation this winter, ventilation will become a major factor we have to face because we just won't be able to turn up that heat to go with that cold air we need to pass through the house.

Mixing Flocks - Another thing we should try to accomplish this winter is not to mix M.s. positive chicks with negative chicks. If a breeder flock is known-positive, set the eggs so that these chicks can be placed in houses by themselves and hopefully on farms where houses are not close together. We usually get by with this practice of mixing flocks in the summer; but in winter, severe problems arise after mixing occurs.

Other Poultry Carriers - It is reported in the literature that ducks, pigeons, guinea fowl ("gennies"), quail, and pheasants can be infected with Mycoplasma organisms and act as carriers. Should this have included also sparrows and starlings, though I could not find specific reference to them. If only a few of the sparrows I see in some houses could be carrying these Mycoplasma, it is understandable why we sometimes have a break we can't explain.

I hope you will take what I have presented as constructive. Just because things have been going well does not mean we have to become complacent. We sure don't want to be jarred back into reality by a condemn sheet.

I do not plan to elaborate on treatments for Mycoplasma today. Those of you who know me, know that I am biased toward good products when it comes to Mycoplasma treatments. Furthermore, I give you credit for knowing what is working for you under your farm conditions. I would warn you to beware of "cure-all" products being added to Marek's vaccine, lest someone make a mistake and destroy the vaccine. I still believe water medication with TYLAN is best and a tetracycline is second best.

I thank you most sincerely for letting me be on your program.

E. COLI INFECTION

W. B. Gross

E. coli is a minor part of the normal gut flora. The many species of organisms in the gut compete for nutrients; they produce products which may be toxic or essential for other organisms. Only a few strains of E. coli exist in the gut at one time and this population tends to be stable. Some of the E. coli. strains are pathogenic and some are not. No strain of E. coli is known to produce disease when confined to the gut of chickens or turkeys.

From the feces, egg shells can become contaminated. The organism may penetrate the egg shell and enter the egg contents. Some strains kill the embryo during incubation. Other strains allow the weak chick to hatch and quickly die. Other strains may cause only a reduction in growth rate. In the hatcher, E. coli spreads to other birds and may become part of the gut flora of the new flock. Some of the chicks develop yolk infections which may result in early losses. This infection is usually eliminated after a couple of weeks and only the gut population remains. From the gut, E. coli gains access to the dust where it can remain for several months. If conditions are right, infections can occur from the dust source. Rodent carriers can contaminate; feed and water can be contaminated from manure.

Many other conditions have E. coli as part of the cause. E. coli from the gut can ascend the ovaduct and cause salpingitis. If contamination of the peritoneal cavity occurs in conjunction with broken yolks, peritonitis results. The predisposing factors for a

cholera like septicemia and for coliform synovitis are unknown. The sequence of events leading to air sac disease are as follows: Chickens become infected with Mycoplasma and/or one of the respiratory viruses such as NDV or IBV (vaccine strains are as effective as field strains). The infections are made worse by environmental or social stresses. When the infection is severe enough, the respiratory tract becomes susceptible to E. coli from the air and infection may progress to pericarditis or death.

E. coli infection can be diagnosed by the isolation of the organism from diseases typical of E. coli infection. Serotyping is not necessary and may lead to confusion since the potential contaminant might also be a pathogen. Serotyping is essential for epidemiological studies.

Many drugs have been effective in the treatment of E. coli infections. Among the problems to be considered are the long duration of infection in a flock as opposed to the short duration in individuals. Chronic infection such as salpingitis and superacute conditions such as peritonitis are not treatable. E. coli is a very adaptable organism and often develops resistance to drugs. It is not possible to clear E. coli from the gut with drugs (what about the house?) The cost of the high doses of drugs that might be required may not be justified.

A program of clean eggs and clean hatchery seems to me to be the most effective low cost prevention of E. coli infection. Adequate feed, water, and temperature help chicks combat E. coli infections. Live virus vaccination after 4 weeks of age is a predisposing factor. Adequate ventilation helps reduce the level of E. coli contamination of the dust. E. coli can be removed from feed by pelleting. Rodent carriers should not be allowed around poultry. Drinking water should be free from E. coli.

E. COLI INFECTION ALMOST ALWAYS REQUIRES A PREDISPOSING FACTOR.

GANGRENOUS DERMATITIS

Stanley G. Moum

Gangrenous dermatitis is an infectious disease of chickens characterized by sudden onset, a sharp increase in mortality and gangrenous necrosis of the skin over the breast and thighs. The disease occurs sporadically in growing chickens from 4 to 16 weeks of age and affects both broiler and replacement stock.

Necrosis is defined as local death of tissue cells within the living individual. Gangrene is defined as the invasion and putrefaction of cecrotic tissue by saprophytic bacteria. By these definitions, it is obvious that necrosis precedes gangrene.

The cause or causes of gangrenous dermatitis are not yet fully understood. Both aerobic and anaerobic bacteria have been isolated from chickens with the disease. Recently, several investigators have reported that the anemia usually seen in conjunction with gangrenous dermatitis can be reproduced in susceptible embryos and chicks with an agent having the characteristic of a virus. This agent is associated with inclusion body hepatitis (IBH). Further investigation is needed before the anemia produced by IBH can be implicated as being part of the syndrome of gangrenous dermatitis.

Environmental factors that may or may not be included in the disease are: heavily contaminated old litter, cannibalism due to poor debeaking, skin injury, anemia, and devitalization of the skin as occurs in staphylococcal infections and in selenium deficiency.

The disease can be reproduced if the skin is first injured or the tissues are irritated with injected chemical irritants and C.l. septicum is injected into the damaged tissue. Inoculated chickens develop gangrenous necrosis of the skin and musculature and death occurs within 12 to 48 hours.

The first sign of gangrenous dermatitis is usually a sudden increase in mortality in the affected flock. Mortality can be as high as 60%. Affected chickens die within 8 to 48 hours after showing signs of depression, lameness, and prostration. Externally, there are patches of gangrenous skin over the breast and thighs and frequently feather loss of sloughing of the epidermis are seen. At necropsy, there is an accumulation of bubbly serosanguineous fluid in the subcutis and the underlying musculature has a cooked appearance. Frequently, the liver and spleen are enlarged and contain large areas of necrosis. The kidneys are usually swollen and the lungs congested and edematous.

Diagnosis of the disease can be made by isolation of Clostridium spp. from the affected area, or by demonstration of gas formation and numerous large filamentous bacilli in the skin, musculature, and liver. The demonstration of saprophytic bacteria coupled with history and clinical findings will differentiate this disease from exudative diathesis, staphylococcal infection, and other diseases involving the skin.

Treatment with antibiotics and other oral medications has been inconsistate in reducing mortality; however, incidence of the disease can be reduced by following one or more of the following practices: complete cleaning of the poultry house, establishment of strict sanitation practices, proper debeaking to control cannibalism, removing

objects from the poultry house that can cause mechanical injury to the skin, and maintaining optimal nutrients in the diet to maintain healthy vigorous body tissue. Care should be taken not to aggravate anemia if it exists in the flock.

VIRAL DISEASES OF CHICKENS AND TURKEYS

R. T. DuBose

The better known viral diseases have been covered in various meetings over the past 10 years. Today we will discuss three that are less well known. VIRAL ARTHRITIS (TENOSYNOVITIS) was diagnosed in a Virginia flock this year by virus isolation. Infection with an adenovirus related to one that causes ADENOVIRUS HEPATITIS SYNDROME in chickens was detected by serologic tests in Virginia turkeys after they exhibited respiratory difficulties. INCLUSION BODY HEPATITIS has not been reported in Virginia, but its presence in the midwest and Canada indicates we should be on the look-out for it.

VIRAL ARTHRITIS (OR TENOSYNOVITIS)

Outbreaks in Virginia of viral arthritis (also called tenosynovitis) were identified by the Veterinary Science Department of Virginia Tech in June and July of 1973. The department's investigation was a joint effort with Industry, the Virginia State Diagnostic Laboratory, Harrisonburg, Dr. C. S. Douglass, Veterinary Extension Specialist in poultry, and the speaker. The virus had been isolated previously in West Virginia, Pennsylvania, Maine, Texas, and Europe.

Viral arthritis usually appears in chickens at four to eight weeks of age, but is not limited to that age range. Inapparent infections (no symptoms or gross lesions) can occur and carriers can be a problem.

Birds other than the chicken are not known to be susceptible. Economic loss results from poor growth and feed efficiency, lack of uniformity and downgrading, condemnations, and, in severe forms, from mortality.

Lameness, and sometimes a disinclination to move when approached, result from inflammation and swelling of the tendons and tendon sheaths above and below the hocks, either on one side or both. In chronic cases, knots may be felt above the hocks where the tendons presumably ruptured from their attachments to muscles of the "drumstick" and later healed. Swelling of the wing joints (elbows) or foot pads are less frequent. Keel blisters may occur. Internal lesions vary with severity and with stage of the disease. The hock and/or elbow joints may contain yellowish to reddish fluid, occasionally a pus-like fluid similar to that seen in mycoplasmal synovitis. Similarities between viral arthritis and mycoplasmal synovitis, and the possibility of both being present, add to difficulties in making a diagnosis.

No treatment is known. Because infection can spread from bird-to-bird and probably from house-to-house, strict Security Management procedures should be followed to prevent mechanical transmission. For broilers, an all-in, all-out program has been recommended. The possibility that the virus is egg-transmitted should be taken into account.

ADENOVIRUS INFECTIONS AND HEPATITIS

Avian adenovirus infections have been detected in many species of birds in North America for over 20 years. Other than demonstrating that it caused quail bronchitis in bobwhite quail, attempts to connect Type I adenovirus with specific diseases of other birds in the U.S.A. were inconclusive. In the poultry industry, Type I adenovirus is usually called "CELO virus." Isolates from quail are called "quail bronchitis virus."

In 1972, researchers at the Indiana Agricultural Experiment Station and Purdue University isolated Type I adenovirus from chickens that were laying eggs of poor shell quality and that had hepatitis. They consistently reproduced the disease by infecting groups of 3-day-old to 8-week-old chickens by various routes with the newly isolated virus.

The original flock had shown a sudden increase in the number of eggs that were misshaped and ridged, but only slight drop in production. Mild respiratory symptoms occurred in the experimentally-infected chickens and inflammation of the trachea and the liver were consistently observed both in field cases and in experimental birds.

Diagnostic work may become more complicated because of similarities between the effects of this disease and infectious bronchitis, vibrionic hepatitis and even fatty liver syndrome.

In cooperation with industry and the State Diagnostic Laboratory, researchers, and the Veterinary Extension Specialist for poultry at Virginia Tech have looked for the presence of unusual viral infections in the state over the last three years. This summer, four turkey flocks with otherwise unexplainable respiratory difficulties were found by the speaker to have been infected with Type I (CELO) adenovirus. Investigation of these infections is continuing.

INCLUSION BODY HEPATITIS SYNDROME

Although this disease has not been recognized in Virginia, its appearance in the last three years in the midwest and in Canada and the possibility that it may be caused by Marek's disease virus or a very similar virus, are grounds for our watching for its presence in our state.

The name of the disease comes from inflammation of the liver

(hepatitis) and from microscopically-visible masses (inclusion bodies) of virus in cells of the liver. Inclusion body hepatitis (IBH) syndrome cause 10% mortality in 9-11½-week-old pullets and 0.1%-7% mortality in 4-7-week-old broilers. The latter figures involved 86 flocks. In some cases, deaths were sudden and visibly sick birds were few. The disease apparently does not occur in turkeys, but a similar condition has been reported in pigeons.

The question can be raised as to whether IBH syndrome is a new disease or is a newly-recognized version of hemorrhagic anemia syndrome, a disease known since 1954. There was evidence that hemorrhagic anemia syndrome was caused by toxins for molds (mycotoxins), and for that reason it was classed as a "mycotoxicosis" in some instances.

IBH hepatitis causes hemorrhages in the liver - from pinpoint or spotty to star-shaped-and sometimes pockets of hemorrhage under the capsule. Necrotic foci (light-colored spots or patches) may be seen in the surface. Hemorrhagic spots or patches occur in the fat and the muscles of the breasts and legs. Anemia is present, and the bone marrow may be pale, yellow or fatty. Changes from normal have been seen in the kidneys and bursa of Fabricius.

No treatment has been suggested at this time, but, as in all diseases, supportive measures (ample feed and water space, adequate ventilation, avoiding chilling) may hold down losses. Researchers in avian diseases are investigating methods of transmission, diagnostic serologic tests, and possible relationships to Marek's disease virus or to hemorrhagic anemia syndrome.

MOLDS AND THEIR TOXINS

J. R. Harris

The presence of molds is universal. The feed ingredients, feed, litter, and environment of poultry can and do support mold growth. Some molds are harmless, even helpful, while some are toxic. Most antibiotics are derived from molds.

Molds are plants that belong to the fungi group. Molds do not have chlorophyll like other plants and, therefore, cannot manufacture their own food. They must live on dead or living plants or animals, either as a parasitic or saphrophytic mold. Molds grow more slowly than bacteria and we do not generally find them growing in conditions where they have to compete with bacteria.

There are two types of problems associated with molds or fungi: (1) mycosis and (2) mycotoxicosis. Mycosis or mycotic infection is a disease involving actively growing molds or fungi. An example is Aspergillosis, or what is sometimes called brooder pneumonia. The respiratory tract is invaded by Aspergillus fumigatus. Another mycosis is caused by the yeast-like fungi, Candida albicans, which affects the crop and intestinal tract. This mycosis is usually a secondary infection as it can often be isolated from the digestive tract of apparently healthy poultry. However, if the poultry is weakened by something else, such as "starve-out" in poults, it will act as an opportunist and start to grow and reproduct by "budding."

The other types of problems from molds are called mycotoxicosis.

These result from animals ingesting feed contaminated by toxins produced by certain molds and called mycotoxins. These mycotoxins or poisons may be present in grains and feed following mold infestation, even though actively growing molds are absent.

This discussion will consider problems involving mycotoxins in poultry, particularly aflatoxins.

MYCOTOXINS THAT HAVE BEEN REPORTED TO AFFECT POULTRY

Mold	Mycotoxin	Primary Effect
Aspergillus flavus	Aflatoxins	bile duct, kidneys, liver pancreas
Aspergillus Ochraceus	Ochratoxin	0.5 ppm depress growth - 4.0 ppm high mortality
Fusarium roseum (Gibberella zeae - sexual state)	F-2 Fusario-toxin	vent swelling - prolapsed cloaca - turkeys Emetic effect in swine
Penicillium palitans	Tremortin A	tremor, small bursa - mimics Gumboro
Fusarium tricinctum	T-2 toxin	necrosis of skin and mouth - mimics pantothenic acid deficiency
Penicillium rubrum	Rubratoxin	not very toxic to poultry

Aspergillus flavus is a common mold, which under favorable conditions for its growth such as adequate oxygen, temperature in range of 50-90°F., relative humidity above 75% and moisture above 14%, may be found on all common cereal grains, oil meals, and meat products. It is the source of the widely studied mycotoxins, the aflatoxins, the exact number of aflatoxins are uncertain but they comprise two groups referred to as B for those that fluoresce blue under ultraviolet light and G for those that fluoresce a yellowish-green under ultraviolet light. The ones designated as B are the most toxic.

Species of poultry vary in their tolerance of aflatoxins. Chicks, ducklings, pheasants, and poults are susceptible to aflatoxins. Chicks

are most resistant while ducklings and poults are most sensitive. Neither of these species receiving one ppm or more of aflatoxin survives a four-week experimental period.

Ochratoxins

Ochratoxins are a group of mycotoxins produced by Aspergillus ochraceus. It is not as prevalent as A. flavus but has been found in grains, particularly if deteriorated. In broilers, 0.5 ppm depresses growth; 4.0 to 8.0 ppm produces high mortality. Leghorns receiving one, two, or four ppm from 14 weeks to one year of age had delayed sexual maturity, reduced egg production, and increased mortality. Chick growth from hens fed ochratoxin was reduced the first two weeks after hatch.

Fusariotoxins

Fusarium roseum produces an estrogenic mycotoxin known as F-2. When Fusarium roseum corn containing 10% of the mold was fed to poults, there was no effect on growth but a swelling of vent and prolapse of the cloaca was seen.

Fusarium tricinctum produces a mycotoxin T-2 that produces increased crop and pancreas weights and lesions of the mouth, feet, and legs mimicking pantothenic acid deficiency. Penicillium pallitans produces tremortin A, a mycotoxin that produces tremor and bursa regression in broilers. It resembles Gumboro disease in its action.

Symptoms of Aflatoxicosis

It is difficult to diagnose aflatoxicosis in the field. Poultry with poor feed conversion and weights and with poor egg production on a good plane of nutrition in the absence of other diseases, such as coccidiosis, should make you suspicious of aflatoxicosis. Birds will

also be pale, anemic, and bruise easily.

Aflatoxicosis is more severe in nutritionally marginal diets such as pullet and breeder replacement feeds and feeding methods.

Gross Pathology

Liver - brown to tan in color, swollen pale kidneys, full distended gall bladder, icterus, yellow color to muscle, small muscle hemorrhages and pale bone marrow. The diagnosis must be confirmed by testing the feed for the presence of aflatoxins.

Control Measures

Control measures are directed at preventing the growth of aspergillus flavus in feed ingredients, particularly corn during harvesting and storage.

1. The addition of grain preservatives that acidify the grain to prevent mold growth is being used. Chemical preservatives such as calcium and sodium propionate, propionic and acetic acid added to grain and feed can help in preventing mold but are not effective against aflatoxins.

2. Destruction of aflatoxin once it has been formed. Research in progress now shows that ammonia, either gaseous or liquid, at levels of 0.5% based on dry corn weight will reduce aflatoxins B_1 to below detectable levels. Feeding experiments on ammonia-treated grain is underway now.

3. What can a serviceman do? Insist that feed mill purchase quality feed ingredients and periodically have grain and feed tested for aflatoxin. Routinely, have bulk feed handling equipment and farm storage bins emptied, cleaned, and disinfected.

PHYSIOLOGICAL EFFECTS OF AFLATOXIN ON POULTRY

Parameter Effect	Aflatoxin Fed		
	Low (.6 ppm)	Medium (2.5ppm)	High (10 ppm)
Growth rate	no effect	decrease	decrease
Feed conversion ratios	no effect	increase	increase
Egg production	no effect	decrease	decrease
Egg size	no effect	decrease	decrease
Liver size (hens)	no effect	decrease	decrease
Liver fat (hens)	no effect	increase	increase
Liver size (chicks)	increase	increase	increase
Liver fat (chicks)	increase	increase	increase
Spleen size	no effect	increase	increase
Pancreas size	no effect	increase	increase
Bursa size	no effect	decrease	decrease
Kidney size	no effect	no effect	increase
Kidney function	no effect	no effect	decrease
Bruising	increase	increase	increase
Capillary fragility	increase	increase	increase
Tissue integrity	decrease	decrease	decrease
Interference with vaccines	increase	increase	increase
Antibody formation	decrease	decrease	decrease
Serum cholesterol	decrease	decrease	decrease
Serum total lipid	decrease	decrease	decrease
Serum triglycerides	decrease	decrease	decrease
Serum phospholipids	decrease	decrease	decrease
Hemoglobin	no effect	decrease	decrease
RBC count	no effect	decrease	decrease
Hematocrit	no effect	decrease	decrease
Bone marrow hyperplasia	no effect	increase	increase
Serum proteins	decrease	decrease	decrease
Serum uric acid	no effect	no effect	decrease
Serum calcium	no effect	decrease	decrease
Serum glucose	no effect	decrease	decrease
Adrenal size	no effect	increase	increase
Semen production	no effect	no effect	decrease
INTERACTIONS OF AFLATOXIN			
Carcass color	decrease	decrease	decrease
Candida albicans	worse	worse	worse
Vitamin D deficiency	worse	worse	worse
Riboflavin deficiency	worse	worse	worse
Vitamin K deficiency	none	none	none
Vitamin E deficiency	none	none	none
Thiamine	better	better	better

ENROLLMENT

Howard C. Agnew
103 Seaview Court
North Wildwood,
New Jersey 98260

Howard Anger
Sterwin Lags, Inc.
P. O. Box 1736
Salisbury, Md. 21801

Win Beane
Poultry Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

Robert L. Biller
Timberville, Va. 22853

Frank Bontz
Rocco Chickens
1620 South Main St.
Harrisonburg, Va. 22801

Dick Boyd
1167 Westmoreland Highway
Harrisonburg, Va. 22801

Roy D. Bradley
Grassy Knoll Hatchery
P. O. Box 6036
Charlottesville, Va. 22906

Robert L. Branner
Route 2
Timberville, Va. 22853

Denver Bragg
Heatwole Hatchery
Kratzer Road
Harrisonburg, Va. 22801

Ed Brown
Norwich Agr. Products
Route 3 Box 465
Salisbury, Md. 21801

Clyde A. Bryant
Amspec Inc.
Route 6 Box 283
Calhoun Avenue
Salisbury, Md. 21801

Robert Cain
91 Pleasant Hill Road
Harrisonburg, Va. 22801

Webb Cassell
Arbor Acres Farm
Mt. Harmon Road
Salisbury, Md. 21801

R. D. Chaney
Route 8
601 Cherokee Trail
Dalton, Georgia 30720

J. A. Cherry
Poultry Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

E. B. Craun
Agriculture Extension Service
County Office Building
Harrisonburg, Va. 22801

Don Cummins
Dow Chemical Co.
614 Irene Ave.
Salisbury, Md. 21801

Clifton S. Douglass
Veterinary Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

Prem Dua
337 Monticello Ave.
Harrisonburg, Va. 22801

R. T. DuBose
Veterinary Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

Richard Eaton
c/o Long Foods, Inc.
New Market, Va. 22844

Robert L. Ford
Box 227
Pen Laird, Va. 22846

Earl Garber
c/o Long Foods, Inc.
New Market, Va. 22844

Jerry Gass
Virginia Poultry Federation
Route 33 East
Harrisonburg, Va. 22801

Calvin Germroth
Rockingham Poultry Marketing Coop.
Timberville, Va. 22853

Guy Getz
Route 1
Broadway, Va. 22815

Paul Grady
Route 6
Harrisonburg, Va. 22801

W. B. Gross
Veterinary Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

Bruce Grover
Green Hills
2406 N. Augusta St.
Staunton, Va. 24401

Herman W. Halterman
Linville, Va. 22834

J. R. Harris
Scott Hall
Box 5307
North Carolina State University
Raleigh, North Carolina 27607

Milton Hendrixson
Goldsboro Milling Co.
Goldsboro, N. C.

C. E. Howes
Poultry Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

R. D. Killingsworth
Eli Lilly & Co.
Apex, N. C.

Harry Lantz
Route 1
Broadway, Va.

Alton Lawson
4 Shenandoah Ave.
Harrisonburg, Va. 22801

A. T. Leighton, Jr.
Poultry Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

Roger Lewis
Rockingham Poultry Marketing Coop.
Timberville, Va. 22853

Bill Little
Arbor Acres Farms
Concord, N. C.

G. W. Litton
V.P.I. & S.U.
Blacksburg, Va. 24061

Ivan L. Long
Dept. of Agriculture
267 Green Street
Harrisonburg, Va. 22801

Raymond Long
Broadway, Va. 22815

John P. Martin, Jr.
Route 6 Box 74
Harrisonburg, Va. 22801

Sam Mason
Eastern Shore Labs
P. O. Box 657
Laurel, Delaware 19956

Norris Miller
Route 1
Mt. Crawford, Va. 22841

Richard Miller
Rocco Feeds
1 Kratzer Road
Harrisonburg, Va. 22801

Jim Mongold
303 S. Main St.
Bridgewater, Va. 22812

Larry F. Moran
1192 Westmoreland Drive
Harrisonburg, Va. 22801

Stanley Moum
Merck & Company
Salisbury, Md.

Al Olinde
Grassy Knoll Hatchery
P. O. Box 6036
Charlottesville, Va. 22906

Howard Ore
861 Furnace Road
Harrisonburg, Va. 22801

Jack Paull
Box Q
Harrisonburg, Va. 22801

Walter Petit
109 Cresent Drive
Harrisonburg, Va. 22801

L. M. Potter
Poultry Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

Fred Rea
Va. Dept. of Agriculture and Commerce
Richmond, Va. 23219

Mark Reid
Route 1
Broadway, Va. 22815

James A. Rhodes
Agri-Bio Corp.
211 South 2nd St.
Altoona, Pa. 16602

Robert Rhodes, Jr.
Rocco Feeds
1 Kratzer Road
Harrisonburg, Va. 22801

Dave Ritchie
Route 2
Dayton, Va. 22821

Matthew Ritchie
Fulks Run, Va. 22830

Alvin Roadcap
Route 2
Broadway, Va. 22815

Jack Roth
State Veterinarian's Office
Animal Health & Dairies
Virginia Dept. of Agriculture
Richmond, Va. 23219

O. P. Saffores
The Amburgo Co.
Philadelphia, Pa.

Thomas D. Scanlan
P. O. Box 991
Harrisonburg, Va. 22801

Pete Secrist
Route 5
Harrisonburg, Va. 22801

Lowell Sharpes, Jr.
Route 3
Broadway, Va. 22815

T. M. Shelton
Grassy Knoll Hatchery
P. O. Box 6036
Charlottesville, Va. 22906

Brown Shipe
Route 2
Broadway, Va. 22815

Suresh Singh
170 Suter St.
Harrisonburg, Va. 22801

John Stone
Route 4
Harrisonburg, Va. 22801

Charles O. Strickler
Rocco Feeds
1 Kratzer Road
Harrisonburg, Va. 22801

Eddie Thomas
Route 1
Harrisonburg, Va.

John Turner
1 Kratzer Road
Harrisonburg, Va. 22801

Winston Turner
1097 Park Road
Harrisonburg, Va. 22801

Harry P. VanKrey
Poultry Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

Bill Weaver
Poultry Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

Lewis Wesley
Poultry Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

R. E. Whetzel
Route 2
Linville, Va. 22834

H. P. Williams
Eastern Shore Labs
P. O. Box 657
Laurel, Delaware 19956

E. L. Wiseman
Poultry Science Dept.
V.P.I. & S.U.
Blacksburg, Va. 24061

Hal Yacowitz
The Amburgo Co.
Philadelphia, Pa.

Christon Zirkle
Route 1
Mt. Jackson, Va. 22842