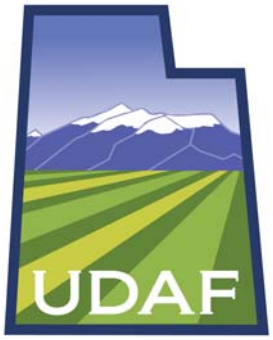

P.O. Box 134
Green River, UT 84525

UtahState UNIVERSITY | COOPERATIVE extension

UTAH GAME BIRD BIOSECURITY AND MANAGEMENT SYMPOSIUM

Green River, Utah
March 5-6, 2009

SPECIAL ACKNOWLEDGEMENT

We are proud to extend a very special thanks to Cargill for their generous contribution to enhance the quality of this meeting.

To all the folks of Cargill:

THANK YOU!

TABLE OF CONTENTS

UTAH GAME BIRD
BIOSECURITY AND MANAGEMENT SYMPOSIUM

March 5-6, 2009
Green River, Utah

March 5 (Thursday)

8:00 – 8:15 a.m.
INTRODUCTION & WELCOME . . . . . *Royd Hatt*, Hatt's Ranch
Bruce King, Assistant State Veterinarian

HATCHERY MANAGEMENT
8:15 – 9:00 a.m.
Incubation/hatching optimization
Egg breakout (if available) . . . . . *Vern Christensen*
North Carolina State University (retired)

ANATOMY, DISEASE, AND DIAGNOSIS
9:00 – 9:45 a.m.
Post mortem examination . . . . . *David Frame*
Utah State University Extension

Eva Wallner-Pendleton
Pennsylvania State University

9:45 – 10: a.m.. . . . . Break

10:00 – 10:30 a.m.
Utah Avian Influenza
Surveillance Plan . . . . . *Bruce King*
Assistant State Veterinarian

10:30 – 11:45 a.m.
Disease management. . . . . *Eva Wallner-Pendleton*
(Emphasis on cholera and coccidiosis) Pennsylvania State University

11:45 – 12:00 p.m.
Laboratory Services, Utah Veterinary
Diagnostic Laboratory System. . . . . *David Frame*
Utah State University Extension

12:00 – 1:30 p.m.. . . . . Lunch

NUTRITION
1:30 – 1:45 p.m.
Feeding for optimal performance. . . . . *Jed Diamond*
Diamond Ranches
1:45 – 2:45 p.m.
Mycotoxins and feed costs . . . . . *Don Giesting*
Cargill Animal Nutrition

2:45 – 3:00 p.m.. . . . . Break

MANAGEMENT
3:00 – 3:45 p.m.
Game bird management. . . . . *Royd Hatt*
Hatt's Ranch

3:45 – 5:00 p.m.
Game bird management. . . . . *Bill MacFarlane*
MacFarlane Pheasants, Inc.,
Janesville, WI

DINNER. . . . . TBA
6:00 p.m.

****

March 6 (Friday)
9:00 a.m. to 12:00 p.m. . . . . Tour of Hatt's Ranch (breeder farm, hatchery, and hunt club)

UTAH GAMEBIRD
BIOSECURITY AND MANAGEMENT SYMPOSIUM

Speaker Biographical Information

Vern L. Christensen
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Dr. Vern L. Christensen received his B.A. from Utah State University in 1971, subsequently earning the M.S. degree from Brigham Young University in 1975 and his Ph.D. from University of Missouri in 1978. His areas of professional interest and expertise are in the embryology, physiology, metabolism, and fertility of avian species. Specific interests focus in molecular events that affect the fertilization and embryo viability of domestic turkeys. For nearly 30 years, Dr. Christensen has been a faculty member at North Carolina State University. He has published over 150 refereed journal publications on the physiology and survival of turkey embryos.

Jed Diamond
Diamond Ranches

Jed Diamond manages a pheasant hunting operation in Syracuse, Utah as part of Diamond Ranches, where he and his father raise cattle and hay and they also guide hunts for elk and deer on a CWMU in the fall. Jed has worked with Hatt Ranch and Cargill in developing his pheasant raising and feeding program. Jed graduated from Clearfield High School where he was active in the FFA program. Jed was recently married and making the transition from bachelorhood.

David D. Frame
Utah State University Extension
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A 1980 graduate of Utah State University with a B.S. in Animal Science, Dr. Frame subsequently received his DVM degree from Oregon State/Washington State Universities in 1984. Dr. Frame completed an avian medicine residency with the University of California, Davis specializing in poultry pathology and diagnostics. He is board certified in the American College of Poultry Veterinarians. Dr. Frame was employed as chief veterinarian for Moroni Feed Company for almost 12 years before joining the faculty of the USU Animal, Dairy, and Veterinary Sciences Department in 1998 as an Associate Professor. He currently serves as the USU Extension Poultry Specialist with an additional assignment as poultry diagnostician for the Utah Veterinary Diagnostic Laboratory. Dr. Frame married Lisa Gilbert of Fairview, Idaho in 1979 and they are the parents of two girls and two boys (none of whom are interested in pursuing a career in veterinary medicine!).

Don Giesting
Business Development Manager
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Dr. Don Giesting is a native of Indiana. He grew up on a mixed agriculture farm in the eastern part of the state. His educational trail included stops in West Lafayette, IN,

where he obtained a B.S. in Animal Science from Purdue University, and Urbana-Champaign, IL where he earned an M.S. and Ph. D. in Swine Nutrition at the University of Illinois.

Don spent three years working in product and technology development for swine and calves at Central Soya Company, before joining Cargill Animal Nutrition (CAN) in 1990. For eight years, he developed and deployed swine products for CAN. In 1998, Don moved to CAN's animal nutrition laboratory, where we led evaluation of ingredients from around the world, analytical and *in vitro* method development, and formulation enhancement deployment, across all species.

In 2001, Don joined Cargill Feed Applications as solutions development manager, researching non-antibiotic feed additive products and tools for a variety of species. He worked with technologies that protect or enhance the value of nutrients for livestock and companion animals, including organic acids, microbials, enzymes, and mycotoxin binders.

In 2003, Don re-joined CAN as a technology manager for the Promote brand, continuing efforts to improve nutrient value through development and application of performance ingredients. In 2006, Don became a business development manager for Promote, and became the business leader for the Promote brand in 2007. Today, the Promote brand includes a range of products to protect and enhance nutrients in forages, feed ingredients, and animal nutrition programs.

Don lives in Minnetonka, Minnesota with his wife, Kate; children, Graham and Anna; and dog, Cornell.

Royd Hatt
Hatt's Ranch
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Royd Hatt is a lifelong resident of Emery County, Utah having been born and raised in Green River. He and his wife, Toni, are the parents of four children. Royd and his family are owners of Hatt's Ranch game bird ranch. Royd has served as president, vice president, and board member of the North American Game Bird Association.

Bruce L. King
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Dr. Bruce L. King was raised in Antimony, Utah on a cattle ranch. He is the third of ten children. He graduated from Piute High School in 1970 and married Valene Oyler in 1975. They have four children, two girls and two boys. After graduating from Colorado State University in 1981 with a Doctors Degree in Veterinary Medicine, he practiced with Dr. Thomas Anderson in Gunnison, Utah for 16 years in primarily a food animal practice. In 1988 he went to work for the Utah Department of Agriculture and Food as a field veterinarian. Dr. King's current position is the Assistant State Veterinarian with an office in the Branch Veterinary Diagnostic Laboratory in Nephi, Utah.

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Owner of MacFarlane Pheasants, Inc. located in Janesville, Wisconsin, Bill has a B.S. in Economics from the University of Houston (1979) and is past president of the North American Gamebird Association. He is the original importer of Manchurian pheasants from China (1989). MacFarlane Pheasants, Inc. hatched over 1.8 million

pheasant and partridge chicks during the 2008 season. They are a nationwide distributor of dressed pheasants (175,000 produced in 2008) and anticipate a sale of 300,000 adult pheasants 80,000 adult partridges this season to hunting preserves and for release.

Eva Wallner-Pendleton
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Dr. Eva Wallner-Pendleton received her DVM in 1979. She completed a residency in avian medicine at the University of California, Davis from 1980-82. From 1982-1989 she served as an avian pathologist at Oregon State University. She completed a Masters project studying fowl cholera and minored in Poultry Science. She spent the next 12 years at the University of Nebraska as an avian pathologist and cooperative extension specialist. She is currently working at the Animal Diagnostic Laboratory at Penn State University. She has always had an interest in diseases of upland game birds and works extensively with production facilities and hunting preserves in Pennsylvania. She has been a featured speaker on game bird health throughout the US. She currently serves as advisor to the North American Gamebird Association and the Pennsylvania gamebird industry on issues of gamebird health and environment.

BASIC CONCEPTS OF GOOD INCUBATOR OPERATION: WHAT CAN THE EMBRYO TELL US?

V. L. Christensen, G. S. Davis, and M. J. Wineland
North Carolina State University

EGG HANDLING PRIOR TO INCUBATION.

The main objective of a good egg handling system is to **control/eliminate harmful organisms** that may be on the eggshell surface, prevent the egg from being **contaminated** through handling and provide the **proper humidity** and **temperature control** for maximum hatchability.

SANITIZING EGGS:

The egg comes in contact with many sources of possible contamination before it gets to the hatchery. Organisms such as ***Salmonella arizona***, other ***Salmonellae,*** **coliforms,** ***Pseudomonas***, etc., may be found in fecal material, floor litter, airborne dust and nest litter. When an egg comes in contact with any of these materials, the eggshell surface is instantly contaminated. Organisms get inside the egg through thousands of tiny pores in the shell as a result of the pressure. **The pressure differential that occurs when the temperature of the interior of the egg cools down is necessary to sanitize the egg to kill bacteria on the eggshell surface before the egg cools.**

EGG HANDLING AND SANITATION PROCEDURES.

Egg hatching quality depends upon the rate of carbon dioxide loss from an egg. Any management practice that reduces carbon dioxide loss from eggs, such as decreased temperature, increased humidity or packaging will also improve hatchability.

Management Practices.

1) Collection.

a. Collect eggs hourly with hens being pushed off the nest at each collection. This will minimize shell-surface contamination and discourage broodiness. An egg in the nest may be broken if a second hen enters the nest to lay.
b. Collect nest eggs and floor eggs separately and discard floor eggs.

2) Disinfection.

a. Disinfect settable eggs immediately after collection (within 60 minutes after being laid).
b. Disinfect either by washing with a quaternary ammonia solution or by fumigation. If the washing method is used, take care to avoid removing the cuticle. A fixing agent such as formaldehyde may

be used in the wash solution to ensure cuticle integrity. This will also help reduce bacterial or fungal growth through pores in the shell. Check washing solution for bacterial or fungal contamination twice weekly.

3) Temperature

a. After disinfection, cool eggs immediately.
b. Maintain the temperature in the egg rooms and egg storage room at 60°F. If the eggs are to be stored more than 4 to 5 days, room temperature should be 55°F. The egg storage room should have a relative humidity of 75%.
c. Maintain egg-handling and storage rooms in a sanitary condition in order to prevent contaminating eggs after washing. Immediately remove residue from broken eggs and disinfect the floor. Wash and disinfect floors and walls of the egg-handling room at the end of each day. Clean and disinfect floors and walls of the holding room weekly or following shipment of eggs to the hatchery.

4) Problems

a. Hairline cracks, which are not visible to the eye, will result in embryonic mortality. Avoid this type crack by using an adequate amount of a soft-nesting material. Collecting no more than 40 to 50 eggs per basket will also help reduce cracks. To ensure this number, use baskets, which will hold no more that 40 to 50 eggs.
b. Mold development in hatching eggs is seen frequently as a problem, particularly during warm weather. Check nesting material, change it often, and keep it dry.

5) Transportation

a. When transporting hatching eggs from farm to hatchery considers the distance, temperature and humidity as part of the management practices. Keep the temperature and relative humidity in the transport van nearly the same as in the egg-holding room. Otherwise, if egg temperature rises, "sweating" or moisture condensation on the shell surface will occur. Moisture on the shell surface will lead to bacterial or fungal contamination inside the egg.
b. The most important point to consider is control of embryonic development during transport. The embryos should be maintained in an undeveloped

state. Embryo development should cease immediately after the egg is laid and not start again until the incubation process is begun.

MAINTAINING HATCHING QUALITY:

Embryonic development has already started in an egg at the time of lay. If the egg is cooled too rapidly and embryo growth (cell division) is stopped before the egg is 6 hours old, the embryo has very little chance to develop. It is likely to be weak, and it may be destroyed easily. Embryo growth should continue for another 6-8 hours after being laid to develop a good, strong embryo that will withstand the stress of shipping and storage. **A temperature of 75-80°F during this period is ideal**. Embryo growth must be stopped by the time the egg is 18 hours old. If embryo growth is not stopped, an over-developed embryo will result, and it may die prior to start of incubation. In dry air, the evaporation of moisture through the pores of the egg is faster than it is in moist air. If the egg loses too much moisture, the thick albumen can start to break down, the egg's air cell can become too large and embryonic mortality can be increased. Pouts that do get out of the shell are likely to be of poor quality. **Relative humidity should be maintained at 75-80% in order to slow the loss of moisture due to evaporation.** If eggs are to be shipped or **stored longer than 5-6 days, relative humidity of 85-95% is recommended.**

CONCEPT OF PHYSIOLOGICAL ZERO:

The concept that a temperature exists at which all embryonic development ceases has existed in poultry science since the 1930's. Early data suggested the temperature was between **65 and 70°F** but more recent data suggest **50 to 55°F**. Eggs stored for 2 to 3 days probably hatch best when stored at the higher storage temperature while those stored for longer than 7 days probably hatch better when stored at the lower storage temperature. Most commercial farms operate their egg storage rooms at approximately 65°F. The concept of a physiological zero is still a subject of much research. As new DNA technologies become available; the storage temperature conditions for eggs will undoubtedly be refined.

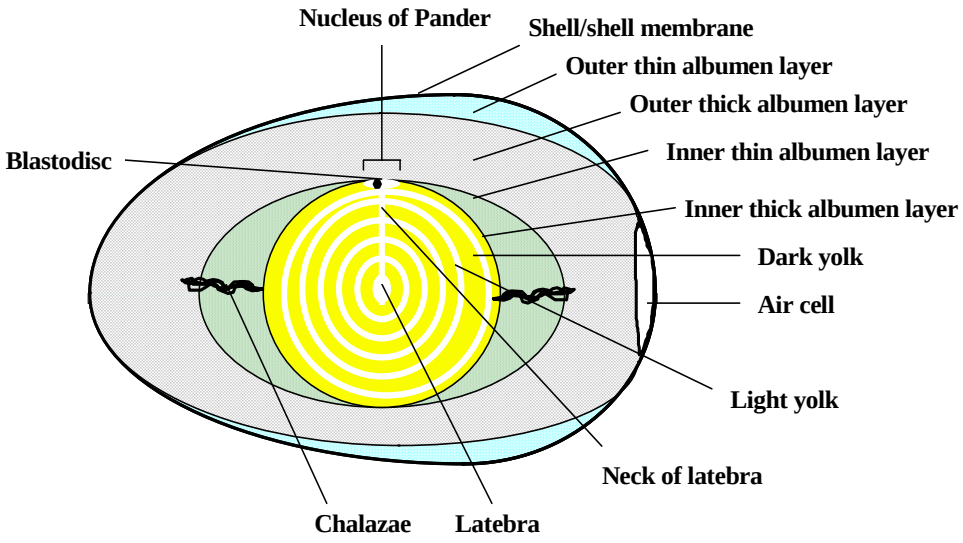
When storage temperatures change, it is important to remember that the relative humidity in the egg storage room must be changed simultaneously. A storage room relative humidity of about **70%** is recommended and is most easily measured using a wet bulb thermometer. For example:

Wet bulb temperature	Dry bulb temperature	Relative Humidity
65-68°F	60-62°F	75%

POSITION OF THE EGG DURING STORAGE:

Hatching eggs are normally stored **with the blunt egg in a vertical position**. In wild species the eggs are stored with the blunt end of the egg in a **horizontal position**. It is not known how the hen accomplishes egg storage of about 12 days in the wild. When similar attempts are made to duplicate horizontal storage in situations of artificial incubation, the eggs do not hatch well. The following examples illustrate some of these physiological concepts involved in egg position during storage.

1) When eggs are stored with **the blunt end in a vertical position**, the yolk will remain next to the air cell. The yolk weighs less than the albumen so it is floating in the egg white. Because of the viscosity of albumen, it requires a long period of time before the yolk will actually float to the top of the egg. This amount of time may be critical to embryo survival during storage because we know that **if the embryo adheres to the membranes**, it dies.
2) When eggs are stored with **the pointed end in the vertical position,** the yolk remains in the center of the egg. It would seem that this would enhance embryonic survival, but **this position does not enhance embryo survival.**
3) When eggs are stored with the **blunt end of the egg in a horizontal position**, the yolk will gravitate toward the top of the egg and eventually will make contact with the eggshell membranes. **This position results in more embryos adhering to eggshell membranes and more embryonic deaths.**



CHALAZAE.**The chalazae are twisted cords of thick albumen that lie at each end of the yolk in the long axis of the egg.** The chalazae at the narrow (pointed) end of the egg consist of two cords while the chalaza at the blunt end of the egg is composed of one cord. At the outer ends of each chalaza the cords merge with the thick

albumen, and serve to stabilize the yolk in the center of the egg; limited rotation of the yolk is possible, however, the chalazae serve to prevent lateral movement. The chalazae stabilize the yolk within the field of gravity.

LATEBRA. **The center of the yolk has a hole which protrudes halfway through called the latebra (see Figure 1)**. The embryo rests on top of the latebra. Because the yolk is not a solid mass, the heavier end of the yolk will always be at the bottom of the egg in the field of gravity. This serves to stabilize the embryo in a vertical direction. The chalazae serve to stabilize the yolk within the albumen laterally. During egg storage it is important to stabilize the yolk with its embryo both vertically and laterally.

It is recommended that eggs be placed with the blunt end in a vertical position during storage. If the eggs are stored in this position for longer periods of time (longer than 7 days), it requires daily turning during storage to prevent embryos from adhering to the eggshell membranes covering the air cell. **Daily turning enhances the survival rates of embryos when eggs are stored for more than 10 days.**

CARBON DIOXIDE AND EGG STORAGE

The loss of carbon dioxide from fertilized eggs results in the loss of embryonic viability eggs during storage. Carbon dioxide is lost by diffusion from eggs from the very moment of oviposition. **Carbonic anhydrase** is an enzyme that is involved in metabolizing carbon dioxide in physiological systems. Carbonic anhydrase is involved in the following reaction:

$$\underset{\text{Bicarbonate}}{HCO_3^-} + H^+ \xleftrightarrow{\text{Dissociation}} \underset{\text{Carbonic Acid}}{HCO_3} \xleftrightarrow{\text{Carbonic Anhydrase}} CO_2 + H_20$$

In the egg, carbonic anhydrase is present in the epithelium of the chorioallantoic membrane (CAM). Carbonic anhydrase is responsible for local acidification of the CAM which allows for the solubilizing of the eggshell to release calcium and other minerals that can then be transported to the embryo. The equilibrium of the chemical equation is reversible. When carbon dioxide accumulates within an egg, the contents become acidic. When carbon dioxide is lost from the egg, the egg contents become more basic.

Within the albumen is a protein containing two different protein molecules, ***i.e.,*** ovomucin and lysozyme. Ovomucin is present in small amounts in egg albumen and contributes to the gelatinous properties of the egg white. Lysozyme is an enzyme that scavenges bacteria within the egg. In the egg at oviposition, ovomucin is bound to lysozyme. As pH and temperature increase, the two protein molecules are cleaved and as they separate, the albumen thins. This concept is presented below:

$$\text{Ovomucin-Lysozyme} \xrightarrow[\text{pH}]{\text{Temp}} \text{Ovomucin + Lysozyme}$$

This thinning allows the yolk to float more freely within the albumen and requires that eggs be turned more often to avoid loss of embryonic viability during storage. **When carbon dioxide loss from eggs is slowed, then the breakdown of ovomucin and lysozyme is slowed and egg storage may be facilitated**.

EGG PROCESSING FACILITY:

A three-stage egg house system is used by most breeder farms and consists of a sanitizing room, a "clean" overnight room and an egg holding room. Floors, ceilings and walls in these rooms should be smooth, cleanable surfaces that can withstand repeated washes with disinfectant solutions.

Sanitizing room. The egg-sanitizing machine used by most of the industry is the Aquamagic. An air intake should be provided for the machine from a clean air source outside of the sanitizing room in order to prevent contamination of the cleaned eggs. An air exhaust system should be provided to remove humidity and fumes from the dryer and from the sanitizing room. The airflow goes from the "overnight" or "clean" room and out the sanitizing room. The machine should be monitored constantly for correct water temperature **(110-130°F)** and disinfectant levels (use manufacturer's recommendations). The machine should be sprayed out after every gathering and should be taken apart and thoroughly cleaned at the end of each day. **Eggs with fecal or muddy material on the shell should not be processed through the machine and must be discarded.** The floor should be kept clean at all times and mopped with disinfectant solution daily.

"Clean" Overnight Room. The Aquamagic egg sanitizing machines are mounted through the wall between the sanitizing room and the sterile overnight room. Sanitized eggs are discharged from the end of the machine directly into the "clean" overnight room. Eggs are placed in flats to cool down overnight and are cased the next morning and moved to the egg holding room.

The "clean" overnight room is totally isolated from the sanitizing room and fully insulated for proper temperature and humidity control. In order to maintain the proper temperatures between 65°F and 75°F, this room must be equipped with a system, which can heat and cool and a thermometer. In order to maintain the proper relative humidity of 75-80%, a humidifier and a humidity indicator are required. A sink for washing hands and a foot dip pan should be placed at the entrance to this room. Access to this room should be limited to the person handling sanitized eggs, and this person should was his hands and put on a clean smock prior to handling eggs. A temperature of 75°F during the day and 65°F during the night should be maintained. The floor must be kept clean at all times and mopped daily with disinfectant solution.

Personnel entering this room must wash and disinfect their hands prior to entering.

Egg Holding Room. This room should be fully insulated with a cooler capable of maintaining a temperature of 58°F and a humidifier capable of maintaining a minimum of 75% relative humidity with accurate thermometer and humidity indicators. There is a connecting door between the holding room and the "clean" overnight room with access limited to the person

operating the "clean" overnight room. This room should have an access door to the outside, with a foot dip pan. For egg pick up, the truck driver picking up the eggs does not enter the room.

Only cased eggs and a three-day supply of cases and flats are stored in this room, so they can absorb moisture. The floor in this room should be mopped daily with disinfectant.

Egg Packing Material Storage Area. Only egg packing materials are kept in this room. No other supplies or trash are permitted. Access to this room is limited to the person operating the "clean" overnight room with all other access doors remaining closed.

ARTIFICIAL INCUBATION OF EGGS

The final destination of the properly managed fertilized egg is the incubator within the hatchery. Modern-day hatcheries generally have egg-setting capacities thousands of eggs. Fertilized eggs have different developmental period depending upon the species so the scheduling of egg settings and removal of hatchlings requires careful planning and monitoring of incubation conditions. Sanitation is imperative in commercial hatcheries as well because conditions for bacterial, mold and viral growth are ideal under incubation conditions. In fact, human vaccine manufacture is often accomplished using fertile poultry eggs. To accomplish successful incubation, an understanding of the basic principles of incubation is essential.

Five principles of artificial incubation are essential for successful hatching of eggs. These factors are valid as well as for the incubation of almost any species of birds. The five principles are **Temperature; Humidity; Turning: Ventilation; and Light/Sound**. Each of these environmental factors must be controlled to successfully develop a blastoderm into a hatchling.

Explanation of each of the five principles

Temperature. When discussing incubation temperature it is important to differentiate the type of incubating machine used. Incubators can be classified based on ventilation and how eggs are set. Ventilation can be by **Still-air** or **Forced-draft. Single-stage** incubators are machines designed to incubate one setting of eggs through to hatching. **Multi- stage incubators** are engineered to contain eggs of multiple ages. Eggs become **exothermic** approximately midway between setting and hatching (approximately 14 days). Energy can be saved if **endothermic** (eggs less than 14 days of setting) are incubated with eggs older than 14 days. Heat from the older eggs is used to provide incubation temperatures for the younger eggs. Temperature requirements differ for each type of incubator.

1) **Still-air Machines.** It is recommended that the incubation temperature for still-air machines be 100.5°F the first week of incubation, 101.5°F the second week, 102.5°F the third and 103°F the fourth week (Insko and Martin, 1936).

   Reducing temperature 2 to 3°F and increasing humidity to a wet bulb temperature of 90°F (70% RH) increased hatchability of eggs (Krueger, 1966).

A suitable alternative may be simply maintaining a constant incubation temperature of 102°F throughout the entire incubation period.

4) **Forced-draft Machines.** Each manufacturer will have specific directions for their machine. It is generally recommended that incubation temperatures be approximately 99.5°F for the initial 25 days of the incubation period.

3) **Critical Range for Incubation Temperatures.** Embryos have either a nonexistent or a very weak thermoregulatory ability. The zone of thermoneutrality is very narrow for an embryo. Therefore, incubation temperatures must be controlled very closely to avoid physiological aberrations and/or death. The **critical temperature for incubating eggs is between 96.0°F and 103.0°F.** Out side of this range is the LD 50(Lethal dose for 50% of the animals). It is important in operating incubators that the temperature of the machine not be less than 96°F nor greater than 103°F. Operating at these temperatures for short periods of time will not harm the embryo, but operation for extended periods may be fatal.

Humidity. The physiological role of humidity in incubation is only partially understood. It is easy to see how **dehydration** may affect embryonic growth because all life must develop in water. The role that **over hydration** plays in incubation and embryonic survival is less easily understood. Experience has proven that a relative **humidity of 54% inside the incubation cabinet is ideal** for embryonic survival. However, incubator humidity must be increased during the final few days of incubation to prevent dehydration during the actual hatching process. If humidity is not increased during the final few hours of hatching, many embryos will stick to the shell because the residual albumen held in the albumen sack will act as glue if desiccated.

1) **Dehydration**. At oviposition a fertilized egg is approximately two/thirds water. In order for optimal growth to occur during incubation, the relative amount of water in an egg must remain at approximately 66%. Water vapor is lost from an egg at incubation temperatures through tiny pores in the shell. Collectively, these pores account for less than 1% of the total surface area of the egg, but they play a very important role in embryonic growth. As development proceeds **water vapor from the embryo diffuses through these pores** into the incubator environment. **Diffusion of gases** is dependent upon a gradient. Water vapor will flow in the direction of the smaller gradient. The rate of flow can be controlled by the size of the gradient (the ratio of the water vapor pressure inside the shell to the water vapor pressure outside of the shell). In hatcheries we deal with water loss as a percentage of weight lost from the egg at the time of setting until hatching begins by breaking the shell. **The ideal percentage water loss for most eggs is 11.5 to 12% of the initial egg mass**. This water loss has been shown to result in an embryo at hatching which is 66% water. Hatchlings with 66% water are not dehydrated.

2) **Over hydration.** It is more difficult to understand the physiology underlying the inability of an embryo to grow when there is too much water in an egg than when there is not enough. The lipids in the yolk provide more than 90% of the energy for embryonic growth. Lipid metabolism creates water as a by-product. If no water were lost from an egg, the relative water content of an egg would increase during development because of the metabolic water formed during energy metabolism. It is thought that the embryo may drown metabolically. It could not drown in the usual sense of the word because the lung of the bird is the shell and its underlying chorioallantoic membrane. For optimal survival rates, water must be lost at a rate that maintains the relative water content of the egg at 66%. **As stated previously, the ideal percentage weight loss for most eggs to fulfill this requirement is again 11.5 to 12.0%.**

Suggestions for Controlling Incubation Humidity:

1) Game bird eggs are more sensitive to water loss than are chicken.

2) 30 to 35 eggs should be marked with a soft-lead pencil and observed throughout an incubation cycle. The following table shows the values that should be seen at different incubation stages.

Day of Incubation	Water loss (%)	Ideal loss (%)	Loss from a 90 g egg
6	**2 to 3**	**2.5**	**2.25 g**
12	**4.1 to 6**	**5.0**	**4.50 g**
18	**6.2 to 9**	**7.5**	**6.75 g**
24	**9.0 to 12**	**10.0**	**9.00 g**

3) Alternative means of monitoring egg weight loss is by observing the air cell of incubating eggs. Excessively large or small air cells indicate problems.

4) **AS A GENERAL RULE, INCUBATE AT 54 PERCENT RELATIVE HUMIDITY UNTIL 24 DAYS, THEN USE 70 PERCENT UNTIL HATCHING.**

5) If additional humidity is needed, wet the incubator floor, place pans of water in the incubators, place burlap wicks in the pans already there. In general, one needs to increase the surface area available for evaporation.

6) **TO REDUCE HUMIDITY, INCREASE THE VENTILATION.**

Ventilation. Proper atmospheric concentrations of oxygen and carbon dioxide are a subject of considerable debate. Basically, the fairest statement is that critical concentrations are unknown. However, it is known that embryos are sensitive to both too little and too much oxygen and carbon dioxide.

1) **Oxygen.** Based on limited research, there are some generalities that can be made about oxygen in the incubator. Normal atmospheric concentrations of oxygen are 20.9% of the environmental air. In scientific terms it makes more sense to discuss oxygen as a partial pressure because barometric pressure can vary from location to location. If we assume that 760 mm of mercury (mm Hg) is the standard condition, then normal oxygen partial pressure will be .209 X 760 = 159 mm Hg. We know that **oxygen toxicity occurs at partial pressure greater than 190 mm Hg and less than 114 mm Hg**. If we assume that most hatcheries operate at about 760 mm Hg then these values **correspond to 25% and 15% of the atmosphere**.

   Oxygen concentrations are maintained in incubators by fans and vents. The fans are controlled by thermostats rather than by an oxygen sensor. Oxygen sensors are expensive and unreliable under incubation conditions. Therefore, it necessitates that the hatchery manager controls the vent openings manually. The proper vent opening may need to be determined by trial and error within each hatchery. Portable oxygen sensors and generators are available and many hatcheries use them to monitor oxygen concentrations -- especially during the actual hatching process. The vent openings can then be adjusted to optimize the oxygen concentrations based on actual atmospheric oxygen measurements.

2) **Carbon dioxide.** We know less about proper carbon dioxide concentrations than we do about oxygen. Carbon dioxide is essential to the development of embryos because normal development does not occur in the absence of carbon dioxide. We also know that carbon dioxide probably has upper and lower limits just as oxygen does. As mentioned above with oxygen, the concentrations of carbon dioxide are also expressed as percentages of the total atmosphere. If we are incubating at 760 mm Hg, then normal atmospheric concentration of carbon dioxide is .0025 X 760 = 2 mm Hg. **The lower toxic limit for carbon dioxide is about .4 mm Hg whereas the upper toxic limit for carbon dioxide is about 4 mm Hg**. Less than 760 mm Hg, these values **correspond to .05% and .5% of the total atmosphere**.

   If carbon dioxide is totally absent from the atmosphere, then normal blood acid/base balance does not occur before hatching and the egg pipping process is delayed. If too much carbon dioxide is found in the incubation environment, then the embryo may die immediately or the shell pipping process may be accelerated.

3) **Oxygen and Carbon Dioxide Concentrations at Different Times during Incubation.** Times of morphologic stages during the embryonic development period are characterized by great sensitivity to carbon dioxide and oxygen whereas others are characterized by almost a totally lack of sensitivity. The most sensitive periods in the development of a embryo are 1 to 6 days of incubation and 21 to 28 days of incubation.

Suggestions for Controlling Respiratory Gases.

1. At both critical times of incubation, less than 15% oxygen (114 mm Hg at 760 mm Hg of barometric pressure) is toxic. Similarly, at both stages 1.5% carbon dioxide (4 mm Hg at 760 mm Hg of barometric pressure) is toxic.

2. At all other times of incubation, embryos can tolerate less than 15% oxygen and up to 7% carbon dioxide.

3. **DO NOT OPEN THE INCUBATOR DOOR MORE THAN NECESSARY WHEN THE DEVELOPING EGGS ARE AT THE CRITICAL STAGES.**

4. Ventilation should be measured carefully when embryos are at the critical stages of development. Newer model incubators now have carbon dioxide and oxygen monitors on them.

5. Ventilation plays and important but undefined role at the final stages of incubation. This effect is probably best demonstrated by the following data depicting survival of embryos in eggs with drilled holes:

Day of Incubation	Hatchability (%)
13	89
14	89.5
15	90.4
16	93.5
17	87.6
18	85.1
19	84.9

Turning. Funk (1934) was probably the first to note the beneficial effects of turning eggs during artificial incubation. **Most eggs are generally turned every three hours day and night.** However, if the automatic turning equipment fails, manual turning at least five times a day is a very satisfactory. **Fertilized eggs should not remain in the same position for longer than 8 hours during the critical developmental time periods (1 to 9 days of incubation).**

1) **Physiology of Egg Turning.** It was demonstrated many years ago that if eggs were not turned, **the embryo would actually adhere to the shell membranes**. It is not known if the adherence kills the embryo but it certainly does not do it any good. It is also known that if eggs are not turned, the fluids from the albumen will penetrate the inner and outer shell membranes of the shell and **prevent the formation of chorioallantoic blood vessels** that serve as the lung of the developing embryo. One can easily visualize an unturned egg with an egg candling light by observing the lack of blood vessel development around the

shell. A third suggested cause of death in unturned eggs is that **the nutrients in an egg become stratified** in the absence of turning. That would render nutrients unavailable to the embryos and possibly cause death.

2) **Frequency of Turning.** Most eggs need turning during only the first 30% of incubation period. After that time turning does not seem essential to embryonic survival. **The minimum amount of turning is two times per day** whereas there **does not seem to be a limit to the number of times an egg can be turned before it affects embryonic survival.**

3) **Dimensions of Turning.** Most eggs are set in the vertical position in egg incubators. How far must an egg be turned and in what planes does it need to be turned? Research indicates that **eggs turned between 30 and 60 degrees in a one-dimensional plane hatch best. Most commercial incubators turn eggs about 45 degrees in a one-dimensional plane.**

Light and Sound. The use of light and sound in incubation are relatively new developments. Because they are so new, there aren't many management guidelines available. Lauber (1961) was the first to observe effects of light in incubators. She was observing the effect of light on the formation of the embryonic eye and noted that the **eggs exposed to the light began hatching nearly 24 hours earlier** than those in total darkness. It is now a common observation that light accelerates embryonic development, but there have been no consistent reports on a beneficial effect on hatchability. Transferring incubating eggs from a dark incubator into a lighted incubator at 24 days of incubation resulted in accelerated hatching and an improvement in hatchability, but exposing incubating eggs to light for the entire incubation period did not.

Sound has similar effects on hatching rates (Sandusky, 1994). The effect of **playing music had a synchronizing effect on hatching times.** The hatchlings seemed to emerge from the shell very close to the same time. More research is needed to confirm this very preliminary report.

Both light and sound may be factors that can be used in the future in our artificial incubation of eggs. However, currently most of our concern should be focused on the previously discussed factors of temperature, humidity, ventilation and turning.

PHYSIOLOGY OF THE EGG DURING INCUBATION

Vern L. Christensen

The technical expertise to control incubation equipment has far exceeded our knowledge of the biological requirements for embryonic growth and survival. Hatchery managers should have a rudimentary understanding of biological bases on which to make judgments for optimal incubator operation. The following section describes some of the biological requirements for embryonic development.

Preparation for Embryonic Development

Although we usually think of embryonic development as an accepted phenomenon, there are many events that are unknown to the general public even to many who operate commercial hatcheries. The size and scope of the commercial hatching business has placed less emphasis on the biology of avian embryonic development and more on efficiencies of scale and the engineering of systems to incubate large numbers of eggs using a minimal amount of labor. The purpose of the next few pages is to acquaint the student of incubation with a brief physiological sketch of the components of an egg and their role in embryonic development.

Figure 1 shows each of the structures that will be discussed so the reader is advised to consult the Figure when reading the physiological function of each component. Some of these structures were discussed previously in the Section on Egg Storage.

Latebra

The latebra is a small portion of "white yolk" in the center of the ovum that is the remainder of white yolk formed in the early stages of ovum development prior to deposition of the pigmented yellow yolk (Burley and Vadehra, 1989). This yolk is less viscous and has a greater proportion of protein to lipid (Burley and Vadehra, 1989). The latebra is connected, via the neck of the latebra, to the periphery of the ovum which is in contact with the blastoderm the **nucleus of Pander**. **This white yolk provides the first nutrients for the blastoderm prior to formation of the embryonic membrane structures (Burley and Vadehra, 1989).**

Chalazae

The chalazae are twisted chords of thick albumen, which lie at each end of the yolk in the long axis of the egg. The chalaza at the narrow (pointed) end of the egg consists of two chords while the chalaza at the blunt end of the egg is composed of one chord (Burley and Vadehra, 1989). At the outer ends of each chalaza the chords merge with the thick albumen, **and serve to stabilize the yolk in the center of the egg; limited rotation of the yolk is possible, however, the chalazae serve to prevent lateral movement** (Burley and Vadehra, 1989). The twisted nature of the chalazae is due to the rotation of the ovum as it travels down the reproductive tract; as albumen is deposited the chords are twisted.

Vitelline membrane

Initially this membrane surrounds the yolk and maintains an osmotic gradient to keep the yolk and albumen nutrients separated. As the embryo ages, **the membrane begins to disintegrate and allow the protein from the albumen to be intermingled with the lipids from the yolk thus providing a balanced diet for growth for the embryo.** Eventually, the yolk sac membrane replaces the vitelline membrane. The yolk sac membrane is not a true biological membrane. It develops from a single layer of cells that extend from the area vasculosa (inner portion of the area opaca). This highly vascularized membrane is fully formed by the ninth day of incubation and encloses the yolk. The inner surface of the yolk sac membrane is highly convoluted which allows maximum contact with the yolk. **One yolk sac membrane function is to break down and absorb yolk for the developing embryo.**

Egg White (Albumen)

The albumen provides several physiological functions during incubation. **It contributes water and solutes, which enter the yolk, and provides albumen protein, which is absorbed via the yolk sac during the last week of incubation. In addition, the early embryo (Blastoderm) uses glucose from the albumen as an energy source. The albumen has an antimicrobial role, which is provided by one of the proteins of which it is composed. Antibacterial proteins inhibit bacterial growth in different ways. Ovotransferrin chelates iron and avidin binds biotin which make these substances unavailable for use by microorganisms. In addition, there are several bacterial protease inhibitors; ovomucoid, ovoinhibitor, cystatin, and ovomacroglobulin. Ovomucin in albumen is a known antiviral agent. Lysozyme**, a bacteriophage, is also present in the albumen. This protein will be discussed in detail later.

Inner and Outer Shell Membranes

The outer shell membrane is thicker than the inner shell membrane (Burley and Vadehra, 1989). The outer shell membrane is joined to the shell by fibers that pass into the mammillary cores of the shell, while the inner and outer shell membranes are joined by fibers that pass from the inner to the outer membrane (Burley and Vadehra, 1989). The **inner shell membrane** also makes contact with the chorioallantoic membrane and **assists in calcium movement** from the shell to the chorioallantoic membrane. **At the large end of the egg the inner and outer shell membranes separate to form the Air Cell, which provides the embryo with its first breath of oxygen during pipping.** The **outer shell membrane** serves as the **lung of the embryo** early in development by resisting the diffusion of respiratory gases and water vapor early in embryonic development.

Shell

The shell provides three important functions for the embryo. First, it protects the embryo from the outside environment of the incubator. It has some insulation value and resists the loss of heat when an incubator door is left open or when a hen is away from the nest. It provides a cushion from shock. If an egg is dropped

or tipped unnecessarily the embryo could become damaged if not contained in a shell. It is also a major shield against bacterial invasion of the stored nutrients. Secondly, the shell **provides important nutrients to the embryo**. It is the major source of calcium and magnesium for the growing tissue mass. Lastly, the **shell is the lung of the embryo**. Tiny microscopic pores that resist or facilitate the loss of carbon dioxide and water vapor from the embryo and the competing aim of providing oxygen to the developing organism cover the shell. All respiration by the shell is accomplished totally by diffusion, *i.e.*, the embryo is totally dependent upon the number and geometry of pores to supply respiratory gases, it cannot increase its respiration rate.

Yolk

Yolk is actually formed in globules. These globules are so named because they are unaffected by the concentration of salt or urea which dissolve or disrupt other structures in yolk (Burley and Vadehra, 1989). However, as their lipid composition is similar to that of yolk low-density lipoprotein (with the only difference being a higher proportion of phospholipid), it may be that these globules supply energy for the growing embryo. There are two types of yolk based on the amount of yellow pigment they contain. Yellow yolk differs from white yolk in two ways; first, it contains pigments from the diet (carotenoids) which give it the yellow color, and second, it contains small particles (yolk low density lipoprotein) not present in white yolk (Burley and Vadehra, 1989). **The function of yellow yolk during incubation is to provide lipids and proteins to the embryo**. Both lipids and proteins are absorbed via the yolk-sac membrane then transferred as an energy source to the embryo. Proteins (and carbohydrates) are the main energy source during the first two-thirds of incubation while lipid metabolism is most intense during the last 7 days of incubation of the embryo when growth is rapid. Surface layers of yolk are formed during the final rapid phase of yolk formation while the ovum is still on the ovary. The surface layers consist primarily of yellow yolk so this type of yolk would be available to the embryo before the white yolk would be.

Water

The egg is composed of 83 to 84% water in altricial species (young hatch helpless) and 72 to 75% in precocial species (young are more independent at hatching). Approximately 12 to 15% of water is lost by the egg is via diffusion. **Water in the egg influences the egg temperature**, the hatching process and the weight of the hatched chick Eggs of small species of birds tend to have proportionally more water in their eggs than other birds. These small birds, which have higher metabolic demands than larger birds are able to leave the eggs unincubated for longer periods of time during trips for food, without the eggs cooling appreciably. Distribution of water in egg components varies through the egg depending on the period of incubation. Water is an important factor involved in **osmotic balance** of the egg components and the embryo. **Water also serves as a solvent for many biochemicals**.

Carbonic Anhydrase

Carbonic anhydrase is an enzyme that is involved in the following reaction:

$$\underset{\text{Bicarbonate}}{HCO_3^-} + H^+ \xleftrightarrow{\text{Dissociation}} \underset{\text{Carbonic Acid}}{HCO_3} \xleftrightarrow{\text{Carbonic Anhydrase}} CO_2 + H_20$$

In the egg, carbonic anhydrase is present in the epithelium of the chorioallantoic membrane (CAM). Carbonic Anhydrase is also found in the albumen of the egg. **Carbonic anhydrase is responsible for local acidification of the CAM which allows solubilizing of the eggshell** (Calcium carbonate) to release calcium and other minerals which can then be transported to the embryo (Narbaitz, 1987). In embryos that have developed primitive and definitive red blood cells, **carbonic anhydrase also serves to regulate carbon dioxide transport and pH** (Tuan, 1987).

Subgerminal fluid

Subgerminal fluid is aptly named, as it is the fluid that is contained in a cavity beneath the early avian embryo **(blastoderm)**. **Subgerminal fluid contains glucose, sodium, potassium, protein and water, which bathe the embryo**. Later in incubation, amniotic and allantoic fluids replace this fluid.

Extra Embryonic Structures During Embryogenesis

Very early in development, membranes emanating from the embryo grow to surround the embryo and or yolk. Figure 2 depicts these structures. There are four of them and each has a unique physiological function. The student of embryology and incubation should have at least a basic knowledge of these structures. This knowledge will aid in identifying incubation problems.

1. Chorion

The sole function of all of the structures is to protect the embryo and provide for its nutritive, excretory or respiratory needs. The chorion is a layer of cells that fuses with the allantois (the bladder of the embryo) very early in incubation. The structure formed is called the chorioallantoic **membrane and forms the circulatory system around the periphery or the egg and also serves as the site of respiratory gas exchange or as the "lung" of the embryo**.

2. Amnion

This layer of cells forms a fluid-filled sac that surrounds the embryo proper. It has three functions:

1. **Protects the embryo from drying.**
2. **Forms a fluid cushion for the embryo.**
3. **Forms an isolated chamber where growth and positional changes can occur easily.**

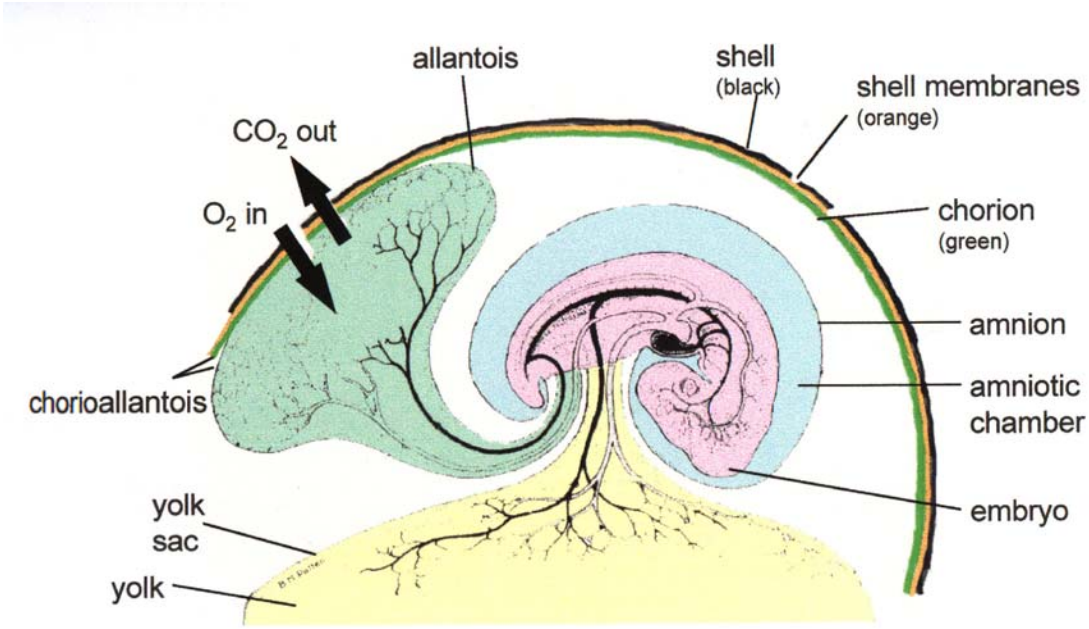
3. Yolk sac

The yolk sac membrane fuses with the chorioallantoic membrane during Embryogenesis. It lies between the food supply, the yolk, and the circulatory system to carry nutrients to the growing tissues. **It contains digestive enzymes and possesses the ability to "digest" food**.

4. Allantois

The allantois is a membrane growing from the yolkstalk of the embryo. It has two general physiological functions:

1. **It participates in the exchange of oxygen and carbon dioxide between outside air and the embryo. It is the embryonic lung.**
2. **It forms a sac in which nitrogenous wastes (Urea, the main breakdown product of protein) are stored. Urea is formed only during the early development stages and is water-soluble. Later in development the embryo forms uric acid which is an insoluble solid and is deposited near the allantoic sac as a white powdery material.**



Adapted from Patten, 1951

HATCHERY RELATED PROBLEMS

Vern L. Christensen

Watch for These Ten Hatchery-Related Problems

Don't blame your hatchery man for excessive early mortality - at least not always. The mortality could be the fault of poor brooder house management.

Having said that, however, there are 10 points for growers to watch for in hatchlings - hatchery related factors that could give producers problems with their poults. They are:

1. **Dehydrated chicks:** They generally result from low humidity during incubation, and early hatch or excessively long holding periods in boxes before delivery. Dehydrated chicks can be identified upon delivery at the farm by **examining the shanks and feeling the chicks**. If a fairly large number of chicks have shriveled legs or shanks and bodies feel hard and look angular, give the birds extra good care the first week. The chicks should have "easy" access to water and feed and they should be kept comfortable. Do not chill or overheat the chicks in the brooder house. **Always check a sample of chicks (50 to 100 at random) upon delivery to determine the state of dehydration and then brood accordingly.**

2. **Weak chicks:** Weak chicks do not have to be dehydrated chicks, and often are not. Weak chicks usually result from higher than recommended temperatures during hatching, inadequate ventilation in the hatchers, over-fumigation at hatching time, infection, rough sexing or setting old eggs. Weak chicks can be identified easily by pressing down on the chicks in the boxes with the palm of the hand. If the chicks are strong, they will offer considerable resistance to the pressure of the hand; if they are weak, they can be pushed down easily. **With a little practice (the touch of the master) you can detect weak chicks upon delivery. Weak chicks need better than average brooder house care.**

3. **Large, soft-bodied chicks:** Large, sluggish chicks usually are the result of high humidity during incubation and hatching. They often have a heavy abdomen and feel soft and full of moisture to the touch. They generally ship better when transported long distances. **These poults usually present no serious brooding problems except that they appear sluggish.**

4. **Rough navels:** The navels of chicks always should be checked upon delivery to the farm. A rough or open navel makes the chick more susceptible to infections. Rough or unhealed navels result when the hatch is late (more than 28 days), incubation temperature has been variable and high, or when excessively high humidity was used during hatching. **Chicks with rough navels upon delivery probably should receive a broad-spectrum antibiotic in the feed or water for the first week to minimize the possibility of infection and morbidity.**

5. **Omphalitis (navel infection):** Omphalitis is the result of filth in the hatchers and/or contaminated chick boxes and chick box pads. *E. Coli, Pseudomonas, Proteus,* or occasionally a *Staphylococcus* usually causes it. Sometimes the yolk sac is involved in addition to the navel. Yolk sac contents change from a yellow-green material to a caseous material or to a yellow-brown watery material when contaminated with *E. coli.* The navel opening often has an offensive odor. **Mortality and morbidity will be high with a high percentage of runts among the surviving poults.**

   An Omphalitis infection means that the hatchery must change its clean-up and egg and hatchery sanitation programs immediately. **A broad-spectrum antibiotic or a nitrofuran may help reduce morbidity and the percentage of runts.** The type of organism involved and drug resistance will affect the type of response one gets to treatment.

6. **Chick delivery.** Errors in the chick delivery system can injure potentially strong, healthy chicks. Damage can occur in several ways; namely, overheating in the delivery van, chilling in the van, poor van ventilation resulting in overheating, chilling, or CO_2 poisoning. Assuming good judgement in programming the load, the driver becomes the "key" to a successful delivery of undamaged chicks. Some truck drivers have no feel for the product being delivered. **The salvage process at the brooder house consists of ample feed and water, and a comfortable brooding temperature, along with a tremendous amount of care and attention.** The amount of loss depends on the damage done to the chicks in transit, and the amount of care given them during brooding.

7. **Improper toe trimming**, wing clipping, snood removal, poultry injection and rough handling during sexing: Quality control and sanitation are the greatest problems a hatchery has with these operations - getting the hatchery personnel to do their jobs properly and uniformly. Improper wing clipping or toe trimming can leave a chronic sore. Many years ago poultrymen recommended wing clipping or toe trimming through a joint taking into account the proper angle, and using a modified beak trimming unit to do the job.

   The injection site and injection process should be kept as sterile as possible. Chicks should be injected according to directions - meaning proper equipment and needles, recommended dosage of antibiotic mixture, injection in the upper portion of the neck and subcutaneously. If the antibiotic mixture is of the type that puts the chicks to sleep, delivery should be delayed until **all** chicks have recovered from the injection.

   There is little that farm management can do with chicks that have been improperly processed except to talk to hatching management.

8. **Chick grading.** All malformed, straddle-legged, and weak chicks should be culled at the hatchery before delivery. A high percentage of abnormal chicks will die or be morbid. Most hatcheries do a good job of removing the abnormal chicks.

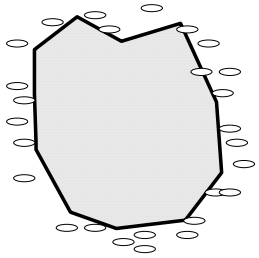
9. **Nutritional deficiencies.** Breeder rations that are marginal in certain vitamins and/or trace minerals can result in hatched chicks that are weak and marginal in vitamin and/or mineral reserves. Those chicks should be fed a prestarter well fortified with vitamins and minerals. Often when young breeder hens have been on a poorly vitamin and mineral-fortified holding ration, the first two or three hatches of chicks will not start and live as well the first week of brooding as later hatches.

Some of the vitamins and minerals which could be deficient in the breeder ration and which could reflect themselves in the young poult are E, K, riboflavin, biotin, folic acid, pantothenic acid and B_{12}. Some of the minerals would be iodine, potassium, manganese and cobalt. Check to see whether the breeder hens are receiving an adequate diet, if not, feed a prestarter. **A prestarter will have more protein, higher vitamin and mineral fortification and higher levels of growth promotant than the regular starter.**

10. **Irregular-sized chicks:** Irregular-sized chicks result from different age of breeder flocks and age size, variations in incubator temperature and humidity. **If the chicks are from a healthy flock, there is little worry since hatching weight is poorly correlated with market weight.**

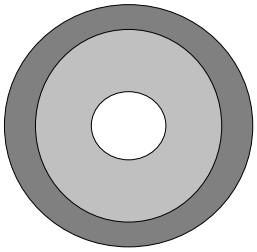
DETERMINING FERTILITY IN AN UNINCUBATED EGG WITH THE UNAIDED EYE

Infertile



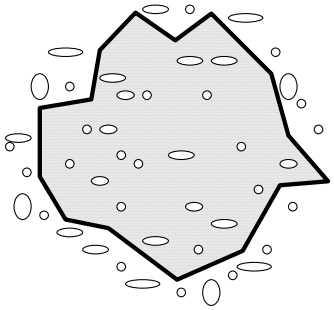
a. The germ spot is a solid white color.
b. The germ spot is relatively small (1/16th")
c. Many vacuoles ("bubbles" or "holes") around the edge of the germ. Sometimes vacuoles are present inside the germ spot.
d. The germ spot is not uniform. It is somewhat circular but has "jagged" or "ruffled" edges.

2. **Fertile**



a. Germ spot is faint. Not a solid white spot. Consists of a faint ring. Germ spot may have a faint or solid white disk in the center of the ring.
b. Germ spot is about 2 times larger than the infertile germ (1/8th").
c. Usually no vacuoles visible in the center of the ring. May be a few on the edge of the ring.
d. Germ spot is very uniform. Is circular with no "jagged" edges.

3. **Early Dead Embryo**



a. Germ spot is a mixture of solid and faint white areas.
b. Germ spot is about the same size as the fertile germ.
c. Usually many vacuoles visible on the edges and in the center of the germ.
d. Germ spot is not uniform. Has irregular edges and is usually not circular in shape.

****<u>NOTE</u>: It is very difficult to distinguish between infertile and very early dead embryos; thus, it is not truly reliable.**

PROCEDURE FOR THE EXAMINATION OF CULL AND DEAD GAME BIRDS (POSTMORTEM EXAMINATION OR NECROPSY)

David D. Frame, DVM, Dipl. ACPV
Utah State University Extension Poultry Specialist

1. Examine outside of birds for parasites, scratches, animal bites, and other signs of injury.
2. Using heavy scissors or tin snips, cut off upper beak and look at sinuses. Gently squeeze sides of beak and check for excess mucus.
3. Using the snips, cut along side of mouth and open the throat to level of where neck enters the body cavity. Observe esophagus for lumps, bumps, atypically colored or textured areas, ulcers, or severe reddening.
4. Cut open the windpipe (trachea) lengthwise and look for excess bloody content, yellowish cheesy material, or excess mucus.
5. Cut open the crop and look at contents. Is it the normal feed? Is it sour-smelling or dry and impacted? Remove contents and look at the crop lining. Is it thickened? Are there reddened or raised areas present?
6. Peel back the skin and feathers from the breast and check for areas of injury, paucity of meat (i.e. starvation), uniformly dry and dark muscles (i.e. dehydration), or presence of tumors.
7. Using a knife, cut down the inner side of each leg until the hip joint is reached. Pull the legs away from body and twist sideways to expose the hip joint. Break one of the leg bones to test the bone strength. Is it soft and rubbery? Does it feel brittle and break too easily?
8. Using the snips, cut away skin and muscle along the rear underside of breast bone to expose the internal organs. Continue cutting along each side of the body cavity until the ribs have been cut in two. Gently raise the breast bone away from the internal organs. Remove the breast by snipping through the bones near the shoulder area, thus exposing the internal organs.
9. Look for cloudy air sacs or cheesy material in the body cavity.
10. Examine heart, liver, lungs, and spleen. Is the heart or liver covered with white or yellowish granular or cheesy material? Is the spleen larger than normal? Are the lungs bright orange-red and spongy, or are they dark red-brown, hard, or compacted?
11. Look at the outer surface of the intestines. Are nodules or areas of hemorrhage present? Slit open the intestines lengthwise and look for worms, bloody content, excess mucus, thickened lining, or other abnormalities.
12. Open up the proventriculus (stomach) and look for pinpoint red lesions, thickening, or enlargement.
13. Slit open the gizzard. Examine contents. Peel away the inner lining and look for reddened or hemorrhagic areas underneath.
14. Examine other areas of the body cavity for abnormalities.
15. Make notes of any abnormalities encountered. If you think your game birds have a problem, contact your area diagnostic laboratory or the USU Extension Poultry Specialist, Dr. David Frame at (435) 283-7586.

Figure 1. External examination of mortality. Examine for parasites, scratches, animal bites, and other signs of injury.

Figure 2. External examination of mortality. Examine eyes, beak, nostrils for unusual growths (i.e. nodules or crusty material) or excess mucus.

Figure 3. External examination of mortality. The application of soapy water helps mat the feathers, keeping them from interfering with the subsequent postmortem examination. Wetting the feathers in this manner also reduces the likelihood of aerosolizing disease-causing agents.

Figure 4. Breast exposed after pulling away skin and feathers. Notice that cuts have been made on the inside of the thighs and the left hip joint has been dislocated by slightly twisting the leg in order to expose the femoral head.

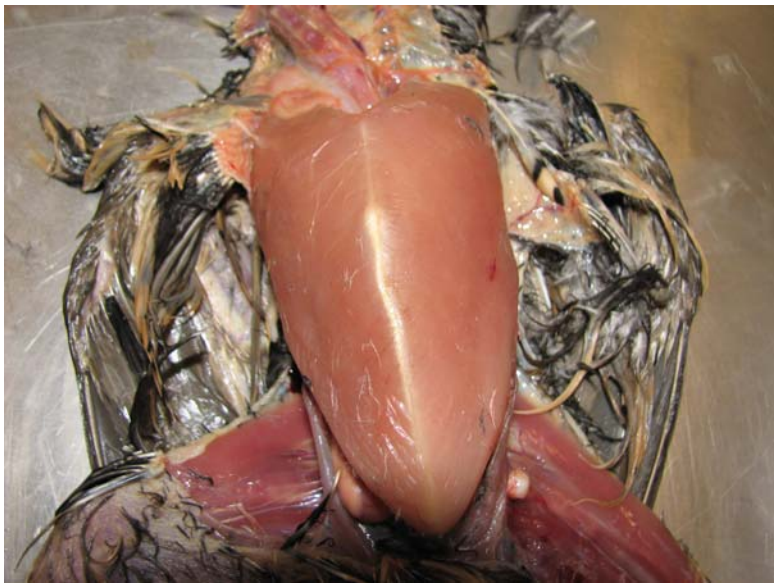


Figure 5. Internal organs in situ. (After removal of the breast.)

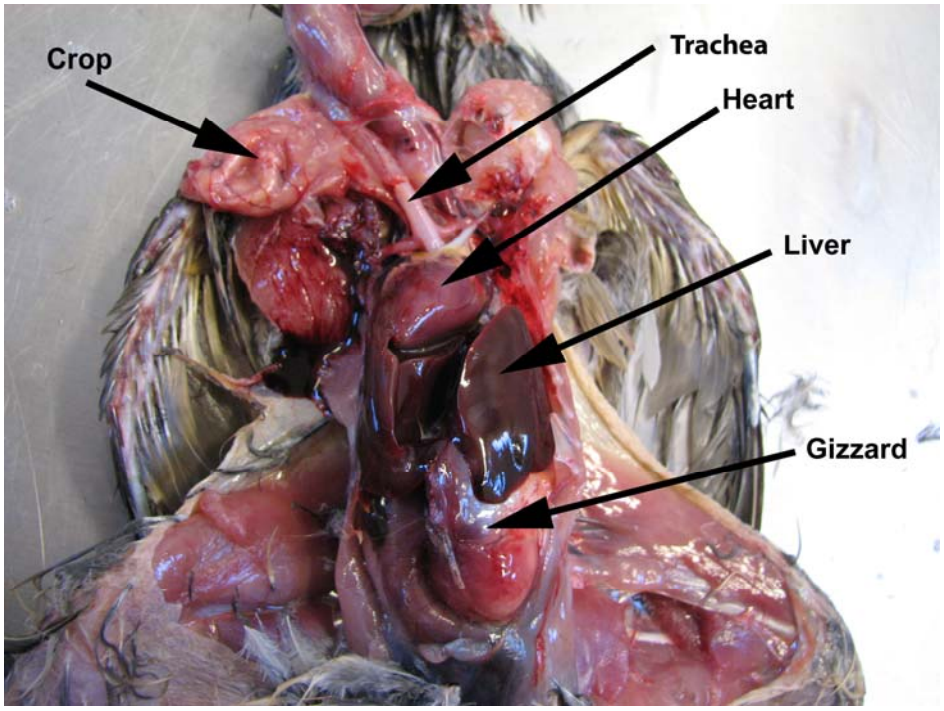
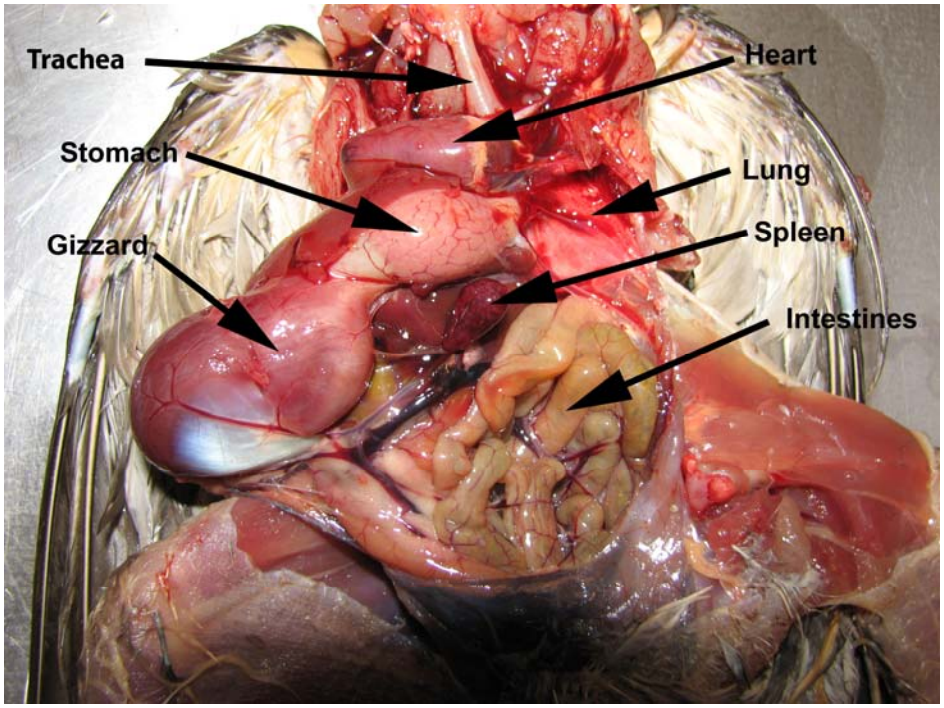


Figure 6. Internal organs in situ. (After lifting away the gizzard and liver.)



UTAH
H5/H7 LPAI AVIAN INFLUENZA SURVEILLANCE, RESPONSE, AND CONTAINMENT PLAN

I. JUSTIFICATION

(A) Avian influenza (AI) is a disease of increasing worldwide importance with growing implications as a human disease threat.
(B) The potential for low pathogenic (LP) varieties of AI to mutate to highly pathogenic (HP) strains, affecting domestic poultry, is significant.
(C) Additional driving forces for a national H5/H7 avian influenza control plan consist of:

1. The World Organization for Animal Health (OIE) has defined notifiable avian influenza (NAI) as described in Article 2.7.12.1:

For the purposes of the *Terrestrial Code*, avian influenza in its notifiable form (NAI) is defined as an infection of poultry caused by any influenza A virus of the H5 or H7 subtypes or by any AI virus with an intravenous pathogenicity index (IVPI) greater than 1.2 (or as an alternative at least 75% mortality) as described below. NAI viruses can be divided into highly pathogenic notifiable avian influenza (HPNAI) and low pathogenicity notifiable avian influenza (LPNAI):

a. HPNAI viruses have an IVPI in 6-week-old chickens greater than 1.2 or, as an alternative, cause at least 75% mortality in 4-to 8-week-old chickens infected intravenously. H5 and H7 viruses which do not have an IVPI of greater than 1.2 or cause less than 75% mortality in an intravenous lethality test should be sequenced to determine whether multiple basic amino acids are present at the cleavage site of the hemagglutinin molecule (HA0); if the amino acid motif is similar to that observed for other HPNAI isolates, the isolate being tested should be considered as HPNAI;
b. LPNAI are all influenza A viruses of H5 and H7 subtype that are not HPNAI viruses.

to include not just highly pathogenic, but all H5/H7 viruses including low pathogenic strains; and
2. Influence of international markets on the economic viability of our nation's poultry industry dictates it is in the best interests of government, human health, and the poultry industry to prevent and control all H5/H7 avian influenza virus infections.

(D) H5/H7 avian influenza is a disease reportable to the State Veterinarian's office by all licensed or otherwise legally practicing veterinarians in the State and all laboratories.
(E) Under a nationally-sanctioned plan, USDA-APHIS would be involved at the request of the Cooperating Official State Agency (Utah Department of Agriculture and Food, specifically, the State Veterinarian's Office) in the cost and control efforts of an outbreak. This Voluntary Cooperative State–Federal Program to control and eradicate H5/H7 avian influenza infections of commercial poultry is state-based and coordinated. In the event of an outbreak of H5/H7 LPAI, the Cooperating Official State Agency is eligible for payment of 100% of the costs of surveillance and monitoring and 100% of the costs of vaccine administration, as determined by this cooperative agreement. This agreement will be made between the Cooperating Official State Agency and APHIS.
(F) The Utah Avian Influenza Surveillance, Response and Containment Plan (i.e., "Plan") will provide for stakeholder input and participation, establishment of passive and active surveillance programs, and planning for a coordinated, pre-planned response in the event of an AI outbreak in the state. The goal is to provide a level of assurance to poultry producers and trading partners and an adequate level of surveillance and response preparedness for government.

II. AVIAN INFLUENZA ADVISORY COMMITTEE

The formation of a state advisory committee has been requested by industry in order to bring all stakeholders such as Utah Department of Agriculture and Food (UDAF), USDA-APHIS, Utah Department of Health, and various producer groups together to provide Plan input. The formation of

this committee is the first essential element to development of a state AI response plan. It is agreed that the AI Advisory Committee in Utah will be comprised of the following participants:
(1) Director of Animal Industry or representative
(2) State Veterinarian
(3) USDA-APHIS Area Veterinarian in Charge (AVIC) or representative
(4) Utah Department of Health representative
(5) Utah Veterinary Diagnostic Laboratory representative
(6) Utah State University Extension
(7) NPIP Contact representative
(8) Utah Egg Producer's representative and/or consultant
(9) Utah Turkey Marketing Board representative
(10) Game bird representative (pheasant, chukar, quail, etc.)
(11) Utah Fancy Poultry Association/Pigeon Breeders representative
(12) Utah Division of Wildlife Resources representative
(13) Department of Environmental Quality representative
This committee will convene once a year to review the State's AI emergency disease management status. Training in emergency disease management, biosecurity, diagnostics, and other relevant topics including exercises in handling different scenarios of outbreaks of H5/H7 AI will be given by appropriate personnel at this annual meeting.

III. MONITORING PLAN

(A) The State will maintain "U. S. H5/H7 Avian Influenza Monitored State" status under the National Poultry Improvement Plan (NPIP) program for avian influenza (see Appendix #1). In furtherance of that requirement the following surveillance activities will be implemented: (B) All commercial producers where LPAI is detected will establish a written flock management agreement developed by APHIS and the State Veterinarian with input from the flock owner and other affected parties. Under this definition, a flock plan sets out the steps to be taken to eradicate H5/H7 LPAI from a positive flock, or to prevent introduction of H5/H7 LPAI into another flock. A flock plan shall include, but is not necessarily limited to, poultry and poultry product movement and geographically appropriate infected and control/monitoring zones. Control measures in the flock plan should include detailed plans for safe handling of conveyances, containers, and other associated materials that could serve as fomites; disposal of flocks; cleaning and disinfection; downtime; and repopulation. Each producer should also sign a *Memorandum of Understanding* (MOU) to support the diagnostic and active surveillance programs by timely submission of appropriate specimens.
(C) The Extension veterinarian (or appointee) will institute an ongoing avian influenza awareness program for all legally practicing veterinarians and pre-outbreak outreach programs for poultry producers and Extension agents in the state regarding the importance of prompt reporting of clinical symptoms consistent with avian influenza.
(D) It should be further understood that:
(1) *No liability shall accrue to the UDAF for damages, losses, or injuries incidental to or arising by virtue of participation in this Plan.*
(2) *The flock owner has the responsibility to adopt and implement the biosecurity measures set forth elsewhere in this agreement.*
(3) *The flock owner has the responsibility of maintaining records of flock morbidity, mortality, and production and shall make such records available for review by the UDAF as requested.*
(E) Serology
(1) *Approved Laboratories*
(a) Utah Veterinary Diagnostic Laboratory, Logan, Utah
950 East 100 North, Logan, Utah

NAHLN Laboratory
Contact person: Dr. Tom Baldwin – (435) 797-1895
Tests available: AGID, ELISA, RT-PCR
Test capacity:
AGID: routine = 300/week; surge = 700/week
ELISA: routine = 500/week; surge = 3000/week
RT-PCR: routine = 400/week; surge = 1000/week
(b) Utah Veterinary Diagnostic Laboratory, 1451 South Main, Nephi, Utah
NAHLN Laboratory
Contact person: Dr. Jane Kelly – (435) 623-1402
Tests available: AGID, ELISA, RT-PCR
Test capacity:
AGID: routine = 100/week; surge = 300/week
ELISA: routine = 500/week; surge = 1800/week
(2) *Testing*
(a) Serologic testing will be used as a screening test for monitoring purposes only. Routine screening for type A avian influenza virus in poultry will be performed using either agar gel immunodiffusion (AGID) or enzyme-linked immunosorbent assay (ELISA). The AGID test is to be performed according to the "National Poultry Improvement Plan and Auxiliary Provisions" §147.9[1]; ELISA test is performed using only federally licensed kits and following the manufacturer's instructions.
(b) A premise or flock will be considered suspect positive if serologic testing reveals antigenic exposure to Type A influenza virus.
(c) A premise or flock will be considered positive only when live (H5/H7) influenza virus is isolated or its presence detected by polymerase chain reaction (PCR) testing.
(d) Positive test results of any type will be reported immediately to the State Veterinarian, as required by Rule in R58-2-2. In the case of a positive serologic test, the State Veterinarian may issue a *Hold Order* and prescribe certain biosecurity measures to be implemented until virus identification is completed and/or assessment of the clinical symptoms exhibited by the flock is made.
i) Hold Order is defined as authority of the State Veterinarian to impose movement restrictions and/or testing requirements appropriate to conditions until a definitive diagnosis or quarantine decision is reached. The Hold Order may involve all or any portion of restrictions defined under Quarantine Measures (see section V) as deemed appropriate to the circumstances by the State Veterinarian.
(3) *Passive Surveillance*
(a) All laboratories that perform diagnostic procedures on avian species (private, State-Federal Cooperative, public health, and university laboratories) will examine all submitted cases of severe, atypical, or otherwise unexplained respiratory disease, gastrointestinal disease, neurologic disease, egg production drops, and high mortality, for avian influenza by both a USDA-approved serologic test and a USDA-approved influenza virus detection test. Results of such testing will be reported quarterly to the State Veterinarian. Positive tests will be reported immediately.
(4) *Active Surveillance*
(a) UDAF in conjunction with USDA, APHIS, VS, Utah Veterinary Diagnostic Laboratory (UVDL), and commercial producer organizations will implement a commercial poultry surveillance program.
(b) A minimum of 11 birds per chicken layer flock are monitored quarterly (90 days) by serologic or other approved tests and reported quarterly to the State Veterinarian. Positive tests will be reported

[1] Laboratory protocol for the AGID test for avian influenza now requires no more than three unknowns be tested in a seven-well format with the center well containing antigen (9 CFR 147.9).

immediately.
(c) A meat-type turkey slaughter plant at which a sample of a minimum of 60 birds be tested each month by USDA-approved serologic protocol (i.e., AGID or ELISA) and reported quarterly to the State Veterinarian. Positive tests will be reported immediately. Plant records shall be maintained for 3 years (9 CFR 146.11.c).

IV. RESPONSE PLAN

The State Veterinarian and the USDA, APHIS, VS Area Veterinarian in Charge (AVIC) will administer an initial containment and control plan developed in conjunction with the Advisory Committee. The Commissioner of Agriculture may request the Governor to declare a state of emergency once state resources have been exhausted or are found inadequate. USDA-APHIS-VS will be invited to develop an Incident Command Center to implement the deployment of necessary state and federal resources to respond to the emergency outbreak.

(A) Definition of H5/H7 LPAI infection

(1) *Isolation of H5/H7 avian influenza virus from poultry*;
(2) *Detection of H5 or H7 subtype AI viral antigen in poultry*; or
(3) *Detection of H5 or H7 subtype AI viral antibodies in poultry that is not a consequence of vaccination*.

(B) Criteria for declaring a premise suspect-positive for AI

(1) *Epidemiologic link with an AI suspect flock or premise. An epidemiologic link is considered to be established if one or more of the following occur(s)*:
(a) Poultry operations employing workers who reside in the same household as person(s) associated with a confirmed AI outbreak elsewhere.
(b) All farms within the same organization that the AI outbreak occurs – particularly if there is sharing of equipment and/or personnel, or if close geographic ties exist.
(c) Poultry farms, companies, or personnel using common facilities or equipment, such as breaker plant, feed mill, egg flats, trucking company, vaccination crews, or other equipment.
(2) *Flock/bird with no clinical signs, no lesions compatible with AI, and no epidemiologic link but AI-positive by one of the following tests*:
(a) AGID
(b) ELISA[2]
(c) Directigen™ Flu A

(C) Criteria for declaring a premise positive for HP/LP AI during a confirmed AI outbreak:

(1) *Flocks/birds showing clinical signs of respiratory disease, sudden unexplained drop in egg production, or lesions consistent with AI (i.e., edema of the head, comb, or wattles; subcutaneous hemorrhage of feet or shanks; hemorrhage/necrosis of comb, wattles, trachea, heart, and/or gut) should be considered suspicious for AI until confirmed or ruled out by appropriate diagnostic tests.*
(2) *Premises inside surveillance zones are considered positive if birds exhibit clinical signs and/or gross lesions consistent with low or highly pathogenic avian influenza virus plus one of the following laboratory tests.*
(a) Isolation and identification of AIV
(b) Positive RT-PCR with H5 or H7 AIV specific primer/probe set
(c) Presence of H5 or H7 AIV subtype-specific serum antibodies
(3) *Premises inside surveillance zones without clinical signs and/or gross lesions, must meet two of the following conditions to be declared positive.*
(a) Directigen-positive (cannot be only criterion to designate a premise as positive even with an

[2] Because of the possibility of false positives using ELISA, all ELISA-positive sera shall be re-tested using AGID.

epidemiologic link)
(b) Isolation and identification of H5 or H7 subtypes of AIV
(c) Positive RT-PCR with H5 or H7 AIV specific primer/probe set
(d) Presence of H5 or H7 AIV subtype-specific serum antibodies
(e) Epidemiologic link. An epidemiologic link is considered to be established if one or more of the following occur(s):
(i) Poultry operations employing workers who reside in the same household as person(s) associated with a confirmed AI outbreak elsewhere. (ii) All farms within the same organization that the AI outbreak occurs – particularly if there is sharing of equipment and/or personnel, or if close geographic ties exist.
(iii) Poultry farms, companies, or personnel using common facilities or equipment, such as breaker plant, feed mill, egg flats, trucking company, vaccination crews, or other equipment.

(D) Initial response plan for an AI-positive flock or premise

(1) *Positive AI serology (AGID or ELISA) without increased mortality or other clinical signs:*
(a) The flock or farm will be placed under Hold Order and strict biosecurity measures will be met. Procedures in Appendix #2 are to be immediately implemented. The State Veterinarian may exercise authority to impose additional measures if needed, such as vehicular traffic restrictions, restrictions of movement of eggs, live birds, dead birds, feed, and other commodities on/off the premises.
(b) All commercial poultry flock owners will be informed of the status of the Hold Order.
(c) Cloacal and pharyngeal swabs from birds in the affected flock shall be sent to UVDL for testing by RT-PCR.
(i) If RT-PCR is positive for either H5 or H7, the UVDL will immediately inform the State Veterinarian and the USDA-APHIS AVIC. Swabs will be forwarded to the National Veterinary Services Laboratories (NVSL) for sub-typing. NVSL will carry out virus isolation for characterization and pathogenicity testing. Initial subtype results should be obtained within 24 to 48 hours. Pathogenicity of the isolate is normally determined within 5 to 20 days.
(2) *Positive AI serology accompanied by significant unexplained on-farm mortality or other clinical signs*
(a) The flock or farm will be quarantined and strict bio-security measures will be met. As a minimum, all procedures in Appendix #2 are to be immediately implemented. The State Veterinarian may exercise authority to impose additional measures if needed.
(b) A quarantine zone around the affected premises will be imposed, and other company-owned or other related flocks will be assessed for risk based on levels of bio-security in place at the time of the outbreak.
(c) Pooled (up to five birds per tube) pharyngeal and cloacal swabs will be collected according to Table 1 and submitted to UVDL for RT-PCR testing.
(d) Tissues (lung, spleen, brains, and/or intestines) from individual dead birds will be sent to UVDL. Positive tissues will be forwarded to NVSL for virus isolation, characterization, and pathogenicity testing. All testing and collection of samples will be performed on-site using appropriate bio-security precautions.

(E) Response following definitive diagnosis of AI (subtype-dependent)

(1) *LPAI subtype H5 or H7*
(a) The flock or farm will be quarantined and strict bio-security measures will be met. As a minimum, all procedures in Appendix #2 are to be immediately implemented. The State Veterinarian may exercise authority to impose additional measures if needed.
(b) Following consultation with USDA and industry parties involved, an infected flock may be depopulated. This depopulation may take place as much as two to four weeks after the initial outbreak in order to reduce the amount of virus spread by infected virus-shedding birds. If there is depopulation then costs would be covered by state/federal indemnity arrangements.

(c) At the discretion of the State Veterinarian and APHIS, poultry that has been infected with or exposed to H5/H7 LPAI may be allowed to move for controlled marketing in accordance with the initial State response and containment plan and in accordance with the following requirements:

(i) Poultry infected with or exposed to H5/H7 LPAI must not be transported to a market for controlled marketing until 21 days after the acute phase of the infection has concluded, as determined by the State Veterinarian;

(ii) Within 7 days prior to slaughter, each flock to be moved for controlled marketing must be tested for H5/H7 LPAI using a test approved by the State Veterinarian and found to be free of the virus;

(iii) Poultry moved for controlled marketing will not be eligible for indemnity.

(d) At the discretion of the State Veterinarian, H5/H7-negative poultry within quarantine zone may be allowed to move for controlled marketing if the flock is tested within 7 days prior to slaughter using an approved test and found to be free of the virus.

(e) Serologic surveillance using sentinel chickens

(i) Place sentinel chickens after infected houses are 100% AI-positive.

(ii) Clearly identify 20 individual birds (and cage locations) per house. Expose some sentinel chickens to the manure pit also.

(iii) Later, place additional sentinels and take blood samples at 4, 8, and 12 weeks post-placement. Then take blood samples every 90 days.

(f) Limited and controlled vaccination of commercial laying hen, turkey breeder, or turkey meat flocks may be used as a method of eradicating the disease, provided that adequate bio-security is in place with approval of the State Veterinarian. (Please refer to Appendix #3 for proposed implementation of vaccination plan.)

(i) Cost of vaccination will be covered by the producer(s) implementing the vaccination program.

(g) Surveillance of all flocks or farms surrounding an infected flock as per section V.A. of this document will be undertaken, and any other company-owned or other epidemiologically related flocks located outside the surveillance zone will also be monitored. Surveillance will be by use of approved serologic tests and/or RT-PCR for H5 or H7 AI.

(i) Cost of testing other company-owned or related flocks will be covered by the producer(s); epidemiologically unrelated surrounding flocks will be tested at state/federal expense.

(ii) Serologic surveillance will continue weekly until the infected flock has been free of active infection for at least 30 days or until all surrounding and epidemiologically linked flocks or farms have been sufficiently tested and found free of active infection for a period of at least 30 days.

(iii) Flock or farm will be tested according to Table 1, taking into consideration special stipulations (if any) contained in the MOU.

(iv) Pooled (up to five birds/tube) pharyngeal and cloacal swabs will be submitted to UVDL for RT-PCR testing. All testing and collection of samples will be performed on-site using appropriate bio-security precautions.

Table 1. Number of birds sampled for serology, PCR, and/or virus isolation on each premise.[a]

No. birds on premise	Minimum number of birds to be sampled
15 or less……	Sample all
16 to 49……..	15
50 or greater……..……	30

[a]Assuming a 95% or greater sensitivity and 99% specificity for the diagnostic testing system used, sampling the indicated number of birds will result in a 95% certainty that at least one positive bird will be detected if at least a 25% prevalence of HPAI virus shedding exists among birds on the premises at the time of sample collection

(2) *Highly Pathogenic Avian Influenza (HPAI)*
(a) The flock or farm will be quarantined and strict bio-security measures will be met.
(b) HPAI is an emergency animal disease and therefore infected flock(s) will be depopulated.
(c) Appraisal of the flock is to occur prior to depopulation as required in Title 4-31-3 of the Utah Code (un-annotated).
(d) Surveillance will be the same as that used for LPAI H5/H7
(3) *LPAI other subtypes (not H5/H7)*
(a) Strict bio-security measures will be maintained throughout the life of the flock.
(b) Control measures to be taken are to be presented in writing by poultry producers to state officials.
(c) Surveillance shall be carried out in adjacent or epidemiologically linked flocks until the infection has been shown to no longer be active. This testing shall be in the form of serology, virus isolation, RT-PCR and/or sentinel birds.
(i) Cost of testing will be covered by the producer(s).
(ii) The producer should be able to take samples, when under the direction of an accredited veterinarian.

V. QUARANTINE MEASURES

Quarantine authority resides with the State Veterinarian, Commissioner of Agriculture, and the Governor. Quarantines on all movement of poultry within, into, and out of one or more of the designated Containment Regions will be imposed by the State Veterinarian upon confirmation of the isolation of live AI virus and subject to approval of the Commissioner of Agriculture. Quarantines will be enforced by Department of Agriculture and Food personnel and local law enforcement officers.

(A) Establishment of surveillance zones

(1) *Three distinct zones, with varying intensities of surveillance will be established*:
(a) *Affected Zone* included the area within 5 miles (8 km) of the index flock;
(b) *Surveillance Zone* including the area between 5 and 10 miles (8 and 16 km) of the index flock;
(c) *Buffer Zone* including the area between 10 and 30 miles (16 and 50 km) of the index flock.
Particular attention to surveillance efforts is to be given to company-owned, other epidemiologically related flocks, common breaker plants, feed mills, vaccination crews, movement of other equipment and personnel, recently purchased breeder stock, or recent participation in exhibition shows or swap meets. These potential epidemiologic links shall be rigorously assessed for risk based on levels of biosecurity in place at the time of the outbreak.
(2) *Affected Zone*
(a) The Affected Zone includes the area within 5 miles (8 km) of the index flock. The target population to include in surveillance will be all commercial and non-commercial poultry operations.
(b) *Commercial poultry operations* (defined as any marketing of poultry or poultry products)
(i) Commercial poultry operations will continue to conduct active serologic surveillance as required by this document and the existing MOU.

(ii) Cloacal and tracheal swabs will be collected by or under the supervision of a USDA-accredited veterinarian from poultry in each house, building, or flock located on the operation. Sick and freshly dead birds are targeted for sampling.

(iii) The total number of birds that will be sampled in each unit is presented in Table 1. Serologic surveillance shall continue weekly for a minimum of 30 days after the last active case of influenza is depopulated, processed, or no longer shedding virus.

(iv) Swab samples will be collected weekly for a minimum of 30 days after the last active case of influenza is depopulated, processed, or no longer shedding virus.

(c) *Non-commercial poultry operations*

(i) An inventory of at-risk non-commercial operations will be developed by UDAF and USDA/APHIS/VS of the affected zone. At-risk operations are defined as those with poultry, waterfowl, pigeons, or ratites.

(ii) All at-risk non-commercial operations will have swab samples collected for RT-PCR testing and/or virus isolation. Both cloacal and tracheal samples will be collected from gallinaceous birds and ratites; only cloacal samples will be collected from waterfowl.

(iii) The total number of birds that will be sampled in each unit is presented in Table 1. Serologic surveillance shall continue weekly for a minimum of 30 days after the last active case of influenza is depopulated, processed, or no longer shedding virus.

(iv) Swab samples for RT-PCR testing and/or virus isolation shall be collected weekly for a minimum of 30 days after the last active case of influenza is depopulated, processed, or no longer shedding virus.

(3) *Surveillance Zone*

(a) The surveillance zone includes the area between 5 and 10 miles (8 and 16 km) of the index flock. The target population to include in surveillance includes all commercial and non-commercial poultry operations. Surveillance is also to include, when considered appropriate, any commercial or non-commercial flocks with epidemiologic links to the index flock that might be located outside of the official surveillance and quarantine zones. Surveillance efforts in this zone will be conducted simultaneously with those in the affected zone.

(b) *Commercial poultry operations*

(i) Testing procedures for commercial poultry operations in the surveillance zone will be identical to those applied in the affected zone.

(ii) Cloacal and tracheal swabs samples for RT-PCR testing and/or virus isolation will be collected from all commercial operations at the initiation of this surveillance plan and again 21 days following initial sampling. The total number of birds to be sampled on each operation is presented in Table 1.

(c) *Non-commercial poultry operations*

(i) Testing procedures for non-commercial poultry operations in the surveillance zone will be identical to those applied in the affected zone.

(ii) Cloacal and tracheal swabs samples for RT-PCR testing and/or virus isolation will be collected from each non-commercial operation at the initiation of this surveillance plan and again 21 days following initial sampling. The total number of birds to be sampled on each operation is presented in Table 1.

(4) *Buffer Zone*

(a) The buffer zone includes the area between 10 and 30 miles (16 and 50 km) from the index flock. Surveillance is also to include, when considered appropriate, any commercial or non-commercial flocks with epidemiologic links to the index flock that might be located outside of the official buffer, surveillance, and affected zones.

(b) An inventory of commercial and non-commercial poultry operations will be developed by UDAF and USDA/APHIS/VS in conjunction with the Advisory Committee. All at-risk non-commercial

poultry operations within 0.3 miles (0.5 km) of commercial poultry operations will be identified, and tracheal or cloacal swab samples collected once for RT-PCR. The total number of birds to be sampled on each operation is presented in Table 1.

(c) Commercial poultry operations will comply with all other requirements as contained in the existing MOU.

(B) Movement and biosecurity

(a) Movement controls will be implemented relating to live birds, dead birds, eggs, egg flats, litter, trucks, equipment, workers, etc., within the affected zone. These orders will remain in place until surveillance is completed.

(b) No avian species may be moved out of or into the quarantined area.

(c) No poultry products or supplies, *excluding properly washed and sanitized and/or monitored for AI: table eggs, egg products, or processed poultry products in properly sanitized containers and vehicles*, may be moved off the affected premises or out of the quarantine area. Eggs going to breaker must be transported in disposable non-returnable flats. Consider having at least a week's storage capacity for breaker eggs that may be prevented from immediately going to the breaker plant.

(d) Trucks or vehicles entering the affected premises must be thoroughly disinfected prior to entering and leaving the affected premises.

(e) If testing within the affected, surveillance, or buffer zone detects any additional positive flocks or farms, the quarantine zone will be extended accordingly.

(f) Only permitted movements will be allowed until surveillance is completed within all three zones.

(g) Industry and regulatory officials may discuss specific details in the event of an outbreak.

(C) Depopulation and disposal

(a) Small populations will be euthanized humanely and disposed by incineration or burial on premise or in an appropriate landfill. Experience dictates that large numbers of birds can overwhelm incinerators and private arrangements for burial or composting. Larger populations will be disposed by burial at an acceptable landfill with attention to transportation routes and disinfection of transport vehicles.

(b) If depopulation is chosen as the preferred method of dealing with an H/LPAI outbreak, the following methods may be used for disposal of the birds.

(i) In-house or approved on site composting may be used for disposal of birds and is the desired method when practical. (Appendix #4)

(ii) Birds that are not able to be composted may be disposed of in an approved landfill in compliance with Department of Environmental Quality requirements. Vehicles transporting carcasses to the landfill (and upon returning) must be covered and sealed to prevent escape of liquid and airborne material, such as blood, feathers, and dander. (See Appendix # 5 for landfill locations, capacity, and availability.) Contact local landfill(s) and find out what it will take to dispose of carcasses in case of a disease outbreak. Do this as soon as possible, before the crisis occurs.

(iii) Smaller flocks may be incinerated using the UDAF T400 Airburner, in compliance with Department of Environmental Quality requirements, if conditions permit.

(D) Euthanasia

Only American Veterinary Medical Association (AVMA) and/or APHIS-approved methods of euthanasia shall be employed.

(E) Premises decontamination and disinfection

(1) *Preparation for cleaning and disinfection*. Following the depopulation or controlled marketing of all poultry infected with or exposed to H5/H7 LPAI on a premises, the following procedures will be completed prior to cleaning and disinfection:

(a) Secure and remove all feathers that might blow around outside the house in which the infected or exposed poultry were held by raking them together and burning the pile;

(b) Apply insecticides and rodenticides immediately after the removal of the birds, before the

house cools;

(c) Close the house in which the poultry were held, maintaining just enough ventilation to remove moisture. Leave the house undisturbed for a minimum of 21 days and for as long as possible thereafter, in order to allow as much H5/H7 LPAI virus as possible to die a natural death.

(d) Heat the house to 100 °F for the 72 hours prior to cleaning and disinfection.

(2) *Cleaning and disinfection*. All premises, conveyances, and materials that came into contact with poultry that were infected with or exposed to H5/H7 LPAI must be cleaned and disinfected. Cleaning and disinfection must be performed on all buildings that came into contact with poultry that were infected with or exposed to H5/H7 LPAI within a premises, including pump houses and service areas. To accomplish cleaning and disinfection, the following procedures should be completed:

(a) *Disposal of manure, debris, and feed*. Clean up all manure, debris, and feed. Compost manure, debris, and feed in the house if possible. If this is not possible, set up a system for hauling manure, debris, and feed to an approved site for burial, piling, or composting. Do not clean out the house or move or spread litter until any H5/H7 LPAI virus that may have contaminated the manure and litter is dead, as determined by the State Veterinarian and in accordance with the initial State response and containment plan. If composting is used as a disposal method, manure and litter should be composted in accordance with State and local regulations. If litter is piled, the litter pile must be covered and allowed to sit undisturbed for an amount of time approved by the State Veterinarian and APHIS and in accordance the initial State response and containment plan. Drying and heat *in situ* over time are effective and may be used in place of composting if weather conditions or conditions in the building are favorable. After use, equipment used to clean out manure, debris, and feed must be washed, disinfected, and inspected at the site to which the manure and litter was transported. In the case of inclement weather, the equipment may be washed, disinfected, and inspected at off-site wash stations at the discretion of the State Veterinarian and APHIS.

(b) *Cleaning of premises and materials*. Manure and all organic material shall be completely removed from infected buildings. If taken to an approved off-site location, it must be transported in a covered and leak-proof container. The sides of the building shall be scraped to remove all residual organic material that might harbor virus. Manure shall be buried or composted on the premises or double bagged and taken to a designated landfill or incinerator according to an approved transportation plan, and taken to an approved off-site location in a covered and leak-proof container. Cleaning and washing should be thorough to ensure that all materials or substances contaminated with H5/H7 LPAI virus, especially manure, dried blood, and other organic materials, are removed from all surfaces. Spray all contaminated surfaces above the floor with soap and water to knock dust down to the floor, using no more water than necessary. Wash equipment and houses with soap and water. Disassemble equipment as required to clean all contaminated surfaces. Special attention should be given to automatic feeders and other closed areas to ensure adequate cleaning. Inspect houses and equipment to ensure that cleaning has removed all contaminated materials or substances and let houses and equipment dry completely before applying disinfectant.

(c) *Disinfection of premises and materials*. When cleaning has been completed and all surfaces are dry, all interior surfaces of the structure should be saturated with an approved disinfectant. U.S. Environmental Protection Agency (EPA)-registered products that have a claim of being effective against influenza viruses should be used. All disinfectants are to be applied according to manufacturer's directions. (See Appendix #6 for EPA-registered disinfectants.)

A power spray unit should be used to spray the disinfectant on all surfaces, making sure that the disinfectant gets into cracks and crevices. Special attention should be given to automatic feeders and other closed areas to ensure adequate disinfection.

(d) *Cleaning and disinfection of conveyances*. Clean and disinfect all trucks and vehicles used in transporting affected poultry or materials before soil dries in place. Both exterior, including the undercarriage, and interior surfaces, including truck cabs, must be cleaned. The interior of the truck

cabs should be washed with clean water and sponged with an approved disinfectant. Manure and litter removed from these vehicles should be and disposed of at an approved site for burial, piling, or composting.

(3) *Activities after cleaning and disinfection.* Premises should be checked for presence of virus before repopulation by PCR testing from drag swabs obtained from equipment, floor, and walls. Placement of sentinel birds may be required before and/or after repopulation at the discretion of the State Veterinarian. If deemed necessary to place sentinel birds before total building repopulation is allowed, 30 clearly identified AI-negative chickens (or turkeys, depending on the facilities) will be placed in the building. If a cage facility, 20 chickens will be dispersed in cages throughout the facilities and 10 chickens will be exposed to the manure pit. Birds will be serologically tested for presence of AI antibodies and PCR-tested for presence of virus weekly for two weeks. The premises may not be restocked with poultry until the disease is deemed to be eradicated by state and federal animal health officials. After repopulation, LPAI surveillance will resume as outlined in Section III (4) (b) and (c).

(F) Biosecurity practices

(1) Ongoing biosecurity practices are to be followed before and after outbreaks according to Appendix #2 and the MOU. Additional guidelines are found at the following web sites:

(a) Commercial turkeys – *Bio-security Principles: Protecting the Utah Turkey Industry*, USU Extension Fact Sheet AG/Poultry Health/Biosecurity/03, http://extension.usu.edu/files/publications/Biosecurity03.pdf

(b) Exhibition poultry producers and exhibitors – *Bio-security Principles: Protecting Your Investment*, USU Extension Fact Sheet AG/Poultry/Health/Biosecurity/01 http://extension.usu.edu/files/agpubs/poulprinciples.pdf

VI. PUBLIC RELATIONS RESPONSE/COMMUNICATION

UDAF and USDA/APHIS/VS will strive to inform the partners of the situation via a contact list including the AI Advisory Committee, Accredited Veterinarians, appropriate industry members, and all signatory parties of an MOU regarding this initial State response and containment plan. (See Appendix #7 for list of contacts.)

(A) Pre-outbreak public awareness activities, to be carried out by Cooperative Extension and UDAF, will be accomplished through appropriate radio and TV communication, fact sheets, group presentations, and web site information: (http://extension.usu.edu/; http://ag.utah.gov/pressrel/AvianFluInfo.html).

(B) All inquiries by public media about serologic findings, outbreaks, or other questions dealing with actual or suspected cases of AI infection in Utah are to be directed to the State Veterinarian's office.

(C) Information released to the public should be timely and include the nature and extent of the emergency; impacted or potentially affected areas of the State; human health implications or lack thereof; and activities being carried out by government officials and industry leaders to respond to the outbreak or mitigate its effects. Released information may also include newspaper inserts or supplements which provide detailed information the public could use, and information about the steps being taken by the state and industry to protect them.

(D) The use of radio and television may include prepared announcements, interviews, question and answer sessions, live footage, and so forth depending on the circumstances.

Appendix #2

MINIMUM BIOSECURITY MEASURES TO PREVENT TRANSMISSION OF AVIAN INFLUENZA

ISOLATION refers to the confinement of animals within a controlled environment.

AIV may be mechanically transmitted by anything that can walk, crawl, or fly from farm to farm.

1. Clean out vegetation around poultry houses to remove shelter and food for possible carriers.
2. Institute a vector control program for insect, mammal and avian vectors. These vectors are important because they can mechanically carry infected feces from one house or premises to another.
   a. Improve barriers to prevent the access of wild birds to poultry houses.
   b. Institute an insect control program. Flies of several species are important in the transfer of AIV.

c. Rodents have implicated in the transfer of AIV. Rodent control and preventing their traffic between houses on a single premises are essential.

3. Prevent the accumulation of standing water. This is a great attraction to migrating waterfowl and shorebirds, both of which have been implicated in AI outbreaks/transmission.
4. Limit sources of food for wild and free-flying birds. Clean up spills when they happen.
5. Do not allow employees to raise their own poultry or attend poultry markets or shows.
6. Freshly laundered clothes for employees should be changed into at the farm. These clothes should be left at the farm at the end of the day.
7. Employees should shower out at the end of the day.
8. The interior of cars/vehicles should be sprayed for flies prior to leaving the premises.
9. Manure and dead birds may not move from the premises unless appropriate bio-security principles are adhered to.

The spread of avian influenza follows the movement of people and equipment.

1. Do not allow movement of people, your employees or other individuals, from your farm to other farms.
2. Conduct business by telephone. Inform other farms of the need for heightened bio-security.
3. Do not let truck drivers, repairmen, or delivery personnel step out onto your facility without new protective foot covering and coveralls. Identify local truck washes that may be used to clean and disinfect vehicles before entering premises and again upon leaving area. Give the locations and addresses to all drivers.
4. Use gates and signs to control traffic.
5. Wash and disinfect all vehicles prior to them leaving the farm. Carry disinfectant spray (e.g. Lysol®), disposable plastic boots, can of fly spray, and alcohol gel hand sanitizer in all company vehicles.
6. Avoid movement of equipment off of the farm. Wash and disinfect prior to equipment leaving the farm.

Appendix #4

PROCEDURE FOR IN-HOUSE COMPOSTING DEAD OR EUTHANIZED BIRDS infected with AIV may be composted in the following manner:

Supplies:

Personal protective equipment (Tyvek® suits, boots, gloves, respirators)
Hand tools (square point long handle shovels, pitchforks, long handle rakes and hoes, stick broom, drill with feeder winch attachment, ladder, hammer, crowbar and cutting pliers.)
Personal needs (toilet facilities, cell phone, food, drinks, paper towels and disinfectant hand wipes.)
Rodenticide and insecticide
Composting thermometers
Carbon source (litter, sawdust, etc.)
Water hose or water supply
Warning signs
Tarp, poly or fleece with anchors
Cleaning & disinfectant supplies, large garbage bags, bucket, brush, hand sprayer
Poly removal supplies (tow rope, fuel source, lighter, disposal approval)

Equipment and Personnel:

Midsize Skid-Steer Loader (~ 1.25 –1.5 Cubic Yard Bucket) and Skilled Operator

Sanitation Equipment:

A high pressure washer must be on site to clean and disinfect equipment and premises.

Composting Procedures:

Layering
Shredding and Piling
Mixing and Piling

Procedure for all methods:

Let birds consume all feed
Raise the feeder and drinker lines

Select Composting Method Based On Depopulation Procedure

If the depopulation procedure concentrates the carcasses in a small section of the house, the layering option may be appropriate. Where carcasses are distributed more evenly over the litter surface, the mix and pile option is recommended.

Layering method:

1. Create a litter windrow that has a 10 to 12 ft. wide base.
2. Scoop the dead birds with the loader and lay them on top of the litter windrow base.
3. Spread the carcasses evenly with a rake or pitchfork until they are about 8 to 10 inches thick.
4. Repeat the layering procedure as needed until the pile is 6 feet high.(If the height of the poultry house prevents a 6 foot high windrow, make only two layers which will be approximately 3 to 4 feet high.)
5. Deposit a 6 to 8 inch layer of litter/sawdust “cap” over the birds with a foot overlap on the sides. *Leave no carcasses or bird parts exposed*.

Shredding and Piling Method:

This method involves shredding carcasses and tilling them into the existing litter base followed by windrowing the mixture. This method may be beneficial for composting large carcasses such as roasters or turkeys. It also does not require the addition of water, as moisture from the shredded carcasses will be adequate to support the composting process.

1. Remove carcasses one bucket-width wide from along the side walls and spread them

evenly in the center of the house.
2. Shred the carcasses using a tiller attached to a skid steer loader or a 3-point hitch, PTO driven unit for farm tractor.
    a) Make at least two passes to ensure adequate shredding
    b) use sharp tines and high rpm
    c) use the best angle and direction of rotation for shredding
3. An alternative to shredding is crushing carcasses with a rubber tire loader.
4. Roll the carcasses into the litter/sawdust windrow.
5. Pile the shredded carcass/litter mixture into a properly shaped windrow (12 to 14 ft. wide and 3 to 5 ft. high). Cap the windrow with litter to cover exposed carcasses.

Mixing and Piling Method:

This method involves mixing carcasses into the existing litter base and forming windrows. (Involves the least time, labor and materials.)
1. Remove carcasses one bucket-width wide from along the sidewall and spread them evenly in the center of the house. (If litter is inadequate and supplemental sawdust is required, this step is not necessary.)
2. Starting with a three-inch minimum litter base, use the feed line as a guide and mix the carcasses with the litter to start the formation of the windrow. Continue to roll the materials from along the sides together to form a windrow 10 –12 feet wide in the center of the house.
3. As with all methods the pile must be covered with a layer of litter or sawdust 4 to 6 inches thick. *All carcasses must be covered!*

Any surplus litter not used in the composting process should also be placed in windrows to inactivate pathogens.

All methods require turning:

1. After 10 to 14 days, the compost temperature will decline. As it drops below ~125^{o}F (52^{o}C), turn the windrows.
2. Turn the windrow inside the house or relocate the stockpile outside, place in shed or windrow and cover it with fleece or poly.
3. Scrape along the edges of the turned windrow and deposit material on the pile.
4. Cap the new windrow with a minimum of four inches of litter or sawdust to cover any exposed tissue on the surface.
5. After turning the compost windrow, the temperature should equal or exceed that in the initial windrow. Monitor and record temperatures.
6. After an additional 2-3 weeks, the compost material may be land-applied.

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PASTEURELLA MULTOCIDA (AVIAN CHOLERA) IN GAME BIRDS

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Historical Points of Interest

- One of four diseases for which Veterinary Division of USDA was created.
- Isolated in 1880 in pure culture by Pasteur (in chicken broth).
- Pasteur used FC organism for classic bacterial attenuation experiments.

Avian Cholera

- This organism can cause many disease syndromes in game birds.
- Caused by a bacterium, *Pasteurella multocida*.
    - Sudden death (acute form)
      Well-fleshed birds, nothing seen outwardly. Owner may suspect trauma death or poisoning. However, death loss may rapidly escalate.
    - Lameness (chronic form).
    - Increase in cull-type birds (chronic form).

How does the disease get into a flock????

- Direct or indirect contact with wild birds, especially waterfowl (pond, stream or other attractant close by) or domestic waterfowl (mallard growers).
- Varmint gets in the pen (skunk, raccoon, cat, dog, mink, or are attracted to dead pile too close to the pens).
- Infected rodents in the pens (holes under feeders, abundant nesting sites). Rodents perpetuate the disease.
- Purchasing older birds from outside that appear recovered from the disease!!
- Returning escapees into the pens.
- Visiting other growers or delivering birds to preserves, then returning to your ranch without C&D of the truck, crates, and showering.

Diagnosis

- Clinical history
- Submitting dead birds to your diagnostic laboratory
- Lesions
    - Preliminary diagnosis
- Bacteriology
    - Cultures of internal organs
    - Results in 24- 48 hours

Treatment

- Sulfonamides (losing efficacy)

- Penicillin (losing efficacy)
- Tetracyclines
- Drug resistant strains becoming very common, especially in upland game birds.
- Success of treatment depends on concurrent diseases, and how long the disease has been present. The longer you wait, the harder it will be to control.
- Long term treatment (begin with water, continue with feed) often necessary as relapses in flocks are common.

Treatment (Work with your veterinarian and extension veterinarian)

- Feed- approved for control of cholera in ducks
    - Chlortetracycline (Aureomycin)
        - 200- 400 g/ton for max 21 days
        - 0 day withdrawal (wd).
    - Sulfadimethoxine and ormetoprim (Rofenaid)
        - "outbreaks"- 454 g sulfa., 272.4 g ormet./ ton
- Feed- with minor species recommendation:
    - Chlortetracycline: 500 g/ton for max 5 days (1 day wd); reduce to 100- 200 g/ ton for longer term- up to 2 weeks (0 day wd).
    - "aid in control"- 227 g sulfa., 132.6 g ormet./ ton.
    - 5 day wd.
- Drinking water- "over the counter":
    - Oxytetracycline: 800 mg/ gallon; 0- 5 day withdrawal (wd).
    - Min 5 days; often need to repeat or continue treatment with feed medication.

Prevention/ Control

- Complete clean-out and disinfection of pen, sanitize feeders and drinkers, bait rodents, mend fences, till under the soil and cover plants. Give some "down" time.

Vaccination??

- Could use autogenous vaccine (killed, from specific farm isolates) if control not achieved by other methods.
- Would require at least 2 injections for adequate protection.
- Serotypes 3,4 have been identified in 3 PSU pheasant cases; these are contained in commercial vaccines.

Biosecurity

- Closed flocks- prevent introduction of sick birds and recovered carriers; limit replacements to chicks, sources with history of no cholera breaks.
- Control rodents / varmints / predators.
- Restrict cat access.
- Cull sick and dying birds ASAP; pick up dead ASAP, and dispose of carcasses properly (don't attract scavengers)!
- Cannibalism control.
- Cull obvious lame or poor-doing birds.
- Chlorinate drinking water to slow down bird to bird spread.

COCCIDIOSIS IN GAMEBIRDS

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Coccidiosis in Game Birds

- Losses close to 40% not unheard of, especially in late season, floor-raised chukars.
- Most problematic in humid environments, multi-age, multi-stage units.
- Along with intestinal parasites, the single most costly disease problem in game bird production, especially in 2008!

Coccidiosis

- Single-celled protozoal organisms that infect the intestines. Kidney species in waterfowl.
- They are excreted as oocysts in the droppings.
- The excreted oocysts cannot immediately infect the bird. They require moisture, oxygen and warmth in the environment.
- Generally resistant to disinfectants.
- Destroyed by high temperatures and ammonia released by composting litter or by freezing.
- Some disinfectants reported to kill them. Mild, wet weather is ideal for cocci survival and infectivity.

Pathogenesis

- Very prolific organism. Single ingested oocyst can result in millions of oocysts in a relatively short time.
- No immunity passed from hen to chick.
- Any age susceptible.
- Development of immunity after infection with persistent cycling of parasite.
- Disease is everywhere birds are raised on floor.

Coccidial Life Cycle

They will undergo multiple generations of reproduction in the intestinal cells, causing much damage to the gut in the process.

Symptoms

- Chukars: Birds act cold, start huddling. Losses from the parasite and losses from piling.
- Watery diarrhea seen. Bloody droppings are not usually present.
- On necropsy, birds show dehydration.
- Intestines usually contain cheesy cores (mid-intestines, ceca depending on the species of oocyst present.
- Watery tan loose diarrhea is seen with coccidiosis in chukar partridges.
- Pheasant cecal cores are commonly observed with coccidiosis.
- Quail Disease (Ulcerative enteritis) often preceded by coccidiosis.

Why are Coccidia Such a Problem in Game Bird Growing????

- Trend towards higher bird densities results in explosive coccidia multiplication. No time for slow immunity to develop.
- Biosecurity between age groups rare on most farms; multiple ages and manure tracking by people.
- Birds (especially chukars) are not as adapted to high intensity rearing, desert species and stress-prone.

Coccidiosis Management

1. Completely clean out and disinfect brooder pens between broods. Use hot water or steam wash down.
2. Do not re-enter the building without dedicated boots, shoe covers, etc. People track oocysts back in!!
3. Foot bath and shoe change between brooders.
4. Use a coccidiostat in the feed (see under medications approved by species).
5. KEEPING BROODER HOUSE DRY WITH VENTILATION, DRINKER MANAGEMENT, AND OPTIMAL BIRD DENSITY IS THE KEY!!!
6. Practice moving from youngest to oldest birds each time.
7. Brood on wire if nothing else works.
8. Rotate coccidiostats to help prevent resistance.
9. Monitor disease in house through necropsies, learn to do fecals with inexpensive microscope. Microscope great for parasite checks as well.

Coccidiosis Treatment Dilemma

Amprolium:

- Drug resistance? Higher dosages needed?

Sulfa drugs:

- Cumulative toxic effect if dosed repeatedly:
- Bone marrow destroyed; damaged immune system, poor feathering, stunting.
- Bleeding disorders, sepsis, coccidiosis rebound effect, dermatitis (B-vitamin deficiency syndrome seen) after multiple sulfa treatments.
- Sulfa should never be given to egg laying breeders!!!
- Shell damage, hatchability of eggs affected results.

Problem with coccidiosis prevention in gamebirds is that the approved drugs are of limited value. Some success seen with addition of chemicals but resistance develops rapidly. With so few drugs, difficult to do shuttle/rotational program to conserve efficacy of the medications.

Rotation: Change coccidiosis program seasonally.
Example: Avatec, first two broods; Clinacox, last two broods

Shuttle: Use different drugs in the same brood
Example: Clinicox, 1-3 weeks; Avatec, 3-8 weeks

Coccidiosis Preventative Medication in Birds

Coccidiosis Control medications in broilers:

Ionophores

- Lasalocid (Avatec™)
- Monensin (Coban™)
- Narasin (Monteban™)
- Semduramicin (Aviax™)
- Salinomycin (Sacox™, Bio-Cox™)

Synthetics (Chemicals)

- Diclazuril (Clinacox™)
- Halofuginone (Stenorol™)
- Nicarbazin™
- Robenidine (Robenz™)
- Zoalene (Zoamix™)
- Nicarb

Turkeys: Monensin, Rofenaid, Zoalene, Clinacox, Stenorol, Avatec

Currently Approved Coccidiostats (Preventatives) For Gamebirds
Amprolium in feed approved for all.

Quail: Salinomycin (Biocox), Monensin (Coban)

Chukars: Lasalocid (Avatec), Rofenaid (potentiated sulfa)

NAGA's Efforts To Develop More Effective Treatment and Prevention Tools

- Board voted in Reno to support a two year research project on coccidiosis prevention and control.
- Determination of coccidia species affecting N. American pheasants, chukars, and quail.
- Coccidiostat susceptibility/safety study with known broiler medications.
- Evaluate potential of coccidia isolates for development of vaccines in chukars, pheasants and quail.

Most Current Drug Studies

Chukar Trials: Very preliminary data!!

- Researchers looked at lesion scores, weight gains, and feed conversions in the studies.
- Using broiler drug dosages in the feed good protection against challenge was seen with Rofenaid, Robenz, Zoalene (at 150 ppm) and Semduramycin/3-nitro combination, Clinacox at 2 ppm.
- Poor protection was observed with Amprolium, Decox, Diclazuril (Clinacox at 1 ppm) and semduramycin alone (regional differences?).
- Incomplete protection with Avatec.
- Several other drugs need to be looked at: Monteban, Maxiban.

Pheasant Studies (Preliminary studies):

- Researchers looked at lesion scores, weight gains, and feed conversions in the studies.
- Rofenaid, Robenz, Diclazuril (Clinacox), Avatec (lasalocid) appear to be effective.
- Decox, 3-nitro, zoalene (150 ppm), semduramycin not as effective.
- Problem with using Rofenaid in the feed: Dangerous to use sulfa in water for treatment. Toxicity can occur.

Quail Coccidiostat Studies

- Robenz, Clinacox, and Rofenaid results were promising.
- Amprolium was very poor. Lesions were as severe at the unmedicated/innoculated controls.

Interesting Comment By the Researchers

- Chukar coccidia challenge dose much lower than in the other two species.
- Innate susceptibility to coccidiosis appears greater in chukars.
- Results shown pertain to coccidia on certain farms throughout the country.
- Be sure to submit specimens from your state to determine species and susceptibility in your area.

Vaccine Development

- Must have separate vaccine developed for each game bird species.
- Vaccines are comprised of live oocysts; good litter management, proper vaccination dose still critical.
- Has had great acceptance with poultry breeders, floor layer people, organic poultry production.
- Administration of coccidiosis vaccines generally in the hatchery.
- Gel-delivered vaccine or spray.

Current recommendations for coccidiosis control:

- Practice preventative management steps described previously.
- Discuss feed medication options with your veterinarian and feed supplier.
- Do try a shuttle/rotational program if possible.
- Relying on one coccidiostat, especially a chemical leads to rapid resistance development, often in one year or less.

GAME BIRD DIAGNOSTIC SERVICES THROUGH THE UTAH VETERINARY DIAGNOSTIC LABORATORY

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Gross necropsy (Nephi)

Histopathology (processed in Logan)
Toxicology (Logan or outside laboratory)

Bacteriology (Nephi)

General – *E. coli*, other enterics, *Staphylococcus*, *Pseudomonas*, etc.
Salmonella – necropsy specimens, environmental drag swabs, chick papers
Rapid Check Test
Delayed enrichment
Slide agglutination test: Group D
All *Salmonella* sent to National Veterinary Services Laboratory, Ames, Iowa for serotyping

Virology

PCR (Logan)
Avian influenza (AI): H5 and H7 subtypes

Serology (Nephi)

Whole blood slide agglutination test – live birds, fresh unclotted blood
Salmonella pullorum/*S. gallinarum*

Serum rapid plate agglutination test
Mycoplasma gallisepticum (MG)
[*Mycoplasma synoviae*] (MS)

Agar gel immunodiffusion (AGID):
AI

Lateral flow assay:
AI (Flu-Detect™, others)

MYCOTOXINS AND THEIR COSTS

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Introduction

Mycotoxins are a threat to the health, performance and economic viability of all commercial animal businesses. More than 200 different mold toxins have been identified, but about six have been shown to be somewhat common and have meaningful physiological impact on animals. These are primarily metabolites of molds of the *Aspergillus, Fusarium and Penicillium* genera. Mycotoxins have multiple metabolic impacts, including damage to internal organs, reductions in egg production and hatchability and suppression of immune status, which can predispose animals to secondary disease impacts. The resultant effects are reductions in animal performance, reproductive efficiency and survival rate, which can all impact the economic viability of game bird enterprises.

While no animal feed can be guaranteed to be free of toxins, three key principles can be applied to reduce the risk and impact of molds and mycotoxins on game birds or other species. These key principles are:

1. Monitor raw materials to avoid contamination.
2. Manage materials appropriately to reduce risk of further toxin formation.
3. Utilize appropriate ingredients to ameliorate contamination when risk is unavoidable.

Molds and Mycotoxins

Molds are common to most plant-based ingredients, they proliferate when conditions (temperature, moisture and access to nutrients) are favorable. Molds consume nutrients from crops and may reduce palatability of feeds made from moldy inputs. The molds generally produce mycotoxins as a defense mechanism in response to stress. Thus, while mold is a necessary prerequisite for mycotoxin production, the level of mold is not a good predictor of mycotoxin production. For example, *Fusarium* molds commonly produce toxins when conditions become wetter and cooler than they prefer for maximal growth. *Aspergillus* species produce aflatoxins when conditions become dry and hot.

Managing "field" toxins, which are produced before plants are harvested, is very difficult. In some years, and in some regions, molds grow and produce toxins, with little opportunity for producer intervention. Plant pests and diseases may exacerbate toxin production, thus, wheats with bunt or rust are more prone to *Fusarium* toxins, and corn affected by rootworms or earworms may be more vulnerable to aflatoxin formation. In much of the U.S., early heavy rains followed by late season dryness led to high incidence of fumonisin and aflatoxin in the 2008 corn crop.

Post-harvest toxin production can result when excessive moisture and pre-existing mold populations are present. Reductions in moisture and temperature can be used to limit mold growth and prevent mycotoxin production, but when conditions change, such as, shipping grain from a cold region to a warm region, resulting in moisture condensation and warm enough temperatures for growth, further mold and toxin production may result.

Major Mycotoxin Risks for Game Birds

Domestic poultry are among the most well understood species in relation to mycotoxin effects, unfortunately, most game bird species are not. Most data from broilers could be extrapolated to game birds with reasonable confidence, but there are studies showing variation among poultry species in sensitivity to different toxins.

Aflatoxin

Aflatoxins are a family of toxic, carcinogenic compounds produced primarily by *Aspergillus flavus* and *A. parasiticus* strains of mold. Aflatoxin B_1 is the most prevalent form found in feeds. Although commonly found in many crops, corn and peanuts are primary sources of aflatoxin in the U.S. In general, aflatoxin risks increase during periods of drought. Aflatoxin effects on poultry are commonly mediated via the liver where they travel after absorption from feeds. They are rapidly metabolized to more toxic and reactive compounds called epoxides. Common effects include poor gain and feed conversion, reduced egg production (due to decreased egg production and embryo toxicity), leg weakness, and in commercial poultry, carcass condemnations. Symptoms include stunting of growth of birds and poor health, related to suppression of the immune system. Ascites is more common in birds challenged with aflatoxin. Effects of aflatoxin are often exacerbated, when additional toxins are present in a feedstuff or a diet.

Among commercial poultry species, ducks are very susceptible to aflatoxin, followed by turkeys, broilers, laying hens and quail (Devegowda & Murthy, 2005). In general, younger birds are more sensitive to effects of aflatoxin than older or mature birds. Ducklings, turkey poults, goslings and pheasant chicks are all more vulnerable than broiler chicks. Research with chukar partridges (Ruff, Huff, & Wilkins, 1990) and bobwhite quail (Ruff, Huff, & Wilkins, 1992) have shown sensitivity to aflatoxin, as well. Game bird producers should be wary of aflatoxin risks, particularly from ingredients produced in the southern U.S. or in drought stricken areas in some years. Aflatoxin can be either a “field” or a “storage” toxin, meaning the mycotoxins can be formed in the field before harvest, but grain can become more contaminated during improper storage or handling (warm, moist conditions) that results in *Aspergillus* mold growth.

Ochratoxin A

Ochratoxin A (OTA) is a very toxic compound for most poultry. It is produced by several species of *Aspergillus* and *Penicillium* species in both tropical and temperate regions. It attacks the kidney and may lead to suppressed intake and growth, slow or poor feathering, rickets symptoms, damage to internal organs and reduced egg production. Eggs may be stained with a characteristic yellow staining of shells, low chick viability and chicks from hens fed diets with OTA may fail to thrive as they grow. In turkeys, high mortality can result from OTA contamination (Devegowda & Murthy, 2005). Bobwhite quail (Ruff, Huff, & Wilkins, 1992) and chukar partridges (Ruff, Huff, & Wilkins, 1990) have also been shown to be highly sensitive to OTA.

Ochratoxin A may also be a “storage” toxin in both temperate and tropical regions when storage conditions permit mold growth. While less common in the U.S. than aflatoxin or some fusarium toxins, OTA can be devastating to game bird operations should a severe contamination occur.

Tricothecenes : T-2 Toxin DAS, DON, nivalenol

Tricothecenes are produced by *Fusarium* species of mold in many parts of the world, including temperate and cool climates. There are a wide array of *Fusarium* toxins and the tricothecenes family, which have a common ring structure in their chemical make up, number into the dozens of different varieties. In general, they tend to act as tissue irritants that may be related to a tendency of some to reduce feed intake. Additionally, when ingested, some tricothecenes, especially, T-2 toxins and the less common DAS (diacetoxyscirpenol) produce necrosis and inflammation of the mouth. In addition to oral lesions, immunosuppression and reduced egg production and egg quality in commercial poultry, T-2 toxin has been shown to affect chukars and quail (Bobwhite and Japanese) (Grizzle, Kersten, Houston, & Saxton, 2005), geese and ducks, in similar ways. Ducklings seem to be remarkably sensitive to T-2 toxins.

Tricothecenes produce immunosuppression that increases the likelihood and effect of secondary infections. Deoxynivalenol (DON), sometimes called vomitoxin, and nivalenol appear to have less impact on birds than mammalian species. Wild, captive ducks were found not to be sensitive to DON, as well. Most *Fusarium* toxins are primarily "field" toxins that do not generally increase in storage.

Fumonisin

Fumonisins are produced by two strains of *Fusarium* fungi. Although relatively less toxic to birds than aforementioned toxins, fumonisin at high levels can reduce performance and may experience immunosuppression. The most common fumonisin, B1 (FB1) is often associated with other toxins, including aflatoxin and other fusarial toxins. Research is ongoing to better understand the contribution FB1 may make to toxic effects of other mycotoxins. Fumonisin contamination of corn, especially in combination with aflatoxin, appears to be a common problem in many states in the mid-south and mid-west this crop season.

Zearalenone

Zearalenone (Z) is an estrogenic compound produced by at least two species of *Fusarium* molds. Poultry are generally resistant to common contamination levels of Z.

Combinations of Mycotoxins

While little work appears in the literature with game birds, effects seen in domestic birds clearly indicate additive or synergistic effects when multiple toxins are present in diets. Some field experts observe detrimental effects at lower levels of toxin contamination, when multiple toxins are involved.

Costs of Mold and Mycotoxin Contamination

Costs of mold and mycotoxin problems are estimated to be in the billions of dollars per year. While mycotoxins have huge impacts on animals, especially in tropical regions, mold growth, without toxin production can greatly reduce the nutrient value of feeds adding cost to production systems. Oils and fats seem to be particularly vulnerable to destruction by molds, leading to losses of essential fatty acids that may exacerbate immune suppression of mycotoxins. Reduced oil content also reduces energy content of moldy grains. Mold damage normally does not reduce crude protein content, but may destroy key amino acids and change dietary amino acid profiles reducing the feeding value of diets. Carbohydrates are the primary fuel of molds and modest changes in starch and sugar content can further decrease the energy value of grains. The effects of molds, separate from mycotoxins may be $10 to $30 per ton of feed.

Estimates of various studies put detectable mycotoxin contamination rates at 25-50% of grain products and more than 75% of some grain byproducts. Ethanol byproducts may contain more than three times more toxins than the corn they originate from because fermentation of the carbohydrate concentrates toxin levels in residual byproducts. These frequencies vary by location, year, point in the supply chain, season, and other factors. Mycotoxin effects on animal physiology is difficult to estimate, but clearly these mycotoxins can readily reduce performance, through reduced intake, poorer efficiency, higher disease rates and mortality. Estimates of 10% performance losses during moderate contamination are common. This may equate to $20 to $30 per ton of feed loss in value.

Beyond liveability and animal performance reductions, losses in reproductive efficiency can be devastating to game bird or other enterprises that require efficient reproduction for viability of the enterprise. Poor hatchability, reduced lay rates, and hatching of chicks with low viability and survivability can produce 5 to 50% increases in cost of production, should mycotoxins be allowed to reach clinically important levels.

Fortunately, frank performance level depressions appear to be uncommon, but periodic spikes in toxins reduce intake and performance, and take a toll on game bird and other poultry enterprises.

Reducing the Risks and Impact of Mycotoxins

How then do game bird and other poultry producers reduce the risk of molds and mycotoxins to their enterprise profitability and animal health and welfare? No single technology can prevent problems. Whether you are purchasing complete feeds or inputs to generate final rations, the principles are the same:

- Avoid contaminated ingredients as much as possible, by appropriate, risk-based monitoring.
- Invest in good handling, storage, and mold inhibition programs to prevent deterioration or contamination increases within feed inputs or final diets.
- Utilize available technologies to limit impact of unavoidable toxin effects.

The growth of molds and production of toxins involve complex interplays among environmental variables (temperature, pH, moisture, relative humidity, and time) and the fungal types present in feed inputs. In general, *Fusarium* molds are “field” fungi that primarily grow in the field and do not readily compete and grow in most storage conditions. Stresses with plant diseases, weather shifts from too dry to too wet, and cool, wet pre-harvest conditions or delayed

harvesting can enhance growth of “field” fungi.

Aspergillus and *Penicillium* are more often “storage” molds that can grow and produce toxins when storage conditions are not optimal, particularly when moisture content is too high and storage sanitation is poor. In general, moisture levels below 14% percent limit growth of molds, however, conditions that produce portions of the grain to become higher in moisture may lead to risk of mold growth, when average moisture levels are in the “safe” zone. Temperature shifts due to sun heating one side of a storage structure can promote moisture migration to the cool side of the structure. Condensation here can produce opportunities for mold growth, even when overall moisture levels are at levels normally deemed to be safe for storage. Transportation of cold, stored grain to warm, humid areas can promote moisture migration or moisture condensation that allow pockets of mold growth and toxin production. In general, corn that has been held in storage in a very cold state, may be subject to mold formation during warmer, more humid summer conditions.

It is important to understand that mycotoxins are metabolites of mold that are produced when conditions faced by the mold are stressful for the mold’s growth or survival. Thus, during periods of rapid, uncontrolled mold growth, the production of toxins may be low. Stresses such as temperature and moisture conditions on the extreme of the mold’s growth range may lead to greater toxin production. Thus, while mold must be present to produce toxins, high levels of mold growth are not required for mycotoxins to form. Ironically, storage conditions that are close to acceptable, but not fully adequate may produce a more optimal condition for mycotoxin formation than truly poor storage conditions.

Monitoring

Because molds commonly grow in only a portion of a field or a mass of grain or feed during storage, and because subtle variations in environmental conditions can trigger mycotoxin production, the observed patterns of mold and mycotoxin contamination are irregular and may be difficult to predict. A contaminated lot of grain may vary from non-detectable to lethal levels within sub-samples from that lot. Of course, moving, grinding, blending the feed inputs spreads the mold or mycotoxins and leads to more uniformity and moderation in levels. As a result, monitoring feed inputs for molds and mycotoxins can be challenging. In general, molds may be best monitored qualitatively. Grain that contains clumps that are damp, warm or moldy should be handled carefully. Any obvious moldy pockets should be destroyed, not used in feed. If grain appears dusty, has an off odor, produces a cloud of dust around collection points or discharge points in a handling system, is likely contaminated with mold, and should be more closely monitored for mycotoxin levels.

Fortunately, technology to measure mycotoxins has become more accurate, more affordable and more toxins can be measured than in the past. Still the testing is expensive, time consuming and difficult, due to the variation in contamination levels. Inadequate sampling programs can make mycotoxin monitoring nearly a waste of time. In general, a lot of grain or feed should be divided into multiple parts. Each part should be sampled by taking a large sample (more than 2 lbs). These samples may be blended to better represent the lot. Finally, multiple samples from the mixture should be measured to increase the chance of detecting toxins, if they are present.

While an in depth discussion of sampling, testing and quality control are beyond the scope of this paper, the following principles are critical. If you are purchasing from a reputable feed source, these practices, or similar ones should be in place. If you purchase or use home raised feed inputs to produce game bird diets, recognize that quality control becomes your

responsibility.

- Know the patterns of toxins being reported in your area or the area of sourcing feed or inputs.
- Monitor grain for visible mold contamination and avoid using visibly moldy products.
- Conduct periodic monitoring of for mycotoxins of potential concern (especially, aflatoxin, ochratoxin, and T-2 toxin) if there is reason to suspect concerns.
- Conduct additional testing of lots found to have a detectible level of toxins, even if the level is below that likely to produce effects on birds.
- Monitor and manage moisture content to help prevent risk of mold and mycotoxin level increases in storage.
- Closely observe animal behavior and performance, as rearing conditions permit, to look for any outward signs that problems may be occurring.
- Conduct additional testing if suspicious signs appear in feed, ingredients or birds.

Managing

Preventing deleterious impacts from molds and mycotoxins requires a basic understanding of the factors that allow for mold production. Ultimately, mold growth and mycotoxin production can only occur when moisture, oxygen, temperature and nutrients are available in a range that permits survival and growth. These conditions vary among genera of fungi, but generally require temperatures above 50^{o}F, moisture content of 14% or higher. Because localized areas in storage can be higher in moisture than others producing micro-environments that allow growth, it is generally preferable to lower average moisture below 13%. It is notable that mold growth produces water as a byproduct of converting carbohydrates to metabolites of molds. Thus, mold growth can become somewhat self-perpetuating when moisture conditions are marginal for growth. Normally access to oxygen and nutrients can not be avoided in feeds or feed inputs.

Use of a concentrated, propionic acid-based mold inhibitor may be indicated when moisture levels exceed 12%, depending on storage time (longer storage increases risk of mold and mycotoxin development) and storage conditions (exposure to sun and shifts in temperature can stimulate moisture migration). Mold inhibitors should be buffered (containing ammonium to raise pH) or should be made from a metal salt of propionic acid, for example calcium propionate. Buffered liquid mold inhibitors (containing ammonium propionate) or dry inhibitors (containing calcium propionate or ammonium propionate on a silica or vermiculite carrier) can be effective in reducing mold growth and mycotoxin production. Levels of 1 to 2 lbs per ton may be adequate at moisture levels up to 13%, higher levels are indicated as moisture levels increase or storage time exceeds 60 days from production to consumption by animals.

When toxins are already present, mold inhibitors will not remove toxins. Likewise, no practical treatment has been developed to chemically or physically remove existing toxins from feed or feed inputs. There are a few products that are recognized as safe feed ingredients for other purposes that have been shown to bind and reduce the effects of mycotoxins. There is no definition of these “mycotoxin binders” in U.S. feed codes, and no product has been approved by FDA to reduce mycotoxin effects in feeds. Research data prepared for other countries shows that some clays, modified clays, charcoal products, enzymes, and yeast cell wall components can reduce mycotoxin effects in some conditions.

Because of the inconsistent levels of mycotoxins, even in controlled test conditions, it is difficult to verify the efficacy of these compounds in animals. Attempts to qualify binders by *in*

vitro or lab-based tests do not appear to be fully satisfactory to predict animal responses. However, it is generally accepted that while *in vitro* binding does not guarantee effects in live animals, it is highly unlikely that products that do not bind effectively *in vitro* will work well in the animal. Because binding in the animal can be affected by the pH of the digestive tract at different points, *in vitro* testing should include binding at an acid pH (pH 2 to 3.5) and at a near-neutral pH (pH 5.5 to 7). Many binders appear to be effective at pH 2, but fail to sustain binding at higher pH. These binders are likely to fail to hold toxins through the higher pH of the small intestine of most birds.

In general, many carefully selected clays (bentonites), sometimes called sodium or calcium aluminosilicates, are effective in binding aflatoxin and reducing, not eliminating, its deleterious effects in animals. Yeast cell wall materials appear to have a broader array of binding effects, including some effect with more polar mycotoxins, but may not be fully effective in removing these toxins from animals. Modified clay products in which a long chain, polar group is bound to clays, appear to have the broadest impact. Table 1 shows results of an *in vitro* study in which different toxins were exposed to different clay, charcoal and yeast cell wall type ingredients that are commercially used in other countries as toxin binders. The binding was in two stages, first at pH 2 to simulate binding in the acid, upper digestive tract, then at pH 6 to see if toxins are released in the higher pH of the lower digestive tract. Efficiency percentages shown in the table represent the percent of toxin in the system that were bound and held through both pH levels. The broadest binding appears to be associated with modified clays, while a non-modified clay was very effective in binding aflatoxin and fumonisin in this assay. At the levels tested, modified, yeast cell walls and charcoal type binders tested gave limited effects against some toxins, but did not match the clay products for any mycotoxins.

Binding of T-2 toxin and DON in this study, as in most studies, was below 80% effective for all products, suggesting limited value in birds. Emerging technologies, including enzymes, bacteria, competing fungi, or plant extracts may hold promise for the future in dealing with these toxins that are most difficult to bind, but generally are limited in cost-effectiveness, consistency, or proof of efficacy in reliable animal models to date.

Conclusions

Avoiding the pitfalls of molds and mycotoxin contamination requires a systematic approach to prevention. Reliable feed suppliers are implementing monitoring and management approaches similar to those described in this paper. Other suppliers are hoping that mycotoxin concerns do not doom the success of their customers. Producers putting together their own diets must understand and develop an effective and cost-effective system to avoid mold and mycotoxin problems. Game birds may vary from commercial poultry in their response to specific mycotoxins or combinations of mycotoxins, but they are definitely vulnerable and should be carefully managed and monitored.

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Table 1. *In vitro* percent binding efficiency comparison.

	Adsorbent inclusion (kg/ton)	4000 ppb Aflatoxin B1	5000 ppb Zearalenone	3000 ppm Fumonisin B1	2500 ppb Ochratoxin A
Clay 1, not modified	5	98	34	98	53
Specialized carbon source	2	0	41	51	58
Modified yeast cell walls	1	39	26	3	67
Clay 2, partially modified	3	76	95	95	93
Clay 3, modified	1	61	95	33	89
Clay 4, modified	1	92	94	93	92

Unpublished, Internal Cargill Data

PROGRESSIVE GAME BIRD MANAGEMENT

Bill MacFarlane

Game Bird Production

- Complacency
  - Low risk, comfortable
- Progressive
  - Open minded, willing to try new methods
- Success
  - Get out of your comfort zone, take risk

Overview to Success

- Awareness & Openness
- Biosecurity
- Genetics
- Consultants
- Organizations
- Protocols
- Marketing
- Denial
- Recognition
- Acceptance
- New Methods

Attributes of a Successful Visionary Approach – Maintaining Bio-security

- Separation
  - Facilities
  - Employees
  - Equipment
  - Species
- Layout Time
  - Facilities
  - Equipment
- Vectors
  - Visitors
  - Pests
  - Birds from other farms

Genetics

- Specialization
  - Meat
  - Flight
- Feed conversion
- Egg production and fertility
- Disease Resistance
- Bird External Appearance
  - Size at maturity
  - Coloration
  - Lack of defects

Consultants

- Bird Health
- Genetics
- Disease Control
- Nutrition
- Equipment

Organizations

- NAGA
  - North American Gamebird Association
- State Gamebird Associations
- Lobbyist
  - Be proactive and be aware of threats to our industry

Protocols

- Development
- Documentation
- Legacy

Protocols (The Manual)

- Anything and everything should be covered
- Medications, feed, contact numbers, etc.
- Cover the who, what, when, where, and why for setup and procedures.

Marketing

- Conceptual Image
- Initial Vision
- Operation Vision
- Attitude
- Customer Satisfaction

Conclusion

These are recommendations that will lead to a successful game bird operation. If you are willing to take the risk, you will reap the rewards.

MANAGEMENT OF PHEASANTS IN THE FLIGHT PENS

Six Weeks of Age until Maturity

Bill MacFarlane

The Plan

- Decide where first birds will go
- Till and plant those pens first
- Determine how many birds to put in each pen
- Set up the pens
- Move out the birds
- Take care of the birds

How to Move Out Birds

Barn Setup:

- Pressure wash crates
- Build catch pen inside barn
- Put pressure washed crates inside

Catching:

- 4 or 5 person catching crew
- 2 or 3 person dumping crew
- Birds are sexed into the crates
- 15 birds per crate
- Use cardboard to drive birds

Preparation of the Flight Pens

- Pen maintenance
- Feed and water
- Shelter and cover
- Miscellaneous Items

Pen Maintenance

- All gates checked and secured
- Division fences checked for integrity
- Roof double checked for holes

Feed Requirements

- 1 feeder for every 100 bird
  - .7 inch of feeder space per bird
- Supplemental feeder and pans in front of the pens
  - 1 feeder
  - 4 feed pans for every pen

Feeding Program

- 0-3 weeks of age: 30% protein
- 3-6 weeks of age: 26%
- 6-14 weeks of age: 20%
- 14-20 weeks of age: 19%
- 20+ weeks: 14%

Water Requirements

- 1 plasson for every 250 birds
- (.1 inch per bird)
- Water pans in the front of pens
  - 1 pan for every 100 birds

Shelter Requirements

- 1 hut for every 100 birds
- Use when cover is <18" tall
- Straw huts

Cover

- Weeds for early birds
  - Lambsquarter
- Corn or corn/sorghum mix later
- Mow paths
  - birds need open space

Planting

- Plant corn early
- Plant corn/sorghum mixture late
- Use a row planter
  - broadcasting can make plants spindly

Square Footages

- Early birds (before 4/15 hatch)
  - 30 square feet for hens
  - 40 square feet for cox

- Late birds (after 4/15 hatch)
  - 18 square feet for hens
  - 24 square feet for cox

Other Precautions Taken for Young Birds

- Straw all 4 corners of pen
- Put feed pans under huts
- Put feed with a coccidiostat in the front feeders
- Add vitamins to the supplemental water pans

When Do You Decide To Move Out Young?

- Weather!
- Health of the flock
- Size of the flock
- Status of the flock behind it

Weather

- 3 days without rain
- Temperature
  - Above 50 degrees when possible
- Be flexible

Health of the Flock

- Don't move sick birds
- Wait 1 week after peeping

Flock Size

- Move crowded birds A.S.A.P.

- What is the flock size behind this group?

Other Considerations

- Time of the year
- Extended forecast
- Health and size of flock behind current flock

Should I or Shouldn't I??

- Temp: 40° F
- Month: April
- No rain expected
- Age: 6 weeks
- Flock size: normal
- Bird health: good

YES

Should I or Shouldn't I??

- Temp: 40° F
- Month: May
- 50% chance of rain
- Age: 6 weeks
- Flock size: normal
- Bird health: good

Always Move Birds Out In the Morning!

- Don't dump birds into pens during the late evening

Care After the Brooder Barns

- Disease Control
- Worming
- Predator control
- Pest control
- Observation

Disease Control

- Dead check on a weekly basis
- Keep accurate records
- Keep feeders clean

Worming Birds

- Worm all second use pens
- Wet pens may need to be wormed more than once
- Dry pens may not need to be wormed at all
- Watch the birds for signs
- Use panacur through the water

Predator Control

- Hawks and Owls
  - catch strays on a daily basis
- Fox and Coyotes
  - trapping program
- Opossum and Skunk
  - prevent dig-ins
  - keep gates closed at all times
- Mink

Pest Control

- Mice and Rats
  - create and implement a plan
- Starlings
  - pre-bait and poison
  - scare devices

Observation

- Drive around pens twice daily (early and late)
- Pay attention to the smallest details
- Keep accurate records

SUMMARY

- ▶ Move out young birds in a timely manner
- ▶ Cover and feeder space are the two key factors in raising birds
- ▶ Check your birds often
- ▶ Use common sense - do not ignore your gut feeling