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## CONTROL OF INFECTIOUS BRONCHITIS

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### Introduction

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Control of Infectious Bronchitis (IB) should be fairly straightforward: Determine which serotypes(s) are present, vaccinate for those serotypes, and do not vaccinate for other serotypes. Unfortunately, the devil is always in the details, and determining that an undefined respiratory disease problem is indeed IB (and particularly differentiating it from lentogenic Newcastle disease), determining the serotype, and designing and implementing the proper preventative programs can be challenging. In this presentation we will discuss the approach to differential diagnosis, determination of the serotype, and tips on designing control strategies. As the author's experience is entirely in North America, this discussion will come from that perspective. I have little experience dealing with IB in the context of velogenic Newcastle Disease (ND) or Mycoplasma-positive breedersss, and simultaneously dealing with those agents certainly impacts the management of IB programs. On the other hand, our inspection service is fairly stringent and unforgiving, and the competitive nature of our business makes us quite sensitive to minor changes in livability or condemnation. Hopefully our Latin American colleagues can still gain a few useful perspectives from our experiences with IB control here in North America.

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**Broiler Performance Data (Region)**
**Live Production Cost**

| | SW | Midwest | Southeast | Mid-Atlantic | S-Central |
|---|---|---|---|---|---|
| Feed cost/ton w/o color ($) | 149.75 | 152.19 | 150.29 | 138.96 | 151.47 |
| Feed cost/lb meat (¢) | 14.22 | 14.80 | 14.04 | 12.45 | 14.60 |
| Days to 4.6 lbs | 43 | 42 | 44 | 41 | 43 |
| Chick cost/lb (¢) | 4.21 | 3.60 | 4.26 | 4.41 | 3.94 |
| Vac-Med cost/lb (¢) | 0.06 | 0.14 | 0.08 | 0.06 | 0.07 |
| WB & 1/2 parts condemn. cost/lb | 0.11 | 0.16 | 0.14 | 0.18 | 0.16 |
| % mortality | 5.19 | 4.72 | 4.71 | 4.63 | 5.21 |
| Sq. Ft. @ placement | 0.83 | 0.84 | 0.81 | 0.81 | 0.84 |
| Lbs./Sq. Ft. | 6.60 | 7.42 | 6.78 | 6.66 | 7.12 |
| Down time (days) | 13 | 16 | 14 | 12 | 12 |

**Data for week ending July 29, 2006**

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## Clinical Appearance of IB and Lentogenic ND in North America

IBV and lentogenic ND are rarely absent from North American broiler populations. Even when more-or-less under control, the birds usually remain under some degree of challenge by resident strains of both viruses, in the face of a "correct" vaccination program. In the case of IB, these resident strains may include (1) the vaccine itself, which likely has been back-passaged in the birds to some degree, (2) the wild type of the vaccine serotype, or (3) a slightly altered but related variant strain of the vaccine serotype. With ND, challenge with the vaccine virus itself is probably common, but wild lentogens undoubtedly coexist as well. Under these circumstances of reasonably good control, with essentially homologous challenge, overt clinical problems usually are due to (1) misapplication of vaccine, resulting in poor protection and/or rolling reactions, (2) immunosuppressive disease, (3) interference phenomena between ND and IB vaccines, with "rolling" ND infections, or (4) mismanagement, especially of temperature, ventilation, diet, or downtime. These endemic cases typically are of mild to moderate severity. The birds may have a slight dry snick or cough and may exhibit increased flicking of the head. The eyes may take on a mild almond shape due to mild conjunctivitis and swelling of Harder's gland, and one may note a few dirty nostrils. If the usual mortality in a normal flock is 0.5 to 0.75/1000/day, then these flocks may approach 1.5 to 2/1000/day. Moribund and dead birds tend to appear as chronically ill "culls", with generalized lesions of secondary colisepticemia. I believe that much of the purulent arthritis and osteomyelitis ("femoral head necrosis") in many operations in winter is due to this combination of resident IBV and ND challenge, immunosuppression, and mismanagement. If the typical total condemnation is 0.75%, cases such as these may reach the 1-to-2 % range. Determining and correcting the multifactor causes in these mild-to-moderate, management-related scenarios with IB can be a real challenge, as much as or more than with the emergence of a new type.

The emergence of a "new" serotype of IB, or the overt failure of a lentogenic ND program, may result in much more severe consequences. Snicking and coughing may be readily audible, even from outside of the house. Obvious conjunctivitis and severe swollen heads may be common, especially with IB complicated by Infectious Bursal Disease (IBD). Affected birds experience a classical progression from foamy suds, to creamy purulent exudate, to caseous material in the air sacs. Mortality may exceed 5/1000/day. Complex-wide weekly average total condemnation may range between 2 and 3.5% or more, with a number of more severely affected individual flocks contributing to that average. Other patterns are entirely possible. The author has witnessed outbreaks of both variant IB as well as lentogenic ND that resulted in much less obvious clinical signs, but severe lesions and condemnations. For example, one outbreak of a variant Arkansas IB strain resulted in essentially no clinical signs other than fever (panting and holding the wings down), and very little mortality, but was accompanied by severe pneumonia and condemnation. As that outbreak progressed, and breaks occurred at earlier ages, more typical but still subtle respiratory signs and mortality became increasingly evident. While these more severe scenarios with IB may be more urgent, the cause is often easier to discern and correct than with the less severe management-related situations.

## Differential Diagnosis of IB and

In most North American practices, the majority of the diseases in our differential list are non-existent (velogenic ND, Avian Influenza, Avian Metapneumovirus, Chlamydia), unusual in market age broilers (pox, aspergillosis, coryza, cholera, primary colibacillosis), probably inconsequential as primary agents (Bordetella avium, Ornithobacterium rhinotracheale, Cryptosporidium, Salmonella), or occur as sporadic outbreaks, typically with highly suggestive signs (Infectious Laryngotracheitis). We generally monitor for and are well aware of the Mycoplasma status of our breederss. While these other diseases remain a threat and should be periodically ruled out, they are generally low on the list. IB and ND are on the top of our list for good reason: a mild to moderate, gradually spreading respiratory disease problem in a broiler complex in North America is usually IB or ND. What we face week in and week out, year in and year out, are the flocks that develop mild to moderate respiratory signs typically over 28 days of age, moderate mortality, and moderate (to sometimes severe) condemnation at processing. We must also constantly bear in mind the possible contributing factors such as Infectious Bursal Disease, Chicken Anemia Virus, other immunosuppressive agents, environmental factors, and so forth. The usual questions then become:

(1) Is this lentogenic ND or IB?
(2) If IBV, what serotype?
(3) Is it vaccine-induced or a vaccine failure?

The diagnosis and management of IB and ND occur on two levels: the individual flock and the population (typically a complex in the integrated industries, or a geographical region in less integrated industries). In the individual flock, when signalment, history, signs, and lesions are compatible with IB or ND, the diagnosis is made by demonstrating the current or past presence of the agent by antigen detection and serology, while simultaneously ruling out the other possibilities. It is clearly best to use both antigen detection and serology for both ND and IBV simultaneously, and not rely on one or the other test or pursue one or the other agent individually. Antigen detection is usually based on fluorescent antibody tests (FA), immunoperoxidase tests (IP), virus isolation in embryonating chicken eggs (VI), or RT-PCR based tests. Serology is typically ELISA or HI, rarely with VN.

Diagnosis of the individual flock can be difficult for a number of reasons. It is expected, especially in the early stages of an outbreak, that at least 40% of individual case workups will fail to establish a clear etiological diagnosis. It can be difficult to apprehend the culprit in every case in a population of birds with a life span of only 6 to 8 weeks, affected by an agent whose presence is fleeting, and which agent frequently strikes in the last week or two of that short lifespan. Timing is critical for recovery of the actual agent by any of the tests mentioned. Often, by the time a grower or field supervisor recognizes that a flock is ill (usually due to secondary bacterial infection), the primary viral agent is already gone. In addition, none of these techniques is 100% sensitive when the agent is present. The use of naive sentinels greatly increases the sensitivity of virus detection in flocks. Finally, the fact that most broilers are vaccinated with live viruses can make positive antigen detection open to interpretation. Serology is difficult to interpret because of previous immunization and the frequently short time available between infection and slaughter. The ability to hold affected birds in isolation for two to three weeks after disease onset can help, as can a regular serological monitoring program for market age birds, to establish the normal patterns for that age, locality, vaccination program, etc.

Because of the difficulties in individual flock diagnosis, and because our responses to control IB and ND are by necessity population-based, the final approach to these diseases in the either the integrated broiler industry or in a dense but non-integrated broiler production area is truly population-based. A single case of mild to moderate respiratory disease rarely attracts much attention and typically is not pursued very far diagnostically. Neither the professional or paraprofessional resources exist in most operations to pursue each and every mild to moderate deviation from normal. Several severe cases, or more commonly a pattern of repeated mild to moderate cases, do attract attention, and a diagnosis is pursued for the group problem. Diagnoses are indeed attempted on individual flocks, but yield clear answers only in a percentage of cases, so multiple affected flocks must usually be exam-

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ined. Likewise, preventative program responses typically are not made on the basis of these individual flock diagnoses, but rather on patterns of repeated diagnoses. Identification of an endemic agent in one or two initial cases, or even a new serotype in one or two cases, is rather thin evidence upon which to make a major program change in a complex. Prudent clinicians should make population program changes to solve a problem only when the nature of the population problem has been clearly established. When agent detection methods are used, how many times is each agent recovered on repeated attempts? It is common to recover more than one agent. Both IBV and NDV may be recovered, and more than one serotype of IBV may be recovered, especially in the case of vaccinated populations. Which agent predominates and is the likely cause of the problem? When using serology, variable titers to both IBV and NDV are common, and to different serotypes of IB on HI testing. What patterns emerge in comparison to the "normal" pre-disease pattern on repeated examinations? Our challenge is to establish the population diagnosis as rapidly as possible, but with as much surety of a favorable outcome as possible.

Program responses are likewise population-based, for a number of cogent reasons. It is logistically impossible for the number of management personnel in the typical complex to administer individually tailored preventative programs to each farm in the complex. To attempt such a course would result in misdirected and fouled programs on a routine basis. Uniformity of programs and economies of scale are an inherent part and advantage of the integrated system. In the case of IB, such an approach is also scientifically unwise, due to the variety of serotypes involved. Use of varied serotypes for different farms in the same hatchery on the same day is an invitation to disaster. Use of multiple serotypes in the field is almost as bad, considering the cross traffic in the typical complex. Finally, with competitive contracts, it is becoming a legal caveat that each and every grower be treated as close to the same as possible.

The situation may be somewhat different with a less integrated structure. Nevertheless, a given (and fortunately for us, a fairly narrow) subset of IB and ND agents tend to predominate in an area at a given time, and most of these concepts should apply to some degree.

## Antigen Detection and Characterization

Virus isolation is the "gold standard". Preferred tissues for lentogenic ND are tracheas or tracheal swabs. Tracheal swabs, trachea, lung, or cecal tonsils are all used for IBV. Embryonated SPF chicken eggs at 9 to 11 days of incubation are the best substrate for both viruses. Samples are best if obtained in the acute phase of the disease. The disease is frequently in the secondary bacterial stage by the time it is recognized, and viral recovery may be difficult in obviously ill birds. A few normal-appearing contacts therefore should be sampled as well. SPF sentinel birds are frequently more sensitive for VI than sick broilers because the timing of examination can be precisely determined and immunity does not interfere. Naive SPF leghorns are exposed to the sick flock for 5-7 days and then submitted. It is particularly effective to submit several birds per day between 5 and 7 days. We have also used sentinels vaccinated by eye drop and held in isolators, in order to rule out the vaccine strains in use. However, the author prefers to use naive, unvaccinated sentinels, for at least 3 reasons. First, the preparation of vaccinated sentinels is laborious, requiring that they be given the vaccine (usually by eye drop, to assure complete coverage) and then held in isolation for several weeks while they respond and clear the vaccine virus. Ideally, they should be tested to assure they are not shedding virus before placement in the field. Isolation space to do this is frequently at a premium. It is also an advantage to be able to place sentinels at a moment's notice, as when a good candidate flock is identified a week before it is scheduled for slaughter. Second, if the vaccine virus is causing the problem, I want to know that. For example, if I repeatedly place naive sentinels with 6-week-old broilers that were vaccinated 3.5 weeks earlier at 17 days of age, and they repeatedly pick up the vaccine virus, something is wrong. Finally, naive sentinels are more likely to pick up variants of vaccine viruses. For example, the 93 GA variant is an Ark that apparently has drifted significantly. The Ark vaccines protect against it reasonably well in laboratory challenge tests, but in the author's opinion, this protection is not quite as good in commercial situations. Sentinels not vaccinated for Ark are more likely to detect this variant. Knowledge that the problem is due to the variant, and not caused directly by the vaccine itself, allows one to defend continued use of the vaccine in the face of criticism of that vaccine, and to avoid the potentially costly error of abandoning that vaccine which, though not performing optimally due to the drift in the challenge variant, is actually preventing disaster.

The specimens should be delivered to the lab and inoculated immediately. If one is close to the lab, and adequate biosecurity can be maintained, submit live birds so that the freshest possible samples can be obtained. If there is a delay, organ samples or swabs should be chilled or frozen. 50% glycerol is also a good preservative. IBV may persist in the cecal tonsils for weeks. While recovery rates from cecal tonsils are higher, the virus isolated may not be the one causing the disease problem. Isolation of ND does not appear to be as problematic as for IB. The technology available for detecting and characterizing IBV has improved immensely over the past 10 years. Formerly, IBV could be difficult to isolate in many laboratories. Even in laboratories that were reasonably successful in isolating the virus, serotyping by cross-neutralization studies was slow, laborious, and expensive. There is now little excuse not to seek the "gold standard" of isolation and identification with today's techniques. As indicated previously, it is advisable to obtain multiple isolations and serotypings in order to establish with surety the primary cause of the outbreak. Two main techniques are now used: monoclonal antibodies and molecular biological techniques, with the molecular techniques now in more common use. The most common approach is to make one or more blind passes in embryonating eggs, and then test the allantoic fluid by RT-PCR. Various techniques are available, depending on the laboratory, including use of serotype-specific primers, RFLP of products from more general primers, and sequencing of PCR products. All concentrate on the S1 gene, whose protein product is the site of serotype specificity and virus neutralization.

Fluorescent antibody or immunoperoxidase techniques are also commonly used in many labs for antigen detection, especially for ND.

## Enzyme-Linked Immunosorbent Assay (ELISA)

Since VI is slower, more expensive, and sometimes negative, serology should be conducted simultaneously. ELISA is probably the most commonly used serological test because of the availability of commercial test systems, which allow large-scale testing at an economical price. Large-scale testing of numbers of flocks is critically important to establish patterns. In the US, two main systems are available: Synbiotics (formerly Kirkegaard and Perry Laboratories, KPL), and IDEXX Laboratories. The author has more experience with the IDEXX system, and further comments regarding specific titers pertain to that system. The two systems are comparable. The Synbiotics system has traditionally given lower responses for similar levels of antibody for NDV, and has a steeper slope across the range of IBV antibody levels, when compared to IDEXX. ELISA titers are useful, but not necessarily easy to interpret. A number of caveats must be borne in mind.

ELISA systems are subject to both internal and external variability. Antibody titers also do not necessarily correlate with protection from challenge. The ELISA responses to live vaccination programs are generally low and variable. In the author's opinion, ELISA systems are not especially useful for monitoring live vaccination programs in broilers or breederss. ELISA systems can be used successfully to monitor the

efficiency of vaccination in breederss where killed vaccines are used. The author finds them most useful in monitoring field challenge in populations of birds, especially broilers. ELISA titer patterns across age ranges (not the actual titers) in US broilers are fairly predictable for NDV and IBV (and IBD as well). They begin with a passive (maternal) titer, which varies with the vaccination program used in the breederss. (Killed vaccines result in higher maternal titers). The titers decline to a nadir, usually around 21 days of age. Then, the titers will begin to increase. The magnitude of the increase reflects the vaccination program, severity of viral challenge, and in the case of IB, serotype of challenge in relation to the vaccine serotype. Therefore, a sample obtained as late in the broiler's life as possible (prior to slaughter) is of the most value in monitoring and diagnosing exposure.

It should be emphasized that such testing programs are most useful in monitoring populations of flocks. The results on any single flock can be misleading, due to a variety of factors. An important factor is the timing of the sample. Many flocks are challenged shortly before slaughter. There may be elevated mortality at the time of slaughter, and excessive condemnations due to acute air sacculitis, but titers will be low because the birds have not had time to develop the IgG response measured by the ELISA test. Ideally, paired acute and convalescent samples should be compared in parallel. In cases of late breaks, isolation facilities can be used to hold a sample of exposed birds for 1-2 weeks after slaughter of the flock, for re-bleeding and testing at that time. Do not look too hard at individual birds. Do not even look too hard at individual flocks. Look at large-scale population patterns and trends. It is also useful to look at titer histograms on a number of flocks. In some cases of late breaks, in which mean flock titers are not especially high, one will repeatedly observe several individual birds with high titers. If this pattern is seen repeatedly, it may suggest that the vaccine is "rolling" in the flock, or that seroconversion is beginning to occur at the time the birds are bled. Holding birds for seroconversion is especially useful in cases such as these.

The manufacturers of the kits are reluctant to publish titers that correlate with exposure, disease, or protection, for good reason. For example, correlating titers with resistance to challenge requires an understanding of the specificity of the antibody, the virulence of the challenge organism, the antigenic characteristics of the challenge organism, the route of challenge, the dose, and the end-point used to determine resistance (death, signs, viral recovery, etc.) Determination of exposure is equally tricky. It is important to learn what is "normal" with the resident vaccination program and challenge, so that "abnormal" becomes easier to determine. The manufacturers stress the importance of establishing "baselines" in each operation. This sounds as if the manufacturers are evading the question, but it is advice that is best heeded. Nevertheless, most new clinicians want and need some sort of a ballpark starting point. The following titers reflect the author's experience in his company, and may or may not be applicable to other areas and situations:

IB:

| Scenario | Expected GMT Range (General Population) |
|---|---|
| 1. Mild vaccine (such as Mass or Conn), Low challenge levels | Very low titers, generally <1000, often much lower |
| 2. More aggressive vaccine (especially Ark), Low challenge levels | Moderate titers, ~1000-2500 |
| 3. More aggressive vaccine (e.g., Ark), Higher but essentially homologous to homologous variant challenge | Moderate titers, 2500-7000 in flocks experiencing challenge |
| 4. Heterologous challenge (i.e., "wrong vaccine!) | High titers, >7000, often 7000-14,000, occasionally 18,000-20,000 |
| 5. Killed vaccine, two doses, peak | >6500 |

ND:

| Scenario | Expected GMT Range (General Population) |
|---|---|
| 1. Mild vaccine (such as C-2), No challenge | Very low titers, generally <300, often <100 |
| 2. More aggressive vaccine (B1), Low challenge levels | Low titers, ~ <1000 |
| 3. More aggressive vaccine (B1), Higher challenge | Moderate titers, 1000-4500 in flocks experiencing challenge |
| 4. More aggressive vaccine (B1), Severe challenge | High titers, >5000, often to 15,000, |
| 5. Killed vaccine, two doses, peak | >10,000 |

In the author's company, we obtain blood samples from 15 birds, from a minimum of 6 flocks per week, at the slaughter plant. (Twenty to 30 birds is preferable from a statistical standpoint, but 15 is an economical compromise, which allows us to test 6 flocks on a 96-well plate, along with the positive and negative controls and two wells for an internal standard we use). If flocks are detected with respiratory lesions on the evisceration line, those flocks are preferentially selected for blood collection; otherwise, random flocks are chosen. If an outbreak occurs, more than six flocks may be sampled in a week. The geometric mean titer of each flock is plotted against time. This system has proven useful in population monitoring.

## HI Testing

HI testing helps to point us towards a particular serotype. HI test results are also subject to variability, especially between labs. Important factors include the incubation time, the number of HA units of antigen used, the proper preparation of the antigen (for the IBV HI test), and the reading of the endpoint. Again, it is difficult to correlate titers exactly with protection, exposure, or disease. In the author's experience with the HI tests at the University of Georgia, Poultry Diagnostic and Research Center, the following are "common" titers:

Mild NDV vaccine, two doses, mild exposure*: < NDV HI)
Arkansas IBV vaccine, moderate exposure: <128 (Ark IBV HI)

Arkansas IBV vaccine, heavy challenge: 256-1024 (Ark IBV HI)
Heterologous IBV challenge: >1024 (Ark IBV HI)
* The literature indicates that a single dose of a lentogen will produce an HI titer of 16 - 64. Ark SE17

One advantage of the HI test for IBV is that it is serotype-specific. A separate test is performed for each serotype. Most labs that do IBV HI can do Mass, Conn, and Ark; Del 072, FLA, SE 17, and others are available in some areas. The HI test measures IgM, so the response is quicker than with ELISA. However, there are some pitfalls to be avoided, the main one being the occurrence of cross-reactions after repeated exposures, and the other being the response to "unknown" or "untested" serotypes. We also find HI to be almost too sensitive. On our Ark DPI-Del 072 program, any significant challenge with either virus results in very high HI titers to both serotypes. Making a final serotypic diagnosis and instituting a vaccine change based only on HI testing is a risky proposition, but the test can be a useful adjunct.

When a naive bird is exposed to a single IBV serotype, say Mass, and then tested by HI for a variety of serotypes, the HI titer for the serotype to which the bird was exposed (in this case Mass) will usually be obviously higher than the titers for the other serotypes, and the answer is fairly clear. However, if a bird is exposed to one serotype, then later to another serotype, the picture is much less clear. Serum from a secondary immune response produces considerable crossreaction in this test, and the titers to a variety of serotypes may be elevated. For example, say a group of broilers was vaccinated for Mass at day 1, and experienced a challenge with Conn at 4 weeks, and you had collected serum every 2 weeks and tested for a whole panel of serotypes. The results might look something like this:

| | Mass | Ark | Conn | Fla | SE17 |
|---|---|---|---|---|---|
| 2 weeks | 32 | 8 | 8 | 8 | 8 |
| 4 weeks | 32 | 8 | 8 | 8 | 8 |
| 6 weeks | 128 | 48 | 256 | 70 | 90 |

The Mass was somewhat elevated early on due to vaccination. When the exposure to Conn occurred, it increased Conn titers the most, but it also "dragged" everything else with it, due to this cross-reaction common with the secondary or anamnestic response. The Mass was increased considerably due to the previous exposure. Some refer to this as "original antigenic sin". In a disease situation, where the challenge is unknown, these results would suggest that Conn might be responsible, or that the culprit is at least more closely related to Conn than to the others, but they do not prove the serotype. Since most broilers are vaccinated, this would be a common problem. However, testing multiple flocks will again help establish a pattern. If you are unable to actually isolate and type the virus for some reason, HI results give a little more information upon which to select a vaccine serotype than a blind shot in the dark. They are also quicker, cheaper, easier, and possibly better than crude challenge tests for making the initial decision. [Note: the described behavior of HI tests should not be construed to mean that vaccination with one serotype, followed by vaccination with a different serotype, will engender widespread cross-protection against challenge with multiple serotypes. Unfortunately, this is not the case; our lives would be much simpler if it were.]

## Summary of the Diagnostic Approach

The occurrence of repeat cases of a respiratory syndrome indicates the potential start of an outbreak. Consideration of signalment, signs, and lesions, along with knowledge of previous and current endemic diseases, leads to an ordered list of differential diagnoses. In North America, IB and ND are typically at the head of that list. Simultaneous examination of several initial cases for the most likely or dangerous differentials will generally begin to rule out the more exotic considerations, leading to a concentration on lentogenic ND and IB. Examination of a series of cases by both antigen detection techniques (preferably VI) and serology (usually ELISA and/or HI) should give an indication of which agent is the primary cause. In the case of IB, determination of serotype is critical. ELISA is of no help in this regard, and HI is frequently of limited help. Identification of the serotype by monoclonal antibody or molecular techniques, preferably again on a series of cases, is absolutely necessary for successful resolution of the problem. It is important to remember that there is generally very little cross-protection among different serotypes of IB. Particularly in commercial situations, one needs to use the homologous serotype to achieve reasonable protection.

## Preventative Measures for IB

The key to control of endemic strains of NDV and IBV is proper selection and application of vaccine. In the case of IBV, selection of the proper serotype is critical. Clearly identify the problem, down to the specific serotype. Multiple attempts at VI may be necessary, even after ELISA and HI serology suggest the possible answer. Isolation of the new type in at least several different flocks is suggested, to make certain that a single isolation is not a fluke. We can sometimes see several unknown variants per year, that are never seen again, and that apparently represent spontaneous mutations that (fortunately) did not become established. Do not start plugging in new serotype vaccines to see if something will work. If the problem is that severe, the virus should be detectable with current technology, given sufficient effort.

Once an IBV problem has been identified and typed, there are 3 possible scenarios for action.

1. If the IBV serotype is different from your current vaccine program, and particularly if the problem is relatively widespread, and a vaccine exists, the answer is simple-switch to the correct vaccine. This scenario frequently is associated with some of the most severe problems, but is probably the easiest to identify and correct. For example, Ark was not recognized in Georgia prior to 1992, and the vaccine was not in use. An epornitic due to Ark occurred in the spring of 1992. Clinical signs and lesions were particularly severe, and HI testing was indicative of Ark exposure, even though the birds had been vaccinated twice with Mass-Conn. The Ark serotype was identified with monoclonal antibody techniques. Institution of vaccine programs with Ark resulted in immediate and dramatic improvement in several complexes in north Georgia that fall.

2. If the IBV serotype is the same as one of your vaccines (and you have confidently ruled out the other differential diagnoses), and particularly if the problem is sporadic, then it is likely that the administration of the vaccine is at fault. Possible problems to be investigated include the timing, dose, passage level or brand, method of administration, care in administration, water quality, and so forth. Proper application is paramount with IBV vaccines. Unlike B1, which is naturally attenuated, the various IBV vaccines are attenuated from wild field strains, and gain virulence upon back passage in chickens. Complete coverage of the entire flock is therefore necessary regardless of the method used. Alternatively, other management factors such as temperature, ventilation, or litter management may be involved, or contributing factors such as immunosuppressive disorders (IBD, CAV, etc.), ORT, Bordetella, cryptosporidiosis, and so forth should be investigated. This is a much more difficult situation to confidently identify and fix.

3. If a new serotype is found, or even a significant variant, for which there is not a vaccine available, the diagnosis is fairly clear, but the solution is difficult. The immediate course should be to perform challenge studies with this virus against the available vaccines, and select the best

vaccine on that basis. Alternatively, cross VN studies may help narrow down the choices of vaccine to test either in the lab or field. In the meantime, efforts could be made to entice a vaccine company to attempt to attenuate the virus and produce a vaccine, as was done with Del 072. This is an extremely difficult proposition, and, due to the government approval process necessary for any live vaccine, would take a considerable amount of time (2 years +) even with the best of luck. In the case of a variant, consider the closest vaccine serotype you have available, and use that vaccine as aggressively as you can. There is little to no cross-protection between different serotypes—that is why they are classed as serotypes. Also, with IBV, mixing blue and yellow will not necessarily give green. Trying to solve a new serotype problem by using random combinations of existing vaccines is not likely to yield beneficial results in the absence of challenge data to indicate that such a combination may give some crossprotection. I believe such instances are very rare.

We have also experienced another, unreported phenomenon that can mimic Scenario 2. The normal course of events expected from a live IBV vaccine is for the birds to become infected, exhibit a mild reaction, shed the virus for a few days, then respond immunologically and clear the vaccine virus. In the case of some serotypes, especially Ark and possibly Delaware 072, prolonged shedding appears to be common. When these vaccines are in use, they tend to be the only serotype recovered from both sick birds and sentinels. However, we have evidence that other serotypes, or wild variants of the vaccine serotype, can be present, and possibly causing the disease problems, but not recoverable because of the presence of the vaccine virus. These variant viruses will challenge the vaccine protection, they will elicit ELISA titers (in our case) in the 4000-6000 GMT range, and they can cause minor to moderate disease and condemnation problems in birds properly vaccinated with the related vaccine strain. They can cause major problems in birds vaccinated only with unrelated strains. The variants become evident when the related vaccine is removed. One would assume that the vaccine virus, being egg-adapted, is preferentially isolated, and in effect masks the presence of the variant when VI is done in eggs. Scenario 3 then becomes more applicable, and continued use of the same vaccines may be the best course of action. Two actual examples of this phenomenon in the field are offered.

We experienced moderate disease and condemnation problems 1996. Extensive serological and VI studies revealed only Ark IBV. These findings eventually persuaded us to blame and abandon the Ark DPI vaccine, and revert to a simple B1-Mass program. The problems actually were due, in retrospect, to a variant Arkansas IBV, named Georgia 93, along with other management issues. As soon as the Ark DPI vaccine was abandoned, and before clinical problems resulted, ELISA titers in market-aged birds rose to high levels. This was due to heterologous challenge, specifically variant Ark (Georgia 93) challenge of Massvaccinated birds. The Georgia 93 variant had not been detected previously in this operation, in spite of a concerted effort at VI that spring. It was detected in profusion as soon as clinical problems prompted us to resume VI attempts. Those problems were severe, with exorbitant levels of condemnation. When the Ark DPI vaccine was replaced, and the problem began to abate, virus isolations again shifted away from Georgia 93 and back to Ark DPI, to the point that Georgia 93 eventually was no longer isolated. It is debatable when or whether the Georgia 93 variant actually disappeared.

We replicated this exact same experience with the test removal of the Ark DPI and Del 072 vaccines in spring of 2000. In that case, we had not detected Georgia 98 (an apparent derivative of Del 072) in our operation, but had reason to believe (based on signs and serology) that flocks were being challenged on our Ark DPI-Del 072 program, possibly with a variant of either Ark or Del 072.

We placed flocks vaccinated with Connecticut only in the affected area. IBV ELISA titers in market-aged birds immediately increased, clinical disease occurred immediately, and the Georgia 98 strain was isolated. Resumption of the Del 072 vaccine resulted in abatement of the problem, a decrease in marketage titers, and failure to isolate any further Georgia 98.

Several other caveats are important in using ND and IBV vaccines. Use enough. Don't cut doses excessively. Use multiple administrations (e.g., day-of-age and field boosting) if necessary. Obviously, give the vaccine correctly. Avoid using more viruses than you need. If your problem is IBV, and evidence indicates that NDV challenge is low, dial the NDV program down to as low a level as you can. This will lessen the vaccine stress on the birds, and afford the IBV vaccines a better opportunity to accomplish their missions with minimal interference.

Avoid multiple serotype combinations of IBV vaccines. For example, if the program has been B1-Mass-Conn for years, and an Ark 99 problem occurs, and there is no evidence of a Conn problem, don't add Ark to the B1-Mass-Conn. Drop the Conn. All of these IBV's compete for the same receptors on the same cells. Give the serotype(s) you really need the best opportunity to immunize the birds, and forget the ones you don't currently have. If the above rule results in unconventional (and not government-sanctioned) combinations, do not hesitate to test these combinations. If possible, single antigens should be mixed by titer, not by "dose".

The concept of interference between vaccines can have real consequences. This is a documented phenomenon with NDV and IBV. In particular, the IBV will interfere with NDV. In US vaccines, the manufacturer must demonstrate the efficacy of vaccine combinations for all of the viruses present. Most manufacturers ensure NDV efficacy by maintaining the NDV titers at least 1.5-2 log 10 higher than the IBV. Most vaccines are released with IBV titers well in excess of 3.5 log 10 and NDV in excess of 6.0 log 10. Cutting the vaccine dose to adjust the reaction (due mainly to NDV) may result in insufficient IBV titers and a "rolling" vaccine reaction due to back passage. Conversely, mixing of vaccines may result in interference between different IBV strains or between IBV and NDV.

The concept of "original antigenic sin" also appears to apply to IBV vaccine programs. We have found that, when we have a serious IBV challenge, we have better success when the needed serotype is given at day 1, and then boosted at 2-3 weeks if needed. Application in the boost alone does not seem to provide the needed control. The Arkansas serotype also appears to be a special case, from two standpoints. Based on the author's field observations, it appears to be especially adept at interfering with B1 NDV vaccine, resulting in rolling NDV reactions. Also, it tends to shed for prolonged periods itself, and a second dose is recommended to end this shed.

In summary, the best approach to controlling IBV is to clearly identify the serotype via multiple isolations and typing, and use the proper vaccine. If isolation and typing are not available, there are other (but unfortunately less desirable) approaches to use as a compromise.

1. Make an educated guess, based on ELISA, HI results, reports from neighboring areas, etc. If your guess is correct, the desired response is likely. However, if you guess incorrectly, not only will the problem persist, it may actually get worse, because now you have introduced a new serotype which was not needed.

2. Vaccinate test birds and hold them in isolation. Then challenge them with your isolate (if you can get it), or even ground-up tracheas from sick birds. Use the vaccine that protects best. This technique is also slow and laborious. Furthermore, if you pick the "wrong" sick flock as the source of the tracheas, the answer could be "wrong" also, since the virus was never identified. Using multiple challenge sources increases the facilities and workload, and secure facilities to contain the test vaccine viruses are hard to locate.

Maternal antibody titers to IBV have less effect on either protection of progeny or response to live vaccines. In the US, about 1/3 of the companies use a killed IBV in the pullet program, and 2/3 do not. The live and killed programs parallel the NDV program, as the NDV and IBV vaccines are usually combined.

# Excerpts from the latest USDA National Agricultural Statistics Service (NASS) "Broiler Hatchery," "Chicken and Eggs" and "Turkey Hatchery" Reports and Economic Research Service (ERS) "Livestock, Dairy and Poultry Situation Outlook"

## Broiler-Type Eggs Set In 19 Selected States Down Slightly

According to the latest National Agricultural Statistics Service (NASS) reports, commercial hatcheries in the 19-State weekly program set 213 million eggs in incubators during the week ending July 29, 2006. This was down slightly from the eggs set the corresponding week a year earlier. Average hatchability for chicks hatched during the week was 83 percent. Average hatchability is calculated by dividing chicks hatched during the week by eggs set three weeks earlier.

## Broiler Chicks Down 1 Percent

Broiler growers in the 19-State weekly program placed 173 million chicks for meat production during the week ending July 29, 2006. Placements were down 1 percent from the comparable week a year earlier. Cumulative placements from January 1, 2006 through July 29, 2006 were 5.23 billion, down 1 percent from the same period a year earlier.

## June Egg Production Up 1 Percent

U.S. egg production totaled 7.38 billion during June 2006, up 1 percent from last year. Production included 6.33 billion table eggs, and 1.06 billion hatching eggs, of which 991 million were broiler-type and 66 million were egg-type. The total number of layers during June 2006 averaged 342 million, up 1 percent from last year. June egg production per 100 layers was 2,159 eggs, down slightly from June 2005.

All layers in the U.S. on July 1, 2006, totaled 341 million, up 1 percent from last year. The 341 million layers consisted of 284 million layers producing table or market type eggs, 54.0 million layers producing broilertype hatching eggs, and 2.82 million layers producing egg-type hatching eggs. Rate of lay per day on July 1, 2006, averaged 72.7 eggs per 100 layers, up slightly from July 1, 2005.

## Egg-Type Chicks Hatched Up 8 Percent

Egg-type chicks hatched during June 2006 totaled 37.7 million, up 8 percent from June 2005. Eggs in incubators totaled 32.1 million on July 1, 2006, down 10 percent from a year ago.

Domestic placements of egg-type pullet chicks for future hatchery supply flocks by leading breederss totaled 183,000 during June 2006, up 5 percent from June 2005.

## Broiler-Type Chicks Hatched Down 1 Percent

Broiler-type chicks hatched during June 2006 totaled 788 million, down 1 percent from June 2005. Eggs in incubators totaled 650 million on July 1, 2006, down 1 percent from a year earlier.

Leading breederss placed 7.64 million broiler-type pullet chicks for future domestic hatchery supply flocks during June 2006, up 7 percent from June 2005.

## Turkey Eggs in Incubators on July 1 Up 5 Percent

Turkey eggs in incubators on July 1, 2006, in the United States totaled 32.2 million, up 5 percent from July 1 a year ago. Eggs in incubators were up 2 percent from the June 1, 2006 total of 31.6 million eggs. Regional changes from the previous year were: East North Central up slightly, West North Central up 2 percent, North and South Atlantic up 16 percent, and South Central and West combined, up 1 percent.

## Poults Placed During June Up 6 Percent From Last Year

The 25.7 million poults placed during June 2006 in the United States were up 6 percent from the number placed during the same month a year ago. Placements were 2 percent above May 2006. Regional changes from the previous year were: East North Central up 1 percent, West North Central up 5 percent, North and South Atlantic up 18 percent, and South Central and West were down 9 percent.

## Broiler Meat Production Estimates for Third and Fourth Quarters Decreased

According to the latest Economic Research Service (ERS) reports, U.S. broiler meat production was up 4.1 percent in first-quarter 2006, and the estimate for the second quarter is 9.1 billion pounds, a 1.9-percent increase from last year. With the current pace of chicks being placed for growout being below a year earlier for the last several months, and a reduction in the number of replacement pullets being placed in the hatching flock, the production estimates for the third and fourth quarters of 2006 were lowered by 100 million pounds each. This makes the production total for the second half of 2006 at just under 18 billion pounds, which would be less than 1 percent higher than in the second half of 2005. The production estimate for 2007 was also reduced a total of 250 million pounds to 36.6 billion

*USDA Reports continued from page 5*

pounds, 1.6 percent higher than in 2005.

Over the last 5 weeks (June 10 to July 8) the average weekly number of chicks being placed for growout was 174 million, down 2.1 percent from the same period in 2005. Chicks being placed for growout at the beginning of July would likely be going to slaughter in the second half of August.

Broiler meat production in May totaled 3.2 billion pounds, up 6 percent from a year earlier. A large portion of this increase was due to May 2006 having one more slaughter day than May 2005. The increase in meat production was due to a combination of a larger number of birds slaughtered (up 4 percent) and an increase in the average weight of those birds at slaughter (up 2 percent). Preliminary data point towards only a small increase in total broiler meat production in June, as an increase in the average weight at slaughter is expected to just offset a decline in the number of birds being slaughtered.

With growth in broiler production expected to slow in the second half of 2006, prices for most broiler products are projected to gradually strengthen compared with the first half of 2006. Prices for whole birds have averaged 61.9 cents per pound over the first half of 2006. This is about 14 percent lower than in the same period in 2005, which was down 5 percent from the first half of 2004. However, whole bird prices have been rising in the last several weeks, and the average price for June was 64.4 cents per pound, the highest this year, but still 11 percent below the price in June 2005. Prices for many other broiler parts have also increased, but like whole birds, prices for most broiler parts are still lower than a year earlier.

With a forecasted increase in broiler meat production of less than 2 percent in the second quarter and a strong export demand, the estimate for 2006 ending stocks was lowered. The estimate for the yearend ending stocks is now 750 million pounds, down 50 million pounds from the previous estimate.

## Turkey Production Rises in May

Domestic turkey production totaled 495 million pounds in ay, up 7 percent from the previous year. Again a large proportion of the production increase stemmed from May having an additional slaughter day compared with a year earlier. The number of turkeys slaughtered was up 6 percent and the average weight at slaughter was 28.6 pounds, up 1 percent from the previous year. Even with the increase in May, turkey meat production for the first 5 months has increased only 2 percent from the same period in 2005. All that increase has come from an increase in the number of birds slaughtered, as the average weights over the first 5 months of 2006 are about even with the same period in the previous year.

Turkey meat production in the second half of 2006 is estimated at 2.85 billion pounds, up 2.7 percent from second-half 2005. The most recent turkey hatchery report showed that the number of poults being placed for growout in June was 6.1 percent higher than the previous year. Over the first half of 2006, the total number of poults placed for growout was 147 million, up 7 percent from the same period in 2005.

Relatively small increases in turkey meat production, along with lower ending stock levels, have meant that whole turkey prices have remained slightly higher than yearearlier levels. Prices for whole turkeys averaged 71.3 cents per pound in the second quarter, up about 5 percent from the previous year. With only a small increase in production expected for second-half 2006, whole bird prices are expected to remain somewhat higher than those of a year earlier through the third quarter, but are expected to fall below their year-earlier level in the fourth quarter. Prices for other turkey products have not increased much due to some weakness in the export market. The export market for turkey products is expected to strengthen somewhat as broiler prices continue to increase.

At the end of May cold storage holdings of turkey products totaled 466 million pounds, about the same as the previous year. However, there was a large difference in the cold storage holdings for whole turkeys and turkey parts. Cold storage holdings for whole turkeys totaled 218 million pounds, down 10 percent from the same period in 2005. For turkey parts, cold storage holdings totaled 248 million pounds, up 11 percent from the same period in 2005.

# Meetings, Seminars and Conventions

## 2006 September

**Sept. 4-6:** ***XVIII International Poultry Symposium,*** Rogow near Koluszki, Poland. Contact: Prof. Ewa Swiercaewska. Phone: +48 22 593 6558; Email: riedel@alpha.sggw.waw.pl

**Sept. 10-14:** ***12th European Poultry Conference,*** Veronafiere Congress Centre, Verona, Italy. Contact: Secretariat XII WPSA European Conference, Department of Food Science, Via San Giacoma 9, 40126 Bologna, Italy. Phone: +39 051 209 4221; Fax: +39 051 251 936; Email: wpsa@alma.unibo.it; Website: www.epc2006.veronafiere.it

**Sept. 12-13:** ***Poultry Production and Health Seminar,*** Memphis, Tennessee. Contact: U.S. Poultry & Egg Association. 1530 Cooledge Road, Tucker, GA 30084-7303. Phone: 770-493-9401; Fax: 770-493-9257

**Sept. 12-14:** ***Iowa Poultry Association Fall Festival,*** Arrowood Resort and Conference Facility, Lake Okoboji, Iowa USA. Contact: Kevin Winchattle, 8515 Douglas Avenue, Suite 9, Urbandale, Iowa 50322. Phone: 515-727-4701; Fax: 515-727-4707. Email: kevin@iowapoultry.com. Website: www.iowaegg.org

**Sept. 13:** ***Delmarva Breeders, Hatchery and Grow-out Conference,*** Location to be determined. Contact: Bud Malone, University of Delmarva. Phone: 302-856-7303

**Sept. 27-29:** ***14th Baltic Poultry Conference,*** Vilnius, Lithuania. Contact: Studentu g. 39, Vilnius, Lithuania. Phone/Fax: +370 5275 7095. Email: bamlab@vpu.it

**Sept. 27-29 :** ***VIV China 2006,*** (Postponed from June 2006-dates not yet specified), Beijing, P.R. China. Contact: VNU Exhibitions Europe B.V., PO Box 8800, 3503 RV Utrecht, The Netherlands. Phone: +31 30 295 2772; Fax: +31 30 295 2809; Email: viv.china@vnuexhibitions.com; Website: sites.vnuexhibitions.com/sites/viv or Mr. Ruifent Xu, CNAVS Trade Fair Office. Phone +86 10 649 50 373; Fax: +86 10 649 50 374; Email: rfxu@china-av.net

## 2006 October

**October 3-4:** ***Poultry Federation Symposium,*** Springdale Holiday Inn, Springdale, Arkansas. Contact: Judity Kimbrell, The Poultry Federation, P.O. Box 1446, Little Rock, Arkansas 72203. Phone: 501-375-8131: Fax: 501-375-5519

**October 9-11:** ***National Meeting On Poultry Health & Processing,*** Clarion Resort Fountainebleau Hotel, Ocean City, Maryland. Contact: Karen Adams, Delmarva Poultry Industry, Inc., 16686 County Seat Highway, Georgetown, Delaware 19947-4881. PPhone: 302-856-9037; Fax: 302-856-1845. For information about meeting rooms and food accommodations at the Clarion Resort Fountainebleau Hotel, contact Kay Windwor, Phone: 1-800-638-2100.

**October 10-11:** ***Alabama Broiler Industry Seminar,*** Auburn University Hotel and Dixon Conference Center, Auburn, Alabama. Contact: Alabama Poultry & Egg Association, P.O. Box 240, Montgomery, AL 36101-0240. Phone: 334-265-2732; Fax: 334-265-0008.

**October 10-14:** ***World Poultry Science Association (WPSA) European Poultry Conference 2006,*** Verona, Italy. Contact: Secretariat - XII WPSA European Converence, Department of Food Science, University of Bologna, Via San Giacomo 9, 40126 Bologna, Italy. Phone: +39 041 209 4221; Fax: +39 051 251 936; Email: epc2006@wpsa.it; Website: www.epc2006.veronafiere.it

**October 12-13:** ***Poultry Protein And Fat Seminar,*** Nashville, Tennessee. Contact: U.S. Poultry & Egg Association, 1530 Cooledge Road, Tucker, Georgia 30084-7303. Phone: 1-770-493-9401; Fax: 1-770-493-9257.

**October 17-20:** ***16th Expoaviga,*** Gran Via 2 exhibition complex, Barcelona, Spain. Contact: Fira Barcelona, Av. Reina Maria Cristina s/n, 08004 Barcelona, Spain. Phone: +34 655 98 53 56; Fax: +34 93 233 21 77; Email agurri.premsa@firabcn.es: Website: www.expoaviga.com

**October 25-26:** ***North Carolina Broiler Breeders and Hatchery Management Conference,*** Statesville, North Carolina. Contact: Mike Wineland. Phone: 919-515-5529

## 2006 November

**November 6-7:** ***4th World Mycotoxin Forum,*** Cincinnati, Ohio. Contact: Bastiaanse Communication, P.O. Box 179, 3720 AD Bilthoven, The Netherlands. Phone: +31 30 229 4247; Fax: +31 30 225 2910; Email: wmf@bastiaanse-communication.com; Website: www.bastiaanse-communication.com

**November 7-8:** ***Alabama Breeders/Hatchery Workshop,*** Auburn University Hotel annd Dixon Conference Center, Auburn, Alabama. Contact: Alabama Poultry & Egg Association, P.O. Box 240, Montgomery, AL 36101--0240. Phone: 334-265-2732; Fax: 334-265-0008

**November 14-17:** ***EuroTier 2006,*** Hanover, Germany. Contact: DLG (Deutsche Landwirtschafts-Gesellschaft e.V.), Eschborner-Landstrasse 122, 60489 Frankfurt-am-Main, Germany. Phone: +49 69 24788 265; Fax: +49 69 24788 113; Email: eurotier@DLG-Frankfurt.de; Website: www.eurotier.de

## 2007 January

**Jan. 22-23:** ***International Poultry Scientific Forum,*** Georgia World Congress Center, Atlanta, Georgia. Contact: International Poultry Scientific Forum. Phone: 770-493-9401; Fax: 770-493-9257. sEmail: poultryscientificforum@poultryegg.org. Website: www.poulryegg.org

**Jan. 24-26:** ***2007 International Poultry Exposition,*** Georgia World Congress Center, Atlanta, Georgia, USA. Contact: US Poultry & Egg Association, 1530 Cooledge Road, Tucker, Georgia 30084 USA. Phone: +1 770 493 9401; Fax: +1 770 493 9257; Website: www.poultryegg.org

## 2007 February

**Feb. 12-14:** ***Australian Poultry Science Symposium 2007,*** University of Sydney, Sydney, Australia. Contact: Poultry Research Foundation, University of Sydney, 425 Werombi Road, Camden, NSW 2570, Australia. Phone:+61 2 46 550 656; Fax: +61 2 46 550 693; Website: www.vetsci.usyd.edu.au/apss

## 2007 March

**March 1-3:** ***5th International Poultry Show and Seminars 2007,*** Dhaka, Bangladesh. Contact: International Seminar, Dr. Q.M.E. Huque, Bangladesh Livestock Research Institute, Savar, Dhaka 1341, Bangladesh. Phone: +8802 770 8324: Fax +880 2 770 8325; Email: qmehuque@bangla.net

**March 7-9:** ***VIV Asia 2007,*** Bangkok, Thailand. Contact: VNU Exhibitions Europe B.V., P.O. Box 8800, 3503 RV Utrecht, The Netherlands. Phone: +31 30 295 2778: _+66 2 229 3737; Fax: +31 30 295 2809; Website: www.viv.net

# Meetings, Seminars and Conventions

**March 7-8:** ***Nebraska Poultry Industries Annual Convention,*** New World Inn & Conference Center, Columbus, Nebraska. Contact: Nebraska Poultry Industries, Inc., University of Nebraska, A103 Animal Sciences, P.O. Box 830908, Lincoln, Nebraska 68583-0908. Phone: 402-472-2051.
**March 12-15:** ***PEPA Convention,*** Loews Coronado Bay Resort, Coronado, California. Contact: Pacific Egg & Poultry Associatioin, 1521 I Street, Sacramento, California 95814. Phone: 916 441 0801; Fax: 916 446 1063.
**March 13-15:** ***Midwest Poultry Federation Convention 2007,*** St. Paul, Minnesota USA. Contact: Midwest Poultry Federation, 108 Marty Drive, Buffalo, Minnesota 55313 USA. Phone: +1 763-682-2171; Fax: +1 763-682-5546;
Email: Nicole@midwestpoultry.com;
Website: www.midwestpoultry.com
**March 27-29:** ***4th Internatioinal Poultry Conference,*** Sharm El-sheikh, Egypt. Contact: Dr. MA. Kosba, Facultry of Agriculture, Alexandria University, Alexandria, Egypt. Phone: +20 35 921960; Fax _20 35 231939; Email: mkosba@hotmail.com

## 2007 May

**May 8-10:** ***Victam International 2007,*** Jaarbeurs Hall, Utrecht, The Netherlands. Contact: Victam International BV, P.O. Box 197, 38600 AD Nijkerk, The Netherlands. Phone: +31 33 246 4404; Fax: +31 33 246 4706; Website: www.victam.com
**May 21-23:** ***VIV Russia 2007,*** Moscow, Russia. Contact: VNU Exhibitions Europe B.V., P.O. Box 8800, 3503 RV Utrecht, The Netherlands. Phone: +31 30 295 2772; Fax: +31 30 295 2809; Email: viv.russia@vnuexhibitions.com; Website: www.viv.net

## 2007 June

**June 28-30:** ***VIV Turkey 2007,*** Istanbul, Turkey. Contact: Richard deBoer, VNU Exhibitions Europe, P.O. Box 8800, 3503 RV Utrecht, Netherlands. Phone: +31 30 295 2714; Fax: +31 30 295 2809;
Email: richard.de.boer@vnuexhibitions.com;
Website: vnuexibitions.com or www.viv.net

## 2007 October

**October 8-10:** ***2007 National Meeting on Poultry Health Processing,*** Clarion Resort Fountainebleau Hotel, Ocean City, Maryland. Contact: Karen Adams, Delmarva Poultry Industry, Inc., 16686 County Seat Highway, Georgetown, Delaware 19947-4881. Phone: 302-856 9037; Fax: 302-856-1845. For information about meeting rooms and food accommocations at the Clarion Resort Fontainebleau Hotel, contact Kay Windsor, Phone: 800-638-2100.

## 2008 January

**January 23:** ***International Poultry Expo 2008,*** Georgia World Congress Centre, Atlanta, Georgia. Contact: UD Poultry & Egg Association, 1530 Cooledge Road, Tucker, Georgia 30084-7804. Phone: 1-770-493-9401; Fax: 1-770-493-9257;
Email: expogeneralinfo@poultryegg.org.
Website: www.poultryegg.org or www.ipe08.org

## 2008 March

**March 5-6:** ***Nebraska Poultry Industries Annual Convention,*** New World Inn & Conference Center, Columbus, Nebraska. Contact: Nebraska Poultry Industries, Inc. University of Nebraska, A103 Animal Sciences, PO Box 830908, Lincoln, Nebraska 68583-0908; Phone: 1-402-472-2051.

**March 5-7:** ***Victam Asia 2008,*** Bangkok, Thailand. Contact: Henk van de Bunt, Victam International B.V., P.O. Box 197, 3860 AD Nijkerk, The Netherlands, Phone: +31 33 246 4404, Fax: +31 33 246 4706, Email: expo@victam.com; Website: www.victam.com or Contact: Mr. Phusit Sasitaranondha, Thailand, Phone: +66 2 640 8013; Fax: +66 2 664 2076; Email: phusit@expolink.net

## 2008 June

**July 29-July 4:** ***XXIII World's Poultry Congress,*** Convention and Exhibition Centre, Brisbane, Australia. Contact: WPC 2008 Congress, Intermedia Convention & Event Management, PO Box 1280, Milton, Queensland 4064, Australia,
Phone: +61 7 3858 5594; Fax: +61 7 3858 5510;
Email: wpc2008@im.com.au;
Website: www.wpc2008.com

## 2008 July

**July 8-12:** ***Poultry Science Association Annual Meeting 2007,*** San Antonio, Texas. Contact: Website: www.poultryscience.org

## 2008 August

**August 10-15:** ***XXIII World's Poultry Congress,*** Convention and Exhibition Centre, Brisbane, Australia. Contact: WPC 2008 Congress, Intermedia Convention & Event Management, PO Box 1280, Milton, Queensland 4064, Australia.
Phone: +61 7 3858 5594; Fax: +61 7 3858 5510;
Email: wpc2008@im.com.au; Website: www.wpsa.info
**August 17-21:** ***8th International Marek's Disease Symposium,*** Townsville, Queensland, Australia. Contact: Dr. G. Burgess, School of Veterinary & Biomedical Sciences, James Cook University, Townsville, Queensland 4811, Australia.
Phone: +61 7 4781 5472; Fax: +61 7 4781 6833;
Email: graham.burgess@jcu.edu.au
**August 26-30:** ***16th European Symposium on Poultry Nutrition,*** Strasbourg, France. Contact: Groupe Francais de la WPSA, BP 5, 37380 Nouzilly, France. Fax: +33 2 47 56 11 39;
Email: WPSAFrance@aol.com;
Website: www.wpsa.fr

## *Reminder*

All previous issues of the Poultry Informed Professional are archived on our website www.avian.uga.edu under the Online Documents and The Poultry Informed Professional links.

## Broiler Whole Bird Condemnation (Region)

| | SW | Mid-West | S. East | Mid-Atlantic | S. Central |
|---|---|---|---|---|---|
| % Septox | 0.103 | 0.176 | 0.161 | 0.256 | 0.148 |
| % Airsac | 0.063 | 0.063 | 0.044 | 0.043 | 0.064 |
| % I.P. | 0.009 | 0.027 | 0.007 | 0.009 | 0.043 |
| % Leukosis | 0.001 | 0.038 | 0.002 | 0.000 | 0.001 |
| % Bruise | 0.003 | 0.003 | 0.001 | 0.001 | 0.004 |
| % Other | 0.002 | 0.007 | 0.011 | 0.004 | 0.007 |
| % Total | 0.176 | 0.303 | 0.226 | 0.314 | 0.266 |
| % 1/2 parts condemnations | 0.228 | 0.349 | 0.327 | 0.669 | 0.351 |

Data for week ending July 29, 2006

## Broiler Performance Data (Company)
## Live Production Cost

| | Average Co. | Top 25% | Top 5 Co.'s |
|---|---|---|---|
| Feed cost/ton w/o color ($) | 150.23 | 149.90 | 148.04 |
| Feed cost/lb meat (¢) | 14.17 | 12.80 | 12.81 |
| Days to 4.6 lbs | 43 | 43 | 41 |
| Chick cost/lb (¢) | 4.13 | 5.41 | 5.51 |
| Vac-Med cost/lb (¢) | 0.09 | 0.08 | 0.02 |
| WB & 1/2 parts condemn. cost/lb | 0.14 | 0.11 | 0.11 |
| % mortality | 4.81 | 3.53 | 3.57 |
| Sq. Ft. @ placement | 0.83 | 0.72 | 0.72 |
| Lbs./Sq. Ft. | 6.83 | 5.57 | 5.64 |
| Down time (days) | 13 | 14 | 15 |

Data for week ending July 29, 2006

## Broiler Whole Bird Condemnation (Company)

| | Average Co. | Top 25% | Top 5 Co.'s |
|---|---|---|---|
| % Septox | 0.158 | 0.087 | 0.150 |
| % Airsac | 0.065 | 0.041 | 0.027 |
| % I.P. | 0.022 | 0.022 | 0.008 |
| % Leukosis | 0.008 | 0.002 | 0.001 |
| % Bruise | 0.003 | 0.003 | 0.002 |
| % Other | 0.007 | 0.006 | 0.003 |
| % Total | 0.253 | 0.161 | 0.190 |
| % 1/2 parts condemnations | 0.330 | 0.247 | 0.244 |

Data for week ending July 29, 2006

| Broiler Performance Data (Region) Live Production Cost | | | | | |
|---|---|---|---|---|---|
| | SW | Midwest | Southeast | Mid-Atlantic | S-Central |
| Feed cost/ton w/o color ($) | 152.43 | 151.07 | 150.03 | 138.87 | 148.43 |
| Feed cost/lb meat (¢) | 14.24 | 14.58 | 13.74 | 12.41 | 14.52 |
| Days to 4.6 lbs | 42 | 41 | 43 | 40 | 42 |
| Chick cost/lb (¢) | 4.16 | 3.49 | 4.13 | 4.16 | 3.86 |
| Vac-Med cost/lb (¢) | 0.07 | 0.13 | 0.08 | 0.06 | 0.07 |
| WB & 1/2 parts condemn. cost/lb | 0.11 | 0.16 | 0.15 | 0.16 | 0.15 |
| % mortality | 4.46 | 4.45 | 4.00 | 4.67 | 4.84 |
| Sq. Ft. @ placement | 0.83 | 0.86 | 0.80 | 0.81 | 0.84 |
| Lbs./Sq. Ft. | 6.60 | 7.63 | 6.92 | 6.94 | 7.18 |
| Down time (days) | 12 | 16 | 15 | 11 | 12 |

Data for week ending June 24, 2006

### Broiler Whole Bird Condemnation (Region)

| | SW | Mid-West | S. East | Mid-Atlantic | S. Central |
|---|---|---|---|---|---|
| % Septox | 0.106 | 0.177 | 0.168 | 0.227 | 0.125 |
| % Airsac | 0.049 | 0.056 | 0.045 | 0.053 | 0.060 |
| % I.P. | 0.008 | 0.029 | 0.008 | 0.009 | 0.063 |
| % Leukosis | 0.001 | 0.039 | 0.002 | 0.000 | 0.000 |
| % Bruise | 0.003 | 0.003 | 0.001 | 0.001 | 0.004 |
| % Other | 0.009 | 0.008 | 0.011 | 0.005 | 0.007 |
| % Total | 0.174 | 0.311 | 0.235 | 0.295 | 0.250 |
| % 1/2 parts condemnations | 0.225 | 0.334 | 0.346 | 0.572 | 0.340 |

Data for week ending June 24, 2006

### Broiler Performance Data (Company) Live Production Cost

| | Average Co. | Top 25% | Top 5 Co.'s |
|---|---|---|---|
| Feed cost/ton w/o color ($) | 149.99 | 151.09 | 148.54 |
| Feed cost/lb meat (¢) | 14.06 | 12.92 | 12.84 |
| Days to 4.6 lbs | 42 | 43 | 44 |
| Chick cost/lb (¢) | 4.06 | 5.47 | 5.50 |
| Vac-Med cost/lb (¢) | 0.09 | 0.08 | 0.14 |
| WB & 1/2 parts condemn. cost/lb | 0.15 | 0.12 | 0.12 |
| % mortality | 4.34 | 3.50 | 3.90 |
| Sq. Ft. @ placement | 0.83 | 0.72 | 0.72 |
| Lbs./Sq. Ft. | 6.96 | 5.56 | 5.64 |
| Down time (days) | 13 | 13 | 15 |

Data for week ending June 24, 2006

### Broiler Whole Bird Condemnation (Company)

| | Average Co. | Top 25% | Top 5 Co.'s |
|---|---|---|---|
| % Septox | 0.162 | 0.104 | 0.173 |
| % Airsac | 0.068 | 0.068 | 0.032 |
| % I.P. | 0.027 | 0.032 | 0.009 |
| % Leukosis | 0.008 | 0.004 | 0.001 |
| % Bruise | 0.003 | 0.004 | 0.002 |
| % Other | 0.008 | 0.007 | 0.003 |
| % Total | 0.266 | 0.208 | 0.220 |
| % 1/2 parts condemnations | 0.334 | 0.257 | 0.241 |

Data for week ending June 24, 2006

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