By John Wenz, Assoc Prof, Ag Animal Health Program, WSU

WSU and collaborators from University of Idaho, University of California-Davis and South Dakota State University have developed a set of surveys to better understand the impact of COVID-19 and the Coronavirus pandemic on US Dairy Farms. The surveys were initiated to gather preliminary data for a grant proposal submitted on June 4th to the USDA’s “Rapid Response to Novel Coronavirus (SARS-CoV-2) Impact Across Food and Agricultural Systems.” program.
Although the grant has been submitted, 

**stakeholders can still respond to the surveys until July 31, 2020.** The results will be used to identify critical areas for outreach activities with the objective of mitigating potential health risks to those involved in dairy farming. Whether or not one believes the novel coronavirus presents a significant health risk, response to the current and potential future waves of COVID-19 definitely presents an economic risk to the dairy industry. It is anticipated that results will be summarized by August for peer reviewed journal submission as well as distribution through extension partners and lay journals.

There are 3 surveys.

**Allied Industry Professionals** (Veterinarians, Nutritionists, Consultants, Sales Reps):  
https://wsu.co1.qualtrics.com/jfe/form/SV_3Jn5tx9KnPNE2nr

**Dairy Producers/Managers:**  
https://wsu.co1.qualtrics.com/jfe/form/SV_d7gA0pfFIljeRs9

**Dairy Farm Workers** (English and Spanish):  
https://ucdavis.co1.qualtrics.com/jfe/form/SV_czN1s3rjp9FaAHb

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**Dairy: New Guidelines for Passive Immunity in Dairy Calves**

By Dale A. Moore, WSU Extension Veterinarian

The USDA National Animal Health Monitoring System conducted a project on dairies across the US in 2014. Some dairy farmers and Extension folks in Washington got to participate in this project. You might wonder what happened to all the data they collected. The data become a “check” on what and how the dairy industry is doing with regards to animal health and management and are reported at the USDA NAHMS website ([NAHMS DAIRY STUDIES](https://www.aphis.usda.gov/animal_health/nahms/dairy/)). Those data are used to set USDA priorities and create new standards.

I was fortunate to participate in a panel of USDA investigators, academics and others to review the data on calf serum immunoglobulin levels from the calf component of the USDA study, evaluate current literature on the topic, assess different cutpoint associations with calf mortality and morbidity, and come to consensus on new guidelines for IgG, serum total protein and Brix percent cutpoints for farmers and veterinarians to use.

Current standards for deciding successful (greater than or equal to 10 g/L or 5.1 g/dL serum total protein concentration) or unsuccessful transfer of passive immunity (less than 10 g/L serum IgG or 5.1 g/dL serum total protein) were based on mortality rate differences in calves with different values. With the dairy industry dramatically reducing mortality in preweaned calves over the last three decades but remaining steady on morbidity rates, it was time to revisit the cutpoints for evaluating transfer of passive immunity.

Follow-up data on more than 2,300 calves were used in the analyses. After much deliberation, the panel came to consensus on a four-category scale (see the table below) with the idea that over one-third of the farms in the study were already achieving these levels of transfer of passive
immunity. The categories are meant to be used to evaluate the herd or cohorts of calves and to help improve morbidity rates (and lower treatment costs and antimicrobial use).

<table>
<thead>
<tr>
<th>Transfer of Passive Immunity Category</th>
<th>Serum IgG (g/L)</th>
<th>Total Protein (g/dL)</th>
<th>Brix %</th>
<th>Consensus % of calves that should be in this category</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excellent</td>
<td>≥ 25.0</td>
<td>≥ 6.2</td>
<td>≥ 9.4</td>
<td>&gt; 40 %</td>
</tr>
<tr>
<td>Good</td>
<td>18.0-24.0</td>
<td>5.8-6.1</td>
<td>8.9-9.3</td>
<td>~ 30 %</td>
</tr>
<tr>
<td>Fair</td>
<td>10.0-17.9</td>
<td>5.1-5.7</td>
<td>8.1-8.8</td>
<td>~ 20 %</td>
</tr>
<tr>
<td>Poor</td>
<td>&lt; 10.0</td>
<td>&lt; 5.1</td>
<td>&lt; 8.1</td>
<td>&lt; 10 %</td>
</tr>
</tbody>
</table>

(After Lombard et al., 2020)

One question from a panelist was “Why four categories?” These categories were evaluated in several ways statistically and practically. The “survival” curves for the four categories of serum IgG were most telling. There were clear differences in the probability of disease over the first 60 days of life as well as the probability of death over the same time period that decreased with increasing serum IgG.

Eight years ago, I had an inkling that we should be looking at higher cutpoints so I asked a veterinary student to rework some data I had from three calf ranches (about 400 calves total) for her senior paper. She concluded that calves with serum IgG >15 g/L had less than 1% mortality and that no calves that had >16 g/L serum IgG died before weaning on these ranches. It appears that more is better.

There is true disease prevention power in the transfer of passive immunity and it is measurable. The categories and the proportion of calves in a herd that could meet the standards are achievable in dairy herds in the US. These new guidelines do set the bar a little higher than what farmers and veterinarians might be used to but if achieved, have clear benefits in reducing preweaning calf mortality, morbidity and treatment costs.

Reference

Dairy: On Farm Methods to Determine Transfer of Passive Immunity
By Dale Moore, WSU Extension Veterinarian

Although radial immunodiffusion (RID) for serum IgG concentration is considered the gold standard for evaluating the transfer of passive immunity in neonatal calves, it is expensive and not immediately available. Several on-farm methods to assess failure of transfer of passive immunity (FTPI) have been evaluated so that veterinarians, calf raisers and farmers can identify potential colostrum management problems. This article provides and update on the use of these on-farm methods.
The most common methods include various types of refractometers. Other methods, including GGT, whole blood glutaraldehyde coagulation, sodium sulfite and zinc sulfate turbidity tests are not used to any great degree in North America although the zinc sulfate turbidity test is well-correlated to IgG by RID \((r = 0.88)\) (Dunn et al., 2018). For a recent review of all the alternative tests, see Cuttance et al (2019).

The optical, analog clinical refractometer was the first on-farm device suggested to estimate IgG levels in calves (McBeath et al., 1971). Over the last four decades there have been numerous publications on the comparison between serum total protein concentration (STP) and association with calf mortality rate (Donovan et al., 1998; Rea et al., 1996). However, a recent systematic review of the use of a clinical refractometer to identify FTPI reported that most studies used a standard of 10 g/L IgG determined by RID as a cutoff (Buczinski et al. 2018). The authors cautioned that the optimal cutoff to “protect” a calf varies with calf environment and management and that dehydration and inflammation might falsely elevate the serum protein concentrations. Despite this, refractometers are used because IgG makes up a large part of the STP and these refractometers are easy to use and provide quick results.

In a study comparing three different clinical refractometers to RID IgG results, Calloway et al. (2002) calculated correlation coefficients \((r)\) of 0.74 to 0.77. When estimating sensitivity using a RID cutoff of 10 g/L compared with a 5.2 g/dL STP cutoff, the sensitivity range was 0.86 to 0.89 and the test correctly classified over 80% of the proportion of calves with FTPI (based on the 10 g/L IgG cutpoint). However, changes in the prevalence of FTPI affected the proportion of calves correctly classified. Digital and optical handheld refractometers appear to give similar results \((r = 0.97)\) (Wallace et al., 2006). A digital refractometer was used to evaluate STP correlation with IgG as well as with a new rapid test (ZAP) (Renaud et al., 2018). The STP was correlated with IgG \((r = 0.87)\) but the quick test for antibody did not perform as well to detect FTPI.

One study compared four refractometers to evaluate FTPI in beef calves (Vandeputte et al., 2011). When evaluated at room temperature, STP results from all four (one non-temperature compensated, two temperature-compensated and one digital temperature-compensated) were highly correlated with serum IgG concentrations derived from High-performance liquid chromatography (HPLC). Zakian and others (2018) found a strong positive correlation between STP from a digital refractometer and IgG as measured by ELISA \((r = 0.95)\) and between a digital Brix refractometer and IgG by ELISA.

Brix refractometers have been evaluated for on-farm use in estimating total solids in milk, milk replacer, and colostrum, as well as IgG and FTPI. One study compared both the STP values obtained from a clinical refractometer and Brix refractometer values with serum IgG by RID. The Brix values and the STP values had a near perfect correlation. The STP and the Brix values were also highly correlated \((r = 0.93)\) with the IgG values by RID (Deelen et al., 2014). Using a receiver operator curve, the cutoff selected using 10 g/L IgG concentration from RID as a definition of failure was 8.4% Brix. A STP cutoff of 5.2 g/dL had the highest sensitivity with that IgG cutpoint reported in this study.

Values from two different types of refractometers (optical refractometer for STP and digital Brix) were compared to IgG results from RID (Elsohaby et al., 2015). Correlations between an optical
clinical refractometer and a digital Brix refractometer with IgG by RID were 0.74 and 0.79, respectively. Using a ROC, the best cutoff for digital Brix was 8.3% and 5.5 g/dL for the STP to assess FTPI (based on 10 g/L IgG cutpoint). Digital and optical Brix refractometer results were compared with IgG by RID and found to be correlated (Thornhill et al., 2015). The RID IgG concentrations correlated with Brix % (r= 0.74 and 0.71) for the digital and optical devices, respectively. The correlation between Brix values with IgG by RID was 0.60 in another study (Dunn et al., 2018).

Hernandez et al. (2016) also evaluated the relationships between Brix% on serum, STP and IgG by turbidimetric immunoassay (TIA). Brix% was correlated with STP (r = 0.91) and with serum IgG (r = 0.79) and STP was correlated with IgG (r=0.82). Brix % was correlated with STP (r = 0.97). Using the FTPI cutoff for IgG of 10 g/L, the Brix cutoff for FTPI was estimated to be ≤ 8.5% and 5.2 g/dL for STP. A study using blood from neonatal Jersey calves described correlations with IgG by RID of 0.78 and 0.79 for Brix and STP, respectively (McCracken et al., 2017).

A very recent study evaluated three sample types (serum, centrifuged plasma and filtered plasma) with three different testing methods (Brix%, ELISA and capillary electrophoresis). The Brix % was well-correlated with IgG by RID, particularly on serum (r = 0.84)(Sutter et al., 2020).

The research is clear. We can use handheld devices that measure serum total protein or Brix % to estimate serum IgG levels. There are just a few caveats to their use.

Factors affecting values obtained by refractometers

Sample centrifugation and hemolysis -- If the sample to be evaluated is not solely serum, there may be falsely elevated STP levels. Centrifugation to separate the serum from the clotted blood is the method of choice to ensure the sample is serum. However, allowing the blood to clot completely before pulling off the serum to test will provide similar results. Wallace et al. (2006) reported that serum collected from blood tubes allowed to clot had a STP content (as determined by a standard digital refractometer) that was highly correlated (r = 0.97) to the STP content of a duplicate sample that was centrifuged. Two investigations of sample type (serum, centrifuged plasma and filtered plasma) have recently been reported (Elsohaby et al., 2019; Sutter et al., 2020). Practically, there was little difference in the ability to detect FTPI based on sample type.

Temperature vs non-temperature compensated refractometers -- Clinical optical refractometers work by measuring the extent to which ambient light is bent by the proteins in a serum sample, but the index of this refraction is influenced by the temperature of the fluid being evaluated (Vandeputte et al., 2011). Temperature-compensated refractometers have been developed to overcome the variation in STP results from the influence of ambient temperature.

Calibration of and cleaning refractometers -- Handheld, optical refractometers require calibration to provide consistent results. They can be set to zero with a sample of distilled water. Milk A film left from one sample can distort the value obtained for the next sample. The prism of each refractometer should be cleaned with distilled water before each use.

Age and breed of the calf -- The serum IgG and STP decreases with the age of the calf (Villaroel et al., 2013). Most consultants recommend that blood be collected on day two (allowing for total absorption of IgG from colostrum ingestion) and up to day 7 of life. Wilm and others (2018) suggested that blood can be collected for IgG levels up to 10 days of life but that it be adjusted by day of age. Correlations of STP between Day 2 and 3 of life were highest with the 24-hr reference value in their study. Jersey calf serum IgG and STP were consistently higher than Holstein calf
serum in one study (Villaroel et al., 2013) and McCracken and others (2017) suggested that a cutoff of 5.5 g/dL STP might be too high for Jersey calves.

Illness – Illness and dehydration may affect the relationship between some of the quick measures of FTPI and IgG by RID. In a study of 27 ill calves (with diarrhea, septicemia, hypoglycemia and hypothermia) Tyler and others found that the zinc sulfate test, sodium sulfite test and serum GGT activity had better sensitivity than STP methods and the authors suggested a higher test endpoint for evaluating FTPI in clinically ill calves. Looking at blood and clinical evaluations of sick calves, Fecteau and others (2013) evaluated the regression relationships between plasma protein concentrations, STP, and zinc sulfate turbidity levels on their prediction of serum IgG. None of the clinical covariates significantly changed the prediction of IgG by zinc sulfate turbidity. However, both hydration status and fibrinogen concentrations affected predictions of IgG by both serum and plasma protein.

Using the Tools
All the handheld on-farm methods can be used as surrogates for direct IgG measurements. Regardless of the type of refractometer used, however, using these tools to assess individual calves for intervention might not be as appropriate as using the values to monitor the farm’s colostrum management program (Godden et al., 2019). Recommendations are to collect at least 12 samples from calves 2 to 7 days of age (for a spot check) but some farms test every calf (McGuirk and Collins, 2004). A threshold of 20% failures upon which to act has been suggested. With new guidelines for assessing failure of transfer of passive immunity based on new cutpoints for IgG, STP and Brix% (Lombard et al., 2020), monitoring, investigating and remediating the potential causes of failures can lead to lower calf mortality, morbidity and treatment costs.

References
Livestock: Musings on Infection Control and Herd Immunity

By Craig McConnel, WSU Extension Veterinarian

A The SARS-CoV-2 pandemic suddenly has made us all aware of the importance of epidemiology and associated terminology. “R0” [R naught] has been particularly prominent in the news as it is the basic reproductive rate of a disease, representing the number of new infections estimated to stem from a single existing infection. As we all know by now, R0 is dependent on population dynamics related to individual contacts, susceptibility, and resistance. And, as with any infectious disease, there are tools that help alter transmission dynamics such as hygiene, personal protective equipment, quarantine, and therapies. In an ideal situation, an effective vaccine will become available that can rapidly increase resistance by helping achieve a level of herd immunity that results in protection for susceptible individuals (immunocompromised, unvaccinated, etc.). An excellent resource for demonstrating the impact of herd immunity is available [here].

At times it seems we overlook the fact that these same epidemiologic principles underlie our approach to livestock infectious disease control. All too often our management focuses on products without necessarily considering their placement within the overall picture of disease...
mitigation. In other words, when was the last time that you and your veterinarian discussed and updated your infectious disease control strategies based on identifiable risks related to contacts and susceptibility? It may be all the more important to consider these risks this year given the uncertainty of markets that may alter how you manage your herd in terms of selling calves, removing cull cows, etc.

Regardless of whether you operate a beef ranch, dairy, or small ruminant flock or herd, there are some obvious fundamental periods of physiologic stress throughout the production cycle (e.g., birth, weaning, parturition, early lactation). Environmental and management stressors such as adverse weather, transportation, co-mingling, overcrowding, and breeding all can influence pathogen exposure and transmission dynamics as well. For the most part, we tend to describe infectious disease relative to specific organ systems and associated pathogens within at-risk populations. For example, primary areas of infectious disease concern for beef or dairy cattle might be as follows:

1. Bacterial and viral enteric disease (neonates)  
   *E. coli*, *Clostridium perfringens*, Rotavirus, Coronavirus, Pestivirus Type 1 & 2 (BVD)
2. Bacterial respiratory disease (weaned, co-mingled, immunocompromised)  
   *Mannheimia haemolytica*, *Pasteurella multocida*, *Histophilus somni*
3. Bacterial reproductive disease (breeding, pregnant)  
   *Leptospira* spp., *Campylobacter fetus*
4. Viral respiratory and reproductive disease (weaned, co-mingled, immunocompromised, breeding, pregnant)  
   Pestivirus Type 1 & 2 (BVD), Bovine Herpesvirus Type 1 (IBR), Parainfluenza Type 3 (PI-3), Bovine Respiratory Syncytial Virus (BRSV)
5. Bacterial mastitis (lactating)  
   *E. coli*, *Staphylococcus aureus*
6. Bacterial general (across production cycle)  
   *Clostridium* spp. (muscle, GI, liver), *Salmonella* spp., *Mycobacterium avium* subsp. *paratuberculosis*, *Moraxella bovis*

Obviously, there are variations on this theme and this is not intended to be an exhaustive list of infectious disease for cattle. Small ruminant systems clearly include other pathogens of concern as well. Nonetheless, the outline above provides a good starting point for demonstrating when and where to focus interventions that can limit pathogen spread, reduce individual animal susceptibility, and enhance herd immunity. Our current personal experiences with SARS-CoV-2 highlight the lengths that must be taken to limit disease in the face of poor herd immunity, and reinforce the need for excellent colostrum management, environmental sanitation, nutritional support, and stress management within our various herds and flocks. Thankfully, we also have vaccines at our disposal to help overcome inherent limitations within livestock infection control. The key is to identify those limitations and position immunization prior to the intersection of stressors and pathogen exposure. By staying up to date on your vaccine protocols and doing your best to manage inherent stressors, you should be able to maintain a level of herd immunity against many pathogens that aids in control or reduction of disease if not outright prevention of infection.

Keep in mind that your vaccination strategy should take into account additional specific considerations such as the type and duration of immune response (cell mediated, humoral) associated with protection against a given pathogen, vaccine compositions (killed, modified live, Gram negative, etc.), and associated restrictions related to the breed and age of the animals, route of administration, or pregnancy status. Overlay these considerations with your insight into current disease prevalence, opportunities for handling animals within your production system, and forthcoming management changes to reassess your current vaccination protocols relative to
identifiable biosecurity risks. At a minimum, consider re-evaluating your **infectious disease management strategies** including vaccination schedules on an annual basis, and do your best to make R0 naught.

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**Dairy: Impact of Heat Stress on Dairy Cattle Well-Being**

By Amber Adams Progar, WSU Dairy Management Specialist [amber.adams-progar@wsu.edu](mailto:amber.adams-progar@wsu.edu)

It has been an unpredictable year so far, including the weather. Regardless of when the hot weather decides to hit, our best defense is to be prepared. We know that heat stress causes decreases in milk production and alters cow behavioral patterns, but what are some of the less known impacts of heat stress on dairy cattle well-being? Below are some interesting study results you may want to consider when deciding how to manage heat stress on your dairy.

1. **Dry matter intake**
   During a 45 day dry-off period, cows that received cooling measures during the entire dry period had an almost 10% higher dry matter intake than cows that received cooling measures for only the first half of the dry period (Fabris et al., 2019).

2. **Immune system cytokines**
   Cows housed in a heat stress environment (temperature-humidity index > 68) had higher plasma concentrations of some key immune system cytokines. Higher concentrations of these cytokines (IL-1β, IL-6, IFN-γ, and TNF-α) indicate that heat stress causes an inflammatory response in the cow’s body (Chen et al., 2018).

3. **Mucosal defense system**
   Housing cows in a heat stress environment also causes a stress-induced activation of the mucosal defense system in lactating cows (Koch et al., 2019). Of course, the more energy the cow’s system uses to cope with heat stress, the less energy that is available for milk production.

4. **In utero heat stress and calf immunity**
   Calves exposed to in utero heat stress have lower rates of peripheral blood mononuclear cell (PBMC) proliferation than calves not exposed to in utero heat stress (Tao et al., 2012). This effect can persist until the calves reach 56 days of age. PBMCs are the cells that make up the immune system, in which a low proliferation rate puts the calf at a health risk.

5. **In utero heat stress and calf growth**
   Newborn calves that experienced in utero heat stress have lower plasma insulin, prolactin, and insulin-like growth factor-I concentrations than calves that did not experience in utero heat stress (Guo et al., 2016). Insulin, prolactin, and insulin-like growth factor-I are key components of calf growth and development.
6. In utero heat stress and calf health
Cows that were cooled during the dry period had heavier calves that also had lower body temperatures at calving than calves from cows housed under heat stress conditions during the dry period. Cooled cows also had calves that were more efficient at absorbing IgG from colostrum and gained 0.44 lb/day more than calves from heat-stressed cows (Laporta et al., 2017).

7. In utero heat stress and calf IgG absorption
Calves born to cows exposed to heat stress during the dry period have lower serum IgG concentrations and higher serum cortisol concentrations than calves born to cows housed in a thermoneutral environment during the dry period (Almoosavi et al., 2020). Remember, cortisol is often referred to as the “stress hormone”.

8. In utero heat stress affects calf future performance
Heifers born to heat-stressed cows produce 16% less milk for the first 35 weeks of their first lactation than heifers born to cooled cows (Monteiro et al., 2016). Almost 20% more heifers from cooled cows reached their first lactation than heifers from heat-stressed cows.

9. Feed supplements and cow performance
Supplements may help cows cope with heat stress. One supplement fed to heat-stressed cows resulted in lower cow rectal temperatures (Fabris et al., 2017). The same supplement fed to cooled cows resulted in 13% higher milk production than heat-stressed cows that were not fed the supplement.

10. In utero feed supplements and calf health
Feeding the supplement mentioned in #9 to cows during the dry period impacted calf health, depending on whether cooling was provided to the cows (Skibiel et al., 2017). Calf serum amyloid A (protein associated with inflammation) was higher in calves born to heat-stressed cows than cooled cows. Furthermore, neutrophil (a type of immune cell) function at 10 days of age was higher in calves born to cooled cows that received the supplement than cooled cows that did not receive the supplement. [For a list of references, email Dr. Adams-Progar]

Sheep: What’s on the Management Calendar for Summer? Ram Evaluation
By Dale A. Moore, WSU Extension Veterinarian

Sometime this summer, producers should be considering selection of breeding rams for the next breeding cycle. Back when I was a baby vet, we worked at the California Ram Sale and “sifted” (for health, teeth and general physical exams) hundreds of rams. What kinds of things did we look for?

A veterinarian can conduct a brief physical examination which tell a lot about a ram’s ability to cover ewes. First, the ram can be evaluated before touching him to assess rapid or abnormal breathing, potentially signs of pneumonia. The ram’s body condition should be about a 3.5 on a scale of 1 to 5 with 1 being thin and 5 overweight. If there is wool, we need to feel the ribs and the spine. Next,
we examine the teeth. If he does not have normal teeth and bite, he might not be able to maintain weight on range. A quick eye examination will see if he suffers from entropion or a rolling in of the eyelid. Because entropion is heritable, ram lambs suffering from this should not be used for breeding. The eyes should also be examined for pinkeye.

In order to do their job, rams need a good foundation. Feet and legs should be evaluated for structural abnormalities and conditions like foot rot. Lymph nodes and the rest of the body should be palpated for abscesses because the most common cause is caseous lymphadenitis, a contagious condition of sheep and goats. While checking for abscesses, the ram can be evaluated for external parasites that should be treated before putting him in with other animals. There should be no evidence of internal parasites, including white conjunctivae or gums, or diarrhea.

Finally, the external genitalia should be assessed. The testicles can be palpated for equal size and dimension and tone. There should be no lumps or bumps in the body of the testicle or on the head or tail of the epididymis (signs of abscesses or Brucella ovis epididymitis, an important reason for culling rams). The scrotal circumference measurement is a proxy for breeding capacity and standards are provided in the resources. The penis and sheath should be examined, first for pizzle rot as well as for warts. Anything that would impair his ability to breed should be considered in a culling or prepurchase decision. If the ram can pass the physical exam, he can next be evaluated for semen quality.

Checking the rams before they are brought home or turned out with the ewes will save time in the breeding season, help ensure a better lamb crop, and avoid the potential for infectious disease spread to the home flock.

Resources

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Swine: Glasser’s Disease
By Dale Moore, WSU Extension Veterinarian

I recently received a request for information from an Extension educator about a case of Glasser’s disease in a 4H pig. He wanted to know what to tell the 4H group about what to do with the rest of the pigs if they had been exposed. It is worth a review of this disease to highlight the identification, treatment, control and prevention steps to take.
Recognized since 1910, Glasser’s disease (porcine polyserositis caused by the bacterium *Haemophilus parasuis* now called *Glasserella parasuis*) is found worldwide and can be a cause of illness or death in weaner or younger pigs. The disease appears sporadically in conventionally raised swine. The bacteria responsible for disease are commonly found in non-clinical pigs in the upper respiratory tract as early as 2 days of age and may be endemic on many farms (Hurtado et al., 2020).

**If it is already present subclinically in a group of pigs, what can trigger it to cause disease?** Colostral antibodies usually protect piglets from the disease until they can fully develop their own immunity at about 2 months of age. Thus, poor colostrum intake can lead to the development of disease. Other triggers include stressors like weaning, transportation or infections with other diseases such as PRRS. In addition, heat or cold stress might play a role (Hurtado et al., 2020). The best explanation for the timing of clinical disease is waning immunity from ingested colostrum antibodies and increasing stressors that could disturb the pig immune response. In addition, disturbance of the nasal microbiota (healthy nose flora) with preventive antimicrobials might also trigger clinical disease (Hurtado et al., 2020).

**If the infection leads to clinical disease, what signs are usually seen?** The appearance of the disease comes on suddenly. High fever, runny nose, difficulty breathing, red eyes, cough, swollen joints, lameness or neurologic signs might precede death or a pig might die suddenly without observed signs. The number of pigs affected in a group is usually low but of those affected, the death rate is high unless treated. One problem in the identification of Glasser’s disease is that it can mimic many other diseases in pigs.

**If we see typical signs, how do know for sure that it is Glasser’s disease?** A veterinarian can often make a tentative diagnosis based on the history and observed clinical signs and lesions. To make sure, a pig that dies should be necropsied and samples for bacterial culture or PCR testing sent to the diagnostic lab. [For an interesting case, see Poad and Ball, 2012, below in the references].

**Can it be treated?** Any sick animal should receive treatment with an antimicrobial prescribed by a veterinarian. If there are a lot of sick pigs, the veterinarian might recommend a mass, in-feed medication through a Veterinary Feed Directive. However, isolates from different farms might have different sensitivities to different antibiotics. A very recent paper reported on trends in antimicrobial resistance of these organisms in the US (Hayer et al., 2020). The proportion of isolates resistant to commonly used penicillin drugs increased to more than 10% over time. The increase in resistance to ceftiofur, enrofloxacin and tulathromycin was even greater. Because of the potential for bacterial resistance, it is worth the effort to submit samples to the diagnostic lab periodically to make more informed decisions on treatment options.

**How can the development of clinical disease be prevented?** There are some vaccines (bacterins) available for sows. The piglets then need to get adequate amounts of colostrum. Or, the piglets could be vaccinated at one to three weeks of age. Finally, the potential stressors need to be reduced or eliminated in the nursery and weaning environments.

References and Resources
1. Department of Veterinary Diagnostic & Production Animal Medicine, Iowa State University. *Haemophilus parasuis* (Glasser’s Disease). Available at: https://vetmed.iastate.edu/vdapm/FSVD/swine/index-diseases/glasser-disease

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**Poultry: Human Salmonellosis Outbreaks and Backyard Poultry**

**By Laura Chen, Branch Chief, WADDL-AHFSL**

In May 2020, the Centers for Disease Control and Prevention (CDC) reported a multi-state outbreak of *Salmonella* Hadar linked to contact with backyard poultry. The investigation is still ongoing but at the latest update, there have been a total of 97 cases with 17 hospitalizations across 28 states reported (CDC 2020). This outbreak is far from an anomaly. Unfortunately, contact with backyard poultry has been linked in multiple outbreaks of Salmonellosis in people. In 2019, CDC identified 1,134 cases of Salmonellosis associated with backyard poultry contact across 49 states with 219 hospitalizations (CDC 2019). In 2018, 334 cases were reported across 47 states with 56 hospitalizations (CDC 2018). Between 2000 and 2018, there were 76 outbreaks of Salmonellosis associated with backyard poultry (CDC 2018).

A significant limiting factor to reducing or eliminating these outbreaks is the fact that chickens can carry non-host adapted (paratyphoid) strains of *Salmonella enterica* in their gastrointestinal tracts, excrete it in their feces, and then transmit it to people. While host-adapted strains of *S. enterica* (S. Pullorum and S. Typhoid) will often cause clinical disease when present, paratyphoid strains’ infections can present asymptomatically. Detection of the bacteria is therefore often limited to surveillance samples. While regulations are well established in the commercial poultry industry to detect the presence of *S. enterica* by surveillance, the same is not true for backyard poultry owners.

With the risk of Salmonellosis in backyard poultry established, the natural follow up question is, how big is the risk?

In the available peer-reviewed studies examining *Salmonella enterica* in backyard chicken flocks, the reported prevalence is generally low. Clothier et al. (2018) cultured intestinal contents and diseased tissues from 2,347 backyard cases accessioned through the California Animal Health and Food Safety Laboratory System and identified only 44 samples (1.7%) positive for *Salmonella*. Along those same lines, McDonagh et al. (2018) cultured feces, cloacal samples, and dust samples from 53 flocks in the greater Boston, MA region and identified only 1 flock to be *Salmonella* positive (1.9%). In Argentina, a research group had similar findings by culture of individual cloacal swabs from 657 backyard chickens; only 4 samples tested positive (0.6%) (Rodriguez et al., 2018).

Unfortunately, the low prevalence of *Salmonella* among backyard chickens should not be interpreted as synonymous with low or no risk because we know outbreaks occur. Beam et al. (2013) surveyed backyard chicken flock owners across three urban areas and found that in two of the three areas, less than 50% of participants were aware of the risk of *Salmonella* from their chicken flocks. In a more recent study, backyard chicken owners in the Seattle area were surveyed on their knowledge of *Salmonella*, animal husbandry, and biosecurity (Kauber et al., 2017). The
authors found that most owners were aware of the association between chickens, chicken products, and *Salmonella*, yet by video assessment, approximately one in four owners still engaged in risky behaviors that could result in disease transmission (Kauber et al., 2017).

Despite the inherent risk present, there are multiple ways that backyard poultry owners can reduce their risk of Salmonellosis and safely enjoy their flock. This includes:

1) Obtain chicks from a reputable source, ideally from a hatchery that participates in USDA APHIS’s National Poultry Improvement Plan *Salmonella* monitored certification program. Such hatcheries undergo environmental sampling for *Salmonella* every 30 days.

2) Maintain appropriate biosecurity when interacting with chickens. This includes having separate footwear and clothing to wear when interacting with the flock; washing hands immediately after interacting with the chickens; and preventing chickens from entering areas where people are eating or drinking.

3) Always cook chicken products to the appropriate temperatures. For poultry, the internal temperature should reach 165°F. For eggs, the yolk and white should be firm; egg dishes should be cooked to a temperature of 160°F.

Looking for more resources related to human health concerns and backyard poultry? Check out the CDC’s webpage: https://www.cdc.gov/healthypets/pets/farm-animals/backyard-poultry.html

References


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**Objectives**: Zoonotic pathogens on dairy farms are a known risk for people who work and live there. Exposure and/or transmission of *Salmonella* serovars, *E. coli* (O157; H7), *Campylobacter jejuni*, and *Cryptosporidium parvum* have been documented to occur in the dairy farm environment. Social ecological factors have been identified as determinants of preventive behaviors of people at risk of infectious diseases. **Methods**: This study described the effect of socio-ecological factors on selected zoonotic bacterial and protozoal diseases in 42 workers of two dairy farms. **Results**: Occupational exposure to *Salmonella* ser. Dublin, *E. coli*, and *Campylobacter spp.* was confirmed. Self-efficacy and negative workplace perceptions were risk factors for...
Salmonella Dublin exposure (OR = 1.43 [95% CI 1.11-2.22] & 1.22 [95% CI 1.02-1.53] respectively). Additionally, safety knowledge and risk perceptions were protective factors of exposure (OR = 0.90 [95% CI 0.79-1.00]). Positive perceptions of supervisors and coworkers was a protective factor of Campylobacter exposure (OR = 0.89 [95% CI 0.79-0.98]). **Conclusion:** Results indicated that the presence of a supporting organizational environment, good communication with supervisors and coworkers, and training on prevention of zoonotic diseases would potentially reduce occupational exposures to zoonotic diseases on these farms.

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**WSDA Corner**

**Moving Cattle**

By Dr. Dana Dobbs, WSDA Field Veterinarian

Are you moving cattle interstate, planning a dairy sale, or simply testing your herd for Bovine Tuberculosis (TB)? If so, here are some important factors to keep in mind.

On average, veterinarians detect responders in one to five percent of cattle tested with the Caudal Fold Test (CFT) for tuberculosis. This will result in a Hold Order to stop movement of any cattle on your farm, and a WSDA regional field veterinarian will be required to follow up with a Comparative Cervical Test (CCT) on the responders.

**Planning ahead is essential**

The CCT must be conducted within 10 days of your veterinarian’s initial CFT injection, or after 60 days. The CCT requires animals be restrained to allow clipping two spots on the animal’s neck, measuring skin thickness with calipers, and injecting two types of tuberculin. Three days later, the skin thickness at the two sites is measured and the reactions are compared to determine TB status. Any positive CCT suspects will require necropsy and additional laboratory diagnostics.

**Avoid added expense, allow time for follow-up testing**

Finally, don’t assume there won’t be any TB responders when conducting your herd testing. A common mistake is to schedule cattle transport trucks at the conclusion of your veterinarian’s initial CFT testing. This can be costly and frustrating when the cattle are not permitted to load due to a Hold Order. Allow two to three weeks for any follow-up testing. Together, we can keep cattle moving in a smooth and efficient manner. Contact the Animal Health Program at (360) 902-1878, or your WSDA field veterinarian with any questions.

**Continuing Education**

**Veterinarians**

**Dairy Antimicrobial Stewardship Webinars!** Our April program in the western states was postponed and will now be offered through distance. The full-day program will now be offered as 4, 2-hour webinars. CE credit is available for up to 7 hours. Specific dates and times:

- July 8, 2020: 6 pm to 8 pm
- July 15, 2020: 6 pm to 8 pm
- July 22, 2020: 6 pm to 8 pm
- July 29, 2020: 6 pm to 8 pm

[https://vetextension.wsu.edu/dairy-antimicrobial-stewardship/](https://vetextension.wsu.edu/dairy-antimicrobial-stewardship/)
Producers

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The Answer will be posted on the VME Homepage, under Newsletters:
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