



Salting the Pad for Bacterial Pathogen Reduction: This Protocol May Have a Small Impact, But Is It Enough to Make a Difference?

By K. Scott McKenzie, Ph.D., Director, MWI Animal Health Technical Services

Mark Twain was purported to have said, “There’s nothing wrong with feeling strongly about something...as long as you don’t confuse that with thinking.” That seems to surmise a great deal of what is now being done in the poultry industry based on processes and products used in bygone years. For example, adding a gallon or so of cresylic acid to the pad or litter to “make it smell like it did when my grandpa put down creosote,” or adding diesel to the pad to “kill just about everything,” or adding salt to the pad to...kill just about...what?

Too many producers (and chemical applicators) are relying on nostalgia to fix disease problems between flocks, at annual cleanout or in response to a very specific disease pressure. Production pressures have dramatically changed the industry over the last two decades—clean-out or between-flock strategies that were once employed may no longer be relevant. Bird growth rates, short out times, litter disposal barriers (i.e., environmental regulations), house design (tunnel versus curtain), chemical ammonia mitigants and the removal of antimicrobials (such as 3-Nitro[®], or roxarsone) have changed much of the chemistry and microbial ecology of broiler and turkey litter and also the clay or sandy pads that support that litter.

There are several challenges facing the use of salt to accomplish anything chemically significant in litter and/or on the pad, besides adjustment of water activity (A_w).

One challenge: Getting salt to contact the target organism is a problem because achieving microbial kills is a contact sport. Salt (sodium chloride) is a solid. Unlike ammonia treatment, using a sulfate *powder* such as Poultry Guard[®] or PLT[®] (or even a liquid sulfate) to come into contact with a *gas* (ammonia vapor or gaseous ammonia), by adding salt granules to kill, let’s say, *Salmonella* or *E. coli*, you are relying on a grain of salt to snuggle up next to a—bacteria? In doing so, you may have more success trying to shoot a swarm of mosquitos with a shotgun.

So, why add salt?

Many producers believe they are changing the pH—in this case, the acidity—of the poultry house floor by adding salt. This isn’t true—there is no hydrogen in salt. Therefore, salt doesn’t bring any pH impact to the floor.

One source of confusion may come from the use of sodium bisulfate (or sodium hydrogen sulfate), a sodium “salt” used to form ammonium sulfate salt when hydrated in poultry litter. When sodium bisulfate (PLT) gets wet in litter, the sodium ionizes and the resultant hydrogen sulfate

then ionizes into hydrogen (hydronium) and sulfate ion (to form ammonium sulfate), locking ammonia up in the acid environment. Poultry Guard also brings “ready to use” acid—twice as much “hydrogen” per active ingredient molecule than sodium bisulfate—which also acidifies the floor and provides sulfate to bind ammonia. Salt does not provide acid and does not affect pH. Period.

So, again, why add salt?

Many producers think they are killing the bacteria by providing chlorine to the floor—like chlorine provided in the hypochlorite in bleach. This is not exactly true, unless the salt is dissolved or ionized in water. Imagine that a granule of salt is the size of a professional football stadium, a bacterium is the size of a pebble in the parking lot and the cheap parking lot spaces are about a 15-minute walk from the stadium gates. How are you going to get that chlorine in the sodium chloride granule in contact with the bacteria?

The answer is obviously to add water and dissolve the salt. But does adding lots of water without a really good plan of attack—without a true knowledge of how much salt or water to use—help or actually hurt bacteria levels? Salt is actually used as a diagnostic tool for poultry cocci testing, with the *Eimeria* oocyst being able to withstand and easily survive soaking in a fully saturated solution of salt for many days¹, although *E. tenella* is the most sensitive (still resistant...) of most poultry-relevant cocci species.

So, there must be a good use for salt by itself? Such as an intervention for Blackhead?

Remember, while salt may not be such a good idea for microbes, salt may be a good idea for worms. If a salt granule is a pro football stadium, then a worm egg is the size of Godzilla—which is more of a fair fight. Wetted salt kills more complex organisms such as *worms* (histomonads) in the soil pad by overwhelming osmotic gradients across cellular or more complex tissue

layers. This can happen with either “dry” contact with a granule or with slow dissolution and ion dissipation through the pad with normal litter moisture levels obtained during flock grow-out.

Managing water activity (A_w) of litter is key to microbial and parasite control with pH and salt (and temperature?) in litter or pads, more so than the common “% moisture” term that is reported to veterinarians, production managers and service techs. Litter A_w can be impacted greatly by the addition of salt, an important concept discussed further below.

So, what does kill a microbe in litter?

Temperature can be key to controlling pathogens in litter. Very early studies on heat sensitivity of *Eimeria* (coccidia) to heat and drying (i.e., low A_w and/or % moisture) showed that all coccidian oocysts did not respond the same to moderately high temperature gradients². Composting can help increase temperature and further impact moisture and A_w of litter, and increase ammonia release (remember that ammonia is moderately basic or “high” pH). While some heat can kill microbes, it is the length of time and amount of heat combined that determine kill efficiency. And, remember, all microbes are sensitive to heat available in a poultry production barn, compost or no compost.

Will disinfectants kill microbes in litter or on the pad?

Some of the most modern chemical classes that are effective against many viruses, fungi and bacteria have shown very poor efficacy in the field against pathogenic oocysts, including quats, cresylic acid, bleach and sodium hydroxide³. Bleach was mostly ineffective even if used straight⁴ or diluted 50:50 in the lab⁵ (depending on exposure time), while iodophore at high concentration, formaldehyde (either in saline or

CONTINUED ON NEXT PAGE



with a detergent), and ammonia did show some efficacy against oocysts³, as have phenols at high exposure times^{5,6} and some peracetic acid products. All of these disinfectants work better against unsporulated oocysts, similar to killing *Clostridium* spp. bacteria early versus waiting until they form a spore coat. It should be noted that most all of the testing cited in the article has been done in the laboratory in clean conditions or *in vitro*, not simulating the high organic load in true poultry production.

The very frustrating take-home message for many producers and poultry professionals is that there is a clear disconnect between pathogen control in the barn, pathogen disinfection in the hatchery, specific microbes listed on the EPA approved label of registered disinfectants, applications rates to disinfect (especially to fog) based on non-porous surfaces versus the “real life” surfaces in the field. Truth be told, throw in marketing claims, word of mouth, and legacy products no longer available, and it is no wonder producers throw down salt hoping to get some result.

...it will take much more disinfectant than the typical 1/2-ounce-per-gallon rate used in hatcheries to impact anything in a chicken house or turkey barn.

Even successful scientific approaches to field-validating poultry pathogen control products and processes have left integrators continuing to guess what to believe⁷. When looking at EPA-registered disinfectants, always ask about the percent organic load the disinfectant was tested against and the label rates matching those tests. Regardless of the product, it will take much more disinfectant than the typical 1/2-ounce-per-gallon rate used in hatcheries to impact anything in a chicken house or turkey barn.

There has been another “sodium” that has recently made research news as a cocci oocyst control compound. Metam sodium (MS, a.k.a. sodium N-methyldithio-carbamate) is a widely used soil pesticide and is used to control vegetation, fungus, insects and nematodes. In a study published in 2010, 300 parts per million (ppm) metam sodium was applied to 2-3-week-old litter containing either *E. maxima*

and/or *E. acervulina* in an aqueous, soupy slurry⁸. The 12-hour minimum exposure time was needed for the MS to diffuse and penetrate into the oocyst and to reduce output from infected birds. The authors did concede it was likely that even higher concentrations and longer exposure times would be needed to justify further consideration into the practical use of MS, in addition to safety testing for bird exposure approval and investigation of possible residues⁸.

So it appears that moisture—either existing moisture if litter is already wet after birds are gone or by adding water to litter or the pad—is critical for getting salt and disinfectants to work.

So it appears that moisture—either existing moisture if litter is already wet after birds are gone or by adding water to litter or the pad—is critical for getting salt and disinfectants to work. But can adding water to the pad or litter without a proven disinfectant and strategy (sequence, timing, concentrations of chemicals, applications rates or other moisture added from pesticide application) actually *increase* microbial content by increasing the A_w of the litter?

The short and confusing answer is: Maybe, but it depends on the target organism.

Poultry live production can take a page from the food side of this business to at least recognize that much of microbial growth in foods is controlled by pH, water activity (A_w), temperature, oxygen level and time (or timing). Most of the critical microbial growth or survivability in litter can also be impacted by these five parameters, and is further impacted by the proper use of disinfectants alone or paired properly with other chemical interventions. This is an important concept to consider for 2018 and beyond: multiple chemical pathogen hurdles in a well thought-out sequence, timing being crucial, with variation based on seasons (temperature and humidity) and specific economic pressures or pathogen focus (i.e., dermatitis versus *Salmonella* at the plant, profitable sales of paws versus ammonia mitigation or insect pressures).

Salmonella is increasingly being considered as a main driver in deciding what processes to implement and which products to use between flocks and/or at cleanout (or at least in the brood end or in the brooder hub). Very early studies of *Salmonella* death in used litter showed two very clear possibilities⁹:

1. There is a range of moisture measured by A_w that contributes to the low survivability of *Salmonella* (between 0.9 and 0.4), and;
2. High pH due to ammonia already in the litter likely dissolved in the moisture available to the *Salmonella* by proximity and in the surrounding moisture.

Later studies confirmed these observations at acidic pH (pH=4) and water activity at 0.84 (below 0.91)¹⁰. In another study, litter pH levels at or below 3.4 were required to achieve significant *Salmonella* kills in litter with moisture around 24 percent (A_w was not reported)¹¹. In this study, an equal application rate of 100 lb/1,000 ft² of the sulfuric acid product (i.e., Poultry Guard[®]) achieved greater pH reduction than sodium bisulfate (i.e., PLT[®]), presumably due to the greater amount of acid (twice the amount of hydrogen) per molecule or the “freely available” cationic hydrogen versus the bisulfate sodium dissociation and intermediate hydration requirement.

So, the percent moisture, A_w and shifting of pH (either up or down) of the litter all play a strong role in the “natural” Salmonellacidal activity. How, then, can *Salmonella* even survive any between flock time, outside of the host chicken or turkey?

Part of the reason is that the entire floor of the house or barn is not the same—not the same moisture, pH or levels of microbes. Poultry professionals, custom applicators and growers tend to view the floor as a single uniform unit.

Researchers at the University of Maryland¹² identified prime areas of *Salmonella* in a broiler and layer farm to be areas with low airflow and, thus, higher percent moisture and A_w . In a follow-on study, the same research group found that high levels of both *E. coli* and *Salmonella* were found in broiler barn areas with A_w greater than 0.9 and moisture content greater than 35 percent¹³. But high moisture does not always correlate to higher levels of a pathogen.

Eimeria oocysts must sporulate to become infective to the turkey or chicken. The cocci oocyst is excreted in an

undifferentiated form and sporulates based on levels of oxygen, moisture and heat. Sporulation of *E. maxima* is most efficient in dry litter (16% moisture) versus wet litter (62% moisture)¹⁴. The low oxygen content and/or oocidal ammonia level attained in most wet litter may prevent sporulation or damage/kill oocytes. So cocci like dry litter, not wet, for sporulation. This is in contrast with observed high cocci-challenged barns where the litter is likely wet, meaning there could be a higher survival of oocysts in wet litter, but the delay in sporulation means that *timing* of cocci intervention in the barn is critical. Waiting to treat dry areas with an *Eimeria* killing intervention will reduce the efficacy of the chemicals.

Finally, what data do we have on the sequence of applying chemicals to the poultry house or barn floor?

Recent research shows that there is a difference in the response of salt and acid sequence for Gram positive (such as *Clostridium*, Strep, Staph, MS and MG) versus Gram negative bacteria (such as *E. coli* and *Pseudomonas*). *Salmonella* were sensitive to acid treatment whereas *Listeria* was more sensitive to salt/osmotic challenge (low A_w)¹⁵. More important, the *sequence of adding acid, waiting 2-3 days*, then adding salt proved most effective for both classes of microbes. The opposite has been reported in prior studies, showing that in certain conditions Gram positives are resistant to salt alone due to osmoadaptive mechanisms. Susceptibility to chemical interventions may also be determined by temperature, growth phase of the pathogen, oxygen, etc.

To summarize, take-home points to consider include the following suggestions:

- Some pathogens might have higher concentrations in wet areas (i.e., *Salmonella*) while others will be more evenly or randomly dispersed (i.e., cocci/*Eimeria*, *Clostridium*, MS or MG) with different levels of sporulation, survivability and infectivity.
- Not all areas of the floor are equal—focus more of the dry chemical application to wet areas or areas with poor ventilation, leaving the bulk of that area’s work to solid pH/drying agents, and then adding salt later.
- Focus more of the wet chemical application to dry areas while passing over wet areas under water lines and low air-flow areas, for example.

CONTINUED ON NEXT PAGE

- The sequence of applying salt is critical. It is best applied after a pH shift or modification (acid or alkaline), 2-3 days after the microbe is damaged by either acid or basic chemistry.
- Perform chemical intervention strategies within 36 hours after birds are gone. Prevent sporulation of *Eimeria* and *Clostridium*—these spores are simply harder to kill than the weaker, thinner vegetative form.
- Consider a split cost structure with contract broiler growers on litter amendments—use half up front (50-75 lbs/1,000 ft²) for acidification pathogen control important to the plant and the other half (50-75 lbs/1,000 ft²) for ammonia control just prior to new flock.
- Add an acidic iodine disinfectant with an approved EPA *Salmonella* (and *E. coli*, *Pseudomonas*, *Clostridium*, etc.) label, heavy organic load testing and heavy organic load label instructions to further drive down pH and achieve contact in litter. One example is Dyne-o-Might[®], having both an organic acid (cited as critical for killing pathogens like *Salmonella*¹⁶) and an inorganic acid.
- Prevention of water leaks may be one of the most critical tools in preventing bacterial pathogen survival, regardless of the thoroughness of decaking.
- For high health-challenged barns or farms, consider once per year moving pH up (alkaline) instead of down with hydrated lime (not Ag Lime, which is calcium carbonate and will not affect pH), likely in concert and working with ammonia liberation after composting or windrowing. Use an aldehyde or phenolic disinfectant with alkaline pH and hydrated lime.

⁷Tamashi, G., "Testing Disinfectants for Efficacy" (1995) *Rev Sci Tech Off Int Epiz*, 14(1): 75-79.

⁸Fetterer, R.H., et al. "Metam Sodium Reduces Viability and Infectivity of *Eimeria* Oocysts" (2010) *J. Parasitology*, 96(3): 632-627.

⁹Turnbull, P.C.B., and Snoeyenbos, G.H., "The Roles of Ammonia, Water Activity and pH in the Salmonellacidal Effect of Long-Used Poultry Litter" (1973) *Avian Diseases* 17(1):72-86.

¹⁰Payne, J.B., Osborne, J.A., Jenkins, P.K., and Sheldon, B.W., "Modeling the Growth and Death Kinetics of *Salmonella* in Poultry Litter as a Function of pH and Water Activity", (2007) *Poultry Science* 86:191-201

¹¹Payne, J.B., Kroger, E.C., and Watkins, S.E., "Evaluation of Litter Treatments on *Salmonella* Recovery from Poultry Litter", (2002) *Journal Applied Poultry Research* 11:239-243

¹²Mallinson, E.T., et al "A Management Technique to Identify Prime Locations of *Salmonella* Contamination on Broiler and Layer Farms", (2000) *Journal Applied Poultry Research* 9:364-370.

¹³Eriksson, C.L., et al "Effect of Dry Litter and Airflow in Reducing *Salmonella* and *Escherichia coli* Populations in the Broiler Production Environment" (2001) *Journal Applied Poultry Research* 10:245-251.

¹⁴Waldenstedt, L., et al, "Sporulation of *Eimeria maxima* Oocysts in Litter with Different Moisture Contents" (2001) *Poultry Science* 80:1412-145.

¹⁵Tiganitas, A., et al., "Study of the Effect of Lethal and Sublethal pH and A_w stresses on the inactivation or growth of *Listeria monocytogenes* and *Salmonella typhimurium*" (2009) *International Journal of Food Microbiology* 134:104-112.

¹⁶Chung, C.C., and Goepfert, J.M., "Growth of *Salmonella* at Low pH" (1970) *Journal Food Science* 35:326-328.

3-Nitro[®] is a registered trademark of Pfizer/Alpharma
 PLT[®] is a registered trademark of Jones Hamilton
 Poultry Guard[®] is a registered trademark of Trinico Ag
 Dyne-o-Might[®] is a registered trademark of Preserve International

References

¹Ryley, N.G., and Ryley, J.F., "Effects of Saturated Sodium Chloride Solution on Coccidial Oocysts", (1978) *Parasitology*, 77: pp 33-39.

²Chang, K. "Effects of Temperature on the Oocysts of Various Species of *Eimeria* (Coccidia, Protozoa)", (1937) *Am. J. Hyg.* 26: 337-351.

³Campbell, I., et al "Effect of Disinfectants on Survival of *Cryptosporidium* Oocysts", (1982) *Veterinary Record*, 111(18): pp 414-415.

⁴Fayer, R., "Effect of Sodium Hypochlorite Exposure on Infectivity of *Cryptosporidium parvum* Oocysts for Neonatal BALB/c Mice" (1995) *Applied Environ Microbiol* 61(2): 844-846.

⁵Guimaraes, Jr., J.S., et al., "In vitro Evaluation of the Disinfection Efficacy on *Eimeria tenella* Unsporulated Oocysts Isolated from Broilers" (2007) *Brazilian J. Veterinary Parasitology*, 16(2): 67-71

⁶Williams, R.B., "Laboratory Tests of Phenolic Disinfectants as Oocides Against the Chicken Coccidium *Eimeria tenella*", *Veterinary Record* (1997) 141, pp. 447-448.