



Avian mycoplasma myths

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Avian mycoplasma myths – Chris Morrow

- 1) *Mycoplasma synoviae* (MS) does nothing
- 2) MS does not have airborne spread
- 3) It is not MS because there is no joint involvement
- 4) You must use F strain in the first stage of eradicating MG.

That MS does nothing is an interesting view. There is great variation in the potential of MS strains to cause disease but avian Mycoplasma experts worldwide agree that MS is a pathogen, it has its own chapter in Diseases of Poultry describing its pathogenic potential and some of the overt clinical effects emerge in various regions from time to time. The rumour that MS does nothing seems to originate from people trying to sell MS positive stock (usually with few clinical problems but subclinical effects can be more costly). There are certain areas in the world where MS cannot be controlled (and there is no synovitis) and the only solution to date is to argue that the local strains do nothing. Interestingly new MS syndromes are often described in these areas (Well they are not going to be described as often where MS is adequately controlled).

Additionally in layer operations around the world there is often prophylactic administration of anti-mycoplasmal drugs, often routinely in-feed during lay. Sometimes these antibiotics are being marketed as increasing egg production in a non-specific way. They are lucky antibiotics that have a zero withdrawal time. Actually these antibiotics are probably suppressing MS making diagnosis more difficult (MS does nothing because of constant antibiotics!) and the antibiotics may also be helping to control *Brachyspira* sps effects, *Avibacterium paragallinarum* and possibly more dramatic bacterial infections like *E. coli*, Salmonella (especially SE and *S. gallinarum*) and *Pasteurella multocida*. In some areas even breeders receive this sort of prophylaxis during lay. These antibiotics also depress seroconversion to MS (and *S. pullorum* etc) in direct and indirect ways. But if antibiotics were the total solution to mycoplasma infections then the poultry industries would have solved these problems in the 1950s when antibiotics first became available and we would not be discussing mycoplasmas in the 21st century.

The idea that a syndrome cannot be caused by MS because no joint problems can be seen (or that a syndrome must be MS because joints are affected) is perpetuated because of poor knowledge of MS in Asia and elsewhere, in part associated with the lack of local diagnostic confirmatory tests and lack of expertise to rule in or out MS. Serology is over-relied-upon and is of little use in many cases especially in vaccinated flocks (for starters you would want to see the appearance of disease be associated with MS seroconversion). Mycoplasma culture and identification in tropical laboratories is difficult and PCR has also got problems (quality control, an understanding of sensitivity in the case of MG diagnosis, and the need for strain identification where birds have been vaccinated with live mycoplasma vaccines). Sometimes following hatchability and airsacculitis in pips is the best technology to monitor your mycoplasma control in breeders.

MS is often said to be an emerging pathogen. We become more aware of its effects as we get greater control of MG. Where MG is common it is common to assume all clinical cases that look like avian mycoplasmosis are MG. In Australia as we started to use ts-11 to control MG we found that we had some chronic respiratory disease cases where, on investigation, we found MS. Indeed the parent strain of MSH was isolated from one such case. MS strains have been found that are able to cause egg production losses (reduce total eggs and increase FCR) including egg production drops

during lay, make respiratory vaccine reactions worse, vertical transmit and indeed nearly anything (except neurological effects associated with some strains of MG) that has been seen with MG. MS in the laboratory does not appear to be able to cause primary uncomplicated respiratory disease like MG (not since the 1970s but this may be because of improvements in laboratory housing). Some strains of MS do cause infectious synovitis (IS) but there are published examples of MG strains being isolated from joints with IS in Japan and Australia. MS is a great mimic of MG. Clinically we can usually only conclude that we have mycoplasmosis without further diagnostic workup. MS has also been recently described as causing glass top eggs, triggering *E. coli* peritonitis in layers at the beginning of lay and triggering Enterococcus associated joint amyloidosis.

Glass top eggs syndrome (Egg apical abnormality) emerged in the Netherlands in 2000 and has become a very important economic loss to egg producers in Western Europe, Turkey, Korea and Japan. It is probably also in other places (the lack of MS control under OIE avian mycoplasma guidelines has certainly contributed to this. Something some countries insist on). MSH has been shown the laboratory to decrease EAA eggs in challenge trials. In field trials in Japan the condition did not reappear on farms that used MSH vaccine.

MS can be vertically and horizontally transmitted. The horizontal spread can be direct or indirect and can even occur in the hatchery. There have been no successful experiments or studies that I know of looking at indirect spread (spread between birds that are not in-contact) but field experience and using *M. hyopneumoniae* studies as a model means all Mycoplasma experts consider this is possible. I have seen cases where MS spread twice over 2 km from a commercial layer site to GP farm. (Indeed ILT did the same). This transmission was airborne with no other means possible and MS strain identification on isolates from the layer and breeder farm supported this conclusion.

I have come across the myth that MS is not airborne in both the US and UK. Some Mycoplasma experts have often played down the role of airborne transmission when advising chicken farmers on the assumption that there is nothing the farmer can do about the isolation of farms after they have been built so it was better to emphasise biosecurity features that the farmer could influence (visitor entry etc). This has been mis-interpreted at times by some producers as MS cannot have airborne transmission. Indeed the advice to isolate sheds of birds on multiage GP farms by 400 yards was, I suspect, a guess by an early mycoplasma expert in response to the question "What distance is well isolated?". We built GP multiage farms based on this distance all over the world and it works adequately for MG control but MS seems to be harder to contain.

With the introduction of live vaccines that can increase the resistance of chickens to infection by wild strains (the ability of killed vaccines to do this is very limited) we now have something we can practically do to prevent the infection of flocks with wild avian mycoplasma strains. So we no longer have to ignore airborne transmission as something that we are powerless against except using antibiotics. Vaccinate if the risk is too high from your neighbours.

Most transmission studies in the laboratory have looked at in-contact horizontal spread. Here the challenge is very strong and may overwhelm vaccinal immunity. Are these results directly applicable to the field situation? The example of MSH in the laboratory only being able reduce EAA incidence by 50% in a strong in-contact challenge model (40% incidence in the control group with all groups receiving concurrent IM and IT field IBV) compared to complete prevention in the field in Japan (4-10% EAA eggs) suggests not. Industrial poultry production does not mix flocks and allow in-contact

challenge to occur nowadays. The minimum separation between groups would be about 1 m and over this distance challenge is greatly reduced (see the control birds in Feberwee's studies on transmissibility of MG strains that were placed at 65 cm from infected birds and did not become infected). Useful immunity in the field only needs to be strong enough to stop airborne transmission with biosecurity stopping in-contact transmission. Given that immunogenicity (and protection against in-contact challenge) and pathogenicity appear to be positively correlated in naturally occurring MG strains and vaccines derived from them then a problem occurs demanding stronger protection. For F strain published studies show a loss of about 7 eggs per hen housed in vaccinated flocks that are not challenged compared to unchallenged unvaccinated flocks. This is an expression of residual pathogenicity and other probable losses due to poorer FCR have not yet been published. The mutagenesis event involved in the development of the ts vaccines appears to have uncoupled the relationship between immunogenicity/protection and pathogenicity as these vaccines are apathogenic (for example comparison of vaccinated and unvaccinated flocks that are not challenged shows no difference in production). For this reason I consider ts vaccines to be a generation further in development of live Mycoplasma vaccines.

The idea that the immunity provided by ts-11 is not strong enough to protect against strong field challenge is largely the experience (but not published) of Kleven but not the experience of Whithear. It is confounded by the in-contact pen trials looking at protection against the spread of R strain which I am arguing is not relevant to the field situation. I am not saying that F strain cannot displace field strains and that then ts-11 can displace F strain but it will be faster to just use ts-11. Maximum effect of ts-11 will not be necessarily seen till all flocks on a site have been vaccinated in my experience. (It is important to not assess maximal returns in field trials until the whole farm is vaccinated). The flocks need to be protected from challenge until at least three weeks after vaccination as this time is needed for immunity to develop (and from antimycoplasmal antibiotics). Rearing off site can help here or the mycoplasma challenge from surrounding flocks can be dampened by antibiotics to surrounding during a transition period until all flocks are vaccinated rather than using F strain. US registration of ts-11 means that a lot of birds in the US (and outside the US using product from the US) are not vaccinated till 10 weeks of age. This means flocks are unprotected till 13 weeks. In contrast flocks in Australia and elsewhere get earlier protection by vaccinating from 3 to 6 weeks of age. This is just a registration abnormality in the US.

When I went to Vet school the emphasis was on MG eradication and freedom and all parts of the avian industries had bought into this idea. As time has gone on even breeder operations are now reluctant to kill MG positive flocks in many parts of the world. The layer industry has not tried to control MS at all in many areas and incidence is often more than 80+% of flocks and these have become a MS reservoir much feared by the meat breeder and turkey industries in these areas. Our farm set ups based on 400m as a protective distance have been realized to be failing us in our control of MS. Mycoplasma freedom has a place but sometimes it is impossible to achieve especially as Mycoplasma free flocks are totally susceptible to becoming infected. Vaccination can increase the resistance of a flock to infection with wild Mycoplasma strains and has a place in situations where the risk of reinfection with wild Mycoplasma strains is greater than the farmer is willing to accept. Thus the idea of using vaccination on the way to eradication is not for the majority of poultry farmers in poultry dense areas as free flocks are a liability and the value of production continuity and reliability is more important than having a flock that you can show the world is mycoplasma free. Live mycoplasma vaccination can be seen as insurance.

The follow-on benefits from mycoplasma control by vaccination is reduced antibiotic dependence and usage and a lessening in the severity of non-mycoplasma diseases. Australia has reaped this benefit from mycoplasmal vaccination for the last two decades :- Death of a final myth; that you cannot produce poultry without antibiotics.