Swine Mycoplasmas (scores for M. hyopneumoniae)

Control Tools

Diagnostics availability

Commercial diagnostic kits available worldwide

Many available worldwide. Most swine producing countries diagnostic labs.
No diagnostic kits for M. hyorhinis and M. hyosynoviae.

GAP: No diagnostic kits for M. hyorhinis and M. hyosynoviae.

Commercial diagnostic kits available in Europe

Freely available in Europe particularly the standard PCR.

GAP: Validation of PCR assays to diagnose a broad spectrum of M. hyopneumoniae field isolates.

Diagnostic kits validated by International, European or National Standards

Unknown.

Diagnostic method(s) described by International, European or National standards

Unknown.

Commercial potential for diagnostic kits in Europe

Likely to be improved by the development of novel laboratory and pen-side tests.

GAP: Validation tests for oral fluid.

DIVA tests required and/or available

Not likely to be developed in the near future. Probably need to eradicate the organism and not differentiate vaccine from field strains.

Opportunities for new developments

New methods of vaccinations or multiple vaccinations at weaning. Quantification of M. hyopneumoniae may help to better assess the epidemiology and the efficacy of (new) control measures.

GAP: Development of better vaccines and the most appropriate route of administration for M. hyopneumoniae, and effective vaccines for M. hyorhinis and M. hyosynoviae.

Vaccines availability
Commercial vaccines availability (globally)

Widely available. Most commercial vaccine producers have at least one product available.

GAPS:
Development of better vaccines and the most appropriate route of administration for *M. hyopneumoniae*, and effective vaccines for *M. hyorhinis* and *M. hyosynoviae*.

Assessment of vaccine efficacy against *M. hyopneumoniae* field isolates of different virulence.

Commercial vaccines authorised in Europe

Widely available. These include whole cultures or sub-unit vaccines with outer membrane proteins.

A DNA vaccine using a p42 heat stable protein has also been produced.

GAP: Development of better vaccines and the most appropriate route of administration for *M. hyopneumoniae*, and effective vaccines for *M. hyorhinis* and *M. hyosynoviae*.

Marker vaccines available worldwide

None.

Marker vaccines authorised in Europe

None.

Effectiveness of vaccines / Main shortcomings of current vaccines

Most appear effective as they reduce clinical symptoms, lung lesions and production losses. However, they do not significantly reduce the transmission of the pathogen, and cannot prevent the pigs from becoming colonised.

GAP: Improvement of the efficacy of the current vaccines on pathogen transmission.

Commercial potential for vaccines in Europe

Unknown.

Regulatory and/or policy challenges to approval

Probably none required.

Commercial feasibility (e.g manufacturing)

Unknown.

Opportunity for barrier protection

Probably unlikely at the moment.

Opportunity for new developments

May be possible in immunotherapy to prevent the development of the huge cuffing reactions that are present in these cuffing pneumonias.

GAP:
Identify virulence factors of *M. hyopneumoniae* that cause lymphoid cuffing and incorporate in vaccine.
Immune response required to protect against clinical disease caused by *M. hyorhinis* and *M. hyosynoviae*. Development of better vaccines for *M. hypneumoniae* and effective vaccines for *M. hyorhinis* and *M. hyosynoviae*.

### Pharmaceutical availability

**Current therapy (curative and preventive)**

Treatment with existing antibiotics is often effective, but over the years some of the agents have lost some of their activity. Antimicrobial resistance has been shown for fluoroquinolones, macrolides-lincosamides, and tetracyclines. Vaccination is typically preferable to treatment with antibiotics.

**GAPS:**

Improved strategic use of antibiotics to minimize potential for the development of resistance in target mycoplasmas and to reduce the use of antibiotics in general terms.

Improved treatment strategies for minimizing clinical disease due to *M. hyorhinis* and *M. hyosynoviae*.

**Future therapy**

New treatments with anti-inflammatory substances.

**GAP:** Novel vaccines to improve vaccine efficacy.

**Commercial potential for pharmaceuticals in Europe**

Probably will increase as knowledge of cytokine control of inflammation improves.

**Regulatory and/or policy challenges to approval**

None.

**Commercial feasibility (e.g manufacturing)**

Unknown.

**Opportunities for new developments**

Will emerge with new knowledge but at the moment unknown.

### New developments for diagnostic tests

**Requirements for diagnostics development**

Pen-side tests for antigen and antibody and use on non-invasive procedures e.g. saliva or nasal swabs would be useful. Quantification of number of organisms could also be helpful.

**GAPS:**

Validate diagnostic for oral fluid.

Develop diagnostic assays for clinical disease caused by *M. hyorhinis* and *M. hyosynoviae*.

**Time to develop new or improved diagnostics**

Unknown.
Cost of developing new or improved diagnostics and their validation

Unknown.

Research requirements for new or improved diagnostics

Continued molecular improvements and knowledge of genome of mycoplasma and how they regulate the host immune response or avoid it.

**GAPS:**

Detection and quantification of *M. hyopneumoniae* in BALF from live animals.

Better understanding of the regulation of the host immune response.

Technology to determine virus freedom in animals

As above.

New developments for vaccines

**Requirements for vaccines development / main characteristics for improved vaccines**

Continued molecular improvements and knowledge of genome of mycoplasma and how they regulate the immune response or avoid it.

**GAPS:**

Identification of factors that impact immunopathology associated with *M. hyopneumoniae* infection.

Exact immune mechanism required to clear *M. hyopneumoniae*.

Immune response required to protect against clinical disease caused by *M. hyorhinis* and *M. hyosynoviae*.

Time to develop new or improved vaccines

Unknown.

Cost of developing new or improved vaccines and their validation

Unknown.

Research requirements for new or improved vaccines

Continued molecular improvements and knowledge of genome of mycoplasma and how they regulate the immune response or avoid it.

New developments for pharmaceuticals

**Requirements for pharmaceuticals development**

Continued molecular improvements and knowledge of genome of mycoplasma and how they regulate the immune response or avoid it.

**GAPS:**

Improved understanding of antimicrobial alternatives to antibiotics effective against mycoplasma.
Improve vaccines efficacy.

**Time to develop new or improved pharmaceuticals**
Unknown.

**Cost of developing new or improved pharmaceuticals and their validation**
Unknown.

**Research requirements for new or improved pharmaceuticals**
Continued molecular improvements and knowledge of genome of mycoplasma and how they regulate the immune response or avoid it.

**GAP:** Improved knowledge of virulence mechanisms and protective immune responses.

**Disease details**

**Description and characteristics.**

**Pathogen**

Mycoplasmosis is a term frequently used to denote enzootic pneumonia of pigs, but could in fact refer to disease caused by three species of Mycoplasma, i.e. *M. hyopneumoniae*, *M. hyorhinis* and *M. hyosynoviae*. *M. suis* can affect erythrocytes and cause a disease formerly called “eperthyrozoonosis”. The impact of other mycoplasma species found in swine such as *M. hyopharyngis* and *M. flocculare* is not clear so far.

*M. hyopneumoniae* is the primary causative agent of enzootic pneumonia, which is historically one of the most common chronic respiratory diseases of swine. It also plays a primary role in the porcine respiratory disease complex (PRDC). *M. hyorhinis* can cause polyserositis, arthritis, pneumonia and otitis media in piglets, while *M. hyosynoviae* can cause arthritis in fattening pigs.

All of these are pleomorphic microorganisms that lack a rigid cell wall, have very small genomes and limited biosynthetic capabilities. They have adapted to the parasitic mode of life and have pig respiratory tract as a natural habitat. They can be cultivated in artificial media, but are fastidious and require complex media for growth in vitro.

Among the three species, *M. hyopneumoniae* is economically the most important and the most studied.

**GAPS:**

Characterizing the genetic differences between field isolates of all three mycoplasmas.

Studying the relationships/interaction between *M. hyopneumoniae* and *M. hyorhinis* in pneumonia.

**Variability of the disease**

Found only in pigs. May have various antigenic forms (although these have never been formally classified), which may relate to a whole range of surface proteins produced to evade the host defence mechanisms.

There is a high strain variability at genomic level.

**GAPS:**

The role genetics plays in variation of virulence of all three organisms.

Genetic markers for virulence.

Why do *M. hyorhinis* and *M. hyosynoviae* cause disease only intermittently?

**Stability of the agent/pathogen in the environment**
All three Mycoplasma species require the pig respiratory tract and survive for short periods in the environment. *M. hyopneumoniae* can survive approximately 2 weeks in rain water and 1 week air-drying. *M. hyorhinis* can also survive drying for 1 week, but some *M. hyosynoviae* strains can tolerate it significantly better and can survive air-drying for about 1 month.

**Species involved**

**Animal infected/carrier/disease**

Found only in pigs.

**Human infected/disease**

None ever demonstrated.

**Vector cyclical/non-cyclical**

No vectors.

**Reservoir (animal, environmental)**

No vectors, although wild boar and feral pigs can be infected.

**Description of infection & disease in natural hosts**

**Transmissibility**

Disease is easily transmitted by direct contact. Factors that influence transmission and dynamics of the disease include stock density, housing styles, ventilation and climatic condition.

**GAPS:**

Length of transmission of all three mycoplasmas by gilts/sows.

Do other pathogens influence the transmission of the microorganism?

**Pathogenic life cycle stages**

No life cycles.

**Signs/Morbidity**

Principal clinical sign of enzootic pneumonia is an intermittent dry cough which may last for weeks or months. The disease typically has a chronic course and is often detected only at abattoir checks on cranioventral lung consolidation. An acute breakdown usually occurs when naïve herds become affected when coughing may be more pronounced. In the case of concurrent bacterial and/or viral infection, clinical symptoms will also include fever, laboured breathing, lethargy, anorexia and even death.

Outbreaks of polyserositis due to *M. hyorhinis* usually occur in young animals (3-10 weeks of age) with main clinical signs being laboured breathing, anorexia and reluctance to move, swollen joints and lameness. Some pigs may die acutely, but typically, clinical signs begin to resolve after two weeks, with exception of joint swelling and lameness, which may persist up to six months.

Arthritis due to *M. hyosynoviae* suddenly appears in a herd and usually occurs in animals at the age of 3-5 months. The large joints, in often more than one leg, are typically affected. Clinical signs include difficulty in moving, lameness, arched back and inability to get up. Clinical lameness may last for up to 10 days but in some cases protracted course may occur.

**GAPS:**

How do differences in genetics of the isolates impact the severity of clinical disease?
Recent studies have suggested that pigs may be susceptible to a different strain of *M. hyopneumoniae* – what are the cross-protective capabilities? Do pigs become immune or can they be re-infected?

**Incubation period**

The incubation period for *M. hyopneumoniae* infection is usually 10-16 days but may vary largely under field conditions. Often difficult to ascertain because of slow development of infection. In non-vaccinated SPF pigs, the estimated minimal dose of *M. hyopneumoniae* required to induce pneumonia was $10^5$ colour-changing units (CCU) per pig (corresponding to $10^8$ mycoplasmas).

Evidence of the disease due to *M. hyorhinis* infection usually occurs 3-10 days after exposure.

**GAPS:**

What impacts differences in time to disease and seroconversion?

Which is the real prevalence of arthritis cases caused solely by *M. hyorhinis*? Is there any potention between pathogens that can cause arthritis?

**Mortality**

For *M. hyopneumoniae* infection, the mortality is low in uncomplicated infections (<5%). If complicated by secondary infection with other agents of the PRDC it may cause significant mortality.

Mortality is also low for *M. hyorhinis* and *M. hyosynoviae* infections.

**Shedding kinetic patterns**

*M. hyopneumoniae* and *M. hyorhinis* are possibly shed continuously from infected mucosal surfaces of the lower part of the tracheo-bronchial tree and upper portions of the respiratory tract of pigs, respectively. *M. hyosynoviae* is shed primarily during the acute phase of infection - persistently infected animals shed the organism only intermittently.

**GAPS:**

Are the organisms shed continuously from the dams?

Is there a variation in the amount of *Mycoplasma* shed in the long term?

What are the differences in shedding by the sows between different parities for *M. hyorhinis* and *M. hyosynoviae*?

**Mechanism of pathogenicity**

*M. hyopneumoniae* can be seen on the mucosal surface shortly after infection. The organism causes damage of the ciliated epithelial cells and impedes mucociliary clearance, predisposing the affected tissue to secondary infection. Adherence of the organism to the cilia is a prerequisite for colonization of the respiratory tissue. Adherence is a multifactorial process which involves adhesins expressed by *M. hyopneumoniae* and components of the extracellular matrix of the host. Protein P97, which is also referred to as cilium adhesin is directly involved in the adherence of the organism to cilia. The gene which encodes P97 is a component of a two-gene operon and has six paralogs within the *M. hyopneumoniae* genome. These may be differentially expressed making recognition by the immune system difficult. P97 is not the only protein involved in adherence, as *M. hyopneumoniae* can still bind to cilia after adherence via P97 has been blocked. So far, several other proteins, including P159, P216, Mhp271 and P116, have been identified as *M. hyopneumoniae* adhesins. *M. hyopneumoniae* also induces production of inflammatory cytokines and affects phagocytic capabilities of macrophages as well as function of lymphocytes.

*M. hyorhinis* and *M. hyosynoviae* are common inhabitants of the respiratory tract of pigs and exert pathogenicity only after systemic spread. Under specific experimental conditions, infection with specific *M. hyorhinis* strains may lead to lung alterations.

**GAPS:**

Identification of virulence factors that cause lymphoid proliferation for *M. hyopneumoniae*.

Mechanisms (and conditions) by which *M. hyopneumoniae* spreads to inner organs.
The relationship between systemic spread of *M. hyopneumoniae* and virulence of the strain.

Possible mechanism of potentiation of the pulmonary lesions when both *M. hyopneumoniae* and *M. hyorhinis* are present.

Role of *M. hyorhinis* in pneumonia lesions.

Identification of mechanisms of pathogenicity for *M. hyorhinis* and *M. hyosynoviae*.

Mechanisms (and conditions) that enable *M. hyorhinis* and *M. hyosynoviae* to enter the blood stream, invade and adversely affect local target tissues.

### Zoonotic potential

**Reported incidence in humans**

Not reported in humans.

**Estimated level of under-reporting in humans**

None.

**Risk of occurrence in humans, populations at risk, specific risk factors**

Probably none.

**Symptoms described in humans**

Not reported.

**Likelihood of spread in humans**

None.

### Impact on animal welfare and biodiversity

**Both disease and prevention/control measures related**

Establishing of SPF herds which either remain closed or buy certified SPF pigs.

Preventing stress conditions that may facilitate the systemic spread of *M. hyorhinis* and *M. hyosynoviae*.

**GAP:**

All three mycoplasmas impact animal well-being.

**Endangered wild species affected or not (estimation for Europe / worldwide)**

No.

**Slaughter necessity according to EU rules or other regions**

No, only if welfare cases caused by secondary infection.

### Geographical distribution and spread

**Current occurrence/distribution**

Worldwide.
**Epizootic/endemic - if epidemic frequency of outbreaks**
Endemic, no evidence of epizootic strains.

**Seasonal cycle (seasonality)**
Increased when stocking density increased or winter conditions prevail so that ventilation is reduced. A real seasonal cycle is not present when pigs are reared in large populations inside buildings.

**Speed of spatial spread during an outbreak**
Tends to spread slowly.

**Transboundary potential of the disease**
Follows the pig so therefore will spread across boundaries.

**Seasonal cycle linked to climate**
No, all year round occurrence.

**Distribution of disease or vector linked to climate**
No special distribution.

**Outbreaks linked to extreme weather**
Not as far as is known.

**Sensitivity of disease or vectors to the effects of climate change (environmental changes/land use)**
Not as far as is known.

**Route of Transmission**

**Usual mode of transmission (introduction, means of spread)**
Transmission of *M. hyopneumoniae*, *M. hyorhinis* and *M. hyosynoviae* in field conditions occurs most commonly via direct contact with carrier animal. In many herds the transmission chain starts by sow-to-pig exposure. Subsequently the infection is spread between penmates. Groups of pigs can be infected at mixing and moving and particularly weaning. Nose-to-nose contact is by far the most efficient route, although transmission occurs also between animals housed in the same airspace but without direct nose-to-nose contact. Animals infected at a young age may be excreting the agent over long period. *M. hyopneumoniae* can be transmitted over several km in the field conditions and if the source of infection is a huge number of animals. Limits for the long distance transmission of *M. hyorhinis* and *M. hyosynoviae* are not known.

**GAP:** Assessment of the airborne transmission of *M. hyorhinis* and *M. hyosynoviae* on short and long distances.

**Occasional mode of transmission**
Occasionally aerosol spread and possibly fomites if contaminated with nasal discharges.

**Conditions that favour spread**
Moist environment and overstocking and improper ventilation. Location close to infected neighbouring farms or highway increase the risk for airborne transmission.
Detection and Immune response to infection

Mechanism of host response

Host response to infection with *M. hyopneumoniae* consists of pathological reactions in the lung. Acute phase of the disease is accompanied by hyperplasia of the epithelial cells and perivascular and peribronchiolar accumulation of lymphocytes and monocytes. As the disease progresses, an exudate of mucoid material may be found in the lumen of bronchi as well as lymphoid nodules associated with the airways. The peribronchiolar cuff contains macrophages, monocytes, dendritic cells, T-cell and B-cell lymphocytes. These produce IgA and IgG and also cytokines particularly IL-2, IL-4, TNF alpha, and to a lesser extent IL-1 alpha and beta. IL-6 and IL-8 are found in the mononuclear cells of the alveolar septae.

In the acute phase of infection with *M. hyorhinis*, host response is characterized by fibrinous inflammation of serosal membranes, mononuclear cell infiltration and appearance of serofibrinous to fibrinopurulent polyserositis. Acute arthritis is associated with increased amounts of synovial fluid, swollen and hyperaemic synovial membranes and swollen joints. In the chronic stage fibrous adhesions and articular erosions may occur.

Acute lesions due to infection with *M. hyosynoviae* include oedema of synovial membrane, hyperplasia of synovial cells, perivascular infiltration of mononuclear cells and increased volumes of serofibrinous, brownish synovial fluid.

GAPS:

Identification of factors that impact immunopathology associated with *M. hyopneumoniae* infection.

Exact immune mechanism required to clear *M. hyopneumoniae*.

Immune response required to protect against clinical disease and systemic spread caused by *M. hyorhinis* and *M. hyosynoviae*.

Immunological basis of diagnosis

Maternally derived antibodies may protect piglets from *M. hyopneumoniae* infection for 4-8 weeks but it is very variable depending on the sows’ exposure. It may be 20 weeks before the piglets’ immune system mounts an effective response. IgA antibodies usually appear in the tracheal mucosa from 30 days post-infection followed by IgG. The alveolar washes contain IgG from 45 days post-infection and levels peak at about 80 days post-infection. Serum antibody levels develop at about 8-46 days PI and may peak at 70-80 days PI and persist for at least a year.

After experimental infection with *M. hyorhinis* antibodies can be detected six weeks post infection. Antibody levels may be higher in synovial fluid than corresponding serum titres.

In the case of *M. hyosynoviae* infection, maternal antibodies protect piglets for approximately 10 weeks and subsequent decline of the antibody level seems to coincide with increasing number of tonsillar carriers. Active serological response may take place after 10 weeks of age or later and is not necessarily associated with tonsillar infection.

GAPS:

The actual protective immune response against *M. hyopneumoniae*.

The immune response against *M. hyorhinis* and *M. hyosynoviae*.

Main means of prevention, detection and control

Sanitary measures

A lower risk of enzootic pneumonia is associated with separation of production units and different age groups (all in / all out), good stable climate by adequate ventilation, low stocking density in fatteners as well as optimal acclimatization of gilts and optimal parity distribution of breeding sows. The risk for transmission of *M. hyopneumoniae* from the sow to the offspring decreases when pigs are weaned at a younger age.
Mechanical and biological control

Unknown for *M. hyorhinis* and *M. hyosynoviae*. For *M. hyopneumoniae* control is through the purchase of certified free stock, and effective biosecurity. Health status ought to be matched between selling and buying herd as introducing naïve pigs to infected premises may provoke disease outbreak.

First breeding approaches for an *M. hyopneumoniae*-resistant pig line are promising.

**GAP:** Control measures for *M. hyorhinis* and *M. hyosynoviae*.

Diagnostic tools

Clinical history, post mortem appearance and sampling, histopathology and confirmatory laboratory tests. These include culture of fresh tissue using, immunohistochemistry on fixed tissues, immunofluorescence on smears and frozen sections, and conventional and quantitative real-time PCR. In live animals, combination of assessing clinical signs and seroprevalence is indicative of enzootic pneumonia. PCR diagnostics can be used on nasal, laryngeal, tracheobronchial swabs and bronchoalveolar lavage fluid (BALF). Antigen and antibody ELISAs have also been described.

For analysis of genetic variances between different *M. hyopneumoniae* strains, protocols for Random Amplified Polymorphic DNA Analysis (RAPD), Variable Number Of Tandem Repeats (VNTR), pulse-Field-Gel-Electrophoresis (PFGE), Multi-Locus-Sequence-Typing (MLST), Restriction Fragment Length Polymorphism (RFLP) are available.

**GAPS:**

Validation of the use of oral fluid for diagnosis of *M. hyopneumoniae*.

Validation of upper respiratory tract samples (laryngeal or tracheal swabs) to detect infected animals.

Early diagnostic assay for *M. hyorhinis* and *M. hyosynoviae*.

Vaccines

A number of vaccines have been developed for *M. hyopneumoniae*. These include killed organisms or extracts, with adjuvants, administered intramuscularly or intradermally. Experimentally, intradermal, subunit vaccines vector vaccines, DNA vaccines and live attenuated vaccines have been produced. Vaccination reduces the clinical symptoms, lung lesions and the performance losses due to infection. Vaccination of suckling piglets is considered to be protective independently of the sows’ serological status.

Vaccination alone is not sufficient to eliminate the organism from a herd as vaccines are not able to prevent colonization or to significantly limit transmission of the pathogen.

There are no licensed vaccines for *M. hyorhinis* or *M. hyosynoviae*.

**GAPS:**

Development of vaccines for *M. hyorhinis* or *M. hyosynoviae*.

Mechanisms for development of protective immunity conferred by vaccines.

Mechanisms of the development of cell-mediated immunity in the presence of maternally derived immunity.

Assessment of different vaccination schemes (one versus two-shot), the effect of administration route, adjuvant, type of antigen, antigen dose, etc.

Therapeutics

Many different antimicrobials have shown to be effective: tetracyclines, macrolides, lincosamides, florfenicol, pleuromutilins, fluoroquinolones. Acquired antimicrobial resistance, mainly against fluoroquinolones and macrolides-lincosamides has been described. So far, this is not yet a general problem for treatment in practice.

Eradication of *M. hyopneumoniae* was performed in Switzerland by a combination of management measures and strategic antimicrobial medication.

**GAPS:**
Better validation of medication strategies to minimize development of antibiotic resistance.

Defining guidelines for antibiotic administration for the treatment and control of Mycoplasmosis.

**Biosecurity measures effective as a preventive measure**

Supply of pigs from SPF herds is the only way to keep a unit free of *M. hyopneumoniae* when implementing a high biosecurity standard in the receiving herd. Depopulation and repopulation has been used to eradicate the disease from heavily infected herds. Many units now control the disease using 1 or 2 dose vaccines. This reduces clinical disease and greatly reduces the lung lesions at the abattoir and also reduces pericarditis and pleurisy by preventing predisposition to secondary infection.

No effective biosecurity measures for *M. hyorhinis* and *M. hyosynoviae*.

**Border/trade/movement control sufficient for control**

Unlikely to have much effect unless a country has eradicated the disease.

**Prevention tools**

All the antigen testing and serological testing methods can be used. For *M. hyopneumoniae* among the best checks is to use slaughterhouse monitoring of lungs (lung scoring) and to study closely the production records of the farm.

**GAP:** Strategies for *M. hyorhinis* and *M. hyosynoviae*.

**Surveillance**

Best done on individual farm basis.

**Past experiences on success (and failures) of prevention, control, eradication in regions outside Europe**

*M. hyopneumoniae* eradication has been achieved by partial depopulation (all animals younger than 10 months) combined with antimicrobial treatment. It is considered that this may work, especially in smaller herds. There is always a risk for re-infection, especially in pig dense areas, after having obtained *M. hyopneumoniae* free status.

**Costs of above measures**

Vaccination is cheaper than medication and restocking. Restocking and maintaining a *M. hyopneumoniae* free herd is probably economic in a long time perspective.

**Disease information from the OIE**

**Disease notifiable to the OIE**

Not a reportable disease.

**OIE disease card available**

No.

**OIE Terrestrial Animal Health Code (reference)**

Not available.

**OIE Terrestrial Manual (reference)**

Not available.
Mortality due to *M. hyopneumoniae* infection is not high but morbidity can be high and losses through increased FCR, loss of daily weight gain and increased days to slaughter may be high. In addition, there may be carcass and lung losses at the abattoir, increased medication and vaccination charges. One major effect is the increase in the variability of pig sizes which prevents the effective and timely clearance of pens reducing the efficiency of pig flow. Studying SPF herds in several countries suggests that *M. hyopneumoniae* itself decreases growth around 5%. In the case of secondary infections, this figure will be higher. In addition, the impact of concurrent infection with respiratory viruses can further impact production and animal health. In many pig herds, the major part of antimicrobials is used against respiratory disease, in which *M. hyopneumoniae* is many times involved. So, antimicrobial medication could be significantly reduced if *M. hyopneumoniae* infections were not there or under control by efficient vaccination. Much use of antimicrobials predispose to antimicrobial resistance, which may be transposed to humans. So, in this respect, there is a public health link.

**GAPS:**

Few updated information on the economic impact of enzootic pneumonia.

No information on the economic impact of *M. hyorhinis* and *M. hyosynoviae* infection.

**Direct impact (b) cost of private and public control measures**

Only private control measures are likely to be affected as above.

**Indirect impact**

Not likely other than as complications mentioned in Section “Direct impact on production”.

**Trade implications**

**Impact on international trade/exports from the EU due to existing regulations**

None.

**Impact on EU intra-community trade due to existing EU regulations**

None.

**Impact on national trade due to existing regulations**

None.

**Main perceived obstacles for effective prevention and control**

Main obstacle is the failure of farmers to adopt proper management and biosecurity measures such a strict all in /all out by age with proper cleaning and disinfection, drying and repopulation with EP-free stock.

Overstocking is also an important problem in many pig herds as are airborne transmission over short or longer distances.
Buildings are not always well-designed to reduce mycoplasma infections (e.g. large room and pen sizes, open pen partition that favour direct transmission, airflows between rooms, ...).

**GAP:** Lack of harmonization between management practices, building design and equipment.

### Main perceived facilitators for effective prevention and control

The private veterinarian and the progressive farmer who wants to improve productivity.

### Conclusion

(Other information/gaps:)

The relationship between bacterial, viral and mycoplasmal infections has not been fully elucidated.

Pen-side diagnostics, especially if non-invasive techniques, are needed.

**GAPS:**

Increased identification of virulence factors of all three mycoplasmas.

Better understanding of genetics to improve vaccines, diagnostics and control strategies.

New vaccines and appropriate routes of administration.

### Sources of information

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Expert group members are included where permission has been given

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