Biosecurity considerations for pork production units

Sandra F. Amass, DVM, PhD, dipl ABVP; L. Kirk Clark, DVM, PhD

Summary

This paper summarizes and critiques the peer-reviewed literature concerning biosecurity considerations in the pork industry. Manuscripts concerning source of genetic material and segregation procedures were examined to address the risks of introduction of new genetics. Other biosecurity risks reviewed include transmission of pathogens by aerosol, birds, insects, nonporcine animals, and vehicles; and pathogen survival in dead pigs, feed, manure, water, and soil. Many decisions regarding biosecurity protocols on pork production units are currently based on producer and veterinary experience and opinion, not on scientific research. Consequently, research is needed in many areas either to validate current protocols or to develop new scientifically sound biosecurity measures for the pork industry.

Biosecurity, a relatively new word in our vocabulary, is not found in many dictionaries. Its broad meaning is the literal safety of live things, or the freedom from concern for sickness or disease. Saunders Comprehensive Veterinary Dictionary (Blood DC, Studdert VP. *Saunder's Comprehensive Veterinary Dictionary*. 2nd ed. London: WB Saunders, 1999;132) defines biosecurity as "security from transmission of infectious diseases, parasites, and pests." In this literature review, biosecurity is defined as the protection of a swine herd from the introduction of infectious agents (viral, bacterial, fungal, or parasitic).

Preventing the introduction of porcine pathogens into a swine herd is a continual challenge for pork producers and swine veterinarians. The easiest way to transmit porcine pathogens into a herd is to introduce infected pigs. However, biosecurity protocols must take into consideration a multitude of risks for pathogen introduction. The goal of this manuscript is to compile the current state of knowledge regarding biosecurity in the pork industry, and to identify areas that require further research.

Pathogen transmission among pigs

New technologies to enhance the health status of swine herds produce pigs lacking acquired immunity to many swine pathogens. The risk of introducing disease via new genetic material is increased in these immunologically naïve herds. The cost of introducing new genetic material is also increased due to the extra precautions that must be taken to prevent an epizootic. Genetic material can be introduced into a herd by purchasing semen or live breeding animals. No method of introducing genetic material precludes the possibility of transmitting disease from one herd to another. Thus, veterinarians should know the risks and options available to prevent disease entry.

Genetic material can be introduced into a herd in any of three ways:

- introducing SPF stock,
- introducing early-weaned and age-segregated pigs from the breeding facility, and
- with semen.

Specific-pathogen-free (SPF) stock

Current peer-reviewed studies involving SPF stock were not found. Primary SPF animals were derived by hysterectomy. Meyer, et al.,[1] reported that bacteria, fungi, pleuropneumonia-like organisms, viruses, and ascarids were not detected in 6-week-old hysterectomy-derived pigs reared in isolation. In 1955, Young, et al.,[2] reported that cesarean-derived pigs were initially disease free, but not pathogen free. These pigs were referred to as "minimal disease" pigs, rather than "disease-free" pigs, when they were introduced into rearing facilities. In 1959, Young, et al.,[3] reported that clinical signs of atrophic rhinitis and viral pig pneumonia were not observed in progeny of naturally farrowed dams obtained by hysterectomy. However, contemporary controls were not used in Young's study. The authors of this review recommend that, because of their high-health status, minimal-disease SPF stock should be isolated and strategically vaccinated for diseases in the recipient herd to prevent development of clinical disease after they are introduced into the breeding herd.

Early weaned, age-segregated pigs

Clark, et al.,[4] reported that weaning at 14 days of age followed by age-segregated rearing was sufficient to eliminate transfer of *Actinobacillus pleuropneumoniae*, *Pasteurella multocida*, and in all but one case, *Mycoplasma hyopneumoniae,* to progeny of infected dams. *Streptococcus suis* and *Haemophilus parasuis* were not eliminated from these pigs. Transfer of pseudorabies virus (PRV, Aujeszky's disease), but not porcine reproductive and respiratory syndrome virus (PRRSV), was prevented. The authors concluded that early weaning followed by age segregation procedures were sufficient to eliminate clinical signs of *A. pleuropneumoniae*, mycoplasmal pneumonia, and atrophic rhinitis. Elimination of viral disease was dependent on whether sows were shedding virus at or near farrowing. Bacteriologic findings were supported when Dritz, et al.,[5] reported elimination of mycoplasmal pneumonia and *A. pleuropneumoniae* from pigs

SFA, LKC: Purdue University, 1248 Lynn, West Lafayette, Indiana 47907–1248, email: amasss@vet.purdue.edu

This article is available online at http://www.aasp.org/shap.html

weaned at 7–10 days of age. Thus, there is evidence that using early weaned, age-segregated pigs to introduce genetic material has considerable merit in preventing the transfer of some pathogens to high-health herds.

Semen

Experimental addition of pathogen to semen

PORCINE PARVOVIRUS. Insemination of sows seronegative to parvovirus with 50 mL of semen-buffer solution to which $10^{5.5}$ $TCID_{50}$ per 0.1 mL parvovirus was added, resulted in fetal infection by 30 days post insemination. Parvovirus was not recovered from fetuses of control sows inseminated with uninoculated semen-buffer solution.[6]

Porcine parvovirus was reported to be transmissible via semen after insemination of gilts with semen inoculated with porcine parvovirus. Disadvantages of this technique are that the concentration of pathogens in inoculated semen may not reflect the variable concentration of pathogens in semen of boars after natural infection.

Experimental infection of boars prior to semen collection

PRRSV. Porcine reproductive and respiratory syndrome virus has been isolated from semen of experimentally infected boars. In one study, PRRSV was isolated from semen from four of four boars for up to 43 days after experimental inoculation.[7] In another study, PRRSV was isolated from one of nine experimentally inoculated boars 1 week after infection. However, the authors ignored their results and concluded that PRRSV was not shed through the semen (Prieto C, et al. *Proc IPVS*.1994; 98–98). Christopher-Hennings, et al.,[8] reported detecting PRRS viral RNA in boar semen for up to 92 days after infection of boars. Presence of viral RNA does not indicate that the organism is intact, or infectious.

Transmission of PRRSV after insemination was inconclusive. Yaeger, et al.,[9] reported seroconversion of gilts to PRRSV beginning at 3 days post-insemination with semen from experimentally inoculated boars. Swenson, et al.,[10] inseminated gilts with PRRSV-contaminated semen. Six gilts were bred on 3 consecutive days using extended semen from a PRRSV-negative boar. Five gilts were bred on 3 consecutive days using extended semen from the same boar after he was inoculated with PRRSV. Pregnancy rates did not differ (P=.24) between the two groups of gilts. Gilts did not seroconvert to PRRSV, nor was PRRSV isolated from their reproductive tracts or serum.

PORCINE PARVOVIRUS. Porcine parvovirus was isolated from the testes and seminal fluids of ≤ 8-month-old male piglets born to gilts experimentally inoculated with parvovirus before 55 days of gestation.[11] Thus, boars infected in utero may be persistent carriers of parvovirus.

PSEUDORABIES VIRUS. Pseudorabies virus was not isolated from semen samples collected from 9-month-old boars inoculated intranasally with PRV (Hsu FS, et al. *Proc IPVS*.1984; 24–24).

Porcine parvovirus and PRRSV, but not PRV, were isolated from semen of experimentally infected boars. Transmissibility of these pathogens using semen from infected boars was not demonstrated; although, in one case, seroconversion to PRRSV was reported after insemination. The major disadvantages of experimental infection studies are the limited sample sizes and uncertainty regarding whether results reflect natural infection.

Natural infection

BRUCELLA SUIS. Brucella suis was isolated from 63 of 92 semen samples collected from six naturally infected boars.[12] In support of this finding, Lord, et al.,[13] reported isolation of *B. suis* biovar 1 from semen samples collected from multiple naturally infected boars.

PSEUDORABIES VIRUS. Medveczky and Szabó[14] reported isolating PRV from semen from three of 11 naturally infected, vaccinated boars. The herd of origin had been free of clinical PRV for 1.5 years. Rabbit and mouse inoculation studies were used to discriminate between isolation of wildtype PRV or vaccine virus.

As cited above, *B. suis* and PRV have been isolated from semen of naturally infected boars.

Summary

Parvovirus, PRRSV, *B. suis*, and PRV have been isolated from semen of infected boars. Transmissibility of these agents was not reported; however, seroconversion to PRRSV after insemination was reported in one case. Many other agents reportedly have been found in semen or transmissible by insemination; however, these reports were not published in peer-reviewed sources.

Other methods of pathogen spread

Aerosol

Aerosol transmission of pathogens is difficult to document and research due to many uncontrollable variables. Moreover, thoroughly controlled studies do not reflect field conditions.

Survival of pathogens in aerosols

AFRICAN SWINE FEVER VIRUS (ASFV). Aerosols of ASFV survived at relative humidities of 20%–80% when sampled 1 second after aerosol formation. The virus did not survive well at a relative humidity greater than 30% when sampled 5 minutes after aerosol formation.[15]

BORDETELLA BRONCHISEPTICA. Virulent strains of *B. bronchiseptica* were isolated from the air in farrowing and nursery pig housing units.[16]

PSEUDORABIES VIRUS (PRV). Schoenbaum, et al.,[17] reported that PRV survived longer at 55% relative humidity than at 85% relative humidity (P=.017). Survival improved at 4°C (39.2°F) but was not significantly different from survival at 22°C (71.6°F) (P=.18). Infectivity of aerosolized PRV decreased by 50% in less than 1 hour under optimal laboratory conditions.

SWINE INFLUENZA VIRUS (SIV). Three different strains of SIV survived in aerosol for 15 hours at 21°C (69.8°F) and 15% relative humidity.[18]

VESICULAR EXANTHEMA VIRUS (VEV). Aerosols of VEV were stable at a

relative humidity of <30%.[15]

Vesicular stomatitis virus (VSV): Aerosols of VSV were unstable at a relative humidity of >20%.[15]

Transmission of pathogens in aerosols

ACTINOBACILLUS PLEUROPNEUMONIAE. Torremorrell, et al.,[19] documented airborne transmission of *A. pleuropneumoniae* serotype 1 between pigs in two experimental pens separated by a 1-m (3.28 ft)-long air duct. *Actinobacillus pleuropneumoniae* was isolated from eight of eight aerosol-exposed pigs.

FOOT-AND-MOUTH DISEASE VIRUS (**FMDV**). Gloster, et al.,[20] concluded that high virus output, long survival, low dispersion, and large numbers of susceptible animals exposed for many hours were needed for long distance aerosol transmission of FMDV. Multiple outbreaks of FMD were described in which all of the conditions for aerosol transmission were met; however, other modes of transmission could not be ruled out. In another study,[21] experimentally inoculated pigs were reported to shed a maximum of $10^{4.7}$ ID_{50} per animal per hour. Maximum virus recovery occurred about 41 hours after inoculation.

HOG CHOLERA VIRUS(**HCV**). Hughes and Gustafson[22] reported aerosol transmission of hog cholera virus to six of nine exposed pigs. Air was forced by positive pressure from cans containing pigs inoculated with hog cholera virus to cans containing susceptible pigs.

MYCOPLASMA HYOPNEUMONIAE. Risk factor indices for infection with *M. hyopneumoniae* were developed using characteristics of 55 infected herds and 57 uninfected herds.[23] The most important risk factor for infection was the reciprocal of the square of the distance to the nearest farm. Distances within 3.2 km (1.98 miles) had the highest risk.

PORCINE REPRODUCTIVE AND RESPIRATORY SYNDROME VIRUS (**PRRSV**). Wills, et al.,[24] reported that PRRSV was transmitted among pigs without direct contact over short distances in two of five trials. Transmission by aerosol could not be confirmed because their experimental design did not prevent the transfer of feed, feces, and urine among pens. Torremorrell, et al.,[19] documented airborne transmission of PRRSV (strain VR 2332) among pigs in two experimental pens separated by a 1-m (3.28 ft)-long air duct. Virus was isolated from five of five aerosol-exposed pigs. All five pigs seroconverted to PRRSV. Airborne transmission was not documented with a field strain of PRRSV (MN-1b) using the same methods.

PSEUDORABIES VIRUS (**PRV**). In Indiana, probable aerosol transmission of PRV was concluded in an epizootic involving 10 swine herds across an area of about 150 km^2.[25] A Guassian plume diffusion model was used to explain the aerosol spread of virus to nine farms.[26] Conclusions were based on wind speed and direction, herd location, and lack of other modes of spread.[25] In Denmark, an epizootic of pseudorabies was found to be correlated with an unusual predominance of southerly winds, above-normal winter temperatures and precipitation, fewer hours of sunshine, and higher wind speed.[27]

SWINE VESICULAR DISEASE VIRUS (**SVDV**). Sellers, et al.,[28] reported that virus was recovered from the air surrounding pigs experimentally

inoculated with SVDV for 2–3 days during clinical disease.

Aerosol transmission of pathogens under field conditions cannot be definitively proven due to the inevitability of confounding variables. Difficulties in demonstrating aerosol transmission of pathogens in laboratory settings probably result from the small number of pigs used in the trials. Large numbers of organisms appear to be necessary for survival of pathogens over long distances in aerosol form, and small sample sizes preclude this requirement.

In summary, literature suggests that *A. pleuropneumoniae,* HCV, PRRSV, and SVDV can be transmitted by aerosol over short distances, while FMDV, *M. hyopneumoniae*, and PRV can be transmitted by aerosol over long distances.

Rodents

The carrier state of various organisms in rodents has been well documented; however, there is a lack of research concerning rodents' abilities to transmit pathogens.

Le Moine, et al.,[29] reported isolation of porcine pathogens in a field study involving 85 mice (*Mus musculus*) and 40 gray rats (*Rattus norvegicus*) in 15 swine herds. *Bordetella bronchiseptica* was isolated from 11 rats, but no mice. *Salmonella* serotype Typhimurium was isolated from mice, and a group C1 *Salmonella* was isolated from one rat. *Escherichia coli* was isolated from both rats and mice. Rotavirus was identified in feces from both rats and mice. Rats and mice were shown to have seroconverted to transmissible gastroenteritis virus (TGEV).

Brachyspira hyodysenteriae was isolated from cecal scrapings of four mice from three farms infected with swine dysentery.[30] Moreover, mice experimentally inoculated with *B. hyodysenteriae* shed the organism in their feces for up to 180 days after inoculation. Pigs exposed to feces from these infected rodents developed clinical swine dysentery after 11–13 days.

The prevalence of antibodies to encephalomyocarditis virus (EMCV) in rats ranged from 8%–86%.[31] Some laboratory rodents fed EMCV seroconverted to the virus but did not shed virus in feces after 24 hours postinfection. Moreover, control rodents housed with EMCV-infected rodents did not become infected with the virus. The authors concluded that rodents were probably dead-end hosts for EMCV and not involved in the transmission of virus.[31]

Leptospira were isolated from 14 of 128 and 13 of 106 rodents, respectively, trapped at two swine farms. Microagglutination titers to serovars *autumnalis, ballum, bratislava, canicola, hardjo*, and *icterohaemorrhagiae* were detected in these rodents.[32]

Porcine reproductive and respiratory syndrome virus was not isolated from sera, lung, thymus, or spleen of 14 mice and two rats trapped on a swine farm with endemic PRRSV infection. Experimental inoculation

of laboratory mice and rats with PRRSV indicated that rodents were not susceptible to infection with PRRSV. [33]

Pseudorabies virus was not isolated, nor were neutralizing antibodies to PRV detected, in 43 Norway rats trapped on a PRV-positive swine farm. In the same study, both wild and laboratory rats were susceptible to experimental infection with PRV.[34]

Salmonella Typhimurium was isolated from sick brown rats on a Maryland farm.[35]

Toxoplasma gondii was isolated from seven of 1502 house mice, two of 67 white-footed mice, and one of 107 rats trapped on 47 swine farms in Illinois.[36]

In summary, *Bordetella bronchiseptica*, *E. coli*, *Leptospira*, rotavirus, *Salmonella* spp*., T. gondii,* and *B. hyodysenteriae* have been isolated from rats and/or mice. Neither PRV nor PRRSV were isolated from rodents on endemically infected farms. Sampling of few rodents over a limited geographical range may have contributed to failure of pathogen isolation. Rodent-to-pig transmission of *B. hyodysenteriae* was demonstrated under laboratory conditions; however, field transmission by this route has not been confirmed.

Flies, mosquitoes, and ticks

Insects can potentially be vectors of swine pathogens among farms. Flies have been shown to travel 1.5 km between farms.[37]

African swine fever virus

Experimentally infected ticks were allowed to feed on 42 uninfected pigs. All 42 pigs subsequently became infected and died from ASFV.[38] Fifty-four argasid ticks (*Ornithodoros savignyi*) were experimentally infected with ASFV.[39] At 106 days after infection, groups of nine ticks were allowed to feed on six uninfected swine. Three of six pigs developed acute African swine fever. Natural infections of *Ornithodoros savignyi* with FMDV have not been documented. Argasid ticks (*Ornithodoros moubata*) naturally infected with ASFV were collected in Africa. An argasid tick (*Ornithodoros coriaceus*), native to the United States, was shown to transmit ASFV to swine under experimental feeding conditions.[40] Under experimental conditions, *Amblyomma americanum* and *Amblyomma cajennense* maintained ASFV for up to 7 days after feeding on infected swine. However, infected ticks did not transmit the virus to healthy swine after being allowed to feed on them.[40]

Eperythrozoon suis

Stableflies (*Stomoxys calcitrans*) and mosquitoes (*Aedes aegypti*) were allowed to feed on pigs infected with *E. suis* and then immediately, or after at least a 1-hour delay, were allowed to feed on susceptible splenectomized pigs.[41] Transmission of *E. suis* was demonstrated by stableflies in three of 15 pigs, and by mosquitoes in nine of nine pigs after immediate transfer. Transmission was not demonstrated after any delay of transfer. Researchers concluded that the stablefly and mosquito are probable mechanical vectors of *E. suis* under field conditions. The authors believe that studies using nonsplenectomized

pigs are needed before this conclusion can be reached.

Hog cholera virus

Horseflies (*Tabanus lineola* and *Tabanus quinquevittatus*) experimentally transmitted hog cholera virus to susceptible pigs after feeding on infected pigs.[42] Dorset, et al.,[43] reported transmission of hog cholera by houseflies and stableflies. Houseflies transmitted the virus by coming into contact with eyes of sick pigs and then with those of healthy pigs. Stableflies transmitted the virus by biting healthy pigs after feeding on sick pigs. The virus was also transmitted by feeding pigs dead stableflies that had fed on sick pigs. In another experiment, eight of 40 pigs developed hog cholera after a suspension of mosquitoes trapped on a farm during an epizootic of hog cholera was intramuscularly injected into susceptible pigs.[44] Hog cholera was also transmitted experimentally in two of eight pigs after *Aedes aegypti* fed on susceptible pigs after feeding on acutely ill pigs.[44]

Pseudorabies virus

Pseudorabies virus was recovered from houseflies (*Musca domestica*) after flies were experimentally fed the virus.[45] In other studies,[46] pigs were experimentally infected with PRV by exposure to flies that had been fed virus. Transmission occurred after fly exposure to eyes, skin, or ingestion of dead flies. Researchers abraded the skin of the pigs before fly exposure, and natural transmission via this route is questionable. Surface disinfection of the fly eliminated the virus; thus, flies were considered mechanical vectors.

Streptococcus suis

Streptococcus suis type 2 was carried by houseflies (*Musca domestica*) for 5 days after the flies were experimentally fed cultures of the bacteria.[47] Carrier flies contaminated materials on which they were feeding for up to 4 days after infection with *S. suis*.[47]

Swine pox virus

Shope[48] reported that swine pox virus was not transmitted between healthy animals and animals infected with swine pox virus if all pigs were free of lice. However, if pigs were louse-infested, swine pox virus was transmitted after 12–18 days. Swine pox virus was isolated from lice up to 15 days after feeding on infected swine. The authors concluded that the hog louse acts as a mechanical vector, not as an intermediate host.

Transmissible gastroenteritis virus

Transmissible gastroenteritis virus was detected in houseflies originating from a swine unit with enzootic TGEV.[49] In a subsequent study, TGEV was recovered 72 hours after laboratory flies were experimentally infected.[49]

In summary, most evidence of insects as carriers or vectors of pathogens is experimental. Transmission of ASFV, *E. suis*, HCV, PRV, *S. suis*, swine pox virus, and TGEV has been documented under laboratory conditions. Natural infection of insects with ASFV and TGE on farms with enzootic disease have been reported.

Birds

Natural transmission of porcine diseases by birds to swine has not been documented.

Bordetella bronchiseptica

Farrington, et al.,[50] reported that *B. bronchiseptica* was isolated from one of 47 house sparrows and 0 of 54 starlings trapped on a research unit housing infected swine.

Hog cholera virus

Hughes and Gustafson[22] performed a trial in which a pen of pigs infected with hog cholera was connected to two pens of sentinel pigs by separate, screened 1.82 m (6 ft)-long flyways. Fifteen English sparrows were allowed to fly back and forth from infected to noninfected pigs through one flyway. The other flyway was devoid of birds and connected sentinel pigs served as controls. Birds were observed eating with the pigs and bird droppings were found in the pig feeders and waterers. After 6 weeks, pigs in contact with birds developed clinical signs consistent with hog cholera; however, hog cholera infection was not definitively diagnosed. Control pigs remained healthy.

Porcine reproductive and respiratory syndrome virus (PRRSV)

Zimmerman, et al.,[51] reported that Mallard ducks, experimentally exposed to PRRSV in their drinking water, shed PRRSV for up to 25 days post-exposure in their feces. Pigs intranasally exposed to PRRSV isolated from Mallard feces became viremic and could transmit the virus to other pigs.

Streptococcus suis

Devriese, et al.,[52] reported isolating *S. suis* from a backyard-kept duck which died suddenly. The source of the infection was unknown.

Swine influenza virus

Pensaert, et al.,[53] suggested that an influenza A strain originating in wild ducks was responsible for an outbreak of influenza in pigs in Belgium. The strains of influenza isolated from the pigs were related to influenza viruses isolated from wild ducks in North America and Germany. Wright, et al.,[54] compared the origins of gene segments from SIV isolates to that of turkey influenza virus isolates. Gene segments from swine isolates were characteristic of swine influenza viruses; however, 73% of the gene segments from turkey isolates contained genes of swine origin. The authors concluded that genetic exchange and reassortment of influenza A viruses occurred frequently in turkeys and rarely in swine.

Transmissible gastroenteritis virus

Pilchard[55] reported that pigs developed clinical signs of transmissible gastroenteritis after being fed feces of starlings up to 32 hours after the starlings were experimentally fed a suspension of TGEV.

Tuberculosis

Bickford, et al.,[56] reported that they isolated *Mycobacterium avium* from starlings trapped on a farm where swine were infected with avian tuberculosis. The authors hypothesized that the starlings were infected at a nearby poultry farm, and then introduced the infection to the swine herd through fecal contamination.

In summary, *B. bronchiseptica* and *Mycobacterium avium* were isolated from birds trapped on premises that had infected swine. There is some evidence that HCV, PRRSV, and TGEV are transmissible from birds to swine under experimental conditions. Definitive proof of SIV transmission from birds to pigs has not been documented.

Domestic and nonporcine feral animals

Although seemingly probable, no definitive proof exists that pathogens can be naturally transmitted from domestic and nonporcine feral animals to swine. As a precaution, however, perimeter fencing should be sufficient to prohibit entry of domestic strays or feral animals to swine facilities. Perimeter fencing is not sufficient safeguard against raccoons.

Brachyspira hyodysenteriae

Songer, et al.,[57] reported the isolation of pathogenic *B. hyodysenteriae* from a fecal sample of a dog observed to have eaten manure from pigs that had swine dysentery. *Brachyspira hyodysenteriae* could not be isolated from fecal samples of the dog after the dog was removed from the premises. Glock, et al. (*Proc IPVS.* 1978; K.B. 63) reported isolation of *B. hyodysenteriae* from 1–13 days after dogs were inoculated intragastrically with *B. hyodysenteriae* in 14 of 16 inoculated dogs. Twenty-one of 22 pigs fed dog feces collected 1–4 days after dogs were inoculated became infected. Pigs that were fed feces from dogs after day 7 of inoculation did not become infected.

Brucella suis

Brucella suis was isolated from hares in Denmark in the same district as a *Brucella* epizootic in swine.[58] The authors hypothesized that if hares were the source of infection, transmission could occur when swine consume kitchen waste that consists of organs from infected hares.

A watchdog was implicated in the spread of brucellosis to a swine herd.[59] A herd was depopulated due to a brucellosis epizootic. The herd was repopulated with brucellosis-free stock, but became reinfected 2 years later. *Brucella suis* was isolated from organs of an asymptomatic watchdog used to guard the original infected herd, and subsequently the newly populated herd. *Brucella suis* was not isolated from the urine of the dog.

Leptospira interrogans

Leptospira interrogans serovar *pomona* was isolated from the kidneys of five of 14 skunks trapped in and around a swine herd during a leptospirosis outbreak.[60] The author hypothesized that skunks may have contributed to the contamination of the water supply.

Pseudorabies virus

Pseudorabies virus was isolated from six raccoons and two cats found dead on or near farms infected with PRV.[61] Kirkpatrick, et al.,[61] reported transmission of PRV from raccoons to swine after raccoons were experimentally inoculated with the virus. Pigs seroconverted to PRV after having contact with inoculated raccoons. Pseudorabies virus was isolated from nasal discharges of pigs 8 days after pigs were fed the viscera of inoculated raccoons. The probability of natural raccoon-to-swine transmission of PRV is unknown.

Streptococcus suis

Salasia and Lammler[62] reported the isolation of *Streptococcus suis* types 1/2, 4, 9, 20, 22, and 26 from dogs and cats. Devriese, et al.,[63] reported isolation of *S. suis* from a sick fallow deer. Neither set of authors reported whether these animals had any contact with pigs. In 1992, Devriese, and Haesebrouck,[64] reported cases of *S. suis* in horses, a zebra, and cats. None of the equines or felines had contact with swine.

The role of feral animals in the transmission of *S. suis* remains unknown, even though many nonporcine species can become infected with *S. suis*.

Toxoplasma gondii

Antibodies to *T. gondii* were detected in 31 of 74 cats, one of 34 opossums, four of 14 raccoons, and two of seven striped skunks that were live-trapped on swine farms.[65] There was no association between the prevalence of *T. gondii* antibodies in sows from these farms and the prevalence of antibodies in nonswine species. The authors hypothesized that oocysts from cat feces may be a source of contamination for swine.

In summary, *Brachyspira hyodysenteriae* and *B. suis* were isolated from dogs in contact with infected pigs. *Brucella suis*, *Leptospira interrogans*, and PRV were isolated from nonporcine feral animals trapped on premises with infected swine. Pseudorabies virus was transmitted to pigs under experimental conditions by feeding pigs viscera from infected raccoons.

Feed

To prevent introducing foreign animal diseases to the United States, federal law states that "No person shall feed or permit the feeding of garbage to swine unless the garbage is treated to kill disease organisms..." (9 CFR Ch. 1, Part 166- Swine Health Protection, Section 166.2, 1–1–98 Edition). Some individual states forbid feeding both treated and untreated garbage.

Harris, et al.,[66] isolated *Salmonella* from samples of feed and feed ingredients in 46.7% of farms studied. Researchers did not examine whether the presence of *Salmonella* in the feed adversely affected the health of the pigs or was a risk factor for establishing a carrier state in the pigs consuming the contaminated diet.

Lee, et al.,[67] sampled feedstuffs, manure, and cecal samples from pigs at slaughter on two farms for the presence of salmonellae. The first farm fed a liquid diet, which included fish meal found to be contaminated with salmonellae. The second farm fed a purchased diet from which salmonellae were not isolated. No serotype of *Salmonella* was isolated repeatedly from a single source, but on occasion the same serotype of *Salmonella* was isolated from multiple sources. Serotypes did not persist in pigs over time. The incidence of salmonellae in cecal samples collected at slaughter was significantly lower (*P*<.05) for the farm whose feedstuffs were not found to be contaminated with salmonellae when compared to the farm whose feedstuffs were contaminated. However, the incidence and serotypes of salmonellae in pigs at arrival (prior to feed consumption) was not determined. Therefore, an association between contaminated feed and a carrier state in pigs could not be made.

Smith[68] fed two groups of *Salmonella*-free pigs a diet heavily contaminated with *Salmonella* or a diet free of *Salmonella* for a period of up to 50 days, after which both groups were fed a *Salmonella*-free diet. Pigs were euthanized and samples were cultured periodically throughout the trial. Pigs did not become clinically ill during the trial. *Salmonella* was isolated from the mesenteric lymph nodes or rectum of eight of 20 pigs fed the contaminated diet. Seven of 20 pigs fed the contaminated diet shed *Salmonella* in their feces during consumption, but shedding ceased when these pigs were switched to a *Salmonella*-free diet. *Salmonella* was not detected in tissues or manure from the four pigs fed a noncontaminated diet. Incidence of isolation of *Salmonella* from tissues or manure was not statistically different between the two groups, probably due to the inadequate sample size and experimental design. Thus, definitive evidence of feed as a source of *Salmonella* infection for pigs still does not exist.

Toxoplasma gondii oocysts were isolated from two of 491 feed samples from 47 swine farms in Illinois.[36] The authors postulated that these results underestimated the true prevalence of oocysts, estimating that >90% of the detectable oocysts were lost due to sample storage and assay procedures.

In summary, two studies have detected pathogens in the feed of swine. The number of organisms detected in the feed are probably too small to cause infection in pigs consuming the feed, but the risk of infection is unknown. To date, *T. gondii* oocysts and *Salmonella* were the only organisms reportedly isolated from pig feed. Research has not proven that porcine pathogens can be transmitted through contaminated feed.

Vehicles

The risk of pathogen transmission by contaminated vehicles has not been well researched. Common belief is that organisms can be carried on the frame of the vehicle or in caked manure in tire treads.

Transmission of *A. pleuropneumoniae* among nine pig herds was investigated using ribotyping techniques.[69] The finding of identical ribotypes in the infected herd and the suspect herd of origin was evidence for implicating the mode of transmission. Although the authors implicated transmission by vehicles in six of the nine cases, the ribotypes matches could have occurred by chance alone.

Dee and Corey[70] added *S. suis* to swine manure and spread the mix-

ture on a truck tire. *Streptococcus suis* was isolated from the tire tread after the truck was driven for 4.82 km (3 miles) at speeds up to 64.3 km (40 miles) per hour, but not after an additional 12.87 km (8 miles) with speeds ranging from 96.5–120.6 km (60–75 miles) per hour.

In summary, there is no proof that pathogens can be transmitted by contaminated vehicles, but there is some evidence that *A. pleuropneumoniae* and *S. suis* could be transmitted by this route.

Personnel and visitors

People flow into and within production units comprises a large component of biosecurity; however little research is available to support common policies regarding people movement. The length of downtime between human visits to farms is a controversial subject. Most farms have a rule that visitors must be free from exposure to swine for 24–48 hours before entry. Disease research centers such as Plum Island have downtimes ranging from 48–168 hours. The refereed literature includes only two publications describing human transmission of porcine pathogens. First, Goodwin[23] reported that the culture of breath and hair samples from a person exposed to pigs experimentally infected with *M. hyopneumoniae* did not result in reisolation of *M. hyopneumoniae*. Second, Sellers, et al.,[71] sampled people who had been in with contact animals infected with FMDV. More FMDV was isolated from the nose than the mouth of these people. Virus was isolated from the nose of one person at 28 hours, but was not isolated after 48 hours. Nose blowing or washing was not effective in eliminating the virus, and cloth or industrial masks were not effective in preventing inhalation of the virus. Transfer of the virus between people was documented after persons in contact with infected animals spoke to unexposed colleagues in a box for 4 minutes. One year later, Sellers, et al.,[72] reported that FMDV could be transferred by human beings, from infected pigs, to susceptible cattle. Results from Seller's work appear to be the origin for the "48 hour rule" used by many producers even though different viruses and bacteria may be harbored for longer or shorter periods by humans.

Wentworth, et al.,[73] recorded transmission of SIV to human caretakers. In this study, pig-to-human transmission occurred despite the use of Animal Biosafety Level 3 containment practice (coveralls, boots, goggles, gloves, hairnets, and dust masks.). In contrast, the authors[74] could not detect pig-to-human transmission of *S. suis* using throat swab samples collected from farm personnel who were working in close daily contact with infected pigs. Thus, it would appear that the risk of transmitting diseases back and forth between human beings and swine varies with the pathogen. Quantification of the risk of transmission of common porcine pathogens, on an individual basis, is essential.

Whether or not to shower before entry into a production unit is another controversial subject. A shower-in policy ensures that contaminated clothes will not be carried onto the farm and discourages visitors. No reports of the effect of showering on the carriage of bacteria and viruses were found; however, the results of publications on handwashing may aid in decisions concerning showering. Chamber-

lain, et al.,[75] studied the effectiveness of washing hands with nonmedicated soap and water to reduce natural hand bacterial flora and artificially inoculated bacteria. Both a 10-second and a 3-minute wash reduced the numbers of artificially inoculated bacteria tenfold; however, less than half of the naturally occurring bacteria were removed. Washing *increased* bacterial counts on hands that were previously disinfected with 70% alcohol. Deshmukh, et al.,[76] reported that the number of bacterial colonies recovered from washed hands after a 1-minute wash with povidone-iodine followed by use of alcohol foam was less than that after a 5-minute wash with povidone-iodine only. Patrick, et al.,[77] reported the importance of hand drying in reducing the transfer of bacteria by touch after washing hands. Drying hands with a cloth for 10 seconds or using a dryer for 20 seconds reduced the number of bacteria that were transferred to skin or equipment after touch contact by 94%–99.8%. Research concerning the role of personal hygiene in the transmission of porcine pathogens is needed.

Footbaths are often used in transition areas between groups of pigs to prevent disease transmission. No reports regarding effective use of footbaths have been published.

In summary, FMDV and SIV were the only porcine pathogens shown to be transmissible from infected pigs to people. People could spread FMDV to susceptible cattle but spread to pigs was not documented. There were no studies examining the effectiveness of personal hygiene procedures in preventing the transmission, by people, of porcine pathogens.

Pathogen survival

Clinically healthy and ill swine shed bacteria and viruses in secretions and excretions. Organisms from pigs ultimately contaminate the production facility. There are few reports regarding the potential to spread diseases through contact with contaminated premises, manure, water, soil, etc.

Dust, uncleaned rooms

Porcine parvovirus

Mengeling and Paul[78] reported the infection of sentinel pigs with porcine parvovirus after the pigs were placed in an uncleaned room that had previously housed experimentally infected pigs. The room had been vacant of pigs for 14 weeks before sentinel pigs were introduced.

Rotavirus

Fu, et al.,[79] reported the isolation of group A rotavirus from dust from a nursery that had been free of pigs for 3 months.

Flooring

Streptococcus suis

Dee and Corey[70] reported that *S. suis* survived up to 20 hours on clean plastic flooring, less than 4 hours on clean concrete, and less than 2 hours on painted plywood.

Soil

Erysipelothrix rhusiopathiae

Wood[80] reported that *E. rhusiopathiae* experimentally added to soil died out at a logarithmic rate. The longest survival time occurred at 3°C (37.4°F) when the organism was isolated 35 days after the soil was experimentally inoculated with cultures *of E. rhusiopathiae*.

Escherichia coli

Tamasi[81] reported that *E. coli* survived under laboratory conditions in soil columns for 108 days at 8°C (46.4°F) and for 54 days at 20°C (68°F).

Salmonella Typhimurium

Tamasi[81] reported that *Salmonella* Typhimurium survived under laboratory conditions in soil columns for 96 days at 8°C (46.4°F) and for 54 days at 20°C (68°F).

Trichuris suis

Burden and Hammet[82] reported that *T. suis* ova were available to grazing pigs up to 2.5 years after pasture plots were contaminated with pig manure containing *T. suis* ova.

Manure

Ascaris suum

Gaasenbeek and Borgsteede[83] reported that *A. suum* eggs did not survive beyond 16 weeks in experimentally inoculated tubes of pig slurry that were stored submerged in a pig slurry unit. Under simulated field conditions, *A. suum* eggs survived in pig slurry for up to 4 weeks in dry and sunny conditions and at least 8 weeks under moist and shady conditions.

Brachyspira hyodysenteriae

Olson[84] reported that lagoon effluent from a building remained infective for swine dysentery 5–6 days after removal of infected swine from the building. Chia[85] reported that *B. hyodysenteriae* survived up to 48 days in pig manure stored between 0°C–10°C (32°F–50°F).

Pseudorabies virus

Botner[86] reported that, under experimental conditions, PRV survived for 15 weeks at 5°C (41°F) in inoculated pig slurry under anaerobic storage. Survival times decreased as storage temperatures increased.

Water

Mycoplasma hyopneumoniae

The recovery of *M. hyopneumoniae* inoculum from rain and tap water at 17 and 31 days, respectively, may help explain aerosol transmission of the organism during periods of high relative humidity.[23]

Composted carcasses

Morrow, et al.,[87] reported that the process of composting swine carcasses was sufficient to kill *E. rhusiopathiae*, PRV, and some *Salmonella* cultures under experimental conditions. *Salmonella* cultures placed at the top and bottom of the pile survived. *Salmonella* may have been killed had the cultures not been at fixed locations in the pile.

Summary

Porcine parvovirus and rotavirus survived for extended periods of time in facilities vacated of pigs. *Streptococcus suis* survived for short periods of time on flooring. Under laboratory conditions, *E. rhusiopathiae*, *E. coli*, and *Salmonella* Typhimurium survived in soil for extended periods. *Trichuris suis* ova survived for years on naturally grazed pastures. *Ascaris suum*, *B. hyodysenteriae*, and PRV survived for variable periods of time in manure. *Mycoplasma hyopneumoniae* was recovered from experimentally inoculated water for up to 31 days. Carcass composting appeared to be sufficient to kill *E. rhusiopathiae* and PRV.

Cleaning and disinfection

Cleaning, disinfection, and drying of buildings and equipment is of paramount importance to disease control. Cleaning removes organic matter that can prevent many disinfectants from functioning as designed. Disinfecting reduces or eliminates biocontamination of the unit, decreasing the load of bacteria and viruses that build up over time. Drying is important because desiccation kills many organisms. The efficacy of a cleaning and disinfection program can be determined by cultural examination of swab samples collected from the floor or equipment after the area is properly disinfected and allowed to dry.[88]

Most studies of the efficacy of disinfection on the survival of specific porcine pathogens were performed under conditions of optimal pathogen survival (i.e., cell culture or bacterial culture) free of contamination with manure; thus, laboratory results may not reflect field efficacy.

African swine fever virus

Stone and Hess[89] examined the virucidal activity of 11 disinfectants (sodium hydroxide and acetic acid, sodium meta silicate [Fisher Scientific Co., Fairlawn, New Jersey], Roccal™, Weladol™ [Allied Laboratories, Inc., Indianapolis, Indiana], Triton X-100™ [Packard Instrument Co., Downers Grove, Illinois], Amphyl™, pHisoHex™ [Winthrop Laboratories, New York, New York], sodium dodecyl sulfate [Fisher Scientific Co.], LpH™, Environ™ [Vestul Laboratories, St. Louis, Missouri], Environ D™ [Vestul Laboratories], and One-Stroke Environ™ [Vestul Laboratories]) against ASFV. Only One-Stroke Environ was virucidal at concentrations of 0.5%–1%. A minimal contact time of 1 hour with 1% One-Stroke Environ was reported to be effective in decontaminating a room.

Brachyspira hyodysenteriae

Chia and Taylor[85] did not recover *B. hyodysenteriae* after contact with 1200 ppm formaldehyde, 375 ppm phenol, 375 ppm sodium hypochlorite, or 30,000 ppm sodium carbonate.

Porcine parvovirus

Brown[90] reported that a 1:16 dilution of sodium hypochlorite and 5% sodium hydroxide inactivated porcine parvovirus.

Pseudorabies virus

Pseudorabies virus was inactivated after 5 minutes of contact with 70% ethanol, 1:212 dilution of betadine, 1:256 dilution of phenol, 1:200 dilution of quaternary ammonium compounds, 4% formaldehyde, 1:128 dilution of Nolvasan™, 5% sodium hydroxide, and a 1:32 dilution of sodium hypochlorite.[90]

Streptococcus suis

Dee and Corey[70] inoculated plates of Meuller-Hinton agar with *S. suis.* Phenol, quaternary ammonium, formaldehyde, chlorhexadine, 3% iodine, 70% alcohol, and 5% hypochlorite diluted according to recommended levels were swabbed on the agar surface. *Streptococcus suis* did not grow in the presence of any disinfectant except for 70% alcohol.

Swine vesicular disease virus

Blackwell, et al.,[91] reported that 2% sodium hydroxide, 0.04% sodium hypochlorite, and 8% formaldehyde inactivated SVDV in less than 2 minutes under experimental conditions.

Transmissible gastroenteritis virus

Evans, et al.,[92] reported that TGEV was sensitive to 1% Lysol, 2% glutaraldehyde (Cidex™), 1% sodium hypochlorite (Chloros™), 4% solution of an iodophor (Fam™), 36% w/v formaldehyde, and 0.1% peracetic acid. Brown[90] also reported that TGEV was inactivated after 5 minutes of contact with undiluted Cidex™, a 1:32 dilution of sodium hypochlorite, a 1:212 dilution of betadine, and 4% formaldehyde. In addition, Brown reported that 70% ethanol, 1:212 dilution of betadine, 1:256 dilution of phenol, 1:200 dilution of quaternary ammonium compounds, 1:128 dilution of Nolvasan, 5% sodium hydroxide, and a 1:32 dilution of sodium hypochlorite inactivated TGEV after 5 minutes of contact.[90]

Vesicular exanthema virus

Blackwell[93] reported VEV was inactivated after 2 minutes of exposure to 1% formaldehyde, 10% Amphyl™, 1% One-Stroke Environ™, 0.02% Wescodyne™, or 5% benzalkonium chloride.

In summary, African swine fever virus, *B. hyodysenteriae*, porcine parvovirus, PRV, *S. suis* , SVDV, TGEV, and VEV were the only porcine pathogens for which disinfectant efficacy studies were reported.

Guidelines for disinfectant use were included in this review as a reference guide for practitioners (Table 1). In addition to the tabled disinfectants, alcohols are active against bacteria, viruses, and fungi.[94] Chlorhexidine, a biguanide, is bactericidal and has variable antiviral activity. The activity of chlorhexidine is pH dependent and reduced in the presence of organic matter.[94] Iodines and iodophors are bactericidal, fungicidal, virucidal, sporocidal, and tuberculocidal.[94]

Generally, lipid-enveloped viruses and gram-positive bacteria are the most sensitive to disinfectants.[94] Fungi and nonsporulating gram-negative bacteria are slightly more resistant.[94] Nonenveloped viruses, mycobacteria, bacterial spores, and coccidia are the most resistant to disinfectants.[94]

Discussion

Biosecurity has become an important consideration for maintaining the health of swine herds. Unfortunately, the field of biosecurity has not been well researched. Virtually all aspects of biosecurity need to be examined.

Producers can evaluate the effectiveness of current biosecurity programs by recording information regarding movements of people, animals, feed, and equipment to, from, and within their production facilities. Records can proactively alert managers to biosecurity risks or breaches. Records can also help identify the likely source of disease introduction should an outbreak occur. Visitor logs should include:

- names,
- phone numbers,
- reason for visit,
- time since last contact with swine, and
- facilities entered.

Pig movement logs should include:

- the date,
- number of pigs,
- origin,
- destination,
- reason, and
- vehicle used.

Vehicle and semen movement logs should include:

- dates,
- origins,
- destinations, and
- reasons for movement, if applicable.

Manure application logs should include:

- dates,
- origin,
- application site,
- volume, and
- application method.

Research is needed for veterinarians and producers to make informed, cost-effective, scientifically sound decisions regarding biosecurity. The authors propose that research be initiated under controlled laboratory conditions to determine whether pathogen spread by a certain mechanism is possible. Then, field investigations can be performed to determine whether the risk is probable. Biosecurity measures commensurate with the greatest degree of risk can then be prescribed with confidence to producers.

Not knowing the extent to which biosecurity measures need to be employed to prevent the transmission of porcine pathogens is an important problem, because, until that information is known, pork producers will run one of two risks:

- expenditure of funds on unnecessary biosecurity measures, or

Table 1

Properties of chemical disinfectants

Disinfectant class	Spectrum of activity	Inactivating agents	Safety concerns
Quaternary ammonium compounds	Gram-positive bacteria[95] Lipophilic viruses[95] Sporostatic[94]	Hardwater[95] Organic matter[95] Soaps[95] Anionic detergents[95] pH[95]	
Phenols	Bacteria[95] Viruses[95] Fungi[95] Mycobacteria[95]		Highly toxic and corrosive[95, 96] Skin irritation and depigmentation[96]
Halogen releasing compounds (Chlorine)	Bacteria[95] Viruses[95] Fungi[95] Protozoa[95]	Organic matter[95]	Corrosive to skin and metals at high concentrations[96] Irritating to skin and eyes[96]
Aldehydes (Formaldehyde)	Bacteria[96] Fungi[96] Mycobacteria[96] Spores[96] Viruses[95]		Irritating fumes[96] Potential carcinogen[96] Allergen[96]

- insufficient biosecurity measures that place the United States pig population at risk for economically devastating disease outbreaks.

Implications

- Biosecurity considerations are at the forefront of industry issues.
- There is a lack of scientific evidence to support many biosecurity measures currently implemented by the industry.
- The pork industry must investigate biosecurity scientifically to develop effective measures that both meet the needs of the industry and alleviate public concerns regarding zoonoses.

Acknowledgements

References

1. Meyer RC, Bohl EH, Kohler EM. Procurement and maintenance of germ-free swine for microbiological investigations. *Appl Micro*. 1964;12(4):295–300.

2. Young GA, Underdahl NR, Hinz RW. Procurement of baby pigs by hysterectomy. *Am J Vet Res*. 1955; 123–131.

3. Young GA, Underdahl NR, Sumption LJ, Peo ER, Olsen LS, Kelley GW, Hudman DB, Caldwell JD, Adams CH. Swine repopulation. I. Performance within a "disease-free" experiment station herd. *JAVMA*. 1959;134(11):491–496.

4. Clark LK, Hill MA, Kniffen TS, Van Alstine WG, Stevenson GW, Meyer KB, Wu CC, Scheidt AB, Knox KE, Albregts S. An evaluation of the components of medicated early weaning. *Swine Health Prod*. 1994;2(3):5–11.

5. Dritz SS, Chengappa MM, Nelssen JL, Tokach MD, Goodband RD, Nietfeld JC, Staats JJ. Growth and microbial flora of nonmedicated, segregated, early weaned pigs from a commercial swine operation. *JAVMA*. 1996;208(5):711–715.

6. Lucas MH, Cartwright SF, and Wrathall AE. Genital infection of pigs with porcine parvovirus. *J of Comparative Pathology*. 1974;84:347–350.

7. Swenson SL, Hill HT, Zimmerman JJ, Evans LE, Landgraf JG, Wills RW, Sanderson TP, McGinley MJ, Brevik AK, Ciszewski DK, Frey ML. Excretion of porcine reproductive and respiratory syndrome virus in semen after experimentally induced infection in boars. *JAVMA*. 1994;204(12):1943–1948.

8. Christopher-Hennings J, Nelson EA, Hines RJ, Nelson JK, Swenson SL, Zimmerman JJ, Chase CCL, Yaeger MJ, Benfield D. Persistence of porcine reproductive and respiratory syndrome virus in serum and semen of adult boars. *J of Vet Diagn Invest*. 1995;7:456–464.

9. Yaeger MJ, Prieve T, Collins J, Christopher-Hennings J, Nelson EA, Benfield D. Evidence for the transmission of porcine reproductive and respiratory syndrome (PRRS) virus in boar semen. *Swine Health Prod*. 1993;1(5):7–9.

10. Swenson SL, Hill HT, Zimmerman JJ, Evans LE, Wills RW, Yoon KJ, Schwartz KJ, Althouse GC, McGinley MJ, Brevik AK. Artificial insemination of gilts with porcine reproductive and respiratory syndrome (PRRS) virus-contaminated semen. *Swine Health Prod*. 1994;2(6):19–23.

11. Johnson RH, Collings DF. Transplacental infection of piglets with a parvovirus. *Res Vet Science*. 1971;12:570–572.

12. Hutchings LM, Andrews FN. Studies on brucellosis in swine.III.Brucella infection in the boar. *Am J Vet Res*. 1946;7(25):379–384.

13. Lord VR, Cherwonogrodzky JW, Marcano MJ, Melendez G. Serological and bacteriological study of swine brucellosis. *J Clin Microbiol*. 1997;35(1):295–297.

14. Medveczky I, Szabo I. Isolation of Aujesky's disease virus from boar semen. *Acta Veterinaria Academiae Scientarium Hungaricae, Tomus*. 1981;29(1):29–35.

15. Donaldson AI, Ferris NP. The survival of some air-borne viruses in relation to relative humidity. *Vet Microbiol*. 1976;1:413–420.

16. Stehmann R, Mehlhorn G, Neuparth V. Characterization of strains of *Bordetella bronchiseptica* isolated from animal housing air and the airborne infection pressure proceeding from them. *Deutsche Tierarztliche Wochenschrift*. 1991;98(12):448–450.

17. Schoenbaum MA, Zimmerman JJ, Beran GW, Murphy DP. Survival of pseudorabies virus in aerosol. *Am J Vet Res*. 1990;51(3):331–333.

18. Mitchell CA, Guerin LF. Influenza A of human, swine, equine, and avian origin: Comparison of survival in aerosol form. *Can J Comp Med*. 1972;36:9–11.

19. Torremorell M, Pijoan C, Janni K, Walker R, Joo HS. Airborne transmission of *Actinobacillus pleuropneumoniae* and porcine reproductive and respiratory syndrome virus in nursery pigs. *Am J Vet Res*. 1997;58(8):828–832.

20. Gloster J, Sellers RF, Donaldson AI. Long distance transport of foot-and-mouth disease virus over sea. *Vet Rec*. 1982;110:47–52.

21. Sellers RF, Parker J. Airborne excretion of foot-and-mouth disease virus. *Journal of Hygiene, Cambridge*. 1969;67:671–677.

22. Hughes RW, Gustafson DP. Some factors that may influence hog cholera virus transmission. *Am J Vet Res*. 1960; 464–471.

23. Goodwin RFW. Apparent reinfection of enzootic-pneumonia-free pig herds: Search for possible causes. *Vet Rec*. 1985;116:690–694.

24. Wills RW, Zimmerman JJ, Swenson SL, Yoon KJ, Hill HT, Bundy DS, McGinley MJ. Transmission of PRRSV by direct, close, or indirect contact. *Swine Health Prod*. 1997;5(6):213–218.

25. Scheidt AB, Rueff LR, Grant RH, Teclaw RFHMA, Meyer KB, Clark LK. Epizootic of pseudorabies among 10 swine herds. *JAVMA*. 1991;199(6):725–730.

26. Grant RH, Scheidt AB, Rueff LR. Aerosol transmission of a viable virus affecting swine: Explanation of an epizootic of pseudorabies. *Intl J Biometeorology*. 1994;38:33–39.

27. Christensen LS, Mousing J, Mortensen S, Soerensen KJ, Strandbygaard BS, Henriksen CA, Andersen JB. Evidence of long distance airborne transmission of Aujesky's disease (pseudorabies) virus. *Vet Rec*. 1990;127:471–474.

28. Sellers RF, Herniman KAJ. The airborne excretion by pigs of swine vesicular disease virus. *Journal of Hygiene, Cambridge*. 1974;72:61–65.

29. Le Moine V, Vannier P, Jestin A. Microbiological studies of wild rodents in farms as carriers of pig infectious agents. *Prev Vet Med*. 1987;4:399–408.

30. Joens LA, Kinyon JM. Isolation of *Treponema hyodysenteriae* from wild rodents. *J Clin Microbiol*. 1982;15(6):994–997.

31. Tesh RB, Wallace GD. Observations on the natural history of encephalomyocarditis virus. *Am J of Tropical Medicine and Hygiene*. 1978;27(1):133–143.

32. Songer JG, Chilelli CJ, Reed RE, Trautman RJ. Leptospirosis in rodents from an arid environment. *Am J Vet Res*. 1983;44(10):1973–1976.

33. Hooper CC, Van Alstine WG, Stevenson GW, Kanitz CL. Mice and rats (laboratory and feral) are not a reservoir of PRRS virus. *J Vet Diagn Invest*. 1994;6:13–15.

34. Maes RK, Kanitz CL, Gustafson DP. Pseudorabies virus infections in wild and laboratory rats. *Am J Vet Res*. 1979;40(3):393–396.

35. Davis DE. The survival of wild brown rats on a Maryland farm. *Ecology*. 1948;29:437–447.

36. Dubey JP, Weigel RM, Sigel AM, Thulliez P, Kitron UD, Mitchell MA, Mannelli A, Mateus-Pinilla NE, Shen SK, Kwok OCH, Todd KS. Sources and reservoirs of *Toxoplasma gondii* infection on 47 swine farms in Illinois. *J Parasitol*. 1995;81(5):723–729.

37. Denholm I, Sawicki RM, Farnham AW. Factors affecting resistance to insecticides in house-flies, *Musca domestica* L.(Diptera:Muscidae).IV. The population biology of flies on animal farms in south-eastern England and its implications for the management of resistance. *Bulletin of Entomological Research*. 1985;75:143–158.

38. Plowright W, Parker J, Pierce MA. The epizootiology of African swine fever in Africa. *Vet Rec*. 1969;85:668–674.

39. Mellor PS, Wilkinson PJ. Experimental transmission of African swine fever virus by *Ornithodoros savignyi* (Audouin). *Research in Veterinary Science*.1985;39, 353–356.

40. Groocock CM, Hess WR, Gladney WJ. Experimental transmission of African swine fever virus by *Ornithodoros coriaceus*, an argasid tick indigenous to the United States. *Am J Vet Res*. 1980;41(4):591–594.

41. Prullage JB, Williams RE, Gaafar SM. On the transmissibility of *Eperythrozoon suis* by *Stomoxys calcitrans* and *Aedes aegypti*. *Vet Parasit*. 1993;50:125–135.

42. Tidwell MA, Dean WD, Combs GP, Anderson DW, Cowart WO, Axtell RC. Transmission of hog cholera virus by horseflies (Tabanidae:Diptera). *Am J Vet Res*. 1972;33(3):615–622.

43. Dorset M, McBryde CN, Nile WB, Rietz IH. Observations concerning the dissemination of hog cholera by insects. *Am J Vet Med*. 1919; 55–60.

44. Stewart WC, Carbrey EA, Jenney EW, Kresse JI, Snyder ML, Wessman SJ. Transmission of hog cholera virus by mosquitoes. *Am J Vet Res*. 1975;36(5):611–614.

45. Zimmerman JJ, Berry WJ, Beran GW, Murphy DP. Influence of temperature and age on the recovery of pseudorabies virus from houseflies (*Musca domestica*). *Am J Vet Res*. 1989;50(9):1471–1474.

46. Medvecky I, Kovacs L, Kovacs F, Papp L. The role of the housefly, *Musca domestica*, in the spread of Aujesky's disease (pseudorabies). *Medical and Veterinary Entomology*. 1988;2:81–86.

47. Enright MR, Alexander TJL, Clifton-Hadley FA. Role of houseflies (*Muscu domestica*) in the epidemiology of *Streptococcus suis* type 2. *Vet Rec*. 1987;121:132–133.

48. Shope RE. Swine pox. *Archiv fur die gesamte Virusforschung*. 1940;1:457–467.

49. Gough PM, Jorgenson RD. Identification of porcine transmissible gastroenteritis virus in house flies (*Musca domestica* Linneaus). *Am J Vet Res*. 1983;44(11):2078–2082.

50. Farrington DO, Jorgenson RD. Prevalence of *Bordetella bronchiseptica* in certain wild mammals and birds in central Iowa. *J Wildlife Diseases*. 1976;12:523–525.

51. Zimmerman JJ, Yoon KJ, Pirtle EC, Wills RW, Sanderson TJ, McGinley MJ. Studies of porcine reproductive and respiratory syndrome (PRRS) virus infection in avian species. *Vet Microbiol*. 1997;55:329–336.

52. Devriese LA, Haesebrouck F, De Herdt P, Dom P, Ducatelle R, Desmidt M, Messier S, Higgins R. *Streptococcus suis* infections in birds. *Avian Pathology*. 1994;23:721–724.

53. Pensaert M, Ottis K, Vandeputte J, Kaplan MM, Bachmann PA. Evidence for the natural transmission of influenza A virus from wild ducks to swine and its potential importance for man. *Bulletin of the World Health Organization*. 1981;59(1):75–78.

54. Wright SM, Kawaoka Y, Sharp GB, Senne DA, Webster RG. Interspecies transmission and reassortment of influenza A viruses in pigs and turkeys in the United States. *Am J Epidemiol*. 1992;136(4):488–497.

55. Pilchard EI. Experimental transmission of transmissible gastroenteritis virus by starlings. *Am J Vet Res*. 1965;26(114):1177–1179.

56. Bickford AA, Ellis GH, Moses HE. Epizootiology of tuberculosis in swine. *JAVMA*. 1966;149(3):312–318.

57. Songer JG, Glock RD, Schwartz KJ, Harris DL. Isolation of *Treponema hyodysenteriae* from sources other than swine. *JAVMA*. 1978;172(4):464–466.

58. Bendtsen H, Christiansen M, Thomsen A. Brucella enzootics in swine herds in Denmark- presumably with hare as a source of infection. *Nord Vet Med*. 1954;6:11–21.

59. Kormendy B, Nagy G. The supposed involvement of dogs carrying *Brucella suis* in the spread of swine brucellosis. *Acta Veterinaria Academiae Scientarium Hungaricae*. 1982;30(1–3):3–7.

60. Kingscote BF. Leptospirosis outbreak in a piggery in southern Alberta. *Can Vet J*. 1986;27:188–190.

61. Kirkpatrick CM, Kanitz CL, McCrocklin SM. Possible role of wild mammals in transmission of pseudorabies to swine. *J Wildlife Dis*. 1980;16(4):601–614.

62. Salasia SIO, Lammler C. Serotypes and putative virulence markers for *Streptococcus suis* isolates from cats and dogs. *Res Vet Sci*. 1994;57:259–261.

63. Devriese LA, Desmidt M, Roels S, Hoorens J, Haesebrouck F. *Streptococcus suis* infection in a fallow deer. *The Vet Rec*. 1993;132(11):283–283.

64. Devriese LA, Haesebrouck F. *Streptococcus suis* infections in horses and cats. *Vet Rec*. 1992;130:380–380.

65. Smith KE, Zimmerman JJ, Patton S, Beran GW, Hill HT. The epidemiology of toxoplasmosis on Iowa swine farms with an emphasis on the roles of free-living mammals. *Vet Parasitol*. 1992;42:199–211.

66. Harris IT, Fedorka-Cray PJ, Gray JT, Thomas LA, Ferris K. Prevalence of *Salmonella* organisms in swine feed. *JAVMA*. 1997;210(3):382–384.

67. Lee JA, Ghosh AC, Mann PG, Tee GH. Salmonellas on pig farms and in abattoirs. *J Hyg, Cambridge*. 1972;70:141–150.

68. Smith HW. The effect of feeding pigs on food naturally contaminated with salmonellae. *J Hyg, Cambridge*. 1960;58:381–389.

69. Fussing V, Barfod K, Nielsen R, Moller K, Nielsen JP, Wegener HC, Bisgaard M. Evaluation and application of ribotyping for epidemiological studies of *Actinobacillus pleuropneumoniae* in Denmark. *Vet Microbiol*. 1998;62:145–162.

70. Dee SA, Corey MM. The survival of *Streptococcus suis* on farm and veterinary equipment. *Swine Health Prod*. 1993;1(1):17–20.

71. Sellers RF, Donaldson AI, Herniman KAJ. Inhalation, persistence and dispersal of foot-and-mouth disease virus by man. *Journal of Hygiene, Cambridge*. 1970;68:565–573.

72. Sellers RF, Herniman KAJ, Mann JA. Transfer of foot-and-mouth disease virus in the nose of man from infected to non-infected animals. *Vet Rec*. 1971;89(16):447–449.

73. Wentworth DE, McGregor MW, Macklin MD, Neumann V, Hinshaw VS. Transmission of swine influenza virus to humans after exposure to experimentally infected pigs. *The Journal of Infectious Diseases*. 1997;175:7–15.

74. Amass SF, Kreisle RA, Clark LK, Wu CC. A pilot study of the prevalence of *Streptococcus suis* in pigs and personnel at five Indiana swine operations. *J Agromedicine*. 1998;5(1):17–24.

75. Chamberlain AN, Halablab MA, Gould DJ, Miles RJ. Distribution of bacteria on hands and the effectiveness of brief and thorough decontamination procedures using non-medicated soap. *Zbl Bakt*. 1997;285:565–575.

76. Deshmukh N, Kramer JW. A comparison of 5-minute povidone-iodine scrub and 1-minute povidone-iodine scrub followed by alcohol foam. *Military Medicine*. 1998;163(3):145–147.

77. Patrick DR, Findon G, Miller TE. Residual moisture determines the level of touch-contact-associated bacterial transfer following hand washing. *Epidemiology of Infection*. 1997;119:319–325.

78. Mengeling WL, Paul PS. Interepizootic survival of porcine parvovirus. *JAVMA*. 1986;11(1):1293–1295.

79. Fu ZF, Hampson DJ, Blackmore DK. Detection and survival of group A rotavirus in a piggery. *Vet Rec*. 1989;125:576–578.

80. Wood RL. Survival of *Erysipelothrix rhusiopathiae* in soil under various environmental conditions. *Cornell Veterinarian*. 1973;63:390–410.

81. Tamasi G. Factors influencing the survival of pathogenic bacteria in soils. *Acta Veterinaria Academiae Scientarium Hungaricae, Tomus*. 1981;29(2):119–126.

82. Burden DJ, Hammet NC. The development and survival of *Trichuris suis* ova on pasture plots in the south of England. *Res Vet Sci*. 1979;26:66–70.

83. Gaasenbeek CPH, Borgsteede FHM. Studies on the survival of *Ascaris suum* eggs under laboratory and simulated field conditions. *Vet Parasitol*. 1998;75:227–234.

84. Olson LD. Survival of *Serpulina hyodysenteriae* in an effluent lagoon. *JAVMA*. 1995;207(11):1470–1472.

85. Chia SP, Taylor DJ. Factors affecting the survival of *Treponema hyodysenteriae* in dysenteric pig faeces. *Vet Rec*. 1978;103:68–70.

86. Botner A. Survival of Aujesky's disease virus in slurry at various temperatures. *Vet Microbiol*. 1991;29:225–235.

87. Morrow WEM, O'Quinn PO, Barker J, Erickson G, Post K, McCaw M. Composting as a suitable technique for managing swine mortalities. *Swine Health Prod*. 1995;3(6):236–243.

88. Tamasi G. Testing disinfectants for efficacy. *Rev sci tech Off int Epiz*. 1995;14(1):75–79.

89. Stone SS, Hess WR. Effects of some disinfectants on African swine fever virus. *Appl Microbiol*. 1973;25(1):115–122.

90. Brown TT. Laboratory evaluation of selected disinfectants as virucidal agents against porcine parvovirus, pseudorabies virus, and transmissible gastroenteritis. *Am J Vet Res*. 1981;42(6):1033–1036.

91. Blackwell JH, Graves JH, McKercher PD. Chemical inactivation of swine vesicular disease virus. *Brit Vet J*. 1975;131:317–323.

92. Evans DH, Stuart P, Roberts DH. Disinfection of animal viruses. *Brit Vet J*. 1977;133:356–359.

93. Blackwell JH. Comparative resistance of San Miguel sea lion virus and vesicular exanthema of swine virus to chemical disinfectants. *Res Vet Sci*.1978;25:25–28.

94. McDonnell G, Russell AD. Antiseptics and disinfectants: Activity, action, and resistance. *Clin Microbiol Rev*. 1999;12(1):147–179.

95. Jeffrey DJ. Chemicals used as disinfectants: Active ingredients and enhancing additives. *Rev Sci Tech Off Int Epiz*. 1995;14(1):57–74.

96. Bruins G, Dyer JA. Environmental considerations of disinfectants used in agriculture. *Rev Sci Tech Off Int Epiz*. 1995;14(1):81–94.