

Postweaning mortality in commercial swine production II: review of infectious contributing factors

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ABSTRACT: Postweaning mortality is extremely complex with a multitude of noninfectious and infectious contributing factors. In the current review, our objective is to describe the current state of knowledge regarding infectious causes of postweaning mortality, focusing on estimates of frequency and magnitude of effect where available. While infectious mortality is often categorized by physiologic body system affected, we believe the complex multifactorial nature is better understood by an alternative stratification dependent on intervention type. This category method subjectively combines disease pathogenesis knowledge, epidemiology, and economic consequences. These intervention categories included depopulation of affected cohorts of animals, elimination protocols using knowledge of immunity and epidemiology, or less aggressive interventions. The most aggressive approach to control infectious etiologies is through herd depopulation and repopulation. Historically, these protocols were successful for *Actinobacillus pleuropneumoniae* and swine dysentery among others. Additionally, this aggressive measure likely would be used to minimize disease spread if either a foreign animal

disease was introduced or pseudorabies virus was reintroduced into domestic swine populations. Elimination practices have been successful for *Mycoplasma hyopneumoniae*, porcine reproductive and respiratory syndrome virus, coronaviruses, including transmissible gastroenteritis virus, porcine epidemic diarrhea virus, and porcine deltacoronavirus, swine influenza virus, non-dysentery *Brachyspira* spp., and others. Porcine circovirus type 2 can have a significant impact on morbidity and mortality; however, it is often adequately controlled through immunization. Many other infectious etiologies present in swine production have not elicited these aggressive control measures. This may be because less aggressive control measures, such as vaccination, management, and therapeutics, are effective, their impact on mortality or productivity is not great enough to warrant, or there is inadequate understanding to employ control procedures efficaciously and efficiently. Since there are many infectious agents and noninfectious contributors, emphasis should continue to be placed on those infectious agents with the greatest impact to minimize postweaning mortality.

Key words: death loss, infectious, mortality, postweaning, swine

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INTRODUCTION

Postweaning mortality is caused by a complex host of etiologies and risk factors with myriad interactive effects. In an effort to reduce postweaning mortality, contributors must be identified and effective strategies must be generated, evaluated, and implemented. Historically, greater resource allocation has been directed toward preweaning mortality, including research and summary articles, compared with postweaning mortality (Bereskin et al., 1973; Alonso-Spilsbury et al., 2007; Muns et al., 2016; Liu et al., 2018). To reduce mortality most effectively, producers and researchers should focus attention on those contributing factors with the greatest magnitude of effect and that can be controlled. We are unaware of any recent literature review, systematic review, or meta-analysis that has been performed regarding postweaning mortality. While causes of infectious mortality are often categorized by physiologic body system affected, we believe the complex multifactorial nature is better understood by an alternative stratification based on the intervention type applied. This category method subjectively combines knowledge of disease pathogenesis, epidemiology, and economic consequences. The proposed intervention categories are depopulation of affected cohorts of animals, elimination protocols using knowledge of immunity and epidemiology, and a third category of less aggressive interventions. This categorization allows simplification of the innumerable interactions between infectious and noninfectious factors contributing to postweaning mortality that are summarized as “the causal web” proposed by Gebhardt et al. (2020). One of the most complex and important interactive effects is the impact of various noninfectious factors on incidence, severity, and resolution of disease. Our objective is to describe the current state of knowledge regarding infectious causes of postweaning mortality, focusing on estimates of frequency, and magnitude of effect where available. The current document will focus on infectious causes of postweaning mortality without describing detailed

clinical signs or therapeutic, control, or preventative measures.

MATERIALS AND METHODS

The literature search was performed using Web of Science (<https://login.webofknowledge.com/>), which incorporated the Web of Science Core Collection, CAB Abstracts, and Medline databases, and Pubmed (www.ncbi.nlm.nih.gov/pubmed/), as well as the Scopus (www.scopus.com). Search terms included (SWINE OR PIG) AND (MORTALITY OR MORBIDITY OR DEAD OR REMOVAL) AND the respective search item of interest. Additionally, the assessment of bibliographical items resulted in further identification of relevant sources. Articles were restricted to primarily English language; however, non-English articles that contained an English abstract where relevant information could be extracted were included. Once relevant articles were identified, they were filed and categorized according to topic for further evaluation. This review is based on peer-reviewed literature without including conference proceedings and other nonpeer-reviewed literature.

RESULTS AND DISCUSSION

Overview of Infectious Disease

Infectious disease is a common contributing factor to mortality in all growth stages of swine. Mortality categories with the largest proportions include respiratory, gastrointestinal, meningitis/central nervous system signs, and failure to thrive (USDA 2015). Relative measures of incidence and magnitude of effect for infectious causes of postweaning mortality is included in Table 1. Disease is multifactorial in nature, often described as an epidemiologic triad consisting of agent, host, and environment (Dohoo et al., 2003). For the purpose of this endeavor, the epidemiologic triad of determinants of disease can be categorized into infectious and noninfectious factors (Fig. 1). Infectious

Table 1. Summary of infectious factors contributing to postweaning mortality based on incidence and magnitude of potential morbidity and mortality^a

Approach	Infectious agent	Incidence ^b	Magnitude ^c
Aggressive depopulation	Swine dysentery ^d	+	+++
	<i>Actinobacillus pleuropneumoniae</i>	+	++
	Foreign animal disease ^e	–	+++
	Pseudorabies virus	–	+++
Elimination	Enzootic pneumonia ^f	+++	++
	PRRSV	+++	+++
	Swine influenza virus	+++	++
	Nondysentery <i>Brachyspira</i> spp.	++	+
	Coronaviruses ^g	++	++
Infectious agents often managed without depopulation/elimination	<i>Glasserella parasuis</i> ^h	++	++
	<i>Pasteurella multocida</i>	++	++
	<i>Bordetella bronchiseptica</i>	++	+
	Other <i>Mycoplasma</i> spp. ⁱ	++	+
	<i>Actinobacillus suis</i>	++	+
	<i>Lawsonia intracellularis</i>	+++	++
	<i>Salmonella</i> spp.	++	+++
	<i>Escherichia coli</i>	++	++
	Rotavirus	++	++
	Hemorrhagic bowel syndrome	+	++
	<i>Streptococcus suis</i>	+++	++
	<i>Staphylococcus</i> spp.	+++	++
	<i>Erysipelothrix rhusiopathiae</i>	++	++
	Porcine circovirus	+++	+++
	Neurological syndromes associated with viral agents	++	+++

^aQualitative assignment of relative incidence and magnitude of mortality was performed by primary author (J.T.G.) based on summarization of published literature.

^bRelative incidence of mortality attributed to the infectious agent was denoted using a system ranging from + to +++. Infectious agents not currently in domestic swine populations in the United States was denoted by “–”.

^cRelative magnitude of mortality in a population attributed to the presence of the infectious agent was described as + (low potential), ++ (moderate potential), and +++ (significant potential).

^dCaused by *Brachyspira hyodysenteriae*, *Brachyspira hamptonii*, and *Brachyspira suanatina*.

^eIncluding, but not limited to, African swine fever virus, classical swine fever virus, and foot and mouth disease.

^fCaused by *Mycoplasma hyopneumoniae*.

^gIncludes PEDV, TGEV, and PDCoV.

^hFormerly *Haemophilus parasuis*.

ⁱIncludes *Mycoplasma hyosynoviae*, *Mycoplasma hyorhinis*, and *Mycoplasma suis*.

agents can include viruses, bacteria, parasites, and other agents, such as fungi or prions and are discussed in the current report. Noninfectious factors include characteristics of the host and environment that are discussed in greater detail in the accompanying manuscript. It is critical to understand the interwoven nature of these factors and acknowledge the complexity of biology, physiology, and production outcomes.

Reported incidence of specific disease processes for nursery, wean-to-finish, and grow-finish sites is provided in Fig. 2 (USDA, 2016). Mortality has been shown to be significantly greater in

animals experiencing a high health challenge [porcine reproductive and respiratory syndrome virus (PRRSV) and swine influenza virus A (IAV; 19.9%)] compared to moderate health challenge (IAV; 7.7%) or low health challenge (no PRRSV or IAV; 3.3%; Cornelison et al., 2018). Such data illustrates the significant impact that the combination of infectious causes of mortality can have on postweaning mortality. Throughout this review, the terminology of commercial swine production and domestic swine are used interchangeably to describe the swine population raised in a controlled, nonferal manner.

Systems-Based Overview of Infectious Etiologies

One approach to conceptualizing infectious postweaning mortality causes is categorization by physiologic system affected. Major systems affected and associated infectious agents include respiratory (IAV, PRRSV, *Mycoplasma* spp., and *Glasserella parasuis*), enteric (*Lawsonia intracellularis* and *Brachyspira* spp.), and systemic (*Erysipelothrix rhusiopathiae*, *Streptococcus suis*, and porcine circovirus). This approach is commonly used to attempt to understand the interaction between infectious agents resulting in pathology of certain systems, but we will propose and describe an alternative intervention-based approach below.

Porcine respiratory disease complex (PRDC) is often multifactorial, with combinations of viruses, bacteria, and sometimes parasites causing severe losses. Respiratory disease is one of the most common causes of mortality attributed to infectious

causes, with an estimated 47% of nursery mortality, 75% of grower/finisher mortality, and approximately 60% of wean-to-finish mortality associated with respiratory disease (USDA, 2015). Multiple pathogenic mechanisms may be additive or synergistic by enhancing colonization and organism virulence or compromising various host defense mechanisms. Examples include damage to the mucociliary apparatus, immunosuppression, altering cytokine responses, or reducing macrophage function (Yaeger and Van Alstine, 2019). Further description of the polymicrobial nature of PRDC has been described (Choi et al., 2003; Palzer et al., 2008; Opriessnig et al, 2011). Respiratory disease complex is augmented by interactions of infectious and noninfectious factors. Factors such as time between successive batches of pigs in a barn and air quality have been associated with increased incidence and severity of PRDC (Fablet et al., 2012a). This demonstrates the importance of the interaction of noninfectious contributors with infectious insults then resulting in pulmonary pathology and increased mortality. Morbidity estimates for PRDC in a number of different contexts have been reported to range from 1.9% to 40% and mortality from 2% to 20% (Baumann and Bilkei, 2002; Petersen et al., 2008; Hansen et al., 2010).

Enteric disease leads to mortality through impaired gastrointestinal structure or function. Infectious agents capable of damaging the gastrointestinal tract include but are not limited to

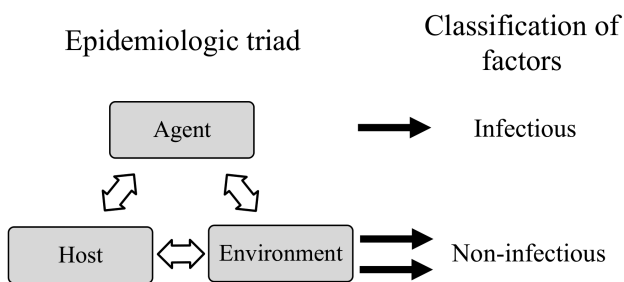


Figure 1. Epidemiologic triad of disease determinants and classification into factors contributing to postweaning mortality.

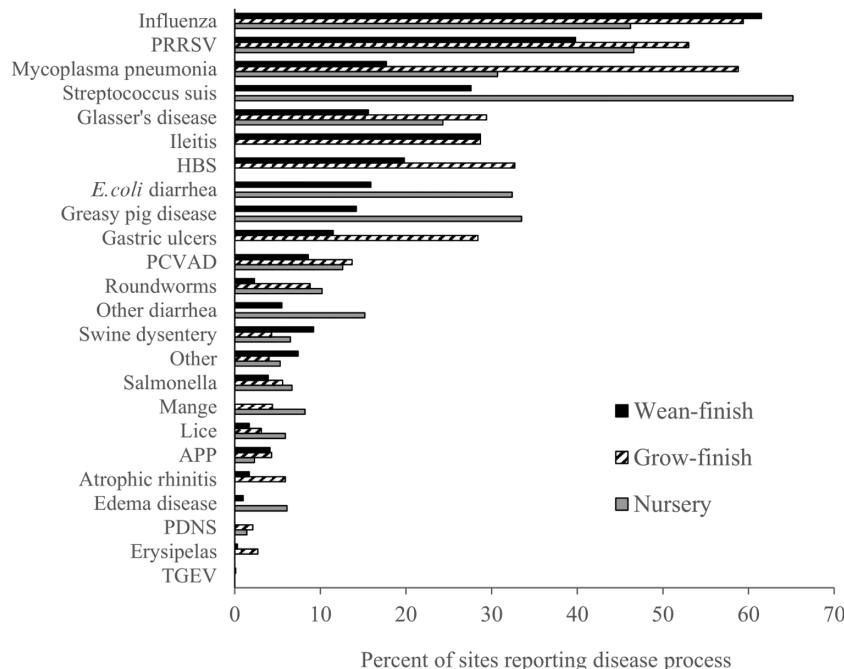


Figure 2. Percentage of nursery, wean-finish, and grow-finish sites reporting specific disease processes in the previous year [adapted from USDA (2016)]. Producer-reported values.

L. intracellularis, *Brachyspira* spp., *Escherichia coli*, *Salmonella* spp., coronaviruses, rotaviruses, and parasites. Similar to respiratory disease, multiple agents are often detected in clinical disease (Thomson et al., 1998; Stege et al., 2000; Thomson et al., 2001; Merialdi et al., 2003; Suh and Song, 2005; Reiner et al., 2011; Viott et al., 2013).

In addition to the respiratory and gastrointestinal system, infectious agents can also affect multiple body systems and result in mortality postweaning. [*E. rhusiopathiae*, porcine circovirus type 2 (PCV2), pseudorabies virus (PRV), foreign animal diseases (FAD)] Many bacterial agents associated with PRDC (*S. suis*, *G. parasuis*, and *Actinobacillus suis*) can also result in systemic disease with arthritis, septicemia, and meningitis. Other postweaning mortality infectious causes include shiga toxin-producing *E. coli* (STEC), which produces a toxin resulting in edema disease. Greasy pig disease, which may or may not be fatal, is a result of toxin production by *Staphylococcus hyicus*. Most of the agents responsible for systemic disease are commonly found in most swine herds. The diseases which may result, including polyserositis, erysipelas, edema disease, or other miscellaneous infections, accounted for 7.3% of pigs necropsied following euthanasia or death as reported by Baumann and Bilkei (2002). Another sporadic pathology associated with septicemia is bacterial endocarditis, which has been reported to be found in 0.019–0.038% of finishing pigs and has been associated with multiple etiologies, including *Streptococcus* spp, *E. rhusiopathiae*, *Arcanobacterium pyogenes*, *Staphylococcus aureus*, and *Streptococcus porcinus* (Katsumi et al., 1997). Although a systems-based approach is commonly used to conceptualize infectious diseases, we hereafter propose an intervention-based framework.

Intervention-Based Overview

Varying levels of intervention can be used to control infectious causes of mortality, including: 1) aggressive depopulation of affected cohorts of animals, 2) elimination protocols using knowledge of immunity, not introducing naïve susceptible host animals for a sufficient period of time, with or without additional control measures, such as herd medication, or 3) less aggressive interventions. Using such an approach to conceptualizing infectious causes of postweaning mortality allows for a clearer understanding of the potential magnitude effect of prolonged presence of the etiologic agent in a swine production system.

Depopulation

The most aggressive approach to control infectious etiologies is through depopulation of affected herds. Historically, these protocols have been implemented with success for *Actinobacillus pleuropneumoniae* (APP) and swine dysentery, as well as others as described by Sasaki et al. (2016). Justification to implement such aggressive measures is dependent upon the realization that satisfactory production efficiency cannot be achieved with the agent present in the herd. In addition to the aforementioned agents, depopulation would likely be used for control and elimination of regulatory-designated FAD or pseudorabies virus if introduced into domestic swine populations in the United States because of higher-level concerns, including access to international trade partners.

Swine dysentery Clinical disease known as swine dysentery is caused by *Brachyspira hyodysenteriae*, *Brachyspira hampsonii*, and *Brachyspira suanatina* (Burrough, 2017; Hampson, 2018a; Rohde et al., 2018; Hampson and Burrough, 2019). The prevalence of *Brachyspira* spp. has been widely reported in the literature, but clinical disease associated with swine dysentery is much less frequent in the United States compared with historical rates (Hampson and Burrough, 2019). Morbidity for swine dysentery can be up to 90% of the population and mortality can be as high as 30% in extreme clinical settings and 50% in experimental settings (Hampson and Burrough, 2019). Depopulation and, in some cases, elimination strategies without depopulation have been implemented with success resulting in a relatively low prevalence in the United States.

Actinobacillus pleuropneumoniae Clinical disease caused by APP is characterized by rapid progression pneumonia with death sometimes within hours (Gottschalk and Broes, 2019). Clinical disease caused by APP has decreased over time, primarily through aggressive depopulation of affected herds, with only a small percentage of sites reporting clinical disease in recent years (USDA, 2016). Vaccination for APP in field trials has been shown to reduce absolute mortality by 3–5% or a relative reduction of 65–83% (Habrun et al., 2002; Del Pozo Sacristan et al., 2014; Table 2). Morbidity is estimated to range from 10% to 100% in clinically affected herds, with mortality ranging from less than 1% to 10% in acute outbreaks (Frank et al., 1992; Pozzi et al., 2011; Sassu et al., 2018). Nevertheless, globally, APP remains an aggressive

Table 2. Efficacy of vaccination in field trials for various etiologies on postweaning mortality^a

	Mortality, %		Absolute difference, %	Relative difference, %
	Control	Vaccinated		
<i>Actinobacillus pleuropneumoniae</i>				
Habrun et al. (2002)	5.8	1.0	4.8	82.8
Del Pozo Sacristan et al. (2014)	4.0	1.4	2.6	65.0
<i>Glasserella parasuis</i> ^b				
McOrist et al. (2009)	1.4–2.1	0.5–0.9	—	—
<i>Lawsonia intracellularis</i>				
Almond et al. (2006)	17.5	1.6	15.9	90.9
Deitmer et al. (2011)	3.3	3.2	0.1	3.0
Peiponen et al. (2018)	6.5	7.4	–0.9	–13.8
<i>Mycoplasma hyopneumoniae</i>				
Bilic et al. (1996)	3.1	0.0	3.1	100.0
Maes et al. (1998)	9.2	9.1	0.1	1.1
Maes et al. (1999)	4.0	3.8	0.2	5.0
Pallares et al. (2000) ^c	3.6	1.4	2.2	61.1
Stipkovits et al. (2003)	9.4	5.1	4.3	45.7
Holyoake et al. (2006)	4.2	3.8	0.4	9.5
Tzivara et al. (2007)	11.9	8.1	3.8	31.9
Tassis et al. (2012)	9.0	6.6 ^d	2.4	26.7
Kristensen et al. (2014) ^e	2.2	2.5 ^f	–0.3	–13.6
Kristensen et al. (2014) ^g	3.0	3.3 ^f	–0.3	–10.0
PCV2				
Cline et al. (2008)	7.8	2.1	5.7	73.1
Fachinger et al. (2008)	8.7	6.6	2.1	24.1
Horlen et al. (2008)	18.4	9.0	9.4	51.1
Kixmoller et al. (2008)	7.5	3.5	4.0	53.3
Desrosiers et al. (2009)	9.5	2.4	7.1	74.7
Neumann et al. (2009)	10.4	5.0	5.4	51.9
Segales et al. (2009)	17.0	9.5	7.5	44.1
Pejsak et al. (2010)	28.8	16.1	12.7	44.1
Takahagi et al. (2010) ^h	20.8	12.1	8.7	41.8
Takahagi et al. (2010) ⁱ	26.5	13.7	12.8	48.3
Takahagi et al. (2010) ^j	14.7	14.1	0.6	4.1
Jacela et al. (2011)	5.9	3.1	2.8	47.5
Venegas-Vargas et al. (2011)	6.9	2.9	4.0	58.0
Young et al. (2011)	7.2	2.6	4.6	63.9
Fraille et al. (2012)	7.0	4.9	2.1	30.0
Lee et al. (2012)	32.2	8.0	24.2	75.2
Potter et al. (2012)	7.0	6.8	0.2	2.9
Han et al. (2013)	26.7	6.7	20.0	74.9
Heibenberger et al. (2013)	11.4	4.0	7.4	64.9
Sidler et al. (2012)	10.0	4.2	5.8	58.0
Velasova et al. (2013)	5.5	4.7	0.8	14.5
Potter et al. (2014)	11.0	7.8	3.2	29.1
Jeong et al. (2015)	16.7	11.1	5.6	33.5
Martelli et al. (2016)	13.4	9.9	3.5	26.1
Villa-Mancera et al. (2016)	26.5	8.3	18.2	68.7
Czyzewska-Dors et al. (2018)	5.3	5.0	0.3	5.7
PRRSV				
Mavromatis et al. (1999)	7.5	3.4	4.1	54.7
Park et al. (2014)	18.3	1.3	17.0	92.9
Jeong et al. (2018)	10.0	3.3	6.7	67.0

Table 2. Continued

	Mortality, %		Absolute difference, %	Relative difference, %
	Control	Vaccinated		
<i>Streptococcus suis</i>				
Pejsak et al. (2001)	4.7	2.3	2.4	51.1
Hopkins et al. (2019) ^k	—	—	—	21.0

^aInformation could not be gathered for etiologies not listed in table. Values provided to illustrate the range in observed responses across studies and not intended to make scientific interpretation. Thus, results of statistical hypothesis testing is not provided.

^bFormerly *Haemophilus parasuis*.

^cAverage of all site types.

^dIntramuscular vaccine treatment including both nursery and finishing periods.

^eNursery portion of publication.

^fAverage of two vaccination treatments using separate commercial vaccines.

^gFinisher portion of publication.

^hRepresents PCV2a-2 portion of investigation.

ⁱRepresents PCV2b portion of investigation.

^jRepresents PCV2a-1 portion of investigation.

^kHopkins et al. (2019) reported that administration of an autogenous *Streptococcus suis* vaccination reduced relative nursery mortality by 21%; however, did not provide specific estimates.

and important etiologic agent with the potential to substantially contribute to postweaning mortality.

Pseudorabies virus Pseudorabies virus, also known as suid herpesvirus 1 or Aujeszky's disease, is an enveloped virus that can lead to reproductive losses, neurological signs, and mortality (Mettenleiter et al., 2019). Seroprevalence historically was 50% or greater in commercial herds (Elbers et al., 1992). The U.S. commercial swine herd was declared free of PRV in 2004 through intense eradication efforts (Pedersen et al., 2013). McNulty (2003) reported up to 20% mortality in pigs aged 14–20 wk. Pseudorabies remains prevalent in Asia (Hu et al., 2016), with highly virulent strains described with morbidity estimates of 30–80% and mortality estimates of 3–60% in commercial settings postweaning (Wu et al., 2013; Yu et al., 2014; Gu et al., 2015; Yamane et al., 2015) and high morbidity and mortality in experimental settings (Tong et al., 2015). Pseudorabies virus has been eradicated from commercial swine in the United States but remains a significant concern internationally, and the possibility of reintroduction is ever present and implications on morbidity and mortality could be significant.

Foreign animal disease Multiple FAD present a risk for significant postweaning mortality, including African swine fever (ASF), classical swine fever (CSF), and foot and mouth disease (FMD) among others. While these diseases are not currently circulating in the United States, introduction may pose massive losses both via pathogenic mechanisms and voluntary depopulation to minimize disease spread. Possible routes of introduction could

include live animals, meat, other swine swine-based products, or fomites, including feed or feed ingredients (Dee et al. 2018; Beltran-Alcrudo et al., 2019; Jurado et al., 2019; Niederwerder et al., 2019). It has been reported that mortality following exposure to CSF ranges from 40% to 48% (Laevens et al., 1999; Dewulf et al., 2000). During an outbreak of ASF in Nigeria in 2001, mortality estimates ranged from 76% to 91% in postweaning pigs (Babalobi et al., 2007). Mortality associated with FMD is much lower at an estimated 2.5% (Pozzi et al., 2019). While FAD are not currently in the United States, introduction of such diseases or novel diseases would lead to significant mortality through pathology or depopulation to control disease spread, loss of trade access, and economic and social implications.

Elimination

When the burden of infectious etiologies is too great to effectively manage, elimination of disease is often attempted. Elimination protocols often involve elimination of offending pathogen(s) from the sow farm producing weaners followed by weaning uninfected pigs to a depopulated, clean site. Sow farm elimination is by not introducing naïve animals for the time period sufficient for immune clearance to eliminate the pathogen and/or reduce shedding to levels that are not infective for susceptible animals when introduced. These protocols often involve periodic vaccination of the herd during the closure period and, with some bacteria, whole-herd antimicrobials prior to the introduction of

naïve replacement animals at the end of the closure (Silva et al., 2019). Infectious organisms that have been successfully eliminated include *Mycoplasma hyopneumoniae* (MHP), PRRSV, coronaviruses, including porcine epidemic diarrhea virus (PEDV), transmissible gastroenteritis virus (TGEV), and porcine deltacoronavirus (PDCoV), IAV, and non-dysentery *Brachyspira* spp. Additionally, lice and mange can be managed with elimination but will not be discussed further as they currently do not lead to significant levels of postweaning mortality when using modern, confinement production practices.

Mycoplasma hyopneumoniae *Mycoplasma* spp. are very common organisms that lead to a number of disease processes, including an important role in PRDC through interactions with coetiologies. Clinical enzootic pneumonia is caused by MHP, historically the most important *Mycoplasma* sp. and a major contributor to PRDC (Pieters and Maes, 2019). The identification of MHP in swine is very common (Maes et al., 2000; Choi et al., 2003; Kukushkin and Okovytyaya, 2012; Merialdi et al., 2012; Vangroenweghe et al., 2015; USDA, 2016). Clinical presentation of MHP can be asymptomatic but also can result in clinical disease without coetiologies. More commonly, disease results due to multifactorial infections (Thacker and Minion, 2012). Vaccination for MHP has variable impact on absolute mortality in field trials, ranging from slightly greater mortality to a reduction in mortality of 4% or a relative reduction up to 100% (Bilic et al., 1996; Maes et al., 1998; Maes et al., 1999; Pallares et al., 2000; Stipkovits et al., 2003; Holyoake et al., 2006; Tzivara et al., 2007; Tassis et al., 2012; Kristensen et al., 2014). Commercial production records from flows that were MHP negative had an absolute finishing mortality that was 1.26% lower compared with MHP-positive flows (Silva et al., 2019). Efforts have been directed toward MHP elimination from sow populations in recent years, and the probability of a sow herd being negative for MHP 1 year following herd closure has been reported to be 83% (Silva et al., 2019). Elimination protocols using whole-herd medication without herd closure have been implemented but with lower success rates (Silva et al., 2019).

Porcine reproductive and respiratory system virus Clinical disease associated with PRRSV in postweaning pigs include respiratory disease, reduced growth rate, and increased mortality (Zimmerman et al., 2019). Coinfections with other respiratory viruses and bacteria are common with PRRSV infections. Disease associated with

PRRSV is very common within the United States (Choi et al., 2003; Tousignant et al., 2015; USDA, 2016). Additionally, clinical disease associated with PRRSV has a very predictable seasonal pattern with greatest weekly incidence occurring during the fall and winter (Tousignant et al., 2015). Porcine reproductive and respiratory syndrome virus seropositive herds were associated with low grow-finish performance [including average daily gain (ADG), gain-feed ratio (G:F), mortality, and carcass weight] compared with PRRSV seronegative herds (Fablet et al., 2018). Porcine reproductive and respiratory syndrome virus status at the time of weaning had a significant impact on postweaning mortality, with positive batches of animals having a median relative increase in mortality of 34% over the expected mortality of the system, meaning that absolute mortality would be 1.8% greater in PRRSV-positive batches of animals compared with PRRSV-negative batches (Alvarez et al., 2015a). Additionally, grow-finish pigs that were PRRSV positive at weaning have been shown to have the greatest mortality, followed by pigs that became PRRSV positive at some point during the grow-finish period and pigs that were PRRSV negative throughout (9.3%, 7.4%, and 6.0% mortality, respectively; Holtkamp et al., 2013). Vaccination for PRRSV in field trials has been shown to reduce absolute mortality by 4–17% or a relative reduction of 55–93% (Mavromatis et al., 1999; Park et al., 2014; Jeong et al., 2018). In one report where a vaccination program was initiated following a PRRSV outbreak in a sow farm, nursery mortality was reported to be 9.3% during the first 18 wk of the outbreak and was reduced to 2.2% 19 wk later following resolution of clinical disease (Kvisgaard et al., 2017). Morbidity rates have been reported to range from 45% to 100%, with 20–100% mortality (Tong et al., 2007; Lyoo et al., 2015; Liu et al., 2017; Dong et al., 2018). Porcine reproductive and respiratory syndrome virus is a very common and frustrating virus that results in respiratory disease among other disorders. The major impact of PRRSV on postweaning pigs is the contribution to PRDC, which can lead to significant mortality.

Swine influenza virus A Clinical disease associated with IAV is common (Loeffen et al., 1999; Choi et al., 2003; USDA, 2016), and morbidity within a population can be high. Synergistic effects have been observed with both bacteria and viruses in conjunction with IAV (Van Reeth and Vincent, 2019). Morbidity and mortality range significantly depending on situation-specific factors, including immunity, other infectious agents present, and

management/control factors being implemented. When acting alone, IAV does not often lead to substantial mortality. Interactions with other infectious agents often are an outcome of IAV infections that can lead to mortality. Influenza status at the time of weaning had a significant impact on postweaning mortality, with positive batches of pigs having a median increase in relative mortality of 13% over the expected mortality of the system (Alvarez et al., 2015a). Infection with IAV is extremely common, and a large percentage of pigs are exposed at some point in their lifetime. Morbidity estimates vary greatly but are of little value without consideration of other infectious agents. Morbidity estimates have been reported to increase by 2–100% when exposed to various IAV biotypes, and mortality has been reported to range from 1% to 10% (Ma et al., 2010; Welsh et al., 2010; Markowska-Daniel et al., 2013). IAV is a common infectious agent providing a significant contribution to PRDC and subsequent mortality.

Nondysentery *Brachyspira* spp. Disease caused by nondysentery *Brachyspira* spp. include porcine intestinal spirochetosis caused by *Brachyspira pilosicoli* (Trott et al., 1996; Hampson, 2018b), as well as colitis caused by *Brachyspira murdochii* and *Brachyspira intermedia* (Hampson and Burrough, 2019). Infection with *B. pilosicoli* has been reported to result in reduced growth performance with watery diarrhea (Hampson, 2018b), with morbidity between 5% and 15% in growing pigs with mortality approximately 1% (Thomson et al., 1998). Additionally, colitis caused by *B. murdochii* and *B. intermedia* is thought to be self-limiting and leads to minimal morbidity and mortality (Hampson and Burrough, 2019). The impact on morbidity and mortality attributed to nondysentery *Brachyspira* spp. is relatively minimal compared to swine dysentery.

Coronaviruses Swine enteric coronaviruses (SEC) are enveloped viruses including PEDV, TGEV, and PDCoV. Porcine respiratory coronaviruses are believed to be derived from TGEV through the deletion of a spike gene and were first described in the 1980s (Saif et al., 2019). The clinical significance of porcine respiratory coronavirus has been much lower compared with enteric coronaviruses, and additional information can be found elsewhere. Enteric coronaviruses replicate within the enterocytes of the small intestine, leading to profound epithelial necrosis, villus atrophy, severe maldigestive, and malabsorptive diarrhea (Saif et al., 2019). The age when pigs are infected impacts on morbidity and mortality, with morbidity and

mortality nearly 100% in pigs less than 7 d of age, 60–85% morbidity with low mortality if infected from 14 to 28 d of age, and lesser morbidity and mortality thereafter (Shibata et al., 2000; Madson et al., 2014). When comparing postweaning batches of pigs either PEDV positive or PEDV negative, it has been shown that PEDV-positive batches have a mean increase in mortality of 2–11% compared with PEDV-negative batches (Alvarez et al., 2015b; Yamane et al., 2016). Swine enteric coronaviruses lead to very high morbidity and mortality rates in young pigs, with less severe impacts postweaning. Nonetheless, SEC can result in substantial postweaning morbidity and mortality.

Infectious Agents Often Managed Without Depopulation/Elimination

Multiple postweaning mortality infectious causes have not been aggressively controlled through depopulation or elimination methods. Reasoning for lack of depopulation or elimination efforts are due to the high efficacy of immunization (e.g., PCV2) or are extremely common inhabitants of swine and/or the environment. Multiple infectious agents within this category can result in mortality without coinfections, while others are commonly associated with coinfections leading to multifactorial disease. However, most of these organisms do have noninfectious risk factors for disease expression. Protozoa such as *Cystoisospora suis* and *Eimeria* spp. can occasionally result in diarrhea postweaning, but clinical disease has much greater significance preweaning (Lindsay et al., 2019). Additionally, nematodes such as *Ascaris suum* and others are common if swine have access to dirt and can be managed with anthelmintic drugs (Brewer and Greve, 2019). Protozoa and nematodes can be minor factors contributing to postweaning mortality in modern, intensive swine production systems but will not be discussed further.

***Streptococcus* spp.** While *Streptococcus* spp. are often present in healthy animals, significant disease can result, most commonly associated with *S. suis* (MacInnes et al., 2008; USDA, 2016). Infection with PRRSV has been shown to increase susceptibility to *S. suis* infection (Thanawongnuwech et al., 2000; Li et al., 2019). Systemic infections (sepsis or septicemia) result in multiple disease processes, including meningitis, polyserositis (epicarditis, pleuritis, and peritonitis), synovitis, arthritis, endocarditis, encephalitis, abortions, and abscesses (Staats et al., 1997). It has been observed that pigs are more likely to die due to *S. suis* if one or more

littermates died during the nursery phase (Hopkins et al., 2018). Additionally, mortality associated with *S. suis* during the study period was shown to be less in offspring from sow's second litter compared with the first (Hopkins et al., 2018). Together, these results suggested that mortality in a farm experiencing *S. suis*-related disease is associated with maternal influence. Vaccines have been shown to reduce relative postweaning mortality from 21% to 51% compared with nonvaccinated cohorts (Pejsak et al., 2001; Hopkins et al., 2019). It has been reported that morbidity ranges are highly variable from less than 0.3–80% but most commonly below 5% (St. John et al., 1982; Staats et al., 1997) with an associated case fatality risk of 2–100% (St. John et al., 1982) based on a number of cofactors. This wide range of morbidity and mortality estimates can be at least partially attributed to the confounded nature of multiple bacterial agents typically involved and difficulty identifying certain pathological manifestations. Treatment has been reported to be successful when performed early with mortality ranging from 0% to 5%, but herd mortality has been reported to be as high as 20% in some situations (Staats et al., 1997; Torremorell et al., 1997; Villani et al., 2003; Hopkins et al., 2018). *Streptococcus suis* is a common infectious agent identified in swine production settings and can lead to substantial postweaning morbidity and mortality.

Glasserella* (formerly *Haemophilus*) *parasuis Glasser's disease is caused by this gram-negative bacterial species, characterized by fibrinous polyserositis and septicemia with tissue localizations in brain, joints, or lungs (Aragon et al., 2019). *Glasserella parasuis* is commonly present and can lead to significant mortality in populations of animals with no previous exposure. This diverse organism is a common inhabitant of the nasopharynx (Smart et al., 1989; Choi et al., 2003; MacInnes et al., 2008; USDA, 2016) but, when sepsis occurs, presents with clinical signs similar to signs observed with *S. suis*. Like *S. suis*, *G. parasuis* may interact with PRRSV to contribute to PRDC (Kavanova et al., 2017), although clear evidence of such an interaction was not observed by Solano et al. (1997). Vaccination of sows with an autogenous *G. parasuis* vaccine has been shown to reduce offspring nursery mortality (1.4–2.1% monthly mortality for control vs. 0.5–0.9% monthly mortality for vaccinated; McOrist et al., 2009). An important consideration when interpreting results of any vaccination trial for this or other pathogens is the potential for underlying publication bias, as well

as confounding factors. Mortality rates can range from 5% to 10% in herds with previous exposure (Nielsen and Danielsen, 1975; Smart et al., 1993; Aragon et al., 2019) and up to 75% in naïve herds (Aragon et al., 2012).

Pasteurella multocida Clinical disease associated with *P. multocida* occurs as either upper respiratory tract disease known as progressive atrophic rhinitis (PAR, commonly serotype D) or lower respiratory tract pneumonia (commonly serotype A). *Pasteurella multocida* can be commonly isolated from the respiratory tract of pigs and is historically very important due to PAR. Progressive atrophic rhinitis is caused by specific toxin-producing strains of *P. multocida* that either alone or in combination with *Bordetella bronchiseptica* can lead to destruction of the nasal turbinates and deformation of the snout (Register and Brockmeier, 2019). The occurrence of PAR is now relatively rare, and elimination strategies have been successful over time.

Generally, *P. multocida* alone is poorly effective at attaching and colonizing lower respiratory epithelium. When damage to the respiratory mucosa and debris-clearing mechanism occurs, colonization can occur leading to pneumonia (Register and Brockmeier, 2019). Thus, pneumonia due to *P. multocida* is largely dependent on other infectious agents to compromise pulmonary clearance or establish the early stages of clinical disease and, then, secondarily colonizes and contributes to disease. Vaccination with both inactivated *P. multocida* and *B. bronchiseptica* antigen has been shown to reduce postweaning mortality (5.3% for vaccinated group vs. 10.1% for unvaccinated group; Stojanac et al., 2013). *Pasteurella multocida*-associated disease has resulted in mortality from 5% to 40% in pigs of all ages (Pijoan and Fuentes, 1987; Cameron et al., 1996; Register et al., 2012). Like many respiratory infectious agents, providing specific estimates of mortality is difficult and dependent on a number of population-specific factors, including the presence of coinfections. The overall incidence of progressive atrophic rhinitis is very low with multisite, all-in/all-out production.

Bordetella bronchiseptica The primary agent associated with reversible, nonprogressive atrophic rhinitis is the gram-negative rod bacteria *B. bronchiseptica* (Brockmeier et al., 2019). The presence of *P. multocida*, with or without *B. bronchiseptica*, results in much more severe progressive atrophic rhinitis (Brockmeier et al., 2019). *Bordetella bronchiseptica* is associated with respiratory disease as a primary cause of pneumonia through colonization enhanced by other infectious agents, such as

IAV and through enhancing colonization of other infectious agents (Loving et al., 2010; Brockmeier et al., 2019). *Bordetella bronchiseptica* is commonly isolated from the respiratory tract of pigs (Choi et al., 2003; Zhao et al., 2011; Kumar et al., 2014) and can contribute to multifactorial disease processes historically, including atrophic rhinitis and most commonly PRDC. Estimates of morbidity and mortality are not available largely due to the complex multifactorial nature of resulting disease.

Other *Mycoplasma* spp. Additional *Mycoplasma* spp. are commonly isolated and associated with disease processes in swine, including *Mycoplasma hyosynoviae* resulting in arthritis in grow-finish pigs, *Mycoplasma hyorhinis* resulting in polyserositis and arthritis, and *Mycoplasma suis* resulting in loss of red blood cells (Pieters and Maes, 2019). While estimates of the impact on morbidity and mortality are not available, the magnitude of effect is believed to be relatively low.

Actinobacillus suis Sepsis due to *A. suis* can be very severe, but *A. suis* has a lower attack rate compared to APP. Isolation of *A. suis* is common in the upper respiratory organism of pigs (Gottschalk and Broes, 2019). Infection with *A. suis* has been described to lead to significant preweaning pig mortality (Sanford et al., 1990). This organism is associated with acute sepsis, serositis, and pneumonia in all stages postweaning, with or without other identified cofactors. The clinical significance is historically less in postweaning populations compared to APP, with little information available regarding estimates of magnitude of effect.

Lawsonia intracellularis Clinical disease due to *L. intracellularis* is known as ileitis, garden hose gut, or porcine proliferative enteropathy (Vannucci et al., 2019). *Lawsonia intracellularis* is a common organism (Chang et al., 1997; Chiriboga et al., 1999; Stege et al., 2000; Jacobson et al., 2005; Biksi et al., 2007; Dors et al., 2015; Weber et al., 2015; USDA, 2016) affecting postweaning pigs. Vaccination for *L. intracellularis* in field trials has shown variable effects ranging from slightly increased mortality with vaccination to reduction of absolute mortality by up to 16% or a relative reduction of 91% (Almond et al., 2006; Deitmer et al., 2011; Peiponen et al., 2018). Morbidity rates in cases of hemorrhagic enteropathy can range from 12% to 50%, and up to 50% of animals affected may result in mortality (Lawson and Gebhart, 2000; Kroll et al., 2005), whereas chronic disease can result in mortality around 1% (Lawson and Gebhart, 2000; Pejsak et al., 2009). *Lawsonia intracellularis* is common in swine herds and, in nonvaccinated populations, can

cause moderate levels of morbidity and mortality as a result of acute and chronic manifestations of disease.

***Salmonella* spp.** Clinical disease due to *Salmonella* spp. are primarily due to *Salmonella enterica* serotype *typhimurium* and *S. enterica* serotype *choleraesuis* (Griffith et al., 2019). Host-adapted *Salmonella choleraesuis* affects postweaning pigs and results in septicemia (Griffith et al., 2019), whereas *Salmonella typhimurium* is more likely to be manifested with diarrhea and dehydration. In the United States, *S. typhimurium* is isolated with greater frequency compared to *S. choleraesuis* (Foley et al., 2008; Griffith et al., 2019). The mortality associated with *S. typhimurium* is reportedly low (Griffith et al., 2019), although specific estimates are not available. Cases of *S. choleraesuis* often result in a morbidity of <10% with a high case fatality risk (Griffith et al., 2019). Pedersen et al. (2015) reported mortality ranging from 20% to 30% in Danish herds following an outbreak of *S. choleraesuis*. Due to the zoonotic nature of *Salmonella*, numerous studies have investigated *Salmonella* prevalence in swine populations, which are widely available in published literature. One recent report describes such changes from 1997 to 2015, reporting an increase in the prevalence of *S. enterica* serovar 4,[5],12:i- (Yuan et al., 2018), and has been associated with clinical disease (Shippy et al., 2018; Arruda et al., 2019; Naberhaus et al., 2019). Other *Salmonella* spp. remain important potential contributors to postweaning mortality with the additional ever-present risk of zoonosis.

Escherichia coli Multiple disease processes in postweaning pigs are a result of the gram-negative bacterium, including postweaning diarrhea, edema disease, and septicemia/endotoxemia, (Fairbrother and Nadeau, 2019). A case-control study comparing 50 Canadian nurseries with and without postweaning *E. coli* was performed and found that the mortality in case farms was 7.7% compared to 1.8% in control farms (Amezcuca et al., 2002). Postweaning diarrhea can result in mortality up to 25% but is more commonly much lower ranging from 1.5% to 2% (Fairbrother and Nadeau, 2019). *Escherichia coli* is a common enteric infectious agent that results in morbidity in postweaning pigs. The magnitude on mortality is often mild to moderate but can be more severe in select cases.

The edema caused by STEC is due to toxin production that enters the circulatory system and damages blood vessel walls, leading to fluid leakage into extravascular tissues (Fairbrother and Nadeau, 2019). Edema disease is relatively infrequent (USDA,

2016). Sporadic mortality can occur with a case mortality rate from 50% to 90% (Fairbrother and Nadeau, 2019). Edema disease secondary to toxin production by STEC is relatively uncommon and is not a major contributor to postweaning mortality.

Rotaviruses Multiple rotavirus groups have been identified, with rotavirus A being the most common and pathogenic to pigs, B being less common, and C primarily affecting preweaning pigs (Shepherd et al., 2019). Rotavirus infections also occur with other etiologies, increasing the severity of disease (Shepherd et al., 2019). Viral replication occurs within the epithelium of the intestine, leading to villus blunting and reduced absorptive capacity. Rotavirus C has been reported to cause 60–80% morbidity in feeder pigs with no mortality (Kim et al., 1999). Morbidity associated with mixed rotavirus infections has been reported to be as high as 70%, with mortality rates as high as 11% (Molinari et al., 2016). Research has also used molecular diagnostic techniques to characterize the presence of rotavirus from postweaning pigs with diarrhea (Martella et al., 2007; Lorenzetti et al., 2011). A common presentation of rotavirus infections occurs within the preweaning period as piglet diarrhea but can be a contributing etiology to postweaning diarrhea also. Rotavirus can result in significant malabsorptive diarrhea in postweaning pigs due to villous blunting and, especially in the presence of coinfections, can be a contributor to postweaning mortality from other causes, such as colibacillosis, salmonellosis, or inanition.

Hemorrhagic bowel syndrome Hemorrhagic bowel syndrome (HBS), also known as intestinal hemorrhage syndrome, porcine intestinal distention syndrome, or bloody gut, presents as sudden death in combination with abdominal distention and red discoloration of the intestine similar to mesenteric torsion (Grafofer et al., 2017; Thomson and Friendship, 2019). The reported distinguishing factor from a mesenteric torsion is that no clear torsion is evident upon initial examination with HBS. Distention of the intestines without mesenteric torsion is believed to be associated with highly fermentable diet, such as liquid whey, leading to increased intra-abdominal pressure and reduced venous blood flow (Thomson and Friendship, 2019), although various other contributing factors have been described, including infectious etiologies (Novotny et al., 2016; Grafofer et al., 2017). Historically, HBS has been reported to account for approximately 2–5% of yearly finishing pig mortality and as severe as 10–20% mortality in the 1960s and 1970s (Straw et al., 2002). HBS as an

independent disease process from torsion remains speculative because, in many cases, thorough evaluation will identify organ volvulus and/or torsion. There is currently no consensus regarding the cause of HBS in finishing pigs. It is clear that mortality does occur due to abnormalities that have been historically classified due to gross appearance as HBS. Further understanding of the complex pathophysiology is necessary, as well as astute postmortem examination, to accurately identify the cause of mortality.

Staphylococcus spp. Although *Staphylococcus* spp. are nearly ubiquitous in commercial swine production, clinical disease is relatively infrequent. Two primary etiologies cause disease in swine, including exudative epidermitis (greasy pig disease) caused by *S. hyicus* and multiple disease processes caused by *S. aureus* (Frana and Hau, 2019). Greasy pig disease due to *S. hyicus* has been reported to cause 14% morbidity in nursery pigs, with 5% mortality (Arsenakis et al., 2018). Additionally, *S. aureus* occasionally is associated with skin infections, septicemia, osteomyelitis, and endocarditis (Frana and Hau, 2019) but is not an infectious agent of major clinical relevance in swine. The primary concern regarding *S. aureus* is swine acting as a reservoir for the zoonotic methicillin-resistant *S. aureus* (Frana and Hau, 2019), and prevalence estimates have been described by Sun et al. (2015). *Staphylococcus* spp. are extremely common organisms; however, the clinical significance today is much reduced compared to historical magnitudes of effect.

Erysipelothrix rhusiopathiae Clinical disease associated with *E. rhusiopathiae* commonly known as erysipelas include septicemia, infectious arthritis, endocarditis, and skin lesions (Opriessnig and Coutinho, 2019). *Erysipelothrix rhusiopathiae* is present globally and is estimated that it can be isolated from the tonsils of 30–50% of apparently healthy pigs (Opriessnig and Coutinho, 2019). The yearly incidence from 1972 to 1979 in Minnesota ranged from 0 to 99 cases per 1,000 farrow-to-finish pigs (Wood et al., 1984). More recently, outbreaks in naïve populations can have mortality ranging from 20% to 40% in extreme cases (Opriessnig and Coutinho, 2019). The estimates of morbidity and mortality in nonnaïve outbreaks are variable and have not been clearly determined. Thus, *E. rhusiopathiae* remains a threat for significant morbidity and mortality in naïve or improperly vaccinated populations but is not commonly a cause of significant postweaning mortality.

Porcine circovirus Porcine circovirus has multiple types, including the nonpathogenic type 1

and PCV2 and, recently, type 3 has been identified as associated with clinical disease (Palinski et al., 2016; Arruda et al., 2019; Segales et al., 2019). Manifestations of porcine circovirus-associated disease (PCVAD) include PCV2 systemic disease (PCV2-SD, formerly postweaning multisystemic wasting syndrome), PCV2 reproductive disease (PCV2-RD), porcine dermatitis and nephropathy syndrome, and subclinical infections (Segales et al., 2019). Porcine circovirus type 2 has been commonly demonstrated to be present in PRDC-affected swine along with multiple other coetiologies (Kim et al., 2003; Hansen et al., 2010; Wellenberg et al., 2010; Ouyang et al., 2019). It is currently believed that respiratory disease associated with PCV2 are due to PCV2-SD and not primary lung pathology (Fablet et al., 2012b; Tico et al., 2013; Segales et al., 2019).

Widespread use of PCV2 vaccines is due to their efficacy in reducing clinical disease and mortality. A meta-analysis was published in 2011 comparing the efficacy of PCV2 vaccines at reducing postweaning mortality, which found that vaccination results in an absolute reduction of nursery-finish mortality of 5.4% and reduction of finish mortality of 4.4% (Kristensen et al., 2011). Across field studies identified in this review, the magnitude of impact on absolute mortality associated with PCV2 vaccination ranged from 0% to 24% or up to 75% relative reduction (Cline et al., 2008; Fachinger et al., 2008; Horlen et al., 2008; Kixmoller et al., 2008; Desrosiers et al., 2009; Neumann et al., 2009; Segales et al., 2009; Pejsak et al., 2010; Takahagi et al., 2010; Jacela et al., 2011; Venegas-Vargas et al., 2011; Young et al., 2011; Fraile et al., 2012; Lee et al., 2012; Potter et al., 2012; Han et al., 2013; Heibenberger et al., 2013; Sidler et al., 2012; Potter et al., 2014; Jeong et al., 2015; Martelli et al., 2016; Villa-Mancera et al., 2016; Czyzewska-Dors et al., 2018). Herds with high PCV2 antibody titers have been associated with reduced grow-finish performance (combined ADG, G:F, mortality, and carcass weight) compared to herds with lower PCV2 titers (Fablet et al., 2018). PCV2-SD had been shown to result in morbidity ranging from 1.6% to 60% and mortality ranging from 3% to 56% (Jemersic et al., 2004; D'Allaire et al., 2007; Horlen et al., 2007; Carman et al., 2008; Nielsen et al., 2008; Neumann et al., 2009; Pejsak et al., 2010; Woodbine et al., 2010; Shelton et al., 2012; Segales et al., 2019). PCV2 is a very common infectious agent that can lead to significant morbidity and mortality in unvaccinated populations.

Neurological syndromes associated with viral agents In recent years, an increase in the incidence of neurological syndromes has been observed and, in many cases, associated with diagnosis of viral infectious agents. This is still an area of ongoing research efforts, but multiple viruses recently have been associated with neurological syndromes, including porcine sapelovirus (Schock et al., 2014; Arruda et al., 2017b), porcine astrovirus type 3 (Boros et al., 2017; Rawal et al., 2019; Matias Ferreyra et al., 2020), and porcine teschovirus (Bangari et al., 2010; Deng et al., 2012; Carnero et al., 2018). The recently described atypical porcine pestivirus has been associated with congenital tremors (de Groof et al., 2016; Postel et al., 2016; Schwarz et al., 2017) but is not believed to be a major contributor to postweaning mortality (Gatto et al., 2019). Other viruses that can cause neurological conditions include PRV, classical swine fever, Japanese encephalitis virus, and PRRSV among others (Madson et al., 2019), but the current focus will be pertaining to porcine sapelovirus, porcine astrovirus type 3, and porcine teschovirus.

A recent investigation into neurological signs, including ataxia, paresis, and paralysis in 11-wk-old pigs, associated with porcine sapelovirus reported a morbidity of 20% and a case fatality rate of 30% (Arruda et al., 2017b). Porcine astrovirus type 3 has been reported to result in 1.5–4% mortality in weaned pigs (Boros et al., 2017), and a separate investigation reported a 75% case fatality rate (Arruda et al., 2017a). Porcine teschovirus has been reported to result in morbidity ranging from 3% to 60% with a case fatality rate of approximately 60% (Bangari et al., 2010; Deng et al., 2012; Carnero et al., 2018). There are a number of neurologic cases that never have a confirmed cause, and a significant amount of information remains unknown regarding the prevalence and clinical impact of these viruses. Finally, it is human nature to lump and categorize observations based on previous experiences and personal bias based on similar clinical presentation. Such diagnoses many times do not have sufficient evidence to derive such interpretations. This is by no means specific to neurologic diagnoses. It is important to recognize limitations in our knowledge base and accept that establishing a clear diagnosis is often challenging. This becomes very important when evaluating mortality data and attempting to make clinical decisions among the complexity of interactions between infectious and noninfectious factors contributing to mortality.

CONCLUSION

In conclusion, various strategies have been used to control infectious disease in swine, including depopulation, elimination, and less aggressive measures. Depopulation and elimination practices have been shown to be successful and clearly have a significant impact on morbidity and mortality reduction in affected populations. Important diseases and their infectious agents that have been a focus to eliminate through herd depopulation or elimination should continue to be a focus to have the greatest impact on minimization of postweaning mortality.

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