Rapid Communication: Postmortem lesions and heart weights of in-transit-loss market pigs in Ontario[1]

K. Zurbrigg,*[2] T. van Dreumel,† M. F. Rothschild,‡ D. Alves,§ R. Friendship,* and T. L. O'Sullivan*

*Department of Population Medicine, Ontario Veterinary College, University of Guelph, 50 Stone Rd E, Guelph, ON, Canada N1G2W1; †Veterinary Pathology Consultant, Guelph, ON, Canada; ‡Department of Animal Science, Iowa State University, 2255 Kildee Hall, Ames 50011; and §Veterinary Epidemiology Consultant, Elora, ON, Canada

ABSTRACT: In-transit losses (ITL) of market-weight pigs are defined as pigs that die or pigs that become nonambulatory during loading and shipping from the farm to the abattoir. The low proportion of ITL in market pigs implies that individual pig factors may influence ITL, in addition to commonly considered environmental or transport factors. Postmortem examinations of in-transit-loss pigs (n = 85) from 1 Ontario, Canada, abattoir indicated the cause of death to be acute heart failure as a result of cardiac lesions that developed prior to transport. The presence of preexisting cardiac lesions may explain why no or only a few pigs die in a trailer even when the entire load is exposed to extreme temperatures and other common transport risk factors.

Key words: cardiac. heart failure, in-transit loss, swine

 J. Anim. Sci. 2017.95:5532–5536
doi:10.2527/jas2017.2089

INTRODUCTION

In-transit losses (**ITL**) of market-weight pigs is defined as pigs that die during transport from the farm to the abattoir or pigs that become nonambulatory during transport and are euthanized on arrival to the abattoir (Ellis and Ritter, 2005). In-transit losses are not fully explained by commonly cited risk factors such as temperature, trailer stocking density, and journey length (Haley et al., 2008). The low percent of market pigs that die during transport (Haley et al., 2008), compared with the large number of pigs exposed to known in-transit-loss risk factors suggests that individual pig factors may influence losses during transport. Existing health problems in in-transit-loss pigs have not been well studied, and postmortem examinations are not routinely performed on in-transit-loss pigs. Health conditions existing prior to transport, in particular cardiac lesions, could make a market pig more susceptible to dying or becoming nonambulatory during transport and may be a missing component in explaining the variability of ITL. The purpose of this paper is to describe the lesions observed in postmortem examinations of in-transit-loss pigs including their cardiac weights and weight ratios and to determine if it is plausible that any of the described findings could make a pig more susceptible to becoming an ITL.

MATERIALS AND METHODS

Sample Collection

The postmortem examinations and the collection and examination of hearts for this study occurred between June 2012 and April 2015. The in-transit-loss pigs examined were collected from 1 federally inspected pig abattoir in Ontario, Canada. At the time of collection, the abattoir was 1 of the 3 largest in Ontario, receiving approximately 15,000 to 20,000 pigs per week. Pigs were identified by a tattoo that associates a pig with a geographic farm site and an

[1]This project was funded through Ontario Pork's Research Funding (06/13), Agricultural Adaptation Council's Ontario Farm Innovation Project Funding (0012), and the State of Iowa and Hatch funding. The authors would like to thank Peter Physick-Sheard, University of Guelph, for his consultations early in the project regarding data collection. The authors would also like to thank the pathologists and staff of the AHL and the staff at the abattoir, for your assistance in coordinating the collection and examination of carcasses and hearts.

[2]Corresponding author: Kzurbrig@uoguelph.ca

Received September 13, 2017.
Accepted October 3, 2017.

owner. Confidential information was known only to the abattoir staff; therefore, researchers did not have access to farm management or farm production data or the genetic information of the herds involved.

Carcass Postmortem Examination of In-Transit-Loss Pigs and Collection of In-Transit-Loss Hearts

Agreements were established with the abattoir to collect in-transit-loss pigs for postmortem examination. The in-transit-loss pigs examined were a convenience sample of all in-transit-loss pigs at the abattoir based on the ability of the abattoir's head of receiving to notify the project leader that an ITL had occurred. In-transit-loss carcasses were then delivered within 3 h of notification to the Animal Health Laboratory, University of Guelph, Guelph, ON, Canada, where the pathologist on duty completed a postmortem examination. Date of death, BW, sex, type of ITL (dead on truck or euthanized), and gross pathological lesions observed during the postmortem examination were recorded.

Initial postmortem examinations of 27 in-transit-loss pigs (data not shown nor included) indicated that cardiac lesions were the predominant pathology observed in the in-transit-loss pigs. As a result, a detailed examination of the heart was initiated on all subsequent ITL collected in addition to the postmortem examination of each carcass. All subsequent hearts were removed intact and stored in 10% formalin for future examination by 1 pathologist (TVD) who followed a standard protocol (outlined below).

Standardized Heart Examination Protocol

After a minimum of 7 d of fixation in formalin, visible clots were removed from major vessels, noncardiac tissue was trimmed away, and the aorta and pulmonary artery were trimmed to their bases. The heart was then weighed, and the weight recorded as total heart weight (**THW**). The aorta and pulmonary artery and left and right atria were assessed for the presence of dilation. The heart was then sliced transversely, approximately one-third of the distance between the base of the heart and the apex, and a digital photo was taken of the cross section of the base.

The presence of thickening and/or dilation of the left and right ventricles were recorded. If clots were found in the chambers, they were removed and weighed. The weight of the clots was subtracted from the THW. The atria were removed from the ventricles. The right ventricle (**RV**) was removed from the left ventricle (**LV**) and septum by cutting along the coronary grooves. The RV and then the LV plus septum (**LV+S**) were weighed separately, and these weights recorded. Heart valves were examined and scored for the presence or absence of any thickening and/or nodularity.

Four transmural sections of approximately 0.5 cm thickness and 1 cm^2 were taken from each heart perpendicular to the long axis of the LV. They included 1) ventricular septum taken from approximately one-third the distance between the aortic valve and the left ventricular apex, 2) posterior LV wall approximately half the distance between the mitral valve annulus and the left ventricular apex, 3) anterior LV wall approximately 2 cm lateral to the anterior descending coronary artery, and 4) RV wall at the apex. In addition, a transverse section was taken of the proximal aorta dorsal to the aortic valves. Slides were routinely prepared and stained with hematoxylin and eosin, and the presence of any lesions was recorded.

Statistical Analyses

The statistical program used for all analyses was Stata 14 (StataCorp LLC, College Station, TX). The number of unique farm sites, the bivariate variables of the number of in-transit-loss pigs collected in cool/cold and warm/hot weather months (weather type), the sex of the pigs examined, and the type of ITL were calculated. The percentage of cases where a specific lesion was observed during carcass postmortem examination was calculated as the count of the specific lesion divided by the number of carcasses examined. The frequency of gross and histologic cardiac lesions observed was calculated. A Fisher's exact test was used to compare the frequency of lesions between ITL type, sex, and weather type.

The average TWH, LV+S, RV, BW, heart weight (**HW**):BW ratio, and LV+S:RV ratio were calculated. A *t*-test was used to compare the HW and weight ratios between ITL type, sex, and weather type.

RESULTS

Carcass Postmortem

The in-transit-loss pigs examined (n = 85) came from 56 different farm sites. One ITL had an unrecorded farm site ID. Forty-four percent of the pigs examined were collected during warm- and hot-weather months (May–September) and the other 56% were collected during cool- and cold-weather months (October–April). The ratio of males to females in-transit-loss pigs that had a postmortem examination completed and where sex was recorded was 45:23 (17 pigs were missing the sex data). The ratio of the type of ITL (euthanized on the truck on arrival to the plant vs. dead on arrival to the plant) was 33:52. The frequency of postmortem observations were 12% (12/85) gastric lesions (gastrosplenic or colon torsion, ulcers, colitis, and hepatic tears); 16% (14/85) limb fractures; 18% (15/85) respiratory lesions (other than pulmonary edema, e.g., tracheitis, pneumonia, pleural adhesions); 22% (19/85) congestion of liver, spleen, or kidneys; 73% (62/85) pulmonary congestion and edema; and 100% (85/85) cardiac lesions (pericarditis, endocarditis, dilation and/or hypertrophy of ventricles, dilation of aorta and pulmonary artery, and dilation of atria).

Significant differences in the postmortem observations were found between ITL type. Limb fractures were greater for pigs that were euthanized (36% [12/33] euthanized vs. 4% [2/52] dead on the truck; $P = 0.0001$) and congestion of organs other than the lungs was greater for those that died on the truck (6% [2/33] euthanized vs. 33% [17/52] dead on truck; $P = 0.004$). More in-transit-loss pigs had pulmonary edema and congestion (84 [31/37] vs. 65% [31/48]; $P = 0.05$) and congestion of liver, spleen, or kidneys (35 [13/37] vs. 13% [6/48]; $P = 0.02$) in warm/hot weather than in cool/cold weather, respectively. There were no significant differences between postmortem findings by sex.

Gross Cardiac Lesions

Characteristic gross lesions of in-transit-loss hearts consisted of variable severity of overall cardiac enlargement, LV hypertrophy (100% [85/85]), RV hypertrophy (36% [28/77]), RV dilation (98% [83/85]), dilation of the atria (64% [54/85]), dilation of the pulmonary artery and aorta (87% [74/85]), thickening/nodularity of the atrioventricular valves (40% [34/85]), and pericarditis (18% [15/85]). There were no differences in the gross lesions by ITL type, sex, or weather type.

Histologic Lesions

Six of the 85 in-transit-loss hearts were missing histological data and were not included in the summary of histologic lesions. Myocardial fibers with hypertrophy (54% [43/79], degeneration (15% [12/79]), disorganization (32% [25/79]), atrophy and fatty replacement (42% [33/79], nuclear rowing/multinucleation (86% [68/79]), and interstial fibrosis (16% [13/79]) were observed in addition to the lesions of medial hyperplasia (73% [58/79]) and perivascular fibrosis (44% [35/79]) of the intramural coronary arteries. There were no statistical differences in the presence of these lesions between type of ITL, sex, or weather type.

Heart and Body Weights And Weight Ratios

The average HW and HW ratios are listed in Table 1 along with the comparison of the average values for hearts collected in cool/cold and warm/hot weather. Hearts collected in cool/cold weather had greater HW and weight ratios. There were no statistical differences in average HW and HW ratios by sex or ITL type.

DISCUSSION

Carcass Postmortem Examination

The health status of a finishing pig prior to transportation affects a pig's physiological response to stress (Ellis and Ritter, 2005). Preexisting lung or cardiac disease could increase the risk of a pig becoming nonambulatory or dying during transport to the abattoir (Bergmann et al., 1988; Carr et al., 2005; Johnson et al., 2013). Our findings are in agreement with those of several researchers who have evaluated the lungs of in-transit-loss pigs and concluded that respiratory health was not a risk factor (MacGregor and Dewey, 2003; Carr et al., 2005; Sutherland et al., 2008) and that the pulmonary congestion and edema observed were acute lesions (Clark, 1979; Bergmann et al., 1988). The hypertrophy and dilation observed in the in-transit-loss pigs represent remodeling of the heart and vessel walls to compensate for compromised cardiac output. The postmortem observations of gastrosplenic torsion and hepatic tears could be a result of enlargement of these organs due to congestion from cardiac insufficiency (Robinson and Robinson, 2007).

It has been suggested that the relative low HW:BW ratio of modern domestic pigs (von Engelhardt, 1966; Niewold et al., 2000) may result in compensatory cardiac remodeling occurring to maintain cardiac output due to their well-muscled, fast growth (Yang and Lin, 1997). The cardiac lesions observed in in-transit-loss pigs together with the frequency of pulmonary congestion and edema and a lack of other significant findings suggest that the primary cause of death was acute heart failure. This is similar to the findings reported in the few studies on in-transit-loss pigs where postmortem examinations were completed (Clark, 1979; Bergmann et al., 1988). Although it is commonly assumed that in-transit-loss hogs die of heat stress/heat stroke, it is difficult to distinguish from heart failure on postmortem examination (Leon and Kenefick, 2012). In addition, the diagnosis of heat stress is often assumed without confirmation by postmortem examination, because it occurs during hot weather (Barton-Gade et al., 2007; Sutherland et al., 2009; Haley et al., 2010; Nannoni et al., 2016). The need for higher cardiac output to provide evaporative cooling during elevated temperatures can result in collapse, muscle weakness, and clinical

Table 1. Average heart weights and heart weight ratios for 85 in-transit-loss finishing pigs from an Ontario, Canada, abattoir and the comparison of these measures between hearts collected in cool/cold (October–April) and warm/hot (May–September) weather months

Weight variable	Total ITL[1] average	Cool/cold weather average (SD) $n = 48$	Warm/hot weather average (SD) $n = 37$	P-value
Total heart weight, g	465.9 (80)	489.9 (85)	434.7 (60.9)	0.001
Left ventricle plus septum, g	282.7 (45.7)	291.8 (49.1)	271.0 (38.3)	0.04
Right ventricle, g	107.8 (22.5)	114.4 (23.6)	99.1 (18)	0.002
Left ventricle plus septum: right ventricle ratio	2.67 (0.36)	2.58 (0.35)	2.77 (0.35)	0.018
BW, kg	122.8 (9)	125.6 (10)	119.2 (7)	0.001
Total heart weight:BW ratio, g/kg	3.78 (0.54)	3.89 (0.56)	3.65 (0.50)	0.05

[1]ITL = in-transit losses.

signs of heart failure in humans with cardiac remodeling (Székely et al., 2015). Similarly, in-transit-loss pigs during hot weather are often observed to be short of breath, have blotchy areas of pale and reddened skin, be weak, or be down and unable to rise prior their death.

No literature describing differences in organ congestion or limb fractures by ITL type could be found. It is plausible to speculate that both these differences are explained by the length of time a pig was experiencing cardiac insufficiency until death. Pigs that died during transport may have had a greater level of cardiac dysfunction than those that were euthanized, and therefore, greater congestion of the organs was observed. Pigs that were euthanized had cardiac lesions but perhaps did not have dysfunction until after the fracture and the ensuing stress from that event occurred. Alternatively, these pigs may be experiencing symptoms of cardiac dysfunction (down, distressed, anoxic, and ataxic) that makes them more susceptible to fractures during transport.

Gross Cardiac Lesions

The dilation and hypertrophy in the ventricles of in-transit-loss hearts were typical of compensatory changes occurring over weeks to months. The observation of RV dilation in in-transit-loss pigs is in agreement with the findings of Bergmann et al. (1988), who suggested that this lesion resulted in acute heart failure of pigs during transport. Gross RV dilation has been associated with RV dysfunction and poor prognosis in humans and pigs (Greyson et al., 2000; Madias, 2011). The moderate to marked ventricular hypertrophy observed in the hearts of in-transit-loss pigs was also reported by Liu et al. (1994). These researchers observed thickened ventricular wall measurements in the pigs and likened the findings to manifestations of hypertrophic cardiomyopathy (**HCM**), a hereditary disease that can result in sudden death in humans and cats (Liu et al., 1994; Maron and Maron, 2013). It is plausible to speculate that pigs with ventricular dilation and/or hypertrophy would be less tolerant of the physical exertions experienced during loading and transport and would be more likely to succumb to acute cardiac failure.

Histologic Lesions

The lesions observed (medial hyperplasia and perivascular fibrosis of the intramural coronary arteries), hypertrophy and cellular disarray, and nuclear rowing/multinucleation are similar to those associated with HCM in humans (Hughes, 2004) and an HCM-like condition in pigs (Clark, 1980; Liu et al., 1994; Shyu et al., 2002). Additionally, thickening of the walls of intramural coronary arteries in pigs has also been noted in association with endocardiosis (Robinson and Robinson, 2007) and disorganization of cardiomyocytes has been associated with early histologic changes of remodeling due to pressure overload in the pig heart (Nediani et al., 2000).

Heart Weights, BW, and Ratios

The THW:BW ratio of a pig decreases with increasing BW as the pig ages (Yang and Lin, 1997; van Essen, 2017). Calculating the THW:BW ratio may indicate cardiac hypertrophy. Although the average THW:BW ratio for in-transit-loss pigs examined in this study fell within the normal THW:BW ratio reference ranges for pigs (0.32–0.48% [Robinson and Robinson, 2007] and, for 125-kg pigs, 0.28–0.42% [Wiseman et al., 2007]), the number of pigs used to establish these values was low (8 and 12, respectively), no ages were given, and the ranges were wide. The average THW:BW ratio for the in-transit-loss pigs was higher than the range listed for 6- to 12-mo-old crossbred pigs without HCM-like cardiac lesions ($n = 64$; 0.33–0.35%; Liu et al., 1994). Several researchers have found increased THW:BW ratios in pigs with cardiac lesions (Liu et al., 1994; Huang et al., 2001). The average THW:BW ratio for in-transit-loss pigs was lower than the range listed for 6- to 12-mo-old purebred research pigs with HCM-like cardiac lesions ($h = 55$; 0.39–0.53%; Liu et al., 1994).

The gross examinations of 85 in-transit-loss carcasses from 1 Ontario abattoir indicated the primary cause of death in the majority of pigs to be acute heart failure. It is plausible to speculate that pigs with the severity of cardiac lesions observed are likely unable to respond to the increased cardiac workload required during sorting, loading, and transport to the abattoir. Further research is needed to elucidate whether the compensatory lesions are a result of a primary cardiomyopathy or are due to the low THW:BW ratio of modern, fast-growing pigs.

LITERATURE CITED

Barton-Gade, P., L. Christensen, M. Baltzer, and J. Valentin-Petersen. 2007. Causes of pre-slaughter mortality in Danish slaughter pigs with special emphasis on transport. Anim. Welf. 16:459–470.

Bergmann, V., A. Grafe, and F. Spremberg. 1988. Transportbedingte Herz-Kreislauf-Insuffizienz und Myokardveranderungen beim Schwein. Mh. Vet. Med. 43:472–474.

Carr, S. N., J. P. Gooding, P. J. Rincker, D. N. Hamilton, M. Ellis, J. Killefer, and F. K. McKeith. 2005. A survey of pork quality of downer pigs. J. Muscle Foods 16:298–305. doi:10.1111/j.1745-4573.2005.00022.x

Clark, E. G. 1979. Necropsy survey of transport stress death in Saskatchewan market weight hogs. In: Proc. 22nd Annu. Meet. Am. Assoc. Vet. Lab. Diag., San Diego, CA. p. 53–60.

Clark, E. G. 1980. Intramural coronary arteriosclerosis and luminal stenosis in Saskatchewan swine. In. Proc. Int. Pig Vet. Soc. Congr., Copenhagen, Denmark. p. 352.

Ellis, M., and M. Ritter. 2005. Transport losses: Causes and solutions. In: Proc. Allen D. Leman Conf., St. Paul, MN. p. 176-178. http://purl.umn.edu/143195. (Accessed Sept. 2, 2016.)

Greyson, C., Y. Xu, L. Lu, and G. G. Schwartz. 2000. Right ventricular pressure and dilation during pressure overload determine dysfunction after pressure overload. Am. J. Physiol. Heart Circ. Physiol. 278:H1414–H1420.

Haley, C., C. E. Dewey, T. Widowski, and R. Friendship. 2010. Relationship between estimated finishing-pig space allowance and in-transit loss in a retrospective survey of 3 packing plants in Ontario in 2003. Can. J. Vet. Res. 74:178–184.

Haley, C., C. E. Dewey, T. Widowski, Z. Poljak, and R. Friendship. 2008. Factors associated with in-transit losses of market hogs in Ontario in 2001. Can. J. Vet. Res. 72:377–384.

Huang, S. Y., J. H. Lin, E. C. Lin, P. C. Yang, and H. L. Tsou. 2001. Effects of birth season, breed, sex, and sire family on cardiac morphology determined in pigs (*Sus scrofa domestica*) by use of echocardiography. Comp. Med. 51:545–549.

Hughes, S. E. 2004. The pathology of hypertrophic cardiomyopathy. Histopathology 44:412–427.

Johnson, A. K., L. M. Gesing, M. Ellis, J. J. McGlone, E. Berg, S. M. Londergan, R. Fitzgerald, L. A. Karriker, A. Ramirez, K. J. Stalder, A. Sapkota, R. Kephart, J. T. Selsby, L. J. Sadler, and M. J. Ritter. 2013. Farm and pig factors affecting welfare during the marketing process. J. Anim. Sci. 91:2481–2491. doi:10.2527/jas.2012-6114

Leon, L. R., and R. Kenefick. 2012. Pathophysiology of heat-related illnesses. In: P. S. Auerbach, editor, Wilderness medicine. 6th ed. Elsevier, Philadelphia, PA. p. 215–231.

Liu, S. K., Y. T. Chiu, J. J. Shyu, S. M. Factor, R. Chu, J. H. Lin, H. L. Hsou, P. R. Fox, and P. C. Yang. 1994. Hypertrophic cardiomyopathy in pigs: Quantitative pathologic features in 55 cases. Cardiovasc. Pathol. 3:261–268. doi:10.1016/1054-8807(94)90012-4

MacGregor, E., and C. Dewey. 2003. In-transit loss of Ontario's pigs: Range of losses by producer, trucker, packing plant and season. In: Proc. 34th Annu. Meet. Am. Assoc. Swine Vet., Orlando, Florida. p. 43–45.

Madias, J. E. 2011. Right ventricular dilatation: An often neglected component in the electrocardiographic assessment of patients with heart failure. Europace 13:1217–1218. doi:10.1093/europace/eur129

Maron, B. J., and M. S. Maron. 2013. Hypertrophic cardiomyopathy. Lancet 381:242–255. doi:10.1016/S0140-6736(12)60397-3

Nannoni, E., G. Liuzzo, A. Serraino, F. Giacometti, G. Martelli, L. Sarca, M. Vitali, L. Romagnoli, E. Moscardini, and F. Ostanello. 2016. Evaluation of pre-slaughter losses of Italian heavy pigs. Anim. Prod. Sci. 57:2072–2081. doi:10.1071/AN15893

Nediani, C., L. Formigli, A. M. Perna, L. Ibba-Manneschi, S. Zecchi-Orlandini, C. Fiorillo, V. Ponziani, C. Cecchi, and P. Liguori. 2000. Early changes induced in the left ventricle by pressure overload. An experimental study on swine heart. J. Mol. Cell. Cardiol. 32:131–142. doi:10.1006/jmcc.1999.1060

Niewold, T. A., G. J. van Essen, M. J. Nabuurs, N. Stockhofe-Zurwieden, and J. van der Meulen. 2000. A review of porcine pathophysiology: A different approach to disease. Vet. Q. 22:209–212. doi:10.1080/01652176.2000.9695060

Robinson, W., and N. Robinson. 2007. Cardiovascular system. In: G. Maxie, editor, Jubb, Kennedy and Palmer's pathology of domestic animals. 5th ed. Elsevier Saunders, New York, NY. p. 1–101.

Shyu, J. J., C. H. Cheng, R. A. Erlandson, J. H. Lin, and S. K. Liu. 2002. Ultrastructure of intramural coronary arteries in pigs with hypertrophic cardiomyopathy. Cardiovasc. Pathol. 11:104–111.

Sutherland, M. A., K. Erlandson, J. F. Connor, J. L. Salak-Johnson, P. Matzat, J. F. Smith, and J. J. McGone. 2008. Health of non-ambulatory, non-injured pigs at processing. Livest. Sci. 116:237–245. doi:10.1016/j.livsci.2007.10.009

Sutherland, M. A., A. McDonald, and J. J. McGlone. 2009. Effects of variations in the environment, length of journey and type of trailer on the mortality and morbidity of pigs being transported to slaughter. Vet. Rec. 165:13–18. doi:10.1136/vetrec.165.1.13

Székely, M., L. Carletto, and A. Garami. 2015. The pathophysiology of heat exposure. Temperature (Austin) 2:452. doi:10.1080/23328940.2015.1051207

van Essen, G. J. 2017. Cardiovascular proportionality of modern pigs: Are we breaking the allometric scaling laws? PhD Diss., Erasmus University, Rotterdam, the Netherlands.

von Engelhardt, W. 1966. Swine cardiovascular physiology – A review. In: L. K. Bustad, R. O. McClellan, and M. P. Burns, editors, Swine in biomedical research. Frayn Printing, Seattle, WA. p. 307–327.

Wiseman, T. G., D. C. Mahan, J. C. Peters, N. D. Fastinger, S. Ching, and Y. Y. Kim. 2007. Tissue weights and body composition of two genetic lines of barrows and gilts from twenty to one hundred twenty-five kilograms of body weight. J. Anim. Sci. 85:1825–1835. doi:10.2527/jas.2006-407

Yang, T. S., and J. H. Lin. 1997. Variation of heart size and its correlation with growth performance and vascular space in domestic pigs. Anim. Sci. 64:523–528. doi:10.1017/S1357729800016155