Early Embryonic deaths and Mummified Foetuses in Swine

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I. Introduction.

Swine is a prolific breeder in comparism with all other farm animal as it gives 2 litters per year with the average about 10–12 piglets at each farrowing. Much greater number of ova are shed at each heat, but some ova fail to be fertilized and few of the fertilized ones undergo early embryonic deaths. This is a physiological adjustment in order to give the sow better chances of carrying her young ones to full term and also suckle them satisfactorily. The embryonic mortality rate may be defined as the percentage of fertilized ova that fail to develope as living embryo or foetus at any specified stage of the gestation period.

The embryo that die very early in the gestation, undergo resorption but if death occurs late, mummification of foetuses takes place which could be seen at parturition. If the extent of early embryonic death or mummification results in marked reduction of the litter size, the condition is regarded as pathological.

Incidence.

Normal sow under good condition would shed about 20 ova at one heat period, after conception and gestation, the average of litters is about 12 piglets. This difference made of up unborn piglets are due to some being mummified foetuses, and other embryos dying early in gestation. It is also possible that some ova and some zygotes die before implantation. The incidence of early embryonic deaths and mummified foetuses have been reported by several workers. The first investigation of embryonic death in the sow goes back to Hammond's work on this problem in 1914 and 1921, and later Corner's work in 1923.

In 1914 Hammond observed and marked differnce between the number of corpora lutea in the ovaries and the number of pig embryos which were

present in the uteri of slaughtered sows : On further investigation Hammond, (1921) found an apparent embryonic mortality of 32.6 per cent in swine slaughtered between the fourteenth and sixtieth days of gestation. These observations were confirmed by Warwick, (1928) and Crew, (1925–1926) and the problem of embryonic and foetal mortality has since come to be recognized as perhaps the greatest single unsolved problem affecting litter size in swine.

As shown by Squires et al, (1952) ; Self et al (1955) Rathasabapathy et al. (1956) and Lerner et al. (1957), the greater portion of the deaths occur prior to twenty-fifth day of gestation. Gossett and Sorensen, (1959) observed a mortality of 28.9 percent at 25 days and 33.9 per cent at 40 days. Carroll et al, (1962) stated that early embryonic losses of 25 to 30 per cent and late foetal death of 5 to 10 per cent are a major factor in litter size.

Causes.

A Genetic factor

1. Uterine capacity

Settergren (1966) stated that the exact reasons that limit the capacity of the uterus to accommodate foetuses are not known but it has been assumed the implantation site per foetus is an important factor. The genetic basis is reflected in the significant differences of the number of living piglets born in the different swine breeds and even in different families in the same breed.

According to Lerner et al. (1957) the intra-uterine migration in gilt was apparent in 41.7 %. In this study, 44.7% of the corpora lutea were found on the right and 55.3% on the left ovary. The total embryos recovered 50.6% were in the right and 49.4% were in the left uterine horn. Embryonic mortality enhanced the equalization of the normal embryos between two horns since 50.3 %

were found on the right and 49.7% on the left. These data suggest that intra-uterine migration of ova does exist and its purpose may be to allow optimum spacing for each developing embryo. Cornor, (1923) has stated that over crowding of embryos and subsequent uterine insufficiency is an important but not the only factor in causing embryonic mortality in swine. It is also probably natures device to reduce the litter size so that the sow can provide the necessary nutrition for growth of healthy off-spring. Ratnasabapathy et al (1956) stated that foetal atrophy was enhanced if the uterine length was less than 45 cms.

2. Inbreeding

Inbreeding increases the embryonic mortality rate and cross-breeding reduces prenatal death loses (Squiers et al 1952) This depression is likely to be due to the combined losses resulting from maladjustment in the uterine enviroment and to a lessor extent an increase in the numher of genetically defect embryos. It is also believed that certain family-lines have special predisposition for higher incidence of early embryonic losses.

Sang (1956) states that the reduction litter size on inbreeding is such that one pig would be lost before weaning for every 20% increase of inbreeding. King & Young, (1957) stated that the decrease in litter size in sows due to inbreeding was caused by reduction in ovulation rate, defined as the number of ova shed at one oestrus and not by embryonic mortality.

3. Disposition for embryonic deaths or mummification.

These are nmerous reports which show that lines and families differ in ovulation rate. No difference in fertilization rate have been reported.

Embryonic survival appears to be a factor which can be influenced by the individual sow and by families, although, of course, not all authors have observed this. Ratnasabapathy et al. (1956) and Reddy, Lasley & Mayer (1958) reported that there was a significant variation in the foetal death rate of the daugters of different sires and advise that sire selection be made on the basis of the uterine capacity and efficiency of their daughters, Lasley (1957) observed that litters, characterised by low embryo mortality rate early in gestation, tended to have high embryo mortality rate late in gestation, and that genetic factors may have contributed to this. Baker et al. (1958) reported individual differences in the ability of gilts to maintain embryos, these being apparently genetic and environmental. Perry (1959) could devide his sows into two groups according to their embryonic loss. The sow with high embryonic loss, equal to 38.5% of the copora lutea showed no correlation between the number of ova and the rate of loss, where as in the sows with low embryonic mortality (24%) these two factors were correlated. Pond et al. (1960) found the percentage 4.97, 1.95 and 1.08 of late foetal mortality and stillbirths on 551 litters of Berkshire, Chester White and Yorkshire pigs respectively.

4. Chromosomal aberrations.

Genetic factors, especially a simple recessive autosome gene may cause death of the foetus. Recent work carried out by Smidt ated by Hugh (1965) showed evidence of early embryonic mortality in swine caused by infertile boars with chromosomal aberrations, Henriesson ated by Settegren (1966) found a case of translocation of chromosome pair in a boar. This boar had given small litters about half the normal size. It can be supposed these small litters were due to high rate of foetal mortality but this probably occured in the relatively earlier stage of pregnancy.

5. Defective ova.

Genetic defects of the fertilized ova or embryos have been less difficult to study in pig than in the cow because the sow is multiparous and therefore when a few ova or embryo die others remain alive. With the environment of the genetic tract in multipara the same for all ova some failure of fertilization or death of the embryo is probably due to innate defects or to differences between each ovum or embryo in respect to endurance, viability and capacity for growth good eggs survive and poor eggs succumb. The veiw that faulty implantation is responsible for pathological ova is untenable. (Rafferty-Machlis and Hartman, (1953)

The production of abnormal ova may be related to anomalies in the development of the graafian follicle (Laing 1949). Since multiple ovulations are some what inherited this genetic condition may be releted to the hormonal control of ovulation and to the production of normal, abnomal or nonviable ova.

6. Hormonal imbalance.

It is generally realised that hormonal deficiencies or imbalances may affect reproductive processes, and consequently there have been a number of attempts to improve fertility by supplementing the suspected defective endocrine functions.

The significance of progesterone in the reproductive processes influencing embryonic mortality has been investigated. Glasgow and Mayer (1956) had found a decreased level of pregnanediol excretion at the time when the embryonic mortality was greatest. Bredeck and Mayer (1956) had shown that urinary estrogens in sow were increased during the period when pregnanediol excretion was at a minimum.

Perry (1954) stated that early embryonic mortality may be greater in the large litters due to an excess of progesterone out put by the larger number of corpora lutea apparently does not apply in cases of late embryonic mortality. He had studied in Berkshire breed which had the smallest average litter size but the highest percentage of mummified foetuses. Loy et al. (1958) demonstrated an apparent lack of association between the number of corpora lutea and the total progesterone content of the luteal tissue, and they considered that this fails to support the hypothesis that the relatively greater embryonic mortality in sows with high ovulation rates may be due in part to an excess of progesterone. Reddy et al. (1958) and Spies et al. (1959) had given exogenous progosterone to the sow in the early gestation as a means of decreasing embryonic mortality with variable result. The use of hormone therapy (progesterone or estrogen or combination of two hormones) to decrease prenatal death losses had not given consistent results.

Results of the work on endocrine factors in embryonic mortality raise several guestions related to their effect on uterine growth and their role in providing an optimum environment for maximum embryonic survival,

7 Immunological interference between zygote-dam.

It is possible by electrophoresis to distinguish differences in the β-globulins or transferrines in sera of cattle and it has been shown that these differences are genetically controlled. Individuals can thus be classified according to their transferrin type. Ashton & Fallon (1962,) concluded from serveral series of observations that dam-foetus incompatibility associated with the transferrin type of the sire and dam, could result in loss of embryos. Conneally et al (1963) observed that maternal-foetal incompatibility due to iso-immune antibodies had a significant effect on foetal mortality.

The apparent relationship between transferrin types and reproductive performance in cattle have been studied. This stimulated similar studies in sow. Kristjansson (1964) has presented fertility from 350 mating in pig from which it appears that one particular transferrin mating class exhibited significantly poorer fertility than all the other classes

B. Acquired

1. Non-genetic defects in germ cells

a Age of ova and sperm

In the pig Dziuk & Henshaw (1958) stated that spermatozoa stored for three days before insemination produced zygotes much more susceptible to early mortality. Stratman et al (1958) observed no significant difference in fertilization rate following insemination using semen **stored for no or 12 hours.** In 1959 Dziuk reported that, with fresh semen, 88% of his sows had fertilized eggs at 4 days and 70% had embryos at 35 days; when semen was stored for 48 hours before insemination, the 4 day figure was 80% and the 35 day figure 35%. First, Stratman & Casida (1963) made observations, which partly confirmed these finding, as shown below

	Age of Semen	
	6 hours	54 hours
3 – day fertilization rate %	61.0	39.3
% CL as 25 day embryos	48.1	17.6
Embryonic servival	78.9	44.8

In natural mating if several services are performed that towards the end of the series immature sperm may be ejected which through incomplete fertilization will cause atrophy of the foetus or lead to the development of a weak embryo.

In the longer estrous period makes the time of mating more critical and the means of determination of the begining of estrous is of importance in experiments. Hancock (1959) detected estrous by means of a vasectomised boar. The sows were served at varying periods after detection of estrous. As the interval from detection of estrous to service increased, there was an increase in the number of ova with more than two pronuclei, a condition which would lead to abnormal development of the embryo and its probable death. Bomsel-Helmreich (1962) observed the service after the 44^{th} hour of estrous was associated with 4.1% heteroploid embryos.

b. **Seasonal and high environmental temperature affecting.**

Season of the year has no effect upon fertility but may influence embryonic mortality. Poor litters occur more frequently into Winter than in Spring and early Summer. (Braude et al. 1954).

It is generally recognized that swine, because of their poor sweat glands development suffer discomfort at high environmental temperatures. To determine the effect of high environmental temperature upon gilts immediately after breeding. Warnick et al. (1961) maintained five groups in combinations of pasture and shade with subsequent expose to, 15.6°C (60° F) or 32.2°C (90°F). The gilts with the poorest performance (10.4 embryos) were those which were held at 32.2°C for a period of 3 days after breeding. The group held at 15.6°C average 13.6 embryos. The data indicated that temperatures of 32.2°C may be even more detrimental to embryo survival after 3 days post-breeding.

2. Defects in Maternal environment.

Olds & Vandenmark (1957) made an excellent review of the literature regarding the role played by variation in the maternal environment provided by the genitalia for sperm, ova, embryo or fertility.

It is known prenatal deaths increase and litter size increases with the old dam. This way prcbably be seen as a lower capacity of the uterine wall to support the foetus and the development the placenta. Even low virulent non-specific infections in the uterus can deteriorate the maternal uterine environment and give rise to foetal death (Settergren 1966).

3. Infections

The different kinds of infection and especially viruses, have been shown to give rise to foetal mummification which sometimes have reached enzootic proportions.

A. Virus

During recent years a series of viral diseases of domestic animals had been shown to influence the fertility and foetal developments. In swine at least four viruses which had been shown cause foetal deaths or influence foetal growth, which some others are also suspected to cause the same.

1. Swine Fever (Look in Swine Fever vaccination)

2. Aujeszky's disease or Pseudorabies.

This disease reported as a cause of foetal death in swine in Ireland by Gordon et al. (1955).

In a farm with 500 sows an outbreak of diseases occured in small swine up to 4 weeks old, after 2 months the number of sows give birth to mummified

foetuses. In certain case foetuses from whole litters were mummied and other time only appear foetuses were normal and living signs of Vitamin A & D deficiency were noticed in the farm and could have predisposed to the infection.

In Holland, young pigs were attacked by Aujeszky's disease which later brought about abortion in the sow 2 weeks before time of delivery and also later from of embryos deaths and mummified foetuses the aborted sows, the virus has been isolated and aborting sows had antibody in blood, which was not go in the case of normally farrowing sows in the same farm (Terstra, 1961).

Also from Hungary has been reported abortion 2-3 weeks before normal farrowing time in connection with an outbreak of Aujeszky's disease. Experimental infection gave malformed foetuses mummification or maceration.

3. Japanese B encephalitis and Hemagglutinating virus.

In Japan a great number of foetal death sometimes during abortion, and at other times during parturition in swine have occured in connection with or outbreak of Japanese B-encephalitis among human and horses. Many of the foetus were mummified. From small pigs which died has been isolated a virus which was identified as Japanese B-encephalitis virus (Burns 1950)

Later a similar syndrome could be brought forth with a swine pathogenic hemagglutinating virus.

4. SMEDI Virus.

During the last few years in the U.S.A., there were occasions in which a virus has been isolated from swine which was the cause of stillbirth, mummification, embryonic death and infertility. This has got the name SMEDI-Virus after the initials of the English words for the observed symptoms. (Stillbirth, mummification, embryonic death and infertility). The disease has been newly described by Dunne et al. 1965

B. **Bacterial infections**

Swine erysipelas – which attacks pregnant sows causes often abortion if the infection occurs early in the pregnancy. With infections during the later part of

pregnancy the foetus born during the normal time are often dead and eventually mummified or weak piglets may be born which often die. It is therefore of the greatest importance that in an outbreak of erysipelas in a farm a prophylactic treament of the pregnant sows be undertaken.

Brucellosis – among swine is an economically very important cause of prenatal mortality which occurs rather often in the greater part of the world. As a rule the disease causes abortion of the foetus or give rise to weak piglets if they seldom give rise to foetal mummification.

Leptospirosis – Michna (1962) obsesved Leptospira canicola infection in two piggeries with infertility problems. In experimental work on a small number of pigs, artificial infection of two sows in the fourth and fifth week of pregnancy resulted in abortion 4 – 7 days later. McErlean (1964) described an outbreak of porcine infertility characterised by apparent embryonic death and abortion, in which leptospirosis was diagnosed.

c. Mycotic infection

In England it has been reported that Mycotic infection has been demonstrated in the aborted and stillborn foetus and they also demonstrated in the fodder.

4. Nutritional factors

The influence of the general nutrition of the sow on embryonic viability is not clearly understood, but a deficiency of specific nutrients does not appear to be cause of high prenatal death rates commonly observed. However, the level of energy intake is established to be related to embryonic survival. The physiological cause of high prenatal death losses occuring in sow maintained on a high energy plane of nutrition is not known., but it is likely related to both the daily intake of energy during early pregnancy and to the body composition of the dam.

There have been a number of studies on embryonic death in relation to feeding. Robertson, Casida, Grummert Chapman (1951) studied the influence of high and low protein (15% and 11.25%,) and of restricted and unrestricted feeding. The different protein levels had no clear effect on embryonic survival, where as there was a significantly greater degree of embryonic survival in the restricted than in the unrestricted feeding groups, the rates being 67% and 43% respectively. Tribble et al. (1956) fed diets with 16% and 12% protein and obtained slightly better survival of embryos with the lower protein diet, which was compensated for by the higher ovulation rate of the gilt on the 16% protein diet.

Palludau (1961) stressed that inadequat level of Vitamin A in the diet of pregnant sows may cause abortion, absorption and other abnormalities of the foetuses

Casida (1953) cited data showing that in swine a lack of Vitamin B 12 with full feed for gilts during their breeding period resulted in a higher embryonic death rate than if sows and gilts were fed Vitamin B12 and kept on a low plane of nutrition during the breeding period and early gestation.

Apparently swine are the only animal in which a deficiency of calcium may affect reproduction. Davidson (1930) reported that sows developing a severe calcium deficiency had a tendency toward an increase in foetal atrophy.

Robertson et al, (1951) and Christian et al. (1952) reported that a high level of feeding sow cause a higher ovulation rate but smaller litter size in swine. Fatness was also associated with increased mortality of embryos and small litter size.

Overfeeding or high feeding level are reported by Casida (1953) and others to have a definite effect on increasing the early embryonic mortality. There is some evidence which suggest that embryonic mortality is reduced by feeding high levels of antibiotics (Hignett, 1963)

5. Poisoning

There have been many occasions in with poisoning was suspected to be the cause of foetal mortality. In connection with accumulated material about foetal mummification Skane for some 10 years ago, it was suspected that it could have been caused by dicumerol (Ekstram, 1957). The cause for these suspicious was that in some cases with mummified foetuses died in connection with farrowing and that the picture pointed to dicumeral poisoning. In some cases some cattle died also showing the same symptoms. Dicumeral has still not been succesfully demonstrated in the dead foetuses or organs from the sows.

6. Effect of vaccinations against Hog cholera

Young et al. (1955) had demonstrated that attenuated hog cholera vaccine could produce foetal death with absorption or mummification, or foetal arrest resulting in the developement of anomalies and death in the new-born. At the first third of gestation period malformation most frequently occurred when the animal was infected between the 15th and 25th days. The susceptibility of the embryo in this period was attributed to the absence of a completely formed placental membrane. Foetal conformation is nearly complete at 30 days. Infection with the attenuated hog cholela virus during the first 10 days of pregnancy resulted in absorption of the foetuses. Infection after 25 days produced few gross anomalies but resulted in many stillborn and weak pigs.

Pathological changes

When fertilized ova die, they undergo progressive cytolysis within the zona pellucida, which often remains intact after the cytoplasm and nucleus are dead. The whole then disintegrate and get resorbed or are discharge at the next estrum. Presumably, in those cases in which estrous occurs after a normal cycle the ova die most commonly between the fourth and tenth day of the cycle. Although the embryo may die at any period, it does so commonly at about sixteenth day. In

the embryonic stage, the embryonic tissue proper disappears first and trophoblast remains for a while before degenerating.

Afer a later stage in development, the dead foetus may become a mummified. Mummified foetuses repersent cases where a partially resorbed shrunken, dehydrated, grey-brown conceptus was readily discernible, The size varied from a length of approximately 5 cm. to a length approaching that of a full term foetus. Distinction between a mummified foetus and stillborn piglet was made on the basis of the degree of resorption or dehydration.

Diagnosis

In swine, there is no way of an ascertaining the death of foetus or foetuses except at abortion or at parturition. Small litters of 4 to 8 could indicate early embryonic mortality. Small mummified foetuses are commonly noticed at parturition, along with normal foetuses. When gestation is prolonged, death and mummification of all the litter could be suspected.

Prevention

Early embryonic deaths and mummified foetuses in swine are inevitable. But when the incidence is high or the litter size is small, it becomes an uneconomical factor. If hereditary factors are involved such family lines should not be used for breeding purposes. Where boars are responsible for such high incidence of early embryonic deaths, they should be dispensed with. Cross-breeding offers a means of increasing litter size significantly. In proved efficiency could be obtained by selecting replacement gilts from large litter culling sow that produce small litters, and breeding on both the 2nd and 3nd day of estrous, avoiding animals and times of breeding with a high incidence of inherited defects and by following a good disease control in feeding levels and management practices not only at the time of breeding but at all stage of reduction so as to produce large healthy litters at farrowing.

Conclusion

Much research work is needed on methods of improving fertility in the sow. Embryonic mortality is clearly established to be of great importance but the exact causes are not well known. A better understanding of factors affecting implantation may prove to be of considerable assistance. The relative importance of environmental and genetic influences on specific components of the reproductive cycle, in addition to the assessment of the litter size at farrowing is another branch of study that needs further investigation. The study of the variation in litter size at farrowing may contribute some knowledge toward preventing early embryonic death.

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