The Genetic and Non-Genetic Aspects of Leg Weakness and Osteochondrosis in Pigs - Review -

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ABSTRACT : Leg weakness in pigs is one of the most serious problems in the pig industry. Leg weakness is responsible for an increase in the rate of culling of breeding pigs, which results in economic loss in the pig industry. Many researchers have investigated the cause of leg weakness, and that of osteochondrosis, in pigs, as well as in other mammals. For evaluating leg weakness, subjective scoring criteria have been applied, but the scoring systems varied widely. Subjective scoring systems have also been used in evaluating osteochondrosis lesions in leg joints. The lesions were scored optically, radiographically and histologically. Leg weakness and osteochondrosis are to some extent heritable traits. The heritability of leg weakness and joint lesion scores was found to range from 0.01 to 0.42. Leg weakness and osteochondrosis are associated with production traits (lean percentage and back fat depth). Nutritional aspects of leg weakness and osteochondrosis have also been extensively studied. Although the energy level of the diet may affect leg weakness and osteochondrosis, other nutritional factors seem to have very little effect. Confinement conditions also have an effect on leg weakness and osteochondrosis. Although studied extensively, the cause and etiology of leg weakness and osteochondrosis remain uncertain. It could be that there are multiple causes of leg weakness. (*Asian-Aust. J. Anim. Sci. 2001. Vol. 14, No. 1 : 114-122*)

Key Words : Leg Weakness, Osteochondrosis, Heritability, Pigs, Nutrition

INTRODUCTION

Leg weakness in pigs is one of the most serious problems in the pig industry. Leg weakness leads to the reduction of the longevity of breeding pigs. Due to leg weakness, the rate of premature culling of breeding animals has increased, and this had led to a decrease in the rate of improvement of pigs in breeding programs. Consequently, leg weakness in pigs is responsible for large economic loss in the pig industry (Hill, 1990b). Furthermore, the leg weakness problem involves the issue of pig welfare, because lame pigs probably suffer severe pain associated with specific lesions (Nakano and Aherne, 1993). Many researchers have investigated the cause of leg weakness in pigs from various points of view in an effort to find a solution to this complicated problem.

This review covers the causes and methods of evaluation of leg weakness and osteochondrosis. In addition, the genetic aspects of leg weakness and joint lesions in pigs essential to be considered for the improvement of leg soundness in pigs are described. Other aspects related to leg weakness in pigs will also be mentioned.

THE CAUSE AND ETIOLOGY OF LEG WEAKNESS

Although leg weakness in pigs has been studied extensively by many researchers, the exact cause of leg weakness remains unclear (Nakano et al., 1987). Many researchers have investigated the relationship between nutrition and leg weakness. It has been suggested that the energy level of the diet might affect leg weakness (Grøndalen, 1974a; Jorgensen, 1995). However, other nutritional factor, such as the level of Ca, P or vitamins, seems to have very little or no effect on leg weakness if these levels satisfy the minimum requirements. It has also been suggested that mechanical overloading may be responsible for leg weakness (Grøndalen and Grøndalen, 1974). Additionally, the conditions of confinement of pigs seem to affect leg weakness (Elliot and Doige, 1973; Fredeen and Sather, 1978; Sather and Fredeen, 1982).

Many researchers have studied the genetic aspects of leg weakness also. It has been shown that leg weakness and the severity of joint cartilage lesions (osteochondrosis) are to some extent heritable traits (Smith, 1966; Reiland et al., 1978; Bereskin, 1979; Webb et al., 1983; Lundeheim, 1987; Rothschild et al., 1988; Lundeheim and Rydhmer, 1990; Kadowaki et al., 1998).

Although osteochondrosis is not exactly the same condition as leg weakness, it has been suggested that leg weakness is closely associated with osteochondrosis (Grøndalen, 1974a; Reiland, 1978; Goedegebuure et al., 1980; Nakano et al., 1987; Hill, 1990a). To the contrary, other researchers found no relationship between leg weakness and osteochondrosis (Thurley, 1969; Goedegebuure et al., 1988; Jorgensen, 1995). Lundeheim (1987) has suggested that there is significant relationship between osteochondrosis and leg

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weakness only when the joint lesion is severe.

Osteochondrosis is a syndrome or group of syndromes involving degenerative joint disease or limb deformity in young, fast-growing pigs of both genders (Hill, 1990a) Grøndalen (1974b) described osteochondrosis as a primary, noninflammatory disturbance that leads to a failure of endochondral ossification in joint cartilage and growth plates. He suggested that osteochondrosis might be the same condition as that called osteochondritis in humans and dogs. In human and dogs, osteochondritis gives rise to osteochondritis dissecans and finally leads to osteoarthrosis significantly associated with lameness.

It has been shown that osteochondrosis occurs in nearly 100% of growing young pigs (Grøndalen, 1974b; Reiland, 1978b; Lundeheim, 1987). Grøndalen (1974c) has shown that osteochondrosis occurs in both growing pigs and young breeding pigs at the same joint sites, and it is less common in mature breeding pigs. He suggested that after sexual maturity, a lesion that has not affected the surface of the joint cartilage may heal completely or be repaired, and that lesions that have reached the surface of the joint cartilage give rise to osteoarthrosis. Osteoarthrosis and proliferous osteitis can apparently give rise to lameness in pigs (Grøndalen, 1974b; Blowey, 1992). Histological and histochemical investigations focusing on osteochondrosis have also been performed by several researchers (Ekman and Rodriguez-Martinez, 1991; Ekman and Heinegard, 1992; Ekman and Carlson, 1998). Ekman and Carlson (1998) have suggested that the etiology of osteochondrosis may be associated with several factors: trauma, heredity, rapid growth, nutrition and local anemia. These studies showed that the early lesions of osteochondrosis are very similar among species. However the etiology and the cause of osteochondrosis still remain uncertain. Further investigation of osteochondrosis and leg weakness is required. Further investigation of the etiology of osteochondrosis is also necessary.

EVALUATION OF LEG WEAKNESS AND JOINT LESIONS (OSTEOCHONDROSIS)

Evaluation of leg weakness

In order to evaluate locomotive ability or leg weakness, subjective scoring criteria have been used by many researchers (table 1). The number of criteria varies widely. It could be said that the number of criteria for leg weakness scoring may affect the accuracy of measurement of leg weakness. It may also affect the outcome in estimation of the heritability of leg weakness if these values are used.

For the purpose of attaining a more objective measurement of leg traits, Calabotta et al. (1982) used 16 mm motion pictures of pigs walking on a treadmill for measuring the gait characteristics of pigs. They concluded that the correlation coefficient for the relationship between each of various photographic characteristics and the structural soundness scores was generally very low.

Evaluation of joint lesions (osteochondrosis)

Concerning the evaluation of joint lesions of osteochondrosis, post-mortem measurement of joint cartilage has been performed by many researchers.

Table 1. The number of criteria in leg weakness scores and joint lesion scores

Authore	Score position	No of criteria
Leg weakness scores		
Fredeen and Sather (1978)	Front leg	5 (1-best to 5-extreme crippling)
	Hind leg	5 (1-best to 5-unable to stand)
Bireskin (1979)	Whole limb	11 (0-worst to 10-best)
Webb et al. (1983)	19 leg traits	Aggregate score of 19 leg traits
Lundeheim (1987)	Whole limb	3 (1-servere to 3-normal)
Rothschild et al. (1988)	Front leg structure	9 (1 worst to 9 best)
Joint lesion scores		
Grøndalen (1974b)	Each joint	3 (normal mild severe)
Grøndalen (1974d)	Each joint	5 (1-normal to 5-severe)
Goedegebuure et al. (1980)	Proximal humerus	4 (0-normal to 3-severe)
	Distal humerus	4 (0-normal to 3-severe)
	Proximal femur	4 (0-normal to 3-severe)
	Distal ulna	6 (0-normal to 5-severe)
Empel and Shested (1986)	Distal ulna and growth plate	4 (4-normal to 1-severe)
Lundeheim (1987)	Front and hind joint	6 (0-normal to 5-severe)
Jorgensen (1995)	Elbow and stifle joints	5 (1-normal to 5-osteochondritis dissecans)

Like leg scores, various scoring criteria have been used for evaluating the severity of osteochondrosis in the joint cartilage (table 1). Although scoring criteria were used for the evaluation, these scores seem to be more objective ones than leg weakness scores, because pathological diagnostic criteria are usually applied. These methods can be used to evaluate joint lesions precisely. Nevertheless, it is necessary to slaughter pigs to evaluate the condition of the joint cartilage. To obtain more precise information on joint lesions, Hill (1990a) has suggested that it is necessary to examine most of the major joints. Hill et al. (1984a) suggested that examination of radiographs of bone slabs was useful for diagnosing small lesions or for diagnosing growth plate osteochondrosis. Hill et al. (1984b) suggested that histological and histochemical methods of evaluation of growth cartilage and bone are the most reliable methods for diagnosing osteochondrosis especially in case in which the lesions are very small. Enpel and Sehested (1986) have shown that computer tomography (CT) is effective for measuring joint lesions in the case of osteochondrosis in the growth plate of the ulna in postmortem investigations.

Concerning the investigation of joint lesions in live pigs, Hill (1990b) has shown that osteochondrosis in live pigs can be diagnosed by radiography in case in which the lesions are relatively large. However, since anesthesia is required to obtain the radiographs, wide adoption of radiography seems to be difficult.

Although arthrocentesis and evaluation of synovial fluid have been extensively used for diagnosing osteochondrosis in horses (van Pelt, 1974; Tew, 1982), it seems that these methods have not been used for pigs. Hill (1990b) suggested arthrocentesis might be difficult for such diagnosis in the case of pigs since this method requires highly aseptic conditions, whereas pig skin is contaminated with a large amount of microorganisms. Hill et al. (1984b) has shown that synovitis is not a primary change associated with osteochondrosis in pigs.

Kramer et al. (1999) showed that ultrasonography is useful for diagnosing osteochondrosis in stifle joints of dogs, although clinical and radiographical investigation was necessary. Takahara et al. (2000) concluded that osteochondritis dissecans of the humeral capitellum in humans can be detected by ultrasonography. Although it seems that there have been no reports on ultrasonographic evaluation of osteochondrosis in pigs, this procedure may be very applicable since it is not an invasive method and there is no need for radiographical apparatuses.

Various methods for evaluating leg weakness and osteochondrosis have been investigated. However, the main feasible ways are still the subjective scoring method for leg weakness and post-mortem evaluation of cartilage osteochondrosis in pigs.

THE GENETIC ASPECTS OF LEG WEAKNESS AND OSTEOCHONDROSIS

Breed and sex differences

Breed differences in leg weakness and the severity of joint cartilage lesion have been reported by many researchers. Grøndalen (1974c) has shown that Yorkshire pigs have a lower incidence and a lower severity of osteochondrosis and arthrosis than Landrace pigs.

Grøndalen (1974d) has also shown that there is a difference in severity of osteochondrosis between lines of the Yorkshire breed. Similar results have been reported by several researchers (Webb et al., 1983; Lundeheim, 1987). Goedegebuure et al. (1980) have investigated the severity of osteochondrosis in six breeds of slaughter pigs (Belgian Landrace, Duroc, Dutch Landrace, Dutch Yorkshire, Hampshire and Pietrain), and it was shown that Duroc has significantly more severe joint lesions in the front limbs, and that Belgian Landrace has significantly more severe lesions than the other breeds in the hind limbs. As for crossbred pigs, Reiland and Anderson (1977) investigated the difference in severity of osteochondrosis between different cross-bred pigs and he did not find any significant difference between them.

Van der Wal et al. (1987) have investigated the difference in leg weakness and severity of osteochondrosis between boars and gilts of four breeds (Duroc, Pietrain, Yorkshire and Dutch Landrace), and they concluded that there was a significant interaction between breed and sex in the degree of leg weakness and osteochondrosis.

As for the sex difference, it has been suggested that boars have poorer leg soundness and severer joint lesions than gilts (Grøndalen, 1974b; Nakano et al., 1979; Goedegebuure et al., 1988). However, there were several results which have shown no difference between sexes (Grøndalen, 1974c; Grøndalen and Vangen, 1974; Jorgensen, 1995).

Estimation of heritability

Smith (1966) has estimated the heritability of leg weakness scores through analysis of data obtained for MLC testing station boars, and reported that the estimated heritability was not significantly different from zero. Bereskin (1979) reported that estimated values for the heritability of leg traits were between 0.15 and 0.38, although the standard errors were relatively large (table 2-1). Webb et al. (1983) have estimated the heritabilitiy of leg action scores and aggregate leg scores in Large White and Landrace boars. The estimated heritability of aggregate leg scores was 0.17 and 0.19 in the Large White and Landrace breeds, respectively (table 2-2).

In Sweden, Lundeheim (1987) estimated the heritability of leg weakness scores and joint scores in the Swedish Yorkshire and Landrace breeds, and the values of were between 0.11 and 0.35 (table 2-3). Lundeheim and Rydhmer (1990) also estimated the heritability of leg weakness and joint score in the Swedish Landrace breeds. They mentioned that later estimations of the heritability yielded lower values than the former estimations. Van Steenbergen et al. (1990) estimated the heritability of fourteen leg scores by residual maximum likelihood (REML) procedure and the values ranged from 0.01 to 0.31. Other results obtained in estimation of heritability were similar to the above-mentioned values (Jorgensen, 1987; Liu et al., 1991; Kadowaki et al., 1998).

With respect to realized heritability, Rothschild and Christian (1988) have shown that realized heritability of front leg scores obtained from five generations of divergent selection were 0.29 and 0.42 in high soundness and low soundness lines respectively.

Generally, the estimated heritability values ranged from low to intermediate values. Additionally, it seems that the values for heritability of Joint lesion (osteochondrosis) scores are slightly higher than those of leg weakness scores. Although various scoring criteria have been used for evaluation of leg weakness and joint lesions, the effect of the number of criteria on the estimated heritability of leg and joint scores seems to be unclear. Webb et al. (1983) have suggested that bias in estimated heritability of trinomial traits might be smaller than Binomial traits. Actually, nearly all of the leg and joint scores are

Table 2-1. Heritability of leg weakness

Trait	Sire		Sire+dam	
	h^2	s.e.[1]	h^2	s.e.
Front soundness	0.27	0.21	0.28	0.11
Rear soundness	0.15	0.24	0.38	0.11

[1] s.e.: Standard error.
Pooled estimates for Duroc and Yorkshire populations (Berskin, 1979).

Table 2-2. Heritabirity of leg traits in performance tested boars

Leg trait	Breed	at 27 kg		at 91 kg	
		h^2	s.e.[6]	h^2	s.e.
Leg action[1]	LW[4]	0.136	0.042	0.082	0.028
	LR[5]	0.049	0.055	0.179	0.037
Aggregate leg score[2]	LW	0.192	0.045	0.172	0.030
	LR	0.125	0.061	0.194	0.038
Conformation score[3]	LW			0.172	0.029
	LR			0.218	0.039

[1] Leg action is scored subjectively on a scales from 1 to 5.
[2] Aggregate leg score is derived as the sum of the scores (0, 1 and 2) for 19 individual traits.
[3] Conformation score is scored on scales from 1 to 10.
[4] LW: Large White.
[5] LR: Landrace.
[6] s.e.: Standard error of the heritability (Webb et al. 1983).

Table 2-3. Heritability in Swedish Landrace and Yorkshire pigs

Trait	Breed	h^2	s.e.
Elbow joint score[a]	Landrace	0.21	0.03
	Yotkshire	0.25	0.04
Knee joint score[b]	Landrace	0.33	0.04
	Yotkshire	0.35	0.05
Leg weakness score[c]	Landrace	0.14	0.03
	Yotkshire	0.11	0.03
Incidence of uneven digits[d]	Landrace	0.11	0.03
	Yotkshire	0.11	0.03

[a] The severity of elbow joint cartilage lesions was scored on a scale from 0 to 5.
[b] The severity of knee joint cartilage lesions was scored on a scale from 0 to 5.
[c] The leg weakness observed was scored on a scale from 1 to 3.
[d] All or nothing trait (Lundeheim, 1987).

Table 2-4. Heritability in a Swedish Landrace pig population

Trait	Period	h^2
Leg weakness score[a]	1	0.27
	2	0.40
Elbow joint score[b]	1	0.27
	2	0.28
Knee joint score[c]	1	0.19
	2	0.31

[a] The severity of elbow joint cartilage lesions was scored on a scale from 0 to 5.
[b] The severity of knee joint cartilage lesions was scored on a scale from 0 to 5.
[c] The leg weakness observed was scored on a scale from 1 to 3 (Lundeheim and Rydhmer, 1990).

Table 2-5. Realized heritability estimates for front structure and front movement from five generations of divergent selection

Line	Front structure		Front movement	
	h^2	s.e.	h^2	s.e.
Selected for soundness	0.29	0.02	0.31	0.07
Selected for weakness	0.42	0.04	0.38	0.04

Front structure and movement scores: 1-worst to 9-best (Rothchild and Christian, 1988).

trinomial or more than trinomial traits. Lundeheim (1987) suggested the estimated heritability of leg and joint scores might be less biased and less underestimated than the heritability of binomial traits. Van Steenbergen et al. (1990) linearly scored leg traits on a scale from 0 to 9 with 0.5 unit increments (19 levels) and the estimated heritability values were similar.

Phenotypic and genetic correlation

Several researchers have investigated the relationship between production traits and leg weakness and joint lesion traits. Bereskin (1979) estimated the genetic correlation between leg traits and daily gain before testing and in the testing period, between leg traits and back fat, and between leg traits and loin eye area (table 3-1). Van der Wal et al. (1980) have shown an unfavorable phenotypic correlation between the relative weight of ham and morphological lesion scores for the femur. Webb et al. (1983) have reported a significant unfavorable genetic correlation between leg weakness scores and ultrasound back fat depth in Large White and Landrace boars. Lundeheim (1987) found an unfavorable genetic correlation between leg constitutional traits and growth rate, and between constitutional traits and lean percentage (table 3-2). Jorgensen (1987) has reported an unfavorable phenotypic correlation between leg weakness and lean percentage. Johansen et al. (1993) found an unfavorable phenotypic correlation between osteochondrosis and performance traits in a line selected for lean tissue growth late in a low protein feeding regime

Table 3-1. Genetic correlation between leg traits and production traits

Leg trait	Production trait				Rear
	W/A	ADG	BF	LEF	F-L score
Front F-L score	0.035	0.334	0.350	-0.327	0.805
Standard error	0.159	0.171	0.178	0.210	0.138
Rear F-L score	0.087	0.093	0.052	-0.186	-
Standard error	0.139	0.158	0.166	0.191	

W/A: Pig weight per day of age at start of test; ADG: Average daily gain on test; BF: Average back fat thickness; LEA: Loin eye area; F-L: Feet and legs.
Each genetic correlation was estimated from sire+dam components (Bereskin, 1979).

Selection experiments and practical selection programs for leg soundness

Reliable estimations of genetic parameters associated with leg traits have become available only recently. Moreover, low to intermediate heritability values and an unfavorable correlation between leg traits and production traits have been found. Therefore, it has been suggested that selection for leg soundness or for reduction of joint cartilage lesions is difficult (Nakano et al., 1987; Hill, 1990b). However, several experiments on selection for leg soundness have been reported and there are several breeding programs in which leg traits are included in the selection criteria.

In the United States, divergent selection in 5 generations for and against front leg soundness was fulfilled during the period from 1980 to 1985 (Rothschild and Christian, 1988; Rothschild et al., 1988; Goedegebuure et al., 1988). After the divergent selection, Rothschild and Christian (1988) reported that the mean scores for low, control and high lines were 3.02±0.17, 5.67±0.21, 7.91±0.21, respectively ($p<0.05$). They also reported that high line pigs (selected for soundness) had lower osteochondral lesion scores for the proximal radius and ulna than control or low line pigs had, but the severity of osteochondrosis in other

Table 3-2. Genetic correlation between joint scores and leg weakness scores

Trait	Elbow joint score		Knee joint score		Leg weakness score	
	L	W	L	W	L	W
Elbow joint score[a]	-	-	0.36	0.44	-0.50[e]	-0.49
Knee joint score[b]	0.36	0.44	-	-	-0.41	-0.55
Leg weakness score[c]	-0.50	-0.49	-0.41	-0.55	-	-
Incidence of uneven digits[d]	-0.06	-0.08	-0.17	-0.08	0.01	0.26
Growth rate during the test	0.10	0.19	0.29	0.03	-0.26	-0.35
Lean percentage	0.22	0.17	0.28	0.08	-0.43	-0.09
Carcass length	-0.24	0.00	0.13	0.16	-0.21	-0.67

[a] The severity of elbow joint cartilage lesions was scored on a scale from 0 to 5.
[b] The severity of knee joint cartilage lesions was scored on a scale from 0 to 5.
[c] The leg weakness observed was scored on a scale from 1 to 3.
[d] All or nothing trait.
[e] Because the joint scores are form o-best to 5-severe, whereas the leg weakness scores are form 1-worst to 3-best (Lundeheim, 1987).

joints and growth plates was not significantly different between the lines.

As for correlated responses, a significant difference in back fat thickness was found between high and low soundness line pigs in generation 5 (Rothschild et al., 1988). Additionally, Draper et al. (1992) have reported the following significant differences between the three lines.

1) The total weight of extensor muscles of the forearm was heavier in low soundness line pigs than in control or high leg soundness line pigs ($p<0.05$).

2) Although there was no significant difference in the weight of the bones of the front leg, there were several significant differences in the length of front leg bone between these lines of pigs. The humerus was shorter in the high soundness line pigs than in the control or low soundness line pigs ($p<0.05$). The metacarpal bones were significantly shorter in the high soundness line than in the control or low soundness line pigs ($p<0.01$).

3) The head of the humerus was wider in the high soundness line pigs than in the low soundness line pigs.

They concluded that selection for different degrees of leg weakness resulted in significant changes in bone and soft tissue structures.

In Sweden, leg weakness scores and elbow and knee joint scores have been included in the official Swedish pig progeny testing index as a restriction factor since 1988 (Lundeheim and Rydhmer, 1990). After several years of selection for increased daily gain and lean percentage, the realized selection differential was for reducing osteochondrosis lesions and no unfavorable genetic trend with respect to leg weakness was observed (Lundeheim et al., 1994).

Johansen et al. (1993) reported that in a study involving selection for an increase in lean tissue growth rate in pigs fed diets with low and high dietary protein levels, the correlated response of reduction of osteochondrosis lesions was found in the line under selection with the high dietary protein level.

Considering these selection results, it seems that selection for leg weakness is to some extent effective. However, some problems may exist. There are only limited numbers of reports of genetic parameters associated with leg traits. The observed correlated responses reported by Lundeheim et al. (1994) and Johansen et al. (1993) were inconsistent with the previously predicted unfavorable genetic correlation between leg traits and lean percentage. Because the genetic correlation between leg traits and lean percentage is a highly negative value, it might be very important to estimate a more precise genetic correlation. Additionally, further investigations examining the correlation between leg traits and production traits are needed.

THE EFFECT OF NUTRITION ON LEG WEAKNESS AND OSTEOCHONDROSIS

Ca-P levels

Many researchers have investigated the effects of calcium and phosphate on leg weakness and the severity of osteochondrosis. It is well known that calcium and phosphorus deficiencies cause rickets (NRC 1979). However, it has been shown that calcium and phosphorus supplementation has no effect on leg weakness (Kornegay and Thomas, 1981; Calabotta et al., 1982; Barczewski et al., 1990), or the severity of osteochondrosis (Kornegay et al., 1990).

Reiland et al. (1991) investigated the relationship between hypophosphatemia and osteochondrosis, and they concluded that although hypophosphatemia can cause rickets, it was not an etiological factor involved in the development of osteochondrosis.

Energy level of the diet and the feeding regime

Several researchers have reported that the energy level of the feed has no effect on leg weakness (Nakano et al., 1979; Calabotta et al., 1982; Piedrafita et al., 1991). To the contrary, Grøndalen (1974a) has shown that pigs fed *ad libitum* had poorer leg condition than pigs fed by restricted feeding. Jorgensen (1995) found a significant difference in some leg traits between pigs fed diet with different energy levels.

Grøndalen (1974e) have shown that the feeding regime (*ad libitum* or restricted) has no effect on the severity of osteochondrosis. Jorgensen (1995) found that the energy level of the feed has no effect on osteochondrosis. However, Goedegebuure et al. (1980) found that pigs fed *ad libitum* had severer joint lesions than pigs fed by restricted feeding. Because there is inconsistency among these results, it seems that the effect of the energy level of the diet on leg traits and osteochondrosis remains unclear. One possible reason for this inconsistency may be environmental differences in terms of the place where pigs were measured or the time period when the measurements were made.

Vitamins

Several researchers have investigated the effects of dietary vitamin levels on leg weakness. Vitamin A supplementation was found to have no effect on osteochondrosis (Reiland, 1978a; Blair et al., 1992). Reiland (1978a) reported that vitamin D supplementation has no effect on osteochondrosis. Grøndalen (1974a) found that vitamin E supplementation has no effect on osteochondrosis. Neilsen and Vinther (1982) have suggested that vitamin C supplementation might prevent leg weakness or osteochondrosis. However, Nakano et al. (1983) found that vitamin C supplementation had no effect on

joint cartilage condition. Webb et al. (1984) have shown that biotin supplementation can improve pig hoof strength. However, several reports have indicated that biotin supplementation has no effect on limb soundness in pigs (Grandhi and Strain, 1980; Bane et al., 1980; Hamilton and Veum, 1984).

Other nutrients

Van der Wal (1986) has reported that a significant reduction of leg weakness occurred in pigs fed a diet in which 0.3% NaCl was replaced with 0.43% $NaHCO_3$. However, Ernst et al. (1990) found that there was no significant improvement in clinical signs of leg weakness upon supplementation of the diet with $NaHCO_3$.

OTHER FACTORS

Elliot and Doige (1973) have shown that pigs penned individually had a significantly reduced cross-sectional area of cortical bone and significantly decreased breaking strength of bone in comparison with pigs confined in a group. Fredeen and Sather (1978) reported that limb joint cartilage damage was greater in the case of pigs confined individually than in the case of pigs confined in groups. Sather and Fredeen (1982) found that individually confined barrow pigs had a high frequency of leg weakness and a high frequency of joint cartilage lesions, although no significant difference was found in female pigs. The authors suggested that a possible reason for these differences is the difference in the degree of activity between pigs under different confinement conditions; pigs housed in groups are generally more active than those housed individually.

Additionally, it was suggested that local overloading can cause osteochondral lesions (Grøndalen and Grøndalen, 1974; Nakano et al., 1979).

CONCLUSION

Although leg weakness has been extensively studied by many researchers, the exact cause and etiology of leg weakness and osteochondrosis still remain unclear. It could be that there are multiple causes of leg weakness.

It has been shown that leg weakness and osteochondrosis are heritable in pigs, from a low to intermediate extent. Since there are genetic factors related to leg weakness, improvement of leg soundness and reduction of osteochondrosis may be feasible in pigs. In order to improve leg soundness in pig breeding programs, precise estimation of the associated genetic parameters is required.

Several environmental factors such as the feeding regime and the confinement conditions might be associated with leg weakness, and improvement of environmental factors may reduce leg weakness problems.

Additionally, it seems that a more sophisticated and objective scoring method for leg weakness or osteochondrosis is necessary for evaluating the symptoms more precisely.

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