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Turkey Sire Effects on Embryonic Survival and Physiology[1]

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Abstract: Sire effects on turkey embryonic survival and growth are not well understood. In avian species, the sire may play only a minor role in embryonic growth as dam effects, mediated through physical and functional qualities of eggs, are thought to be the main determinants. Very little is known of separate dam and sire influences on embryonic survival. The hypothesis was proposed that sires from lines with different BW and embryonic survival rates when mated to an unrelated dam line would produce embryos with different survival, growth and metabolism. Sires from a line with light BW but good embryonic survival (LBW) or sires from a heavy BW line and poor embryonic survival (HBW) were mated to dams of the same unrelated line. Sires from the dam line were included as a control group (Controls). Hens were randomly assigned to sires and inseminated identically at weekly intervals with semen from the assigned sire line. Eight biweekly settings of eggs were placed into incubators to test embryonic survival rates among the sire lines. Tissues were sampled at designated intervals during the experiment to assess the physiological basis for embryonic survival. Contrary to our hypothesis, embryos from the HBW sire line had the best survival compared to LBW and Control. Although LBW poults were from smaller sires, they weighed more than HBW poults. During development LBW sire embryos stored greater amounts of glycogen and lactate in liver and muscle. No effects were seen in cardiac tissue. BW differences were related to greater yolk, but the differences in organ weights and metabolism were clearly related to sire. Thus, sire DNA may direct organ growth and function and influence embryonic survival.

Key words: Sire, dam, embryonic survival, intermediary metabolism

Introduction

Imprinted genes were noted first about 10 years ago (Haig, 1993). They violate Mendelian rules of inheritance because only one of the genes from the parent has input and the matching gene from the other parent is silenced. In embryonic human growth it has been proposed that the predominant direction of sire DNA is toward growth but that of dam DNA is growth-limiting to protect the life of the mother (Haig, 1993). Thus, a balance between input and silencing is created for the human fetus to grow, survive and spare the life of the mother. The trade-off between egg size and number is a central tenet of avian ecological theory and only a limited number and size of eggs may be created to spare the life of the mother (Bernardo, 1996). Comparisons have been made among pedigreed sibling turkey sires, and heavier sires produced heavier hatchlings with no changes in egg weight or eggshell conductance that are the major components of the maternal growth influence (Christensen *et al*., 2000b). In addition to the heavier BW at hatching, poults from heavier sires also exhibited altered metabolism (Christensen *et al*., 2000a,b). Thus, sire effects on embryonic growth exist in turkeys, but very little is understood about them.

Embryonic viability decreases with selection of turkeys for heavier posthatch BW while lines selected for increased egg production have reduced BW and good embryonic viability (Nestor and Noble, 1995). Embryonic mortality in the selected lines occurs predominantly late in development (Christensen *et al*., 1993) at a time known as the plateau in oxygen consumption (Rahn, 1981). Therefore, the hypothesis was proposed that sires of different BW and embryonic survival rates mated to hens of an unrelated line might exhibit sire effects for embryonic viability, growth and metabolism.

Materials and Methods

Sires from two lines of turkeys with different BW and embryonic survival rates were grown to sexual maturity (Christensen and Nestor, 1994). Dams and sires from a commercial line of turkeys noted for its high hatchability were grown simultaneously under the same conditions as the sires. The sires exhibiting high hatchability and light BW were from the Egg line (LBW) whereas those exhibiting low hatchability and heavy BW were from the F line (HBW) (Nestor and Noble, 1995). Sires from the commercial line served as a control (Control). Sires from the LBW line at 30 wk of age

[1]The mention of trade names in this publication does not imply endorsement of the products mentioned nor criticism of similar products not mentioned.

Table 1: Hatchability and frequency of embryonic mortality (% of fertile eggs) during incubation of turkey embryos from sires of different lines

Sire line[1]	Hatchability	Week 1 mortality	Week 4 mortality	Pipping mortality
LBW	71.4b	5.8	6.6[a]	14.6[a]
HBW	81.7[a]	6.0	4.2[b]	6.2[c]
Control	73.7[b]	7.1	6.3[a]	11.3[b]
$\sqrt{}$ ± SEM	75.6±0.9	6.3±0.5	5.7±0.5	10.7±0.7
Sire	0.0001	NS	0.05	0.0001

[1]LBW = low body weight line; HBW = heavy body weight line; Control = commercial line of turkey breeders.

Table 2: Time (h) to attain and time (h) remaining at a stage of development in embryos and poults produced by sires of different lines

	Stage of development				
	Internal pipping		External pipping		Hatching
Sire line[1]	Time to attain	Time at stage	Time to attain	Time at stage	Time to attain
LBW	602	15.5[b]	616[b]	21.9[b]	637[b]
HBW	604	18.4[a]	619[a]	25.3[a]	647[a]
Control	601	17.7[a]	616[b]	26.3[a]	643[a]
$\sqrt{}$ ± SEM	602±1	17.3±0.8	618±1	24.1±1.1	642±1
Probability	NS	0.02	0.06	0.09	0.03

[1]LBW = low body weight line; HBW = heavy body weight line; Control = commercial line of turkey breeders.

weighed less than half (10.9 kg ± 0.1) that of HBW (27.9 kg ± 0.1) or Control (30.7 kg ± 0.1) lines. All sires were photostimulated at 26 wk of age (14 h of light/day) to initiate semen production, and the dams were photostimulated (15.5 h of light/day) at 30 wk of age to produce eggs. Each sire line (12 sires per line) was randomly assigned to 8 pens with 6 hens per pen. Artificial inseminations were performed weekly using 200 million viable cells per insemination dose. Fertilized eggs were collected 5 times daily, sanitized and placed in a refrigerated room (12.5 C and 75% RH). Eggs were accumulated for eight 14 d periods (8 Trials) prior to setting. Eggs were weighed prior to setting and incubated using standard procedures described previously (Christensen *et al.*, 2000a). Following 28 d of incubation, all nonhatching eggs were opened and examined for embryonic development, and true fertility and estimated time of death were determined. From these data, hatchability of fertile eggs was computed.

Beginning at 25 d of incubation, the times that eggs hatched were observed through the hatching process. Eggs were examined at 6 h intervals with a candling light and categorized according to their morphology at each time of examination (Christensen *et al.*, 2001). The morphological stages were internal pipping (IP), external pipping (EP) and hatching (Christensen *et al.*, 2001). The time that each embryo attained a stage of development as well as the time that the embryo remained at that stage were calculated. The data from staging were used to indicate the sampling time for each treatment. When half of a treatment group attained a stage, in Trials 2, 5 and 7, randomly selected individuals were sampled. Eight embryos or poults were selected at each stage from each sire treatment per trial for a total of 72 samples for the entire experiment. The plateau stage in oxygen consumption has significant effects on cardiac hepatic and intestinal tissues (Christensen *et al.*, 1993), therefore, heart, liver, intestine and blood samples were collected from each and weighed immediately (nearest 0.01 g). Heart and liver were placed immediately into cold 7% perchloric acid preparatory to assaying for glycogen and lactate. Jejunal tissue was placed into cold saline and frozen immediately for measuring maltase and alkaline phosphatase (ALP). Blood was collected into a vial containing 10 mg of EDTA then placed into an ice bath prior to centrifugation at 700xg for 15 min. The plasma was decanted and frozen (-22 ^{o}C) until assayed for glucose, lactate dehydrogenase (LDH) and creatine kinase (CK). All assays were performed as described previously (Christensen *et al.*, 2003).

Statistical analysis was performed using SAS Institute (1998) software. The embryonic viability data and the egg functional quality data were analyzed using the GLM procedure of SAS with two main effects, sire treatment (LBW, HBW and Control) and wk of lay (8 biweekly sets of eggs) arranged as a 3 x 8 factorial. The tissue data were analyzed using the sire treatments and three stages of development EP, hatching and 3 d posthatching (3 d) as the main effects in a 3 x 3 factorial arrangement. Trial was considered a fixed variable in the tissue analysis, but no interactions due to trial were noted so the data were pooled across trials for presentation. Means determined to differ significantly

Table 3: Body weights (g) with and without yolk and yolk weights of embryos and poults produced by sires of different lines

	Stage of development[2]			
Sire line[1]	EP	Hatching	3-d	√
Body weight with yolk				
LBW	70.8[d]	67.0[c]	85.1[b]	
HBW	70.8[d]	65.3[d]	92.1[a]	
Control	71.1[d]	66.2[cd]	92.3[a]	
√				
√ ± SEM	74.6 ± 0.6			
Probabilities				
Sire	NS			
Day	0.0001			
Sire x Day	0.0001			
Body weight without yolk				
LBW	57.3	53.3	81.5	64.0
HBW	55.5	55.4	85.6	65.5
Control	54.7	55.0	88.1	65.9
√	55.8[b]	54.6[b]	85.1[a]	
√ ± SEM	64.2 ± 0.5			
Probabilities				
Sire	NS			
Day	0.0001			
Sire x Day	NS			
Yolk weight				
LBW	13.5[b]	12.1[b]	2.3[d]	
HBW	15.2[ab]	9.4[c]	2.2[d]	
Control	16.5[a]	12.2[b]	1.9[d]	
√ ± SEM	9.5 ± 0.3			
Probabilities				
Sire	NS			
Day	0.0001			
Sire x Day	0.05			

[1]LBW = low body weight line; HBW = heavy body weight line; Control = commercial line of turkey breeders.
[2]EP = externally pipped embryo at approximately 27 d of incubation; Hatched = hatched poult at 28 d of incubation 3d = 3 days posthatching.

were separated using the least square mean procedure (SAS Inst., 1998). Probability level of $P < 0.05$ was considered significant.

Results

Hatchability was highest in the HBW sire treatment compared to the LBW or Control (Table 1). Embryos from the HBW sires survived better because fewer embryos died during wk 4 of incubation than in the LBW or the Control lines. More HBW embryos survived pipping than LBW with Controls being intermediate.

Sires effects were noted in the times of hatching (Table 2). Embryos from all sires attained internal pipping (IP) at 602 h of incubation, but HBW and Control embryos remained at IP for approximately 2 h longer than LBW. At external pipping (EP) LBW embryos remained at EP between 4 to 5 fewer h than did those from HBW or Controls. However, hatching times for LBW were nearly 10 h earlier than those of HBW or Controls.

The interdependence of egg weight, conductance and the length of the incubation period has been suggested (Ar and Rahn, 1978; Christensen *et al.*, 2003). The interdependence was measured mathematically by a constant computed as the product of eggshell conductance (mg H_2O/d/mmHg) and incubation period (h) divided by the egg weight (g). Neither egg weight nor eggshell conductance differed among the sire treatments, but because of the difference in hatching times, HBW (5.32) and Control (5.36) sires exhibited lower constants than LBW (5.56).

At EP embryonic BW did not differ among sire lines (Table 3). LBW sires produced heavier poults at hatching than did HBW, but neither differed from the Control. By 3 d posthatching, poults from LBW sires weighed less than did those from either HBW or Control. Embryonic BW without yolk examined at all days differed

Table 4: Organ weights (mg) of embryos and poults produced by sires of different lines

	Stage of development[2]			
Sire line[1]	EP	Hatching	3-d	√
Heart weight				
LBW	329	377	559	422
HBW	304	391	584	426
Control	298	374	564	412
√	310[c]	381[b]	569[a]	
√ ± SEM	420±9			
Probabilities				
Sire	NS			
Day	0.0001			
Sire x Day	NS			
Liver weight (mg)				
LBW	1,189[e]	1,498[d]	3,395[b]	
HBW	1,060[f]	1,585[c]	3,445[a]	
Control	1,085[f]	1,631[c]	3,365[b]	
√ ± SEM	2,028±21			
Probabilities				
Sire	NS			
Day	0.0001			
Sire x Day	0.04			
Skeletal muscle (mg)				
LBW	2,728	2,840	3,920	3,163[b]
HBW	2,691	3,185	4,390	3,422[a]
Control	2,726	3,038	4,333	3,365[a]
√	2,715[c]	3.021[b]	4,214[a]	
√ ± SEM	3,317±39			
Probabilities				
Sire	0.009			
Day	0.0001			
Sire x Day	NS			
Jejunum weight (mg)				
LBW	305[e]	416[d]	1,616[b]	
HBW	309[e]	548[c]	2,076[a]	
Control	286[e]	492[c]	1,971[a]	
√ ± SEM	891±16			
Probabilities				
Sire	0.0001			
Day	0.0001			
Sire x Day	0.02			
Jejunum length (cm)				
LBW	11.1	12.8	20.8	14.9[b]
HBW	11.7	15.0	22.9	16.5[a]
Control	11.5	14.4	22.2	16.1[a]
√	11.4[c]	14.1[b]	22.2[a]	
√ ± SEM	15.8±0.2			
Probabilities				
Sire	0.001			
Day	0.0001			
Sire x Day	NS			

[1]LBW = low body weight line; HBW = heavy body weight line; Control = commercial line of turkey breeders.
[2]EP = externally pipped embryo at approximately 27 d of incubation; Hatched = hatched poult at 28 d of incubation 3d = 3 days posthatching.

Table 5: Hepatic glycogen and lactate concentrations (mg/ g of wet tissue mass) and their ratio in embryos and poults produced by sires of different lines

	Stage of development[2]			
Sire line[1]	EP	Hatched	3-d	√
Glycogen				
LBW	14.0	8.3	42.6	21.6[a]
HBW	13.0	5.3	32.7	17.0[b]
Control	8.8	8.9	40.8	19.5[ab]
√	11.9b	7.5[c]	38.7[a]	
√ ± SEM	19.4 ± 0.9			
Probabilities				
Sire	0.05			
Day	0.0001			
Sire x Day	NS			
Lactate				
LBW	0.29[c]	0.29[c]	0.37[b]	
HBW	0.26c	0.29[c]	0.44[a]	
Control	0.31c	0.28[c]	0.42[a]	
√ ± SEM	0.33 ± 0.01			
Probabilities				
Sire	NS			
Day	0.0001			
Sire x Day	0.04			
Ratio				
LBW	52.0c	29.1[d]	113.8[a]	
HBW	51.2c	18.2[e]	76.8[c]	
Control	31.0d	32.5[d]	95.0[b]	
√ ± SEM	55.5 ± 2.0			
Probabilities				
Sire	0.03			
Day	0.0001			
Sire x Day	0.02			

[1]LBW = low body weight line; HBW = heavy body weight line; Control = commercial line of turkey breeders.
[2]EP = externally pipped embryo at approximately 27 d of incubation; Hatched = hatched poult at 28 d of incubation 3d = 3 days posthatching.

only by day of incubation. Therefore, different BW with and without yolksac must have been due to residual yolk. A sire by day of age interaction affected residual yolk weights. The HBW and Control sire embryos utilized less yolk attaining EP, but at hatching LBW and Control had more yolk than did the HBW. No differences in residual yolk were noted by 3 d posthatching.

Data for organ weights were analyzed statistically as both absolute and relative weights. The analyses were similar so only absolute weights are presented. Embryonic heart weights did not differ among sire treatments nor did they differ posthatching as the poults grew (Table 4), but significant sire by age interactions affected both liver and jejunum weights. At EP, LBW embryo liver weighed more than either HBW or Control, but at hatching, livers from LBW weighed less than HBW or Control. At 3 d posthatching, livers from HBW weighed more than either LBW or Control. Skeletal muscles weighed less in poults and embryos from LBW than from HBW or Control poults. Jejunum weights of poults did not differ at EP, but at hatching and 3 d posthatching jejuna from LBW weighed less than either HBW or Control. Jejuna from LBW were also significantly shorter than those from HBW or Control. Jejunum maltase and alkaline phosphatase activities increased from EP to 3 d posthatching, but the activities did not differ among the sire treatments (data not shown).

Sire did not affect cardiac glycogen or lactate concentrations (data not shown), but HBW sires consistently depressed hepatic glycogen concentrations compared to LBW or Control (Table 5 and 6). Conversely, hepatic lactate concentrations were depressed in the LBW compared to other sire lines. Thus, the ratio of hepatic glycogen to lactate was elevated in LBW and Control sire treatments compared to HBW. Skeletal muscle glycogen was not affected by sires, but poults from LBW sires had elevated lactate in skeletal muscle compared to HBW and Controls.

Table 6: Skeletal muscle glycogen and lactate concentrations (mg/g of wet tissue mass) and their ratio in embryos and poults produced by sires of different lines

	Stage of development[2]			
Sire line[1]	EP	Hatched	3-d	√
Glycogen				
LBW	1.02	0.75	1.21	0.99
HBW	1.10	0.72	1.00	0.94
Control	1.04	0.76	1.09	0.96
√	1.05[a]	0.75[b]	1.10[a]	
√ ± SEM	0.96±0.03			
Probabilities				
Sire	NS			
Day	0.0001			
Sire x Day	NS			
Lactate				
LBW	0.84	0.95	1.53	1.11[a]
HBW	0.79	0.91	1.44	1.04[b]
Control	0.78	0.90	1.50	1.06[b]
√	0.80[c]	0.92[b]	1.49[a]	
√ ± SEM	1.07±0.01			
Probabilities				
Sire	0.01			
Day	0.0001			
Sire x Day	NS			
Ratio				
LBW	1.22	0.79	0.77	0.93
HBW	1.41	0.80	0.69	0.97
Control	1.33	0.84	0.73	0.97
√	1.32[a]	0.81[b]	0.73[b]	
√ ± SEM	0.95±0.02			
Probabilities				
Sire	NS			
Day	0.0001			
Sire x Day	NS			

[1]LBW = low body weight line; HBW = heavy body weight line; Control = commercial line of turkey breeders.
[2]EP = externally pipped embryo at approximately 27 d of incubation; Hatched = hatched poult at 28 d of incubation. 3d = 3 days posthatching.

Mean blood glucose concentrations of embryos of the LBW sire treatment were elevated compared to the remaining sire lines, and embryonic blood activity of lactate dehydrogenase (LDH) in the LBW sire line was depressed compared to HBW and Controls (Table 7). No differences were detected among sire lines for CK although its concentration increased through the hatching process.

Discussion

The HBW sire line was hypothesized to yield poor embryonic survival because they had been selected for heavier BW (Nestor and Noble, 1995), but HBW sires unexpectedly produced better embryonic livability in the current study than did LBW sires. Improved livability was noted primarily during the plateau stage in oxygen consumption (Rahn, 1981). Thus, we may conclude that selection of sires for heavier BW may influence embryonic survival positively.

The LBW poults at hatching weighed more than the HBW, which was a second unexpected result of the current study. Although the data indicated poult BW with residual yolk was opposite of that seen in the sires, the differences were related to amounts of yolk. Poults from the HBW line utilized more yolk during development than did poults from the LBW line. Thus, hatchlings from LBW weighed more only because they had more residual yolk. No differences existed in tissue mass, therefore, greater yolk utilization suggests that metabolism may be associated with sire and affect embryonic survival. In embryos from HBW sires, a greater amount of the energy for growth was derived from aerobic metabolism (lipids) than in poults from LBW sires that relied more on anaerobic (glycogen or gluconeogenesis). Different

Table 7: Blood plasma glucose concentration and lactate dehydrogenase and creatine kinase activities in embryos and poults produced by sires of different lines

	Stage of development[2]			
Sire line[1]	EP	Hatched	3-d	√
Blood Glucose (mg/dL)				
LBW	286	311	343	313[a]
HBW	268	298	337	300[b]
Control	274	306	325	302[b]
√	276[c]	304[b]	335[a]	
√ ± SEM	305±2			
Probabilities				
Sire	0.03			
Day	0.0001			
Sire x Day	NS			
Lactate Dehydrogenase Activity(U/L)				
LBW	559	690[b]	486	578[b]
HBW	664	812[a]	580	685[a]
Control	621	858[a]	487	655[a]
	615[b]	786[a]	518[c]	
√ ± SEM	640±14			
Probabilities				
Sire	0.02			
Day	0.0001			
Sire x Day	NS			
Creatine Kinase Activity (U/L)				
LBW	661	990	1,273	975
HBW	628	798	1,107	845
Control	567	858	1,282	902
√	619[c]	882[b]	1,221[a]	
√ ± SEM	907±31			
Probabilities				
Sire	NS			
Day	0.0001			
Sire x Day	NS			

[1]LBW = low body weight line; HBW = heavy body weight line; Control = commercial line of turkey breeders.
[2]EP = externally pipped embryo at approximately 27 d of incubation; Hatched = hatched poult at 28 d of incubation. 3d = 3 days posthatching.

metabolisms may also have caused LBW poults to hatch 10 h earlier. Thus, sire had no effect on embryonic tissue mass, but changes occurred in metabolism and yolk utilization that may have affected livability.

The predominant direction of human sire DNA (androgenome) is toward growth, but dam DNA (gynogenome) is growth-limiting to protect the life of the mother in a process called imprinting (Haig, 1993). The trade-off between egg size and number is a central tenet of ecological theory to spare the life of the avian mother (Smith and Fretwell, 1974; Bernardo 1996). Major differences in growth of turkey embryos may be mediated through the gynogenome via egg size and eggshell properties controlling growth although persistent minor differences have been shown within sires (Christensen *et al.*, 2000a,b). Embryonic growth is a highly conserved trait across avian species (Byerly, 1932; Byerly *et al.*, 1938; Ricklefs, 1987). Embryonic growth curves from crosses produced from reciprocal mating of lines of different BW were nearly identical to those of the dams and indicated little influence of the sire (Ricklefs, 1987). Therefore, based on data from the current study it is speculated that the major effect on turkey embryonic BW is mediated through the gynogenome rather than the androgenome as in humans (Haig, 1993).

The weights of selected organs differed among the sires in unique ways despite the lack of sire effects on BW. Embryos from LBW sires reduced hepatic, jejuna and skeletal muscle tissue weights but not cardiac compared to HBW sires. The reduction in growth or function in one or all of these physiological systems may reduce survival of LBW embryos. Therefore, we may conclude that the androgenome in turkeys directs

differential embryonic organ growth thereby possibly affecting survival.

Growth curves for embryonic organs are highly conserved biological traits (Ricklefs and Starck, 1998), but little is known of gender-related differences. Schmalhausen (1930) hypothesized that embryos may regulate organs physiologically between growth and function when presented with life-threatening situations. Regulation between growth and function may explain the current study because the increased yolk at hatching and reduced liver and skeletal muscle weights of LBW sire embryos were accompanied by an enhanced ability to accumulate glycogen (or increase the glycogen to lactate ratio in vital tissues).

The plateau stage in oxygen consumption presents a paradox for turkey embryos (Dietz *et al.*, 1998). Embryos must continue to grow and function without oxygen and without ventilating carbon dioxide. If embryos were suffering hypoxia, glycogen concentrations would be depressed, and lactate concentrations would be elevated (Garcia *et al.*, 1987). Embryos and poults from LBW sires during the plateau in oxygen consumption had persistently greater amounts of hepatic glycogen than lactate and had greater lactate concentrations in skeletal muscle than did HBW indicating that they were adjusting to hypoxia during the plateau. Poults from LBW sires also elevated plasma glucose concentrations and depressed LDH activity compared to HBW. Both LDH and CK are elevated under hypoxia and can be indicative of cardiac muscle damage. (Pearce and Brown, 1971). LDH plays a major role in gluconeogenesis in avian embryos as it converts pyruvate to glucose-6-phosphatase in the Cori cycle during recycling of lactate to glucose (Pearce and Brown, 1971). Avian cardiac tissue lacks enzymes to recycle lactate so the liver performs the process. If the hepatic tissue Cori cycle enzymes were not functional, it would have been evident in depressed cardiac tissue glycogen to lactate ratios, but that was not the case and the embryos with the greatest mortality rate had the greatest hepatic glycogen to lactate ratios. Thus, it is clear that although sire affects the metabolism and growth of embryonic tissues, the ability to create and store glycogen is not impaired (Lilja and Olsson, 1987; Christensen *et al.*, 2000a). The possibility also exists that embryos from some sires are unable to obtain glycogen from selected tissues, but that speculation remains to be investigated.

The HBW sires fathered poults with heavier and longer jejuna than LBW, but no differences were detected in either specific or total maltase and ALP activities. Intestinal growth requires large amounts of energy and other resources at the late stage of incubation (Fan *et al.*, 1997) so these data may indicate the LBW poults are conserving growth of intestinal tissue to allow growth and function of other tissues.

The data from the current study provide evidence that genetic imprinting (Haig, 1993) may be evident in turkeys and that HBW sires improved embryonic livability without affecting embryonic body weight. The improved livability may occur through improved yolk use, heavier liver and skeletal muscle weights and the abilities of selected tissues to accumulate glycogen and recycle lactate compared to LBW sires. These actions lengthened the incubation period of HBW poults by 10 h longer than LBW poults. Thus, genes from a heavier sire can actually improve embryonic livability under the conditions of the current study. These results are in contrast to the effects seen previously when HBW sires were mated to hens of the same heavy line (Nestor and Noble, 1995).

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