

Physiological Factors Associated with Weak Neonatal Poults¹ (*Meleagris gallopavo*)

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Abstract: Management related deaths in turkey hatchlings remain a problem for turkey growers. The etiologies of such deaths also remain obscure. The hypothesis proposed by the current study was that weak poults identified by their characteristic flip-over behavior would differ physiologically from normal poults in a way that interferes with development of critical systems such as the cardiovascular and nervous system. Eight groups of 500 poults (from a 20-wk laying cycle) of the same breeder flock were examined. Each placement was in the same brooder house operated at identical conditions. Observations of “flip-over” poults were made at 6-h intervals for the initial wk of age. Identified poults (n = 12) were sacrificed and sampled immediately. Flip-over poults differed from controls only in depressed heart weight and plasma thyroid hormone concentrations. The identified poults did not differ from controls in body weight, liver weight, yolk weight, blood glucose or organ glycogen concentrations. In a second trial, reduced heart weights were noted along with reduced capability to maintain cardiac glycogen concentrations. The reduced heart weight or function may result in failure to provide nutrients for proper brain function. The data suggest that management to aid weak neonatal turkey poult deaths may need to focus on conditions fostering heart growth and function.

Key words: Flip-over, weak poults, heart, thyroid

Introduction

The growth and livability of turkey poults are continuing concerns for commercial turkey growers. When infectious diseases are discounted, the major mortalities during brooding and rearing are weak poults and “starve-outs” (Minnesota Agricultural Statistics Service, 1986). As many as 10% of the poults may suffer some sort of problem due to management during the initial stages of life. Barnes (1994a,b) has estimated that management-related problems during brooding and growing may account for mortality greater than 6% and growth depressions of 10 to 30%.

Poult mortality is a difficult variable to study because it occurs at less than a 5% rate (Ferket, 2002), and it is nearly impossible to identify potentially weak poults at the time of hatching. Therefore, conditions to delineate mortality are difficult to recreate in the laboratory. Early poult flip-overs have been characterized by weakened poults falling on their back and being unable to right themselves without assistance (Noble *et al.*, 1999). Poults that flip over more than once appear to lack neurological control, lying on their backs with their legs paddling. If handled, the affected poult moved its head laterally and after several seconds appeared to go to sleep. When placed on litter, the poults have no sense of balance and fall to one side or the another and start paddling their

feet. Poults that flip over have also been observed to chirp loudly. No bacterial agents were present and the concentrations of brain neurochemicals were unaffected (Noble *et al.*, 1999).

The purpose of this study was to characterize further the physiology of poults that flip over compared to normal controls to gain insight into possible causes of one category of weak poults. The hypothesis tested was that weak poults would differ from normal controls in intermediate energy metabolism providing glucose for heart and brain tissue or in thyroid function that is necessary for normal neural and cardiovascular development and function (Nobikuni *et al.*, 1989; Fisher, 1999).

Materials and Methods

Experiment 1: The hypothesis was tested in Experiment 1 that flip-over poults would differ from normal poults in energy metabolism providing nutrients to tissues or in thyroid function. Poults from a genetic line² of commercial turkeys were hatched using standard conditions (Christensen and Donaldson, 1992) at the NCSU Research Laboratory and placed in a curtain-sided house. Eight separate trials of 500 poults each were conducted over a time that spanned one complete breeder cycle for the hens. The house was fan-ventilated with gas

¹The mention of trade names in this publication does not imply endorsement of the products mentioned nor criticism of similar products not mentioned. ²British United Turkeys, PO Box 727, Lewisburg, WV 24901
Abbreviation Key: E-line = line of turkeys that is susceptible to flip-over; EDTA = ethylene diamine tetra acetate; L = Line; IP = incubation period; D = Diet

Table 1: Incubator temperature and humidity profiles used to create “Short and “Control” incubation periods in Experiment 2

Incubation period	Days of incubation	Dry bulb temperature (°C)	Relative humidity (%)
Short	1-7	38.0	50
	8-14	37.8	52
	15-24	37.5	54
	25-28	37.0	75
Control	1-7	37.5	54
	8-14	37.5	54
	15-24	37.5	54
	25-28	37.0	75

heaters. House temperature was maintained at approximately 37.6 °C by using thermostats to control both the heater and curtains. The poults (approximately 500 per hatch, 50 per pen), were placed into pens with 7-8 cm of pine shavings for litter. Each pen contained 320 linear cm of feeder and 165 linear cm of drinker space. Plasson drinkers were supplemented with 2 water jars for the first 7 days of age. Feed was placed in the feeders and on egg flats to assist the poults in finding feed and water. Eight trials were conducted at biweekly intervals. The poults were observed at 6 h intervals for the initial week of age. When flip-over poults were discovered, they were sampled immediately and pen-mate poults that appeared normal were selected randomly at the same time and sampled identically. The house was sanitized but litter was not removed between trials. The selected poults were taken to the laboratory for sampling of body weight (with and without yolk sac) as well as yolk sac weight (Christensen and Donaldson, 1992).

A blood sample was obtained by decapitation and heart, liver and jejunum tissues were dissected quickly and weighed (nearest 0.01 mg). The yolk was removed and weighed, the digestive tract was opened and the presence or absence of feed was confirmed, and the contents were evacuated by flushing with saline before weighing. Following weighing, each heart and liver was placed in cold 7% perchloric acid and evaluated for glycogen content using iodine binding (Dreiling *et al.*, 1987). The blood with EDTA for an anticoagulant was centrifuged (700 x g) under refrigeration (4 °C) for 15 min and the plasma was decanted and assayed for thyroid hormones (Christensen and Davis, 2001) and plasma glucose concentration (Christensen and Donaldson, 1992). A total of 24 poults was sampled (12 flip-overs and 12 controls) over the course of the experiment. The means of each group were compared using a one way analysis of variance of SAS (SAS Inst., 1998). Means were separated by the least square means procedure where appropriate. Significance was based on $P \leq 0.05$.

Experiment 2: Experiment 2 shortened the incubation period, increased the amount of carbohydrate in the diet and evaluated a weak poult susceptible genetic line. Based on results of Experiment 1, the hypothesis tested in Experiment 2 was that weak poults may be the result of carbohydrate deprivation or adequate developmental time needed for proper heart or liver development and function to provide energy for adequate central nervous system development. Shorter incubation periods increased mortality in turkeys (Christensen *et al.*, 2001) and reduced embryonic heart and liver weights and metabolism.

Poults from the same commercial line of turkeys² examined in Experiment 1 (COM) and from the flip-over susceptible line (SUS) (Noble *et al.*, 1999) were included in Experiment 2. Additionally, half of the eggs in Experiment 2 was incubated using a second incubator for only the initial two weeks of incubation. The procedure for incubating these eggs is given in Table 1. Eggs from the shorter incubation period were exposed to 38.0 °C for week 1 and 37.8 °C for week 2. At the beginning of week 3, the eggs were again randomized and returned to the same machine as the control (37.5 °C) for the remainder of development. The treatment shortened the developmental period by approximately 6 h compared to controls. At placement, half of the poults were given a diet containing 50% carbohydrate while the remainder was given a standard diet containing approximately 15% carbohydrate (Table 2). The diets were available *ad libitum* throughout the experiment. Prior data indicated that all poults were eating at 72 h post-hatching (Nicholson, 1992). Therefore, beginning at 72-h post hatching and continuing at 96 and 120 h, four poults from each treatment group combination were selected randomly for sampling. Tissue sampling was done similarly as in Experiment 1.

A total sample size of 96 was used in the statistical analysis ($n = 16$ per treatment combination). The data were analyzed as a completely random design arranged factorially in 2 levels of genetic line (COM and SUS) by 2 levels of incubation period (Short and Control) by 2 levels of diet (Carbohydrate versus Control) and 3 levels of sampling time (72, 96 or 120 h). All possible interactions were tested for significance (SAS Inst., 1998). All means determined to differ significantly were separated by the least square means procedure based on a probability of $P < 0.05$ unless otherwise stated.

Results

Experiment 1: Feed was present in the digestive tract of all birds sampled regardless of their classification as flip-over or control. Neither body, yolk, liver nor jejunum weights differed between flip-over and control poults (Table 3). However, heart weights of the control poults were significantly heavier than those of the flip-over poults. Neither cardiac nor hepatic glycogen concentrations differed between control and flip-over poults (Data not shown). Both thyroxine and

Table 2: Control and carbohydrate diets fed to poults in Experiment 2*

Ingredient	Control diet (%)	Carbohydrate diet (%)
Corn starch	6.9	46.1
Soybean Oil Meal	71.5	41.4
DL-Methionine	2.8	1.8
Dicalcium phosphate	2.2	2.2
Limestone	1.4	1.5
Salt	0.4	0.4
Solka flox	5.2	5.2
Vitamin premix ¹	2.0	2.0
Trace mineral premix ²	1.0	1.0
Cottonseed oil	10.0	1.0
Calculated analysis ³		
Crude protein (%)	35.0	20.2
Metabolizable energy (kcal/kg)	2887	2840
C/P ratio (kcal,kg/%protein)	82.5	140.6

¹Provided the following/kg feed irrespective of the chemical form: vitamin A palmitate 4400 IU; cholecalciferol, 900 ICU; DL-"-tocopherol acetate, 22 IU; menadione, 1.1 mg; thiamine mononitrate, 2.75 mg; riboflavin, 5.5 mg; calcium pantothenate, 154 mg; niacin, 70 mg; pyridoxine HCl, 4.5 mg; biotin, 0.33 mg; choline chloride, 2200 mg; Folicin, 1 mg; vitamin B₁₂, 19.8 µg.

²Supplied in mg/kg feed: Cu (as copper sulfate) 8; I (as potassium iodide) 0.4; Fe (as Ferric citrate) 110; Mn (manganous sulfate) 77; Se (as sodium selenite) 0.2; and Zn (as zinc carbonate) 60.

³From National Research Council, Nutrient Requirements of Poultry, 9th ed., revised. U. S. Academy of Science, 1984.

*Based on 100% available carbohydrate in cornstarch and 12% available carbohydrate in soybean meal (Aldolph and Kao, 1934).

triiodothyronine concentrations were significantly depressed in flip-over compared to controls poults (Table 4), but their ratios did not differ.

Experiment 2: Every variable measured in the experiment displayed a significant 4-way interaction (genetics by incubation period by diet by age at sampling). To better understand the complex 4-way interaction, data will be presented as three or two interactions within each of the sampling times (Table 5). At 72 h, a significant line by incubation period interaction for BW was observed. The COM poults when hatched at the shorter incubation period were significantly heavier than those under the control regimen with no differences among SUS poults. Identical differences were seen when weights with yolk sacs were compared (data not shown). At 96 h post-hatching a significant line by incubation period by diet 3 way interaction for BW was noted. COM poults were affected with no effect on the SUS poults. COM poults given the control incubation period and fed the carbohydrate diet were heavier than those fed the control

diet. Conversely, COM poults hatching from short incubation periods fed control diets were significantly heavier than those from those given the carbohydrate diet. At 120 h, COM poults given the carbohydrate diet were significantly heavier than those given the control diet, but no such response was evident among SUS poults. Residual yolk weights among the treatments did not differ at any time post-hatching (data not shown).

Heart weight paralleled body weight at all three sampling times (Table 6). At 72 h poults from the COM line with short incubation periods exhibited significantly greater heart weight than did those with control incubation periods but no such difference was evident for the SUS line. At 120 h a significant line by diet interaction was noted as COM poults fed the carbohydrate diet had heavier hearts than controls with no corresponding effect in SUS poults. At 72 h COM poults with short incubation periods increased liver weight compared to control incubation period poults but SUS poults did not (Table 7). Poults at 96 h from short incubation periods fed carbohydrate had smaller livers than controls fed carbohydrate. Short incubation period poults on control feed as well as control incubation period poults fed the low carbohydrate had intermediate liver weights. At 120 h post-hatching, poults fed the high carbohydrate had heavier livers regardless of genetic origin.

Cardiac glycogen exhibited a line by incubation period by diet interaction at 72 and 96 h (Table 8). The effect in the SUS line was evident as short incubation periods in combination with increased carbohydrate in the diet increased cardiac glycogen at 72 but not at 96 h whereas the control diet decreased it at both 72 and 96 h post-hatching. The effect on COM poults occurred at 72 h where the carbohydrate diet increased cardiac glycogen of control incubation period poults compared to the carbohydrate diet. At 96 h COM poults from the short incubation period fed the control diet had greater cardiac glycogen than COM poults from the control incubation period fed the same diet. At 120 h post-hatching, a 2-way incubation period by diet interaction and both lines responded similarly to the combination of incubation period and diet. When given the control incubation period and the carbohydrate diet, poults had reduced cardiac glycogen compared to all other treatments. The SUS line poults at 120 h post-hatching did not exhibit elevated cardiac glycogen regardless of diet.

The SUS line poults exhibited greater amounts of hepatic glycogen at 72 and 96 h than COM (Table 9). At 120 h a line by incubation period by diet 3-way interaction affected hepatic glycogen. The SUS line poults hatching from short incubation periods and fed the high carbohydrate diet had more hepatic glycogen than other treatments, but COM poults displayed no such differences. Plasma glucose was consistently elevated in the Commercial line compared to the Susceptible line (COM = 309; SUS = 265 mg/dL) at all times examined post-

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Table 3: Body, yolk and organ weights of “flip-over” turkeys versus controls (N = 24)

Treatment BW	without yolk (g)	Yolk (g)	Heart (mg)	Liver (mg)	Jejunum (mg)
Control	48.4	2.8	317 ^a	1,620	703
Flip-over	38.3	2.2	237 ^b	1,345	563
% ± SEM	40.9 ± 3.3	2.5 ± 0.7	277 ± 19	1,483 ± 150	678 ± 169
Probability	NS	NS	0.03	NS	NS

^{a,b}Columnar means with a different superscript differ significantly (P < 0.05).

Table 4: Plasma thyroid hormone concentrations of “flip-over” turkeys versus controls (N = 24)

Treatment	Thyroxine (ng/mL)	Triiodothyronine (ng/mL)	Ratio (T3/T4)
Control	14.0 ^a	6.0 ^a	0.44
Flip-over	7.2 ^b	3.1 ^b	0.43
%± SEM	10.6 ± 1.2	4.5 ± 0.4	0.43 ± 0.02
Probability	0.008	0.002	NS

^{a,b}Columnar means with different superscripts differ significantly (P < 0.05).

Table 5: Body weights (g) without yolks of turkey poults from commercial and “flip-over” susceptible lines fed high carbohydrate diets following different incubation periods (N = 96)

Line ¹	Incubation period ²	Diet ³	Hours post-hatching		
			72	96	120
Commercial	Control	Carbo	62.7 ^{b,4}	8.9 ^a	92.3 ^{a,5}
		Control		76.2 ^b	83.7 ^b
	Short	Carbo	72.8 ^a	72.9 ^c	
		Control		86.2 ^a	
Susceptible	Control	Carbo	45.1 ^c	46.4 ^d	44.2 ^c
		Control		44.5 ^d	47.3 ^c
	Short	Carbo	41.5 ^c	43.9 ^d	
		Control		44.9 ^d	
		%			
		SEM	1.2	1.1	1.3
		Line (L)	0.0001	0.0001	0.0001
		Incubation period (IP)	NS	NS	NS
		Diet (D)	NS	NS	NS
		L x IP	0.05	NS	NS
	L x D	NS	NS	0.04	
	IP x D	NS	0.05	NS	
	LxIPxD	NS	0.05	NS	

^{a,b,c,d} Columnar means with different superscripts differ significantly (P < 0.05). ¹Commercial = commercial line of turkeys; Susceptible = line of turkeys susceptible to flip-over. ²Control = eggs were incubated normally; Short = eggs were incubated to shorten incubation period. ³Carbo = diet contained 50% carbohydrate; Control = diet contained 15% carbohydrate. ⁴Line by incubation period interaction means in this column. ⁵Line by diet interaction means in this column.

hatching but displayed no other main treatment or interaction effects (data not shown).

Discussion

The hypothesis tested by the current study was that “flip-over” poults would differ from normal controls in abilities to provide necessary energy to the critical tissues or have reduced thyroid function, a condition required for normal heart development (Nobikuni *et al.*, 1989; Fisher, 1999). Weak poults identified by symptoms of the “flip-over” condition described by Noble *et al.* (1999) differed in only

two physiological aspects examined. Heart weights and blood plasma thyroid hormone concentrations were depressed compared to controls. These observations suggest that at least these two physiological systems may be involved in weak poults, i.e., the cardiovascular and thyroid systems. The experimental design, however, did not clarify if these conditions are a cause or an effect. It is clear that an organ cannot grow if the metabolism providing it with nutrients is malfunctioning (Lilja, 1983), and stressors associated with hatching also require additional energy intended for such growth (Davis and

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Table 6: Heart weights (mg) of turkey poults from commercial and “flip-over” susceptible lines fed high carbohydrate diets following different incubation periods (N = 96)

Line ¹	Incubation period ²	Diet ³	Hours post-hatching		
			72	96	120
Commercial	Control	Carbo	383 ^{b,4}		582 ^{a,5}
		Control			453 ^b
Susceptible	Control	Carbo	473 ^a		
		Control		459 ^A	
Commercial	Short	Carbo			248 ^c
		Control			262 ^c
Susceptible	Short	Carbo	278 ^c		
		Control	267 ^c		
		%		265 ^B	
		SEM	15	12	7
		Line (L)	0.0001	0.0001	0.0001
		Incubation period (IP)	0.05	NS	NS
		Diet (D)	NS	NS	0.002
		L x IP	0.01	NS	0.06
		L x D	NS	NS	0.0005
		IP x D	NS	NS	NS
		LxIPxD	NS	NS	NS

^{a,b,c}Columnar interaction means with different superscripts differ significantly (P < 0.05). ^{A,B} Columnar main effect means differ significantly (P < 0.05). ¹Commercial = commercial line of turkeys; Susceptible = line of turkeys susceptible to flip-over. ²Control = eggs were incubated normally; Short = eggs were incubated to shorten incubation period. ³Carbo = diet contained 50% carbohydrate; Control = diet contained 15% carbohydrate. ⁴Line by incubation period interaction means in this column. ⁵Line by diet interaction means in this column.

Table 7: Liver weights (mg) of turkey poults from commercial and “flip-over” susceptible lines fed high carbohydrate diets following different incubation periods (N = 96)

Line ¹	Incubation period ²	Diet ³	Hours post-hatching		
			72	96	120
Commercial	Control	Carbo	2,600 ^{b,4}	2,582 ^{a,5}	
		Control		2,500 ^{ab}	
Susceptible	Control	Carbo	3,085 ^a	2,246 ^b	
		Control		2,699 ^a	
Commercial	Short	Carbo		3,364 ^A	3,641 ^A
		Control			
Susceptible	Short	Carbo	1,581 ^c		
		Control	1,488 ^c		
		%		1,649 ^B	1,738 ^A
		SEM	57	55	86
		Line (L)	0.0001	0.0001	0.0001
		Incubation period (IP)	NS	NS	NS
		Diet (D)	0.03	NS	0.03
		L x IP	0.04	NS	NS
		L x D	NS	NS	NS
		IP x D	NS	0.03	NS
		LxIPxD	NS	NS	NS
			Carb. = 2,035 ^B		Carb. = 2,393 ^A
			Con. = 2,432 ^A		Con. = 2,806 ^B

^{a,b,c}Columnar interaction means with different superscripts differ significantly (P < 0.05). ^{A,B} Pooled main effect means differ significantly (P < 0.05). ¹Commercial = commercial line of turkeys; Susceptible = line of turkeys susceptible to flip-over. ²Control = eggs were incubated normally; Short = eggs were incubated to shorten incubation period. ³Carbo = diet contained 50% carbohydrate; Control = diet contained 15% carbohydrate. ⁴Line by incubation period interaction means in this column. ⁵Incubation period by diet interaction means in this column.

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Table 8: Cardiac glycogen (mg/g of wet tissue) of turkey poults from commercial and “flip-over” susceptible lines fed high carbohydrate diets following different incubation periods (N = 96)

Line ¹	Incubation period ²	Diet ³	Hours post-hatching		
			72	96	120
Commercial	Control	Carbo.	1.87 ^a	1.24 ^{bc}	1.56 ^{b,4}
		Control	1.31 ^b	1.09 ^c	1.98 ^a
	Short	Carbo.	1.42 ^b	1.65 ^{ab}	2.19 ^a
		Control	1.30 ^b	1.74 ^a	1.92 ^a
Susceptible	Control	%			1.68 ^B
		Carbo.	1.22 ^b	0.57 ^d	
		Control	1.91 ^a	1.76 ^a	
		Short	Carbo.	2.21 ^a	1.95 ^a
	SEM	Control	0.56 ^c	1.92 ^a	2.14 ^A
		%	0.07	0.06	0.06
		Line (L)	NS	NS	0.004
		Incubation period (IP)	NS	0.0001	0.05
		Diet (D)	0.01	0.05	NS
		L x IP	NS	NS	NS
L x D	IP x D	0.003	0.04	0.02	
	LxIPxD	0.0001	0.01	NS	

^{a,b,c,d}Columnar interaction means with different superscripts differ significantly (P < 0.05).

^{A,B} Columnar main effect means differ significantly (P < 0.05).

¹Commercial = commercial line of turkeys; Susceptible = line of turkeys susceptible to flip-over.

²Control = eggs were incubated normally; Short = eggs were incubated to shorten incubation period.

³Carbo = diet contained 50% carbohydrate; Control = diet contained 15% carbohydrate.

⁴Incubation period by diet interaction means in this column.

Table 9: Hepatic glycogen (mg/g of tissue) of turkey poults from commercial and “flip-over” susceptible lines fed high carbohydrate diets following different incubation periods (N = 96)

Line ¹	Incubation period ²	Diet ³	Hours post-hatching		
			72	96	120
Commercial	Control	Carbo			1.60 ^c
		Control			2.25 ^{bc}
	Short	Carbo			2.45 ^{bc}
		Control			4.43 ^{bc}
Susceptible	Control	%	2.0 ^B	3.1 ^B	
		Carbo			3.01 ^{bc}
		Control			5.40 ^{ab}
		Short	Carbo		
	SEM	Control			3.70 ^{bc}
		%	5.2 ^A	8.3 ^A	
		SEM	0.5	0.5	0.5
		Line (L)	0.009	0.0004	0.05
		Incubation period (IP)	NS	NS	NS
		Diet (D)	NS	NS	NS
L x IP	L x D	NS	NS	NS	
	IP x D	0.06	NS	NS	
	LxIPxD	NS	NS	0.05	

^{a,b,c}Columnar interaction means with different superscripts differ significantly (P < 0.05).

^{A,B} Columnar main effect means differ significantly (P < 0.05).

¹Commercial = commercial line of turkeys; Susceptible = line of turkeys susceptible to flip-over.

²Control = eggs were incubated normally; Short = eggs were incubated to shorten incubation period.

³Carbo = diet contained 50% carbohydrate; Control = diet contained 15% carbohydrate.

Siopes, 1989). Davis and Siopes (1989) showed a suppressed stress hormone response exists during the period immediately post-hatching in the turkey poult. If a vital supply organ such as the heart or the thyroid is compromised, it may limit other critical tissue development such as brain function.

Neonatal heart abnormalities can be the result of many factors. Some examples are rapid growth, hypoxia, or the occlusion of the vents in the incubator (Lilja, 1983; Bagley and Christensen, 1989; Bagley *et al.*, 1990; Lilja and Olsson, 1987; Julian *et al.*, 1992; Breeding *et al.*, 1994; Buys *et al.*, 1998). A surprising result of the current study was that of equivalent body weights of flip-over and control poults accompanied by depressed heart weights in flip-over poults as compared to controls.

Depressed plasma thyroid hormones at hatching may also be the result of many factors as well. The incubation conditions, time of hatching or the amount of iodine in the maternal diet (McNabb *et al.*, 1984; Christensen, 1995; Buys *et al.*, 1998; Christensen and Davis, 2001) can alter hatching plasma thyroid hormone concentrations. Thyroid hormones play critical developmental and regulatory roles prior to and immediately following hatching (Fisher, 1999). Some examples are the ability to thermoregulate and maintain a constant body temperature over a range of environmental temperatures, proper cardiac development and function (Nobikuni *et al.*, 1989), proper neural development and the proper non-calorigenic metabolism (McNabb *et al.*, 1984). The significance of a single point depression in the thyroid function is questionable. It is difficult to make many inferences from the data presented in the current study because more studies of neonatal thyroid function are needed to elucidate the significance of the hypothyroidism observation.

It was hypothesized in Experiment 2 that the depressed heart weights seen in weak poults of Experiment 1 may be caused by deprivation of energy for proper cardiovascular function. Three treatments were used to deprive energy and simulate the weak poult condition. Line, diet and incubation period or their interaction in the COM poults altered body weights, but none of the treatments affected body weights of the SUS line. As COM line poults grew, heart weights increased concomitantly with body weight, but SUS line heart weights increased minimally for the first 120-h post-hatching. Additionally, increased dietary carbohydrate increased heart weights in COM but not SUS line poults. These observations suggest that the metabolic capacity to provide energy to the heart is impaired in the COM line until 72 h but impairment lasted until at least 120 h in the SUS line. Because avian brain tissue has limited ability for lipolysis and gluconeogenesis (Pearce and Brown, 1971), depressed cardiac function could also reduce its supply capability to provide brain tissue with adequate glucose that could also result in flip-over behavior.

Avian cardiac tissue lacks Cori cycle enzymes necessary

to recycle lactate (Pearce and Brown, 1971). Therefore, lactate must be shuttled by the blood back to the liver to be recycled to glucose-6-phosphate then returned to the heart for glycolysis. Because of the involvement of avian hepatic tissue in cardiac growth, liver tissue was also examined in Experiment 2. Liver weight was enlarged by the high carbohydrate diet. The shorter incubation period increased liver weights more rapidly in the COM line poults than in the SUS line. Liver weight as a percentage of body weight (data not shown) was very similar among the lines suggesting that the differences in hepatic growth were due primarily to increases in body weight and not to individual organ growth. Only line affected hepatic glycogen at 72 and 96 h post-hatching, but at 120 h line, incubation period and carbohydrate in the diet all interacted to affect hepatic glycogen. Thus, the data suggest possible liver involvement in the weak poults.

In summary, the current study characterizes weak turkey poults. Flip-over poults exhibited depressed heart weights and plasma thyroid hormone concentrations compared to controls. The reduced cardiac weight was associated with cardiac energy metabolism in Experiment 2. It is suggested that the treatment of flip-over poults should involve management conditions that foster cardiac health and growth. Suggestions of such conditions from prior research may be the use of differing sodium and chloride concentrations in diets (Frame *et al.*, 2001), altering growth post-hatching (Breeding *et al.*, 1994) or avoiding prolonged holding times prior to removal from the incubator (Christensen and Donaldson, 1992).

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