

Effect of Genetics and Maternal Dietary Iodide Supplementation on Glycogen Content of Organs Within Embryonic Turkeys¹

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ABSTRACT In prior studies it was shown that the growth of turkey embryos was dependent upon maternal dietary iodide as well as genetic selection. The current study posed the question of which organ systems respond to these variables. Embryos from lines selected for 16-wk BW grew at the same rate as unselected embryos from the randombred population serving as the initial source of the selected line until approximately 21 d of incubation (selected = F; randombred control = RBC2). Line differences in growth of F embryos could be accounted for increased liver and heart growth at the expense of muscle growth. Muscle growth increased in the growth-selected line prior to

pipping. Muscle growth was affected less when dams were selected for egg production (selected = E; randombred control = RBC1). Muscle growth was slowed in E line embryos compared to that of RBC1, and liver and heart growth were slowed at internal and external pipping stages in E embryos compared to RBC1.

Early muscle growth was augmented when F dams were fed supplemental iodide. A similar response was observed in E line embryos but occurred at a later stage of development. Measurements indicated decreased tissue glycogen in liver, heart, and muscle of selected lines may be one possible mechanism by which growth or organ function may come in conflict.

(Key words: growth, embryo, genetic lines, thyroid)

1999 Poultry Science 78:890–898

INTRODUCTION

Lilja (1983) proposed that selection of domestic animals for rapid growth alters the pattern or chronology of organ growth. In animals selected for growth, it was noted that growth of supply organs (liver, heart, and intestine) preceded that of demand organs (muscle). Organ growth precedence was proposed to begin early in embryonic development (Lilja and Olson, 1987). Romanoff (1960) proposed that optimal embryo survival would occur when embryonic growth was maximized. Ricklefs (1987) extended these ideas to indicate that longer incubation periods due to lower incubation temperatures cause growth of all embryos to be uniformly depressed. At higher temperatures, when growth is accelerated, embryos hatched sooner and at a lower weight. It was speculated that metabolism, in

embryos growing at a faster rate, may increase faster than the nutrient supply to the embryo. Schmalhausen (1930) suggested that growth and mature tissue function were antagonistic and that the decreasing growth rate of embryos reflected the progressive differentiation of embryonic tissues and the acquisition of organ functional capacity. Little is known of this relationship in domestic turkey embryos although several researchers have addressed similar ideas in chickens (Byerly, 1932; Byerly *et al.*, 1938; Crittenden and Bohren, 1961; Siegel *et al.*, 1968).

The objectives of the studies reported here were to determine: 1) differences in the growth rate and glycogen concentration of supply and demand tissues in turkey embryos whose parents had been selected for increased growth or egg production and 2) the effects of feeding iodide to dams of these lines on organogenesis.

MATERIALS AND METHODS

The breeder turkeys used in these experiments were from lines selected for either increased 16-wk BW (F) or

Received for publication September 10, 1998.

Accepted for publication February 2, 1999.

¹The use of trade names in this publication does not imply endorsement by the North Carolina Agricultural Research Service of the products mentioned, nor criticism of similar products not mentioned.

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Abbreviation Key: E = turkey line selected for 180-d egg production; F = turkey line selected for 16-wk BW; RBC = randombred control

180-d egg production (E) and their randombred control lines (RBC2 and RBC1, respectively) described by Nestor and Noble (1995). Hatching eggs were obtained in May of each year, the poults were hatched in June, and then grown using commercially accepted practices. At 30 wk of age, egg production was induced by photostimulation (15.5 h of light/d, 0500 to 2030 h). Thereafter, the hens were divided randomly by genetic line into 24 pens with 10 hens of the same line per pen. The diets fed have been described elsewhere (Grimes *et al.*, 1989). Hens were artificially inseminated with semen from hatchmate toms of the respective lines. Eggs were incubated in Jamesway 252B incubators following the manufacturer's recommendations.

At the time of photostimulation, two diets were fed to the hens representative of the four strains in a completely randomized design. The control diet contained 0.4 ppm iodine and the iodide diet was identical to the control diet but was supplemented with 4 ppm iodine by addition of potassium iodide.

Experiment 1

At each of four stages of development, the plateau in oxygen consumption (25 to 26 d), internal (26 d),

external (27 d), and hatching (28 d), three randomly chosen embryos from each line by iodide treatment combination were killed by decapitation and weighed (nearest 0.1 g). Because of differences between the strains for incubation periods, the samples were classified by stage of development described by Christensen *et al.* (1982). Three trials of embryo sampling were conducted during the laying period of the hens (Weeks 4, 12, and 16), thus giving a total sample size of nine per line by iodide treatment combination. Following weighing of the whole body plus yolk sac, the liver and heart were dissected, trimmed, and weighed (nearest 0.1 mg). Pipping muscles (*Complexus*) were also dissected and weighed at each stage of development.

Prior to statistical analysis, the data were sorted by stage of development and then analyzed with the two lines (F vs RBC2 or E vs RBC1) and two iodide levels as a factorial arrangement of treatments. All factors were considered completely random. Trial was considered as a fixed factor in the experiment, but as no significant trial interactions were observed, the data were pooled across all three trials. Means differing significantly were separated using the least square means procedure of SAS[®] (SAS Institute, 1988) and probabilities were based on $P \leq 0.05$ unless otherwise stated.

TABLE 1. Body weight of turkey hatchlings and embryo growth rate (percentage of hatchling mass) of genetic lines selected for growth (F) or egg production (E) compared to their randombred controls (RBC1 and RBC2) when dams were fed supplemental iodide

Genetic line ¹	Diet ²	Embryonic growth rate			Hatched BW (g)
		Plateau	Internal pip (%)	External pip (%)	
F	Control	105	105	98	60.4
	Iodide	104	107	102	55.8
	\bar{x}	105 ^A	106 ^A	99	58.1
RBC2	Control	92	96	99	56.7
	Iodide	95	95	100	58.1
	\bar{x}	93 ^B	94 ^B	99	57.4
Probabilities					
Line		0.001	0.0005	NS	NS
Diet		NS	NS	NS	NS
Line × diet		NS	NS	NS	NS
	$\bar{x} \pm \text{SEM}$ (n = 36)	99 ± 2	100 ± 2	99 ± 2	57.8 ± 1.1
E	Control	102 ^{AB}	110 ^B	108	42.3
	Iodide	98 ^B	103 ^C	98	45.8
	\bar{x}	100	107	102 ^B	44.1 ^B
RBC1	Control	109 ^A	122 ^A	116	47.5
	Iodide	91 ^B	92 ^D	106	59.2
	\bar{x}	100	107	111 ^A	53.4 ^A
Probabilities					
Line		NS	NS	0.04	0.003
Diet		0.004	0.0001	0.009	0.010
Line × diet		0.04	0.0008	NS	NS
	$\bar{x} \pm \text{SEM}$ (n = 36)	100 ± 2	107 ± 2	107 ± 2	48.7 ± 1.1

^{A-D}Columnar means (n = 9) with no common superscript differ significantly ($P \leq 0.01$).

¹F = turkeys selected for 16-wk BW; RBC2 = randombred control line for F; E = turkeys selected for 180-d egg production; RBC1 = randombred control line for E.

²Control = basal diet; Iodide = basal diet was supplemented with 4 ppm iodide.

TABLE 2. Liver weights and growth rates (percentage of hatchling mass) of livers from turkey embryos from dams selected for growth (F) or egg production (E) compared to randombred controls (RBC1 and RBC2) when dams were fed supplemental iodide

Genetic line ¹	Diet ²	Embryonic growth rate of liver			Liver weight at hatch
		Plateau	Internal pip	External pip	
		————— (%) —————			(mg)
F	Control	78	70	81	1,228
	Iodide	6	79	81	1,292
	\bar{x}	71 ^A	74	81 ^A	1,260
RBC2	Control	65	70	68	1,346
	Iodide	63	72	71	1,283
	\bar{x}	64 ^B	71	70 ^B	1,315
Probabilities					
Line		0.05	NS	0.02	NS
Diet		0.04	NS	NS	NS
Line × diet		NS	NS	NS	NS
	$\bar{x} \pm \text{SEM}$ (n = 36)	67 ± 2	73 ± 3	76 ± 3	1,230 ± 35
E	Control	82	72 ^B	80	1,031
	Iodide	60	73 ^B	69	1,098
	\bar{x}	71	72	75	1,065 ^B
RBC1	Control	72	81 ^A	85	1,178
	Iodide	51	63 ^C	75	1,400
	\bar{x}	62	72	80	1,289 ^A
Probabilities					
Line		NS	NS	NS	0.01
Diet		0.01	0.0007	0.02	NS
Line × diet		NS	0.0001	NS	NS
	$\bar{x} \pm \text{SEM}$ (n = 36)	66 ± 3	72 ± 6	77 ± 2	1,177 ± 35

^{A-C}Columnar means (n = 9) with no common superscript differ significantly ($P \leq 0.01$).

¹F = turkeys selected for increased 16-wk BW; RBC2 = randombred control line for F; E = turkeys selected for 180-d egg production; RBC1 = randombred control line for E.

²Control = basal diet; Iodide = basal diet was supplemented with 4 ppm iodide.

Experiment 2

Experiment 2 was designed to determine the differences in the glycogen concentrations of selected tissues during observed times of differences in growth in Experiment 1. It was hypothesized that the effects of dietary iodide on growth may be mediated by glycogen metabolism. The tissues collected for weighing (liver, heart, and pipping muscle) in Experiment 1 were placed immediately in a 7% solution of cold perchloric acid following weighing. The tissues were dispersed for 45 s using a ULTRA TURRAX T25 tissue homogenizer.³ The samples were centrifuged at $700 \times g$ for 15 min, the supernatant fraction recovered and stored at 4 C until analyzed using the technique of Dreiling *et al.* (1987). The statistical analysis was performed as described in Experiment 1 using the PC/SAS/STAT® program (SAS Institute, 1989). No experimental trial by line or iodide interactions were noted, so the data were pooled across trials for the final presentation.

RESULTS

Experiment 1

Body Weights. Growth rates of F line embryos at the plateau and internal pipping stages were greater than those of the RBC2 line (Table 1). No other differences in growth rates were observed between F and RBC2 lines.

Maternal iodide supplementation interacted similarly with E and RBC1 lines at the plateau and external pipping stages to affect growth (Table 1). In general, iodide supplementation slowed the growth of RBC1 embryos to a greater extent than it did the E embryos. Both line and iodide effects persisted from external pipping through hatching but the two factors did not interact to affect the means.

Organ Weights. Embryonic liver growth rates were decreased by iodide in F, RBC2, E, and RBC1 embryos at the plateau stage compared to controls (Table 2). At the remaining stages of development, embryonic liver growth rates for F and RBC2 lines were not affected by maternal iodide supplementation. Iodide depressed liver growth rates in E and RBC1 embryos until hatching.

Heart growth of F and RBC2 embryos were affected differently than liver (Table 3). At the plateau stage,

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TABLE 3. Heart weights of turkey hatchlings and embryonic growth (percentage of hatching mass) of genetic lines selected for growth (F) or egg production (E) compared to their randombred controls (RBC1 or RBC2) when dams were fed supplemental iodide

Genetic line ¹	Diet ²	Embryonic growth rate			Heart weight at hatch
		Plateau	Internal pip	External pip	
		————— (%) —————			(mg)
F	Control	80 ^A	82	78	300
	Iodide	62 ^B	83	80	321
	\bar{x}	71	82	79	311
RBC2	Control	68 ^B	79	79	301
	Iodide	75 ^{AB}	73	85	297
	\bar{x}	71	76	82	299
Probabilities					
Line		NS	NS	NS	NS
Diet		NS	NS	NS	NS
Line × diet		0.01	NS	NS	NS
	$\bar{x} \pm \text{SEM}$ (n = 36)	71 ± 2	79 ± 2	80 ± 3	305 ± 7
E	Control	92 ^A	96	90 ^A	219
	Iodide	63 ^B	80	72 ^B	247
	\bar{x}	78	88	81	233 ^B
RBC1	Control	74 ^B	93	93 ^A	260
	Iodide	60 ^B	74	92 ^A	321
	\bar{x}	67	83	92	291 ^A
Probabilities					
Line		0.01	NS	0.02	0.0005
Diet		0.0001	0.0001	0.06	0.004
Line × diet		0.05	NS	0.009	NS
	$\bar{x} \pm \text{SEM}$ (n = 36)	73 ± 2	85 ± 1	86 ± 2	262 ± 5

^{A,B}Columnar means (n = 9) with no common superscript differ significantly ($P \leq 0.01$).

¹F = turkeys selected for increased 16-wk BW; RBC2 = randombred control line for F; E = turkeys selected for 180-d egg production; RBC1 = randombred control line for E.

²Control = basal diet; Iodide = basal diet was supplemented with 4 ppm iodide.

supplemental iodide decreased the growth of heart of F line embryos compared with controls, but no iodide effects were seen with the RBC2 embryos. At the plateau and internal pip stages, iodide similarly depressed heart growth of E line embryos with no effect on those of the RBC1 line (Table 3). Iodide depressed heart growth of E or RBC1 embryos at nearly every stage of development examined.

Muscle growth also exhibited a line by iodide interaction at the plateau stage (Table 4). Iodide reduced pipping muscle growth in F embryos as it did heart weight compared to controls whose dams did not receive supplemental iodide, but no effects were observed for RBC2 embryonic pipping muscles compared to the controls. No other significant differences were observed between the F and RBC2 lines.

In general, maternal iodide supplementation augmented pipping muscle growth in E embryos but depressed muscle growth rates in RBC1 embryos compared to controls (Table 4). At hatching iodide depressed E muscle weight but increased those of RBC1 compared to their respective controls.

Experiment 2

Organ Glycogen Content. Maternal iodide effects were noted for liver glycogen concentrations at internal

and external pipping in the comparison of the F with the RBC2 line embryos (Table 5). At internal pipping, iodide supplementation decreased hepatic glycogen in both F and RBC2 lines but at external pipping it decreased only in F embryos. The level of hepatic glycogen differed significantly between E and RBC1 embryos (Table 5). At external pipping, iodide interacted with line to decrease the concentration of hepatic glycogen in the RBC1 line but exerted no effect on that of the E embryos. No significant effects were noted at hatching.

At internal and external pipping, line interacted with iodide supplementation to decrease cardiac glycogen in RBC2 embryos compared to controls but increased cardiac glycogen of F embryos compared to the controls (Table 6). When the effects on the E and RBC1 lines were examined, line and iodide interacted at all stages of development examined to decrease cardiac glycogen in E embryos but increase it in RBC1 compared to the control.

A line by iodide interaction increased pipping muscle glycogen concentrations in the RBC2 embryos at the plateau stage but had no effect on that of F embryos compared to controls followed by the converse at internal pipping (Table 7). At external pipping, iodide increased the muscle glycogen of both lines of turkeys compared to controls and muscle glycogen was greater in RBC2 than F embryos. The single difference seen for the E/RBC1 comparison was at the plateau stage, in which RBC1

embryos possessed more muscle glycogen than E embryos.

At internal pipping, iodide interacted with F and RBC2 lines to increase blood glucose in F embryos compared to controls but had no effect on RBC2 embryos (Table 8). At hatching the converse was observed; the effect was on RBC2 embryos and there was no concomitant effect on F embryos. The blood glucose concentrations of E and RBC1 line embryos at the plateau and internal pipped stages exhibited a line by iodide interaction. Increased concentrations of glucose in E embryos were observed compared to controls, with depressed glucose concentrations in iodide-treated RBC1 embryos compared to controls. At external pipping, the opposite effects were observed with elevated levels seen in the control E embryos compared to iodide treated and depressed glucose concentrations found in the control RBC1 embryos compared to those from dams receiving the iodide treatment.

DISCUSSION

Embryonic Organ Growth

In the current study, line and maternal iodide supplementation reduced body growth and liver growth rate prior to pipping in lines exhibiting good hatchability,

suggesting a hepatic preparatory role for hatching (John *et al.*, 1987). Growth of embryos and liver is known to be affected by plasma thyroid hormone concentrations (Romanoff and Laufer, 1956; Burke, 1987); however, the effect of line and maternal iodide supplementation was, to the best of the authors' knowledge, not previously known.

Iodide supplementation also depressed F embryo heart growth at the plateau stage and resulted in a general depression in rates of E embryo heart growth compared to controls. The same difference was in contrast to the enhanced heart weights in RBC1 and RBC2 embryos at hatching. Obviously, the growth rate of embryonic cardiac tissue is somehow mediated by both iodide treatment of dams and genetics (Nobukuni *et al.*, 1989; Czarnecki, 1991).

Muscle growth was monitored as an indicator of growth rate in a demand tissue due to maternal dietary iodide (King and May, 1984). The pipping muscle was selected because it grows rapidly and is functional at the stages of development examined (John *et al.*, 1987). Iodide treatment increased growth rate prior to pipping in the E and RBC2 embryos but decreased it in RBC1 and F embryos. It may be that growth of the supply organs (liver and heart) in F embryos requires all available nutrients, leaving very little for the growth and function of the pipping muscle. This explanation is consistent with the

TABLE 4. Pipping muscle weights of hatchling and growth rates (percentage of hatchling mass) of turkey embryos from different genetic lines selected for growth (F) or egg production (E) compared to randembred controls (RBC1 or RBC2) when dams were fed supplemental iodide

Genetic line ¹	Diet ²	Embryonic growth rate of pipping muscle			Pipping muscle weight at hatch (mg)
		Plateau	Internal pip	External pip	
		(%)			
F	Control	75 ^A	85	103	675
	Iodide	51 ^B	84	101	732
	\bar{x}	62	84	102	704
RBC2	Control	57 ^B	94	119	489
	Iodide	67 ^{AB}	72	96	716
	\bar{x}	62	83	107	603
Probabilities					
Line		NS	NS	NS	NS
Diet		NS	NS	0.05	NS
Line × diet		0.001	NS	NS	NS
	$\bar{x} \pm \text{SEM}$ (n = 36)	62 ± 4	83 ± 5	105 ± 8	653 ± 7
E	Control	55 ^{BC}	107 ^B	119 ^B	406 ^B
	Iodide	94 ^A	124 ^{AB}	122 ^B	389 ^B
	\bar{x}	74	116	120	398
RBC1	Control	71 ^B	136 ^A	169 ^A	397 ^B
	Iodide	45 ^C	108 ^B	105 ^B	618 ^A
	\bar{x}	58	122	137	508
Probabilities					
Line		NS	NS	0.08	0.03
Diet		NS	NS	0.002	0.02
Line × diet		0.004	0.01	0.001	0.02
	$\bar{x} \pm \text{SEM}$ (n = 36)	66 ± 6	119 ± 5	128 ± 5	452 ± 3

^{A-C}Columnar means (n = 9) with no common superscript differ significantly ($P \leq 0.01$).

¹F = turkeys selected for increased 16-wk BW; RBC2 = randembred control line for F; E = turkeys selected for 180-d egg production; RBC1 = randembred control line for E.

²Control = basal diet; Iodide = basal diet was supplemented with 4 ppm iodide.

TABLE 5. Liver glycogen in hatchling and rate of glycogenolysis (percentage of hatchling mass) in embryos from turkey hens selected for growth (F) or egg production (E) compared to randombred controls (RBC1 or RBC2) when fed supplemental iodide

Genetic line ¹	Diet ²	Rate of glycogenolysis			Liver glycogen at hatch
		Plateau	Internal pip	External pip	
		————— (%) —————			(mg)
F	Control	486	92	79 ^B	4.7
	Iodide	754	383	237 ^A	3.2
	\bar{x}	620	237	158	3.9
RBC2	Control	423	163	159 ^{AB}	5.3
	Iodide	417	287	140 ^{AB}	4.3
	\bar{x}	419	225	150	4.8
Probabilities					
Line		NS	NS	NS	NS
Diet		NS	0.01	NS	NS
Line × diet		NS	NS	0.05	NS
	$\bar{x} \pm \text{SEM}$	519 ± 54	234 ± 14	157 ± 17	4.3 ± 0.5
E	Control	175	79	64 ^B	8.9
	Iodide	199	63	56 ^B	6.8
	\bar{x}	187 ^B	71 ^B	60	7.9
RBC1	Control	406	309	78 ^B	4.0
	Iodide	371	235	262 ^A	6.0
	\bar{x}	388 ^A	272 ^A	170	5.0
Probabilities					
Line		0.02	0.0004	0.005	NS
Diet		NS	NS	0.009	NS
Line × diet		NS	NS	0.01	NS
	$\bar{x} \pm \text{SEM}$	291 ± 6	163 ± 4	121 ± 2	6.4 ± 1

^{A,B}Columnar means with no common superscript differ significantly ($P \leq 0.01$).

¹F = turkeys selected for increased 16-wk BW; RBC2 = randombred control line for F; E = turkeys selected for 180-d egg production; RBC1 = randombred control line for E.

²Control = basal diet; Iodide = basal diet was supplemented with 4 ppm iodide.

TABLE 6. Cardiac glycogen (mg per heart) of turkey hatchlings and rate of glycogenolysis (percentage of hatchling mass) in embryos from turkey hens selected for growth (F) or egg production (E) compared to their randombred controls (RBC1 or RBC2) fed supplemental iodide

Genetic line ¹	Diet ²	Rate of glycogenolysis			Cardiac glycogen at hatch
		Plateau	Internal pip	External pip	
		————— (%) —————			(mg)
F	Control	274	253 ^B	217 ^B	0.7
	Iodide	260	444 ^A	386 ^A	0.5
	\bar{x}	268	348	302	0.6
RBC2	Control	251	397 ^{AB}	410 ^A	0.6
	Iodide	279	111 ^C	255 ^B	1.0
	\bar{x}	268	294	332	8.2
Probabilities					
Line		NS	NS	NS	NS
Diet		NS	NS	NS	NS
Line × diet		NS	0.01	0.003	NS
	$\bar{x} \pm \text{SEM}$	266 ± 17	316 ± 30	312 ± 20	0.7 ± 0.1
E	Control	193 ^A	243 ^B	150 ^B	0.3 ^B
	Iodide	388 ^A	492 ^A	510 ^A	0.8 ^A
	\bar{x}	290	368	330	0.5
RBC1	Control	311 ^A	528 ^A	589 ^A	0.9 ^A
	Iodide	141 ^B	179 ^B	239 ^B	0.4 ^B
	\bar{x}	226	354	414	0.6
Probabilities					
Line		NS	NS	NS	NS
Diet		NS	NS	NS	NS
Line × diet		0.0001	0.0002	0.0001	0.007
	$\bar{x} \pm \text{SEM}$	258 ± 15	371 ± 28	378 ± 28	0.6 ± 0.1

^{A-C}Columnar means with no common superscript differ significantly ($P \leq 0.01$).

¹F = turkeys selected for increased 16-wk BW; RBC2 = randombred control line for F; E = turkeys selected for increased 180-d egg production; RBC1 = randombred control line for E.

²Control = basal diet; Iodide = basal diet was supplemented with 4 ppm iodide.

TABLE 7. Pipping muscle glycogen in hatchlings and glycogenolysis rate (percentage of hatchling mass) in embryos from turkey hens selected for growth (F) or egg production (E) compared to their randombred controls (RBC1 and RBC2) fed supplemental iodide

Genetic line ¹	Diet ²	Glycogenolysis rate			Pipping muscle glycogen at hatch
		Plateau	Internal pip	External pip	
		(%)			(mg)
F	Control	29 ^B	22 ^C	51	4.1
	Iodide	44 ^B	482 ^A	190	2.7
	\bar{x}	36	251	121 ^B	3.4
RBC2	Control	36 ^B	148 ^B	149	2.5
	Iodide	140 ^A	92 ^B	192	2.9
	\bar{x}	88	120	170 ^A	2.7
Probabilities					
Line		0.0001	0.01	0.05	NS
Diet		0.0001	0.003	0.006	NS
Line × diet		0.0006	0.01	NS	NS
	$\bar{x} \pm \text{SEM}$	62 ± 5	187 ± 8	136 ± 12	3.1 ± 0.2
E	Control	32	128	129	2.2
	Iodide	33	60	141	2.2
	\bar{x}	32 ^B	94	135	2.2
RBC1	Control	44	160	239	2.4
	Iodide	39	104	136	2.0
	\bar{x}	41 ^A	132	188	2.2
Probabilities					
Diet		0.03	NS	NS	NS
Line		NS	NS	NS	NS
Line × diet		NS	NS	NS	NS
	$\bar{x} \pm \text{SEM}$	37 ± 2	109 ± 17	161 ± 17	2.2 ± 0.2

A-CColumnar means with no common superscript differ significantly ($P \leq 0.01$).

¹F = turkeys selected for increased 16-wk BW; RBC2 = randombred control line for F; E = turkeys selected for increased 180-d egg production; RBC1 = randombred control line for E.

²Control = basal diet; Iodide = basal diet was supplemented with 4 ppm iodide.

suggestion of Lilja (1983) that supply organ growth is given priority in animals selected for rapid growth. The iodide-based growth depression seemed to persist longer in E than F embryos.

Egg weight and length of the incubation period are primary determinants of growth in avian embryos (Starck and Ricklief, 1998). Those researchers suggested further that one determinant of growth rate (or the length of the incubation period) may be a single limiting tissue or organ. On the basis of interspecies comparisons of posthatching growth in birds (Lilja, 1983; Ricklief, 1987), it has been suggested that the rate of growth after hatching is at least partially determined by the pattern of organ growth. A high rate of growth is connected with a pattern in which a large part of early growth is from "supply organs" (esophagus, proventriculus, gizzard, intestines, heart and liver) at the expense of "demand organs" (breast, wings, legs and feathers). These changes begin very early in embryonic development (Lilja and Olsson, 1987). The effects of line and maternal iodide supplementation in the present study may confirm the hypothesis of Lilja (1983) as well as that of Schmalhausen (1930) that growth and organ function come into conflict when growth occurs too slowly or too rapidly.

Catabolism of embryonic tissue is a major source of energy in the perinatal period (Donaldson *et al.*, 1992). Thus, the observed growth rate in liver, heart, and muscle tissues may reflect the catabolism of tissues to provide

energy for hatching. Thyroid hormones play a major role in supplying glycolytic energy for pipping and hatching (Wittmann and Weiss, 1981) as well as forming glycogen in muscle (Nobukuni *et al.*, 1989) and heart (Czarnecki, 1991). This role is important because cardiac and skeletal muscles do not possess gluconeogenic capabilities as do the liver and kidneys (Watford, *et al.*, 1981). The data suggest that the dam's iodide level may have a sparing effect on embryonic liver tissue in the line selected for growth, perhaps by reducing catabolism.

Tissue Glycogen Concentrations

The hypothesis was proposed that the provision of glycogen to the vital tissues, a process mediated at least in part by thyroid hormones (Nobukuni *et al.*, 1989), may be responsible for the differences in tissue growth seen in Experiment 1. The reduction or accrual of glycogen seen in various tissues in the current study may be the result of several metabolic processes. Possible mechanisms could be glycogenolysis, rate of gluconeogenesis, or rate of glycogen synthesis. It cannot be determined from the data in the current study which of the mechanisms is responsible for changes in tissue glycogen content, but the changes may likely be due to increased glycogenolysis because that mechanism has been reportedly active during the actual hatching process (Wittmann and Weiss, 1981).

TABLE 8. Blood plasma glucose concentrations of hatchlings and embryos from turkey hens selected for growth (F) or egg production (E) compared to randombred controls (RBC1 and RBC2) when fed supplemental iodide

Genetic line ¹	Diet ²	Stage of development			
		Plateau	Internal pip	External pip	Hatched
(mg/dL)					
F	Control	160	228 ^a	227	214 ^{ab}
	Iodide	136	172 ^b	209	242 ^a
	\bar{x}	148	200	216 ^b	228
RBC2	Control	204	213 ^{ab}	232	228 ^a
	Iodide	205	232 ^a	246	198 ^b
	\bar{x}	204	222	240 ^a	213
Probabilities					
Line		0.002	NS	0.04	NS
Diet		NS	NS	NS	NS
Line × diet		NS	0.04	NS	0.02
	$\bar{x} \pm \text{SEM}$	176 ± 6	212 ± 8	227 ± 5	221 ± 5
E	Control	183 ^a	211	193 ^c	248
	Iodide	164 ^b	221	238 ^{ab}	233
	\bar{x}	173	227	214	221
RBC1	Control	121 ^b	226	264 ^a	229
	Iodide	172 ^a	228	223 ^b	213
	\bar{x}	149	227	244	221
Probabilities					
Line		NS	NS	0.04	0.10
Diet		NS	NS	NS	NS
Line × diet		0.05	NS	0.005	NS
	$\bar{x} \pm \text{SEM}$	161 ± 8	221 ± 4	228 ± 7	231 ± 5

^{a-c}Columnar means with no common superscript differ significantly ($P \leq 0.05$).

¹F = turkeys selected for increased 16-wk BW; RBC2 = randombred control line for F; E = turkeys selected for increased 180-d egg production; RBC1 = randombred control line for E.

²Control = basal diet; Iodide = basal diet was supplemented with 4 ppm iodide.

The primary effects of dam iodide concentration and line on embryonic liver growth and glycogen concentration were at the time of internal and external pipping. The internal pipping stage is characterized by hypoxia, hypercapnia, and glycogenolysis (Freeman, 1965). Maternal iodide increased glycogen concentrations of both lines but increased growth only in the F embryos. The data suggest that iodide affects F embryos at internal pipping by contributing to a paucity of hepatic glycogen for liver growth and maintenance compared to that of RBC2. Iodide effects on liver growth of the E/RBC1 comparison at internal pipping also correlated well with the disappearance of hepatic glycogen. The evidence also suggests that some cardiac physiologic mechanism(s) for carbohydrate metabolism may be altered through genetic selection for egg production.

The most dramatic effects seen in the current study involved muscle physiology. Changes in growth priorities may have occurred in the selection of the F embryos. The addition of iodide to the dam affected supply organs (heart and liver) at the expense of the demand organs, in agreement with the suggestion of Lilja (1983). Even when iodide provided additional energy (increased blood glucose concentrations at the plateau stage) to the muscle tissue, the muscle did not respond by increasing growth rate. Muscle growth in the E/RBC1 comparison, in contrast to the F/RBC2 comparison, was affected later in development.

Summary and Conclusions

The data suggest that selection of turkeys for egg production affects embryonic growth and organogenesis differently than does the selection for growth. The interactions of line and maternal iodide supplementation suggest differences occurring in embryonic growth when turkeys are selected for increased growth rates may be partially mediated by thyroid function.

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