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Length of the Developmental Period of Turkey Eggs Affects Cardiac Physiology and Subsequent Embryo Survival[1]

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Abstract: The relationship describing eggshell conductance constants (k) suggests that eggshell conductance (G) is directly related to the length of the incubation period, but inversely with the weight of the egg. Prior studies showed clearly that G is a factor in cardiac health. We tested the hypothesis in the current study that the length of the incubation period may be a factor along with G that affects cardiac physiology and embryo survival. Incubation temperatures were reduced stepwise by 0.2°C in three treatments (37.5, 37.3 and 37.1°C) to prolong embryo developmental periods. The length of the developmental period was increased concomitantly in preliminary trials by 6 and 12 h, respectively by the 37.3 and 37.1°C treatments compared to 37.5°C. Fertilized eggs were incubated using the three temperatures in each of three independent trials. The time of hatching was closely noted and embryo survival was compared among treatments. Embryo heart rates and cardiac physiology in each group were observed. Long developmental periods reduced heart rates in a stepwise fashion and improved embryo survival and cardiac physiology. Thus, cardiomyopathy may be influenced by the length of the developmental period of turkey embryos because longer periods facilitated energy metabolism for myocardial function. Longer developmental periods would be easier to manage than G and may contribute to better turkey embryo viability late in development

Key words: Length of incubation, embryo survival, cardiac physiology

Introduction

Stock and Metcalfe (1984) suggested that depressed metabolism from low oxygen pressure limited organ growth and maturation; thus, the maturation of the turkey embryo may be limited normally by the availability of oxygen. One way to provide additional oxygen to growing tissues may be to lengthen the developmental period thus providing additional time for oxygen to diffuse across the shell. Recently, thicker shells (Christensen *et al.*, 2006a) or increased eggshell conductance (Christensen *et al.*, 2006b) have been shown to be factors in embryo heart development. Eggshell conductance (G) is a component of a multifactorial relationship involving egg weight and the length of the incubation period that is called the conductance constant (k) (Ar and Rahn, 1978; Rahn, 1981). The k varies directly with G and the incubation period but inversely with egg weight. It may be difficult to improve hatchling quality by selecting each egg based on G prior to incubation, but the length of the incubation period can easily be manipulated by altering incubator temperature set points in our modern management systems. Lower temperatures prolong development and may improve survival rates. Therefore, we hypothesize that the length of the incubation period affects heart development and the survival of turkey embryos.

Materials and Methods

Preliminary experiment: The initial study determined the required change in incubator temperature to attain a 6 or 12 h extension of the developmental period for Large White turkey eggs. The eggs were numbered, weighed and then set in an incubator. During incubation they were exposed to different temperatures until the beginning of the 24th day of development when they were moved to the same machine operating at 36.9°C and 75% RH. Beginning at the 25th day of development, poults were counted as they hatched at 3 h intervals until the completion of 28.5 days. The percentage of poults hatched at each of the times was analyzed using SAS software to measure the length of the developmental period resulting from each temperature.

Length of the developmental period, heart rate and embryo survival experiment: Following determination of the treatment temperatures to prolong the developmental period, the effects of longer developmental periods on embryo cardiac energy metabolism and survival were tested. In each of three replicate trials, approximately 5,304 eggs were set randomly in two racks in an incubator. Two racks were placed into each of three incubation cabinets set to operate at 37.5, 37.3 or 37.1°C. Each tray contained 136

Table 1: Time of hatching (h of incubation) of turkey embryos developing in eggs incubated at three different temperatures

	Hours of incubation				
Temperature	648	654	660	666	672
37.1°C	2.7^{b}	9.0^{c}	39.7^{c}	59.9^{c}	100.0
37.3°C	3.4^{b}	25.0^{b}	44.5^{b}	78.7^{b}	100.0
37.5°C	11.3^{a}	46.2^{a}	65.5^{a}	83.1^{a}	100.0
Mean±SEM[1]	5.8±0.4	26.7±0.9	49.9±1.0	73.9±0.9	NA
Probability	0.0001	0.0001	0.0001	0.0001	NS

^{a,b}Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

Table 2: Times (h) to attain a stage of development and the time remaining at that stage of poult embryos incubated at three different temperatures

	Stage of development				
	Internal pip		External pip		Hatched
Temperature	Attain	At	Attain	At	Attain
37.1°C	628	8.1^{c}	636	20.1^{a}	657^{a}
37.3°C	626	11.4^{b}	637	18.1^{a}	655^{ab}
37.5°C	624	14.7^{a}	639	14.1^{b}	653^{b}
Mean±SEM[1]	626±8	11.4±0.5	638±8	17.4±0.1	655±3
Probability	0.1887	0.0001	0.4917	0.0315	0.0588

^{a,b}Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

eggs. The trays were arranged in racks accommodating 13 trays. The trays (26 total trays per treatment per trial) served as the experimental unit for the embryo survival analysis. Embryo survival was recorded as the percentage of hatched embryos counted in each tray at the completion of 28 days of incubation. Eggs on each tray that did not hatch were broken open, examined for embryo development and categorized by approximate developmental age at death. Percentages of embryo survival and death for each G treatment were calculated, transformed by arcsine transformations then analyzed using the general linear models procedure of SAS (1998).

Time of hatching was observed on 13 trays within each trial by counting the number of poults hatched at 6 h intervals beginning at 630 h of development. The rate at which embryos advanced from one stage of development to another was also measured. Embryos were staged (Christensen *et al.*, 2006b) at the same times when hatched poults were counted by using a candling light. The percentage of total poults hatched at each time was the variable analyzed for time of hatching by the general linear models procedure of SAS (1998). The number of hours required to attain a given stage as well as the number of hours the embryo remained at that stage prior to advancing to the next stage were the variables.

Tissues were collected at days 27 and 28 of development by procedures described by Christensen *et al.* (2006ab). Heart rates were measured at days 25, 26 and 27 of development using 15 embryos per trial per treatment (total of 45 per treatment). Five embryos or poults per treatment per trial (a total of 15 per treatment) were weighed (nearest 0.01 g) at pipping (day 27) and hatching (day 28) with and without yolk. Blood samples were collected following decapitation then hearts and livers were quickly dissected, weighed and frozen (-22°C). Blood was centrifuged (700×g) for 10 min then the plasma was recovered and frozen (-22°C). Subsequently, cardiac and hepatic tissues were homogenized in 7% cold perchloric acid and analyzed for glycogen and lactate concentrations (Christensen *et al.*, 2006ab). The blood plasma was analyzed for glucose and lactate concentrations as well as for CK and LDH activities. Tissue data were analyzed by the general linear models procedure of SAS (1998) with individual bird as the experimental unit. Means differing significantly were separated with the least square procedure. Probability was based on $p<0.05$.

Effects of hatching times on neonate viability: Poults from each of the treatments were placed into brooder houses where their mortality was observed for the initial 14 days of life. Also, in a subsequent trial approximately 30,000 poults from the 37.1°C and 37.5°C were placed into brooders to observe the incidence of round heart following long and short incubation periods. Mortalities were noted daily and recorded and were submitted to the diagnostic laboratory where the occurrence of round heart was confirmed or denied.

Results

Preliminary experiment: Significant differences in hatching times were noted as eggs incubated at 37.3°C hatched an average of 6 h after 37.5°C incubated eggs and 37.1°C eggs hatched at an average of 12 h after the controls. Therefore, these two temperatures were selected for the trial to determine the effect of longer developmental periods on embryo cardiac physiology and survival.

Developmental period, heart rate and embryo survival experiment: Embryos in eggs at 37.5°C hatched 6 h earlier than did those at 37.3°C and 12 h earlier than those at 37.1°C (Table 1). The 37.1°C embryos spent less time at internal pipping and more time at external pipping than did those at 37.3°C or 37.5°C. The 37.3°C embryos were intermediate to the remaining treatments at the same stages of development (Table 2). Thus, embryos with longer incubation periods hatched later because of an extended time required to attain internal pipping (the plateau stage in oxygen consumption) and a longer time at external pipping.

Table 3: Heart rates (bpm) of poults and embryos incubated at three different temperatures

	Day of development	
Temperature	25	26
37.1°C	218[b]	223[b]
37.3°C	222[b]	219[b]
37.5°C	231[a]	232[a]
Mean±SEM[1]	224±1	225±2
Probability	0.0008	0.0233

[a,b]Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

Table 4: Embryo survival rates and times of embryo death (% of fertile eggs-pooled means of three trials) of turkey embryos developing in eggs incubated at three temperatures

	Measurement			
Temperature	Embryo survival (%)	Mortality at wk 1 (%)	Mortality at wk 4 (%)	Mortality at pipping (%)
37.1°C	84.5[a]	3.9[b]	5.3[b]	3.4[b]
37.3°C	77.1[b]	4.5[b]	9.3[a]	4.9[a]
37.5°C	76.5[b]	6.6[a]	8.5[a]	3.7[ab]
Mean±SEM[1]	79.4±0.4	5.0±0.2	7.7±0.2	4.1±0.2
Probability	0.0001	0.0001	0.0001	0.0122

[a,b]Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

Table 5: Body weights with and without yolk of poults and embryos incubated at three different temperatures

	Day of development	
Temperature	27	28
Carcass weight without yolk (g)		
37.1°C	53.9[a]	52.5
37.3°C	52.3[ab]	50.4
37.5°C	49.8[b]	53.1
Mean±SEM[1]	52.0±0.6	52.0±0.6
Probability	0.0293	0.1317
Yolk weight (g)		
37.2°C	13.6	8.4[b]
37.3°C	14.7	10.1[a]
37.5°C	15.3	10.8[a]
Mean±SEM[1]	14.5±0.4	9.8±0.2
Probability	0.2218	0.0012

[a,b]Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

Heart rates of embryos at 37.5°C were significantly faster than those at the remaining temperatures at days 25 and 26 of development (Table 3). More (8%) embryos in 37.1°G eggs survived than in the remaining treatments (Table 4). Nearly 5% more deaths near the plateau stage were in 37.3° and 37.5°C than in 37.1°C treated eggs.

Embryo weights differed between temperatures on day 27, as carcasses from the 37.5°C treatment weighed less than those of the remaining two treatments (Table 5). Body weights of the 37.3°C treatment were intermediate and did not differ from either of the remaining treatments. At 28 days of development, poults from 37.5°C treatment weighed less than those from either 37.3°C or 37.1°C. Yolk weights differed only at hatching as the 37.1°C treated poults had significantly less yolk that either of the other treatments. Hearts (mg) weighed more in 37.1°C than 37.5°C embryos and poults at both stages of development (Table 6). The weight of the liver was reduced in both 37.1°C and 37.3°C treatments compared to 37.5°C (Table 7).

Table 6: Heart weights of poults and embryos incubated at three different temperatures

	Day of development	
Temperature	27	28
Absolute (mg)		
37.1°C	310[a]	377[a]
37.3°C	293[ab]	350[ab]
37.5°C	261[b]	339[b]
Mean±SEM[1]	288±4	355±6
Probability	0.0003	0.0277
Relative to body weight (%)		
37.1°C	0.58[a]	0.72[a]
37.3°C	0.56[ab]	0.69[a]
37.5°C	0.52[b]	0.63[b]
Mean±SEM[1]	0.55±0.01	0.68±0.01
Probability	0.0340	0.0020

[a,b]Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

Table 7: Liver weights of poults and embryos incubated at three different temperatures

	Day of development	
Temperature	27	28
Absolute (mg)		
37.1ºC	1,091	1,529[a]
37.3ºC	1,059	1,398[b]
37.5ºC	1,031	1,323[c]
Mean±SEM[1]	1,060±13	1,417±18
Probability	0.1546	0.0001
Relative to body weight (%)		
37.1°C	2.03	2.92[a]
37.3°C	2.03	2.79[b]
37.5°C	2.08	2.49[c]
Mean±SEM[1]	2.05±0.03	2.73±0.03
Probability	0.6214	0.0001

[a,b]Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

Depressed myocardial glycogen and depressed lactate concentrations were seen in 37.1°C and 37.3°C embryos compared to 37.5°C (Table 8). It follows then that the ratio of total glycogen to total lactate was greater in the myocardium of 37.5°C embryos and poults than the lower temperature treatments. Hepatic tissue displayed the opposite response to that of the myocardium but only in hatched poults at day 28. The 37.5°C poults had depressed glycogen and elevated lactate concentrations compared to the remaining

Table 8: Cardiac glycogen and lactate and their ratio in poults and embryos incubated at three different temperatures

	Day of development	
Temperature	27	28
Glycogen (mg/g of tissue)		
37.1°C	0.39	0.26[b]
37.3°C	0.40	0.30[b]
37.5°C	0.46	0.41[a]
Mean±SEM[1]	0.42±0.01	61.8±0.6
Probability	0.1912	0.0020
Lactate (mg/g of tissue)		
37.1°C	1.53	1.38[b]
37.3°C	1.50	1.46[b]
37.5°C	1.61	1.60[a]
Mean±SEM[1]	1.54±0.02	1.49±0.02
Probability	0.1769	0.0022
Ratio (mg glycogen/mg lactate)		
37.1°C	0.26	0.19[b]
37.3°C	0.27	0.21[b]
37.5°C	0.28	0.26[a]
Mean±SEM[1]	0.27±0.01	0.22±0.01
Probability	0.6514	0.0128

[a,b]Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

Table 9: Hepatic glycogen and lactate and their ratio in poults and embryos incubated at three different temperatures

	Day of development	
Temperature	27	28
Glycogen (mg/g of tissue)		
37.1°C	0.19	0.27[a]
37.3°C	0.24	0.25[a]
37.5°C	0.32	0.19[b]
Mean±SEM[1]	0.25±0.04	0.26±0.01
Probability	0.2627	0.0689
Lactate (mg/g of tissue)		
37.1°C	0.37	0.29[b]
37.3°C	0.35	0.36[a]
37.5°C	0.34	0.35[a]
Mean±SEM[1]	0.35±0.01	0.34±0.01
Probability	0.3770	0.0066
Ratio (mg glycogen/mg lactate)		
37.1°C	0.54	0.93[a]
37.3°C	0.69	0.69[b]
37.5°C	1.08	0.55[b]
Mean±SEM[1]	0.77±0.12	0.72±0.04
Probability	0.2227	0.0008

[a,b]Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

treatments (Table 9). Thus, the ratio of the glycogen to lactate in 37.5°C poult liver tissue was reduced compared to the lower temperature treatments.

Plasma CK and LDH activities were affected at day 27 but not at day 28 (Table 10). Embryos and poults from the 37.1°C treatment showed greater CK activity and LDH activity compared to 37.3°C or 37.5°C. The 37.1°C treatment elevated plasma glucose

Table 10: Plasma activities of creatine kinase (CK) and lactate dehydrogenase (LDH) of poults and embryos incubated at three different temperatures

	Day of development	
Temperature	27	28
CK (U/L)		
37.1°C	1,236[a]	1,818
37.3°C	865[b]	2,074
37.5°C	895[b]	2,064
Mean±SEM[1]	998±4	1,986±85
Probability	0.0015	0.3144
LDH (U/L)		
37.1°C	399[a]	566
37.3°C	292[b]	551
37.5°C	289[b]	522
Mean±SEM[1]	326±13	547±14
Probability	0.0008	0.3487

[a,b]Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

Table 11: Plasma glucose and lactate concentrations (mg/dL) of poults and embryos incubated at three different temperatures

	Day of development	
Temperature	27	28
Glucose		
37.1°C	245[a]	269
37.3°C	240[a]	250
37.5°C	221[b]	267
Mean±SEM[1]	235±4	262±4
Probability	0.0466	0.1092
Lactate		
37.1°C	11.2	14.5
37.3°C	11.1	19.2
37.5°C	12.2	17.6
Mean±SEM[1]	11.5±0.4	17.1±0.1
Probability	0.4208	0.1829

[a,b]Columnar means followed by a different superscript differ significantly ($p<0.05$), [1]Overal means±SEM of pooled data from three trials

concentrations at day 27 compared to the other treatments, but the treatments did not affect plasma lactate concentrations (Table 11).

Determination of poult viability from treatments: When poults from each treatment were placed into brooders to determine the incidence of spontaneous cardiomyopathy (Paxton *et al.*, 2005), no differences were seen in the mortality rates (Table 12) in either hens or toms. No differences were noted in the incidence of cardiomyopathy (data not shown).

Discussion

Shorter embryo developmental periods due to slightly elevated incubator temperatures caused more embryos to die at the plateau stage in oxygen consumption than those with longer developmental periods. Thus,

Table 12: Poult mortality resulting from incubating at three temperatures

Trial	Poult sex	Treatment (°C)	7 d mortality	14 d mortality
I (8-14)	Hens	37.1°C	4.5%	4.5%
		37.3°C	5.4%	5.4%
		37.5°C	6.8%	9.6%
	Toms	37.1°C	5.7%	6.5%
		37.3°C	7.4%	8.5%
		37.5°C	4.5%	5.0%
II (8-16)	Hens	37.1°C	0.9%	1.4%
		37.3°C	1.0%	1.1%
		37.5°C	2.0%	2.7%
	Toms	37.1°C	0.9%	1.4%
		37.3°C	1.0%	1.1%
		37.5°C	2.7%	3.4%
III (8-18)	Hens	37.1°C	1.8%	2.7%
		37.3°C	1.6%	2.0%
		37.5°C	2.0%	2.7%
	Toms	37.1°C	3.4%	3.9%
		37.3°C	5.1%	5.1%
		37.5°C	2.7%	3.4%

embryos with shorter developmental periods have a greater probability of dying at the plateau stage in oxygen consumption a time at which the embryos are experiencing hypoxia or hypercapnia (Dietz *et al.*, 1998). Thus, at least some embryo deaths may be caused by insufficient developmental time to oxygenate vital tissues (Christensen *et al.*, 1999).

Failure to oxygenate sufficiently may involve cardiomyopathy due to the failure of energy metabolism of the turkey embryo myocardial fibers (Liao *et al.*, 1996; Christensen *et al.*, 1999). Altered myocardial glycogen and lactate concentrations are symptoms of cardiomyopathy poults (Czarnecki and Evanson, 1980; Czarnecki, 1991; Liao *et al.*, 1996). Earlier studies of cardiomyopathy poults (Czarnecki *et al.*, 1975; Staley *et al.*, 1978; Czarnecki and Evanson, 1980; Mirsalimi *et al.*, 1990; Liao *et al.*, 1996) noted excessive glycogen in various tissues. Studies suggested a failure to provide sufficient energy to the contracting muscle fibers to sustain muscle fibers (Liao *et al.*, 1996). Glycogen granules were observed in lysosomes of cardiomyopathy turkeys which were hypothesized to result from a block in the citric acid cycle preventing the complete breakdown of glycogen and resulting in altered metabolism by the liver, including decreased protein synthesis and increased metabolism of fat, possibly associated with liver damage (Staley *et al.*, 1978). The best explanation for the altered levels was a change in degradation of glycogen (Czarnecki *et al.*, 1978; Liao *et al.*, 1996). Conflicting reports exist, however, for the levels of cardiac glycogen of cardiomyopathy turkeys. Some researchers have reported elevated levels (Czarnecki and Evanson, 1980; Czarnecki, 1991) while others have reported no change (Staley *et al.*, 1978). Yet another (Mirsalimi *et al.*, 1990) reported decreased levels. However, none of those studies accounted for changes in heart size. Embryos in eggs with longer developmental periods in the current study had larger hearts, slower heart rates, lower glycogen and lactate concentrations in the myocardium as well as lower total glycogen to total lactate in myocardium compared to those with shorter developmental periods. Thus, a better energy balance was present in embryos with longer than shorter developmental periods and embryos from the longer treatments survived the plateau stage better. Our data along with the observations of Dawes *et al.* (1959) lead us to conclude that sufficient cardiac energy metabolism is essential to turkey embryo survival during late development.

In contrast to the heart, hepatic tissue showed more glycogen and more lactate in embryos with longer developmental periods than did those in short periods. Our data agree with those or Mirsalimi *et al.* (1990) who saw decreased glycogen to lactate ratios in heart but increased ratios in the liver when myocardial health became limiting. Overall the data suggest that short developmental periods require the embryo to remain at internal pipping in a hypoxic environment for a longer time. Extended time in hypoxia may demand more carbohydrate energy for cardiac activity than the hepatic enzymes can recycle and shuttle back to the heart of the embryo thus resulting in decreased energy available to cardiac myofibers and perhaps death (Liao *et al.*, 1996; Christensen *et al.*, 1999).

A related line of research examined cardiac health in poults by studying CK, LDH and other enzymes involved in providing energy to the heart via the pathways of glycolysis and oxidative phosphorylation. Creatine phosphate carries a high-energy phosphate, which can be transferred to and from ATP by CK in a reversible reaction. It is a source of energy when other means of supplying ATP to cardiac muscle are inadequate (Liao *et al.*, 1996). Simply stated, significant decreases in the speed at which the phosphorylation of ATP from phosphocreatine were found in myocardium from cardiomyopathy turkeys. The amounts of both phosphocreatine and ATP were decreased while free carnitive levels were normal. ATP synthesis via oxidative phosphorylation did not appear to be impaired when concentrations of major marker enzymes were measured. These observations were correlated with decreased contractility of isolated cardiac muscle fibers, indicating that there may be a strong association between decreased energy reserve and decreased contractility in the failing heart, although decreases in

ATP may not occur until late in pathogenesis (Liao *et al*., 1996). CK activity was decreased 40% in myocardium of cardiomyopathy turkeys, the most of any of the energy related enzymes, including those involved in glycolysis (30% depression), the Kreb's cycle (20% depression), and fatty acid oxidation (15% depression) (Mirsalimi *et al*., 1990). Shorter developmental periods depressed CK activity in the current study and elevated LDH as well indicating a greater rate of Cori cycle activity recycling lactate into glucose. Increased CK suggests the embryos at pipping developing in shorter periods experienced greater oxygen debt. Elevated LDH activity implies that embryos developing in shorter periods of time experienced problems recycling lactate to glucose thus decreasing cardiac muscle fiber energy.

Embryo hearts with long developmental periods eggs were heavier and the myocardium showed a better energy balance with more glycogen than lactate for muscular activity than did embryos with short developmental periods. Thus, a possible explanation for the better survival in embryos with slower developmental times would be slower heart rates with a greater stroke volume to maintain blood supplies for growth and development of turkey embryos at the plateau in oxygen consumption (Dietz *et al*., 1998). One contraction of the larger heart in an embryo from a longer developmental period may provide more oxygenated blood to growing tissues than does the more frequent contraction of the smaller heart from embryos developing in shorter periods with decreased glycogen to lactate ratios in the myocardium.

Prior data have indicated that shell thickness and eggshell conductance (G) affected cardiac health (Christensen *et al*., 2003; Christensen *et al*., 2006a,b). The shell provides the major resistance to vital gas diffusion to and from the embryo (Rahn, 1981) and the shell membranes may play a role early in development (Tullett and Board, 1976). Thus, two interrelated factors, G and IP affect the function of the myocardium in the development of the avian embryos and maturity of the hatchling (Ar and Rahn, 1978). Both factors may affect the same mechanisms to improve embryo survival at the terminal stages of embryo development called the plateau stage in oxygen consumption. Thus, we conclude that prolonging the incubation period to improve cardiac health and embryo survival may a more convenient means of improving hatchability than measuring G for each egg.

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

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Abbreviation Key: G = Eggshell conductance measured as mg of water vapor/d/mmof mercury of pressure across the shell. K = Eggshell conductance constant measured as the ratio of the product of G and length of the incubation period in days divided by the egg weight in g; LDH = lactate dehydrogenase; CK = creatine kinase