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Effect of *In ovo* Peptide YY on the Ontogeny of Glucose Transport in Turkey Poults[1]

W.J. Croom, Jr.*, J. Decubellis, B.A. Coles, L.R. Daniel and V.L. Christensen
Department of Poultry Science, North Carolina State University, Raleigh 27695-7608, USA

Abstract: Previous studies in this laboratory have demonstrated that peptide YY (PYY) administration to turkey poults at d25 of incubation enhances intestinal Na-dependent active glucose uptake. This study was designed to further characterize the ontogeny of glucose transport in embryonic and hatchling poults and to investigate the effects of PYY on this process during development. In Trial 1, 20 turkey eggs were randomly selected at days 20, 23, and 26 of incubation, as well as the day of hatch. Hatchlings were cervically dislocated and the body weight, jejunal length and jejunal weight were recorded. Jejunal glucose uptake was estimated by measuring ^{3}H-3-O-methyl-D-glucose accumulation in 2 mm jejunal rings *in vitro*. Jejunal O_2 consumption was measured *in vitro* on jejunal rings using an O_2 probe. In Trial 2, 40 turkey eggs were randomly selected at days 20, 23 and 25 of incubation and injected, via the air sac, with either 0.9% saline or 0.9 % saline plus 400 µg PYY/kg egg weight. Embryos from each treatment were harvested on days 23, 26 and day of hatch. Measurements and analyses on jejunal tissue were conducted as in Trial 1. In Trial 2, embryonic weight and jejunal weight adjusted for body weight increased (p<0.05) with stage of incubation, while adjusted jejunal length decreased (p<0.01). Active and total glucose uptake and jejunal O_2 consumption increased with age (p<0.05). The energetic efficiency of glucose uptake increased (p<0.05) between d26 and hatch. In Trial 2, PYY failed to significantly affect body or jejunal weight, glucose absorption, and O_2 consumption at any stage of development. PYY did however, decrease the efficiency of glucose uptake at d26 and at hatch (p<0.05). In contrast to earlier investigations using higher dosages of PYY, this study demonstrated that *in ovo* PYY administration at 400 µg/kg egg weight has little effect of jejunal function in turkeys.

Key words: Intestine, peptide YY, ontogeny, glucose transport

Introduction
Previous investigations have demonstrated that peptide YY (PYY), a gastrointestinal peptide hormone and a member of the pancreatic polypeptide family of hormones which includes neuropeptide Y and pancreatic peptide (Larhammar, 1996), enhances intestinal glucose absorption (Bird *et al.*, 1996; Coles, 1999). Peptide YY is produced by enterendocrine cells in the lower intestine in response to the presence of free fatty acids in the lumen of the distal small intestine (Larhammar, 1996; Hallden and Aponte, 1997). Bird *et al.* (1996) reported that PYY administration enhanced active glucose transport in the mouse without significant changes in energy expenditure suggesting that PYY may also enhance the efficiency of intestinal glucose absorption.

We have previously demonstrated that *in ovo* administration of PYY increased post-hatch jejunal active glucose transport when administered at 900 µg/kg egg weight in British United Turkey poults (BUT) and 600 µg/kg egg weight in Egg Line and Nicholas poults (Coles *et al.*, 2000). Additionally, Coles *et al.* (1999, 2000) have reported that broilers and turkey poults from eggs treated with PYY demonstrate enhanced growth and feed conversion during the first week post-hatch.

Digestive and absorptive processes in turkey poults are not fully developed at hatch (Suvarna *et al.*, 2005). The inability to fully utilize these processes may significantly contribute to an increased post-hatch mortality and decreased performance (Krogdahl and Sell, 1989; Nitzan *et al.*, 1991; Nir *et al.*, 1993). Croom *et al.* (1999) suggested that *in ovo* administration of PYY *in ovo* may increase the maturation of glucose and other intestinal nutrient transporter systems in incubation and at hatch and enhance nutrient absorption during this critical period of post-hatch growth and development. To date there have been no studies on the effect of PYY on glucose absorption and intestinal growth during incubation. The objective of this study was to determine the effects, in turkey poults, of *in ovo* PYY administration on the ontogeny of jejunal glucose absorption at various stages of embryonic development and at one day post-hatch.

Materials and Methods
Commercial turkey eggs (BUT; British United Turkeys, UK) were obtained from Prestage Farms (Clinton, NC). Developing eggs and poults were treated in accordance with guidelines of the North Carolina State University Institutional Animal Care and Use Committee. In Trial, 1

91 fertile eggs were incubated in a forced draft incubator with automatic egg turner at 38-39°C. On d20, d23, and d26 of incubation, as well as on the day of hatch (d28), 20 embryos or poults were euthanized by cervical dislocation and weighed. Embryos and poults the small intestine was removed from the body cavity and the intestinal mesentery gently dissected away. The intestinal lumen was flushed free of any residual digesta using 4°C HEPES buffer (25 mM HEPES, 4.8 mM KCl, 140 mM NaCl, 2.5 mM $CaCl_2$, 1.2 mM $MgSO_4$, 1.2 mM KH_2PO_4, pH 7.4) and then placed in the ice-cold buffer. The duodenum, jejunum (defined as the tissue immediately distal to the pancreatic ducts and proximal to the yolk sac stalk), and ileal segments were isolated and the unstretched lengths and blotted weights were recorded for each segment. The jejunum was folded into fourths to identify the proximal second quarter, and this segment was isolated in order to measure jejunal glucose uptake and tissue oxygen consumption. The samples were kept in 4°C HEPES buffer solution prior to the assays.

In Trial 2, BUT eggs were incubated as in Trial 1, but were randomly divided into two treatment groups. The control eggs were injected with 0.1 mL of 0.9% saline into the egg air space on d20, d23, and d25 of incubation (controls) or with 0.1 ml of 0.9% plus 400 μg human recombinant PYY(treatment; Quality Controlled Biochemicals, Hokinton, MA). For each group, 20 eggs were injected each day of incubation and incubated an additional 3 days. On d23, d26, and hatch, respectively, embryos and poults were euthanized by cervical dislocation and weighed. Intestinal tissue was isolated and duodenal, jejunal, and ileal weights and unstretched lengths were recorded as in Trial 1. Tissue preparation for glucose transport and tissue oxygen consumption was the same as in Trial 1.

Jejunal glucose transport assay: The jejunal glucose transport assay was modified from the protocol of Fan *et al.* (1996). The isolated jejunal segments were cut into 2 mm rings to yield tissue pieces of approximately 20-40 mg in wet weight. The tissue rings were inverted to optimally expose the villus surface to the assay solutions and stored in 4°C HEPES buffer solution prior to the assay. The rings were then preincubated for 5 min at 37°C in an assay transport buffer containing 25 mM HEPES, 4.8 mM KCl, 140 mM NaCl, 2.5 mM $CaCl_2$, 1.2 mM $MgSO_4$, 1.2 mM KH_2PO_4, 0.5 mM β-hydroxybutyrate, 2.5 mM glutamine, and 0.5 mM 3-O-methyl-D-glucose (3-O-MG), pH 7.4. The transport assay was started by transferring the rings into disposable plastic beakers containing 2 ml of transport buffer with 28 MBq/L ^{3}H-3-O-methyl-D-glucose (^{3}H-3-OMG) (2.9 TBq/mmol and 37 MBq/ml, DuPont NEN, Wilmington, DE).

To differentiate active from passive glucose transport, assays were conducted in triplicate from three adjacent pieces of jejunum from each sample. Total glucose uptake was estimated using transport buffer with ^{3}H-3-O-MG. Passive glucose transport was estimated using transport buffer with 1mM phlorizin (phloretin-2'-b-D-glucoside; Sigma Chemical, St. Louis, MO), a competitive inhibitor that irreversibly binds to the Na+-dependent active glucose transporter (Brot-Laroche and Alvarado, 1983). Assays for total and passive glucose transport were conducted at 37°C. A third incubation with transport buffer plus phlorizin was conducted at 4°C to measure nonspecific glucose uptake. Assays were stopped by transferring intestinal rings into 3 mL of ice-cold 300 mM mannitol. ^{3}H-3-O-methyl-D glucose was extracted from the tissue by incubation in 2 mL of trichloroacetic acid (2.5 g/L) at room temperature for 45 min in a Dubnoff metabolic shaking water bath set at 60 cycles/min. Intestinal rings were removed, blotted, and weighed. The extract was centrifuged at 2000 x g for 10 min at 4°C, and then 1 mL of the decanted supernatant was dispersed in 5 ml of Ecolite (+) scintillation cocktail (ICN Biomedicals, Cleveland, OH) and counted for 10 min in a Beckman LS-7500 scintillation counter (Beckman Instruments, Fullerton, CA). Duplicate aliquots (100μL) of transport buffer, with and without phlorizin, containing approximately 2.8 kBq ^{3}H-3-O-MG and a blank were counted with each set of samples.

Jejunal oxygen consumption assay: Intact jejunal O_2 uptake was measured according to the procedures described by Fan *et al.* (1996) for murine tissue using an oxygen monitor and incubation bath fitted with an oxygen sensor (Yellow Spring Instruments model 5300, Yellow Springs, OH). Oxygen uptake measurements were only conducted on jejunal samples at d26 and at hatch due to the small size of the embryos and intestinal tissue early in the incubation period. Jejunal segments were cut into 5 mm segments and rinsed in 4°C HEPES buffer. Segments were blotted, weighed and transferred to reaction chambers containing 4 ml of reaction media composed of 11 g M199, (Sigma Chemical; St. Louis, MO), 5.96 g HEPES, and 0.36 g $NaHCO_3$ in 0.1 L H_2O, pH 7.4, at 37°C. The samples were constantly stirred and the oxygen uptake recorded for 3 min using a dual channel chart recorder.

Calculations and statistical analyses: Active glucose transport was calculated as the difference between 3-O-MG accumulation in the reactions with and without phlorizin at 37°C. Passive glucose transport was calculated as the difference between 3-O-MG accumulation in the groups containing phlorizin at 37°C and 4°C. Glucose transport values are reported as nmole glucose/min/mg wet tissue. Estimation of O_2 uptake for the entire jejunum was calculated by multiplying O_2 consumption rate per gram of jejunum by 50% of the small intestine weight, as described by Fan

et al. (1996). Oxygen consumption of active glucose uptake is presented as nmole O_2 consumed/min/g tissue. The apparent energetic efficiency (APEE; nmole ATP expended / nmole active glucose uptake) was determined as described by Bird *et al.* (1994) and Croom *et al.* (1998).

Data were statistically analyzed using the General Linear Models Procedure of SAS (1988) with treatment as the main effect. Preplanned comparisons for Trial 1 were: d20 versus hatch, d23 versus hatch; and d26 versus hatch. Preplanned comparisons for Trial 2 were: d23 control versus d23 PYY; day 26 control versus d26 PYY and day of hatch control versus day of hatch PYY. Differences were considered significant at $p < 0.05$.

Results

Trial 1: Embryonic body weight increased significantly with each stage of incubation, most notably from d20 and d23 to hatch ($p < 0.001$; Table 1). Jejunal weight, adjusted for embryonic weight (Table 1), increased with days of incubation ($p < 0.001$). Jejunal length adjusted for embryonic weight decreased ($p < 0.001$) between d20, d23 of incubation and hatch while increasing ($p < 0.001$) between d26 and hatch. Jejunal weight adjusted for embryonic or body weight increased 64% between d26 and hatch ($p < 0.001$; 3.95 ± 0.10 versus 6.49 ± 0.19, respectively).

Total glucose uptake (Table 1) did not change from d20 to d23; however, it increased between d23, d26 and hatch ($p < 0.001$). This increase in total uptake reflects changes in active glucose transport (Table 1). The level of active glucose transport increased 400% between d20 and hatch ($p < 0.001$; 82.84 ± 5.25 vs 363.19 ± 39.9). Passive glucose transport decreased with day of incubation (Table 1). Furthermore, the ratio of active to passive transport (Fig. 1) increased significantly ($p < 0.01$) from incubation to hatch (1/1 at d20, 2/1 at d23, 3/1 at d26, and 10/1 on day of hatch). Jejunal oxygen consumption between d26 and hatch (Table 1) increased 8.6% ($p < 0.01$). APEE decreased from d26 to hatch (Fig. 1; $p < 0.001$); representing a corresponding increase in the overall efficiency of active glucose uptake.

Trial 2: Body weights of embryos increased between d23 and hatch in both control and PYY treated eggs ($p < 0.01$; Table 2), however, no significant differences ($p > 0.05$) were found between control and PYY treatments. A significant increase ($p < 0.05$) in adjusted jejunal weight was observed in poults from PYY treated eggs compared to controls ($p < 0.05$; Table 2). Jejunal length, corrected for body weight, was decreased ($p < 0.05$) by PYY administration at d26 of incubation.

Total glucose uptake was significantly ($p < 0.01$) reduced in the PYY treated poults at hatch. The difference at hatch ($p < 0.01$; 394 ± 10 versus 335 ± 16) was primarily the

result of decreases in active glucose transport (Table 2). Passive glucose transport was decreased ($p < 0.05$) by PYY administration at d23 of incubation. Jejunal oxygen consumption was not affected by PYY administration. The APEE of glucose uptake from the jejunum was calculated for both control and PYY treated eggs. As in Trial 1, the value decreased from d26 to hatch, ($p < 0.05$) representing an increase in overall efficiency of active glucose uptake (Fig. 1). In contrast, PYY administration decreased the efficiency of glucose transport as compared to control at both d26 and hatch ($p < 0.08$ and $p < 0.02$ for d26 and hatch, respectively).

Discussion

In Trial 1, we further described the developmental pattern of jejunal growth and glucose transport processes in BUT turkey embryos (Table 1). During d23 to d26 of incubation, jejunal growth and function is characterized by increases in tissue weight relative to length (intestinal density) and active glucose transport relative to passive transport. In the interval between day 26 and hatch, large increases occur in jejunal growth, glucose transport capacity and jejunal O_2 consumption (Table 1). This is an ontological adaptation to prepare embryos for hatch (Sell *et al.*, 1991). Similar increases in intestinal density and glucose absorption have been reported during this period by Hinni and Watterson (1963). They found these changes in chick embryos to be largely completed during the 3-day period preceding hatch. Morphological studies on poults have documented an increase in the height of epithelial cells and the length and density of epithelial microvilli during this time period (Overton and Shoup, 1964; Grey, 1972). Both changes may be positively correlated with intestinal maturation (Black, 1978). These structural changes are accompanied by dramatic rises in constituent enzyme activity, including phosphatases and disaccharidases on the microvillus membranes, as well as glucose oxidation (Strittmatter, 1973; Black and Moog, 1978).

It is interesting that in Trial 1, the period of maximum increased jejunal growth and increased tissue oxygen consumption (d26 and hatch; Table 1) was accompanied by an increase in the apparent energetic efficiency of glucose transport (Fig. 1). Investigations of enzyme activities in enterocyte intestinal absorptive cell culture systems found evidence for complex hormonal control during maturation (Corradino, 1979; Peterlik *et al.*, 1981). Further studies are needed to more fully understand the physiological basis of these structural and metabolic changes and their impact on absorptive efficiency.

In Trial 2, eggs were injected with PYY to investigate the effects of this peptide on jejunal growth and the development of glucose transport during incubation. PYY is known to modify digestive processes to ensure efficient utilization of ingested food (Hallden and Aponte,

Table 1: Effect of incubation stage on body weight, jejunal weight and length, glucose transport and oxygen consumption at different doses of incubation in BUT turkey poults

Variables	Day of Incubation[1,2]				Significance[3,4]		
	20	23	26	Hatch			
Egg wt (g)	77.60 ± 1.02	78.96 ± 1.44	71.52 ± 1.36	-	c***		
Body wt (g)	25.36 ± 0.55	39.47 ± 0.75	59.61 ± 1.11	63.31 ± 1.32	a***	b***	c**
Jejunal wt (g)/ body wt (kg)	2.86 ± 0.09	2.97 ± 0.06	3.95 ± 0.10	6.49 ± 0.19	a***	b***	c***
Jejunal length (cm)/ body wt (g)	0.33 ± 0.007	0.24 ± 0.004	0.16 ± 0.003	0.20 ± 0.005	a***	b***	c***
Glucose transport	---pmol/min/mg tissue--						
Total uptake	159.06 ± 4.59	145.16 ± 6.52	258.02 ± 9.06	400.02 ± 4.37	a***	b***	c***
Active uptake	82.84 ± 5.25	91.71 ± 5.32	197.46 ± 9.11	363.19 ± 39.9	a***	b***	c***
Passive uptake	76.21 ± 2.86	53.45 ± 4.20	60.56 ± 4.25	36.83 ± 4.52	a***	b***	c***
Active/Passive Ratio[1]	1.14 ± 5.92	2.31 ± 5.92	3.79 ± 5.92	26.64 ± 6.07	a**	b**	c**
Oxygen consumption (nmol/min/mg tissue)	-	-	0.81 ± 0.02	0.88 ± 0.02			c*

[1]Values are least square means ± SEM. [2]n=20/ Day. [3]a= d20 vs. hatch, b= d23 vs. hatch, c= d26 vs. hatch. [4]*= p <0.05, **= p< 0.01, ***= p< 0.001

Table 2: Effects of *in ovo* PYY injections on intestinal parameters and glucose transport at different days of incubation

Variables	Day of Incubation[1,2]						
	Control			PYY			Sig.[3,4]
	23	26	Hatch	23	26	Hatch	
Egg wt (g)	85.69±1.54	83.02±1.55	-	87.04±1.63	85.62±1.36	-	
Body wt (g)	40.67±0.81	69.29±1.54	71.37±2.05	40.48±0.53	70.79±1.07	74.61±1.57	
Jejunal wt (g)/body wt (kg)	3.01±0.07	2.97±0.12	5.19±0.22	2.99±0.08	2.89±0.14	5.39±0.26	c*
Jejunal length (cm)/body wt (g)	0.22±0.006	0.16±0.005	0.18±0.009	0.23±0.08	0.14±0.003	0.19±0.009	b*
Glucose transport	-- pmol/min/mg tissue --						
Total uptake	213.93±9.38	263.00±8.80	394.28±19.6	192.98±8.38	248.41±13.6	334.64±15.9	c**
Active uptake	114.11±8.21	190.37±9.06	328.11±20.1	109.89±7.84	174.32±12.9	270.83±15.6	c**
Passive uptake	99.82±6.63	72.64±3.78	66.17±4.80	83.09±6.79	74.08±3.78	63.81±3.47	a*
Oxygen consumption (nmol/min/mg tissue)	-	0.68 ± 0.05	0.91 ± 0.05	-	0.73 ± 0.04	0.97 ± 0.04	

[1]Values are least square means ± SEM. [2]n=20/ Treatment/ Day. [3]a= d23 Control vs. d23 PYY, b= d26 Control vs. d26 PYY, c= hatch Control vs. hatch PYY. [4]*= p < 0.05, **= p < 0.01, ***= p < 0.001

1997). Peptide YY exerts its effects by inhibiting gut motility and secretions from the intestinal lumen and pancreas (Savage *et al.*, 1987; Hallden and Aponte, 1997). Previous studies from this laboratory, using mice, have documented that exogenous administration of PYY increases intestinal glucose absorption and increases the energetic efficiency of glucose absorption (Bird *et al.*, 1996). Additionally, studies have found that exogenous administration of PYY *in vivo* is trophic to intestinal tissues (Chance *et al.*, 1998). Berg *et al.* (2000) has reported that subcutaneous administration of PYY, at 300µg/kg body weight for 3 days to mice, increases adjusted jejunal weight. Goodlad *et al.* (1990), however, reported that administration of PYY to rats for 3 days had no effect on intestinal cell proliferation. *In ovo* PYY administration to turkey eggs enhanced growth and feed conversion ratios at one week post-hatch for chicks and growth at 3 days post-hatch for poults (Coles *et al.*, 1999, 2000). Both of these responses suggest a potential role for PYY as a regulator of intestinal maturation and nutrient absorption during the first week post-hatch.

In Trial 2, no significant differences were found in jejunal weight and glucose transport between embryos from control and PYY treated eggs, (Table 2), although similar differences were noted in jejunal weight, as in Trial 1, between d26 and hatch. Poults from PYY treated eggs had heavier jejunal weights relative to length at hatch. This increase in intestinal mass was accompanied by significantly less active glucose uptake than the control group at hatch. The energetic efficiency of jejunal active glucose transport (Fig. 2) was also less for the PYY group. Although PYY enhanced jejunal growth in the final stages of embryonic development, it decreased the rate of active glucose uptake as well as the APEE.

The reason for the failure of PYY to enhance the rate and efficiency of jejunal glucose transport in BUT turkey poults is unclear. In the present study, the dosage used was 400µg PYY/kg egg weight. Coles *et al.* (2000) have described significant variation in genotypic response to PYY. They reported that *in ovo* administration of PYY enhanced jejunal glucose transport of hatchlings at 600µg/kg in egg weight Nicholas and Egg Line poults and 900µg/kg egg weight in BUT line poults. Hence, the
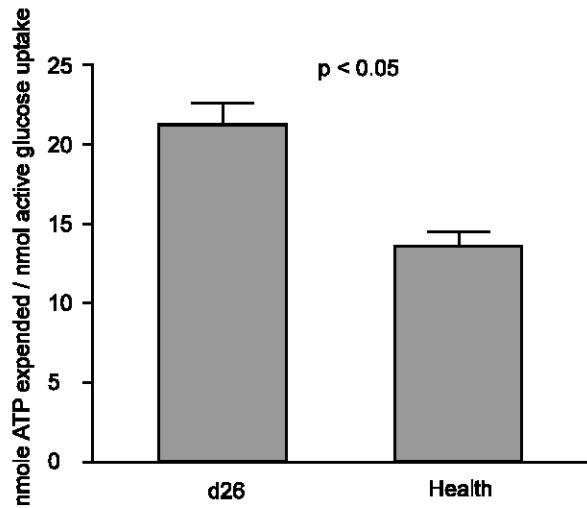
Fig. 1: Apparent energetic efficiency of jejunal glucose uptake at d26 of incubation and hatch in BUT turkey embryos and hatchlings (Trial 1)
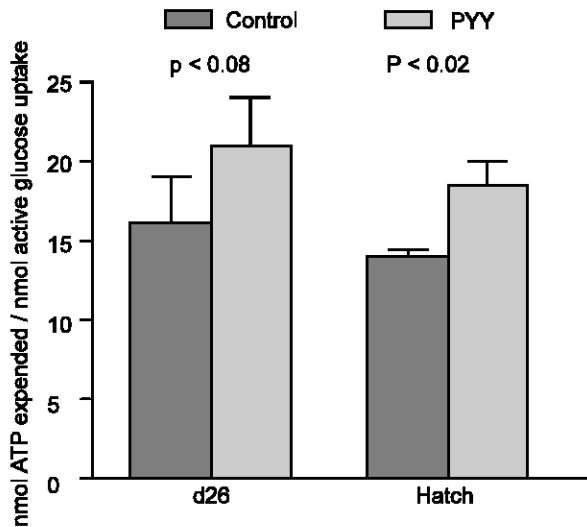

Fig. 2: Apparent energetic efficiency of jejunal glucose uptake in control (saline) and peptide YY (PYY) treated eggs at d26 of incubation and hatch (Trial 2)

dosage of PYY (400µg/kg egg weight) used in Trial 2 may not have been optimal for a positive response in glucose uptake.

Other physiological factors may have contributed to the lack of effect of PYY in Trial 2. The efficacy of *in ovo* administration of PYY into the air sac is predicated on the assumption that sufficient quantities of the peptide is absorbed by the embryonic membranes of the egg and systemically transferred to the embryo. Unknown differences in embryonic membrane permeability between turkey lines may explain, in part, the genotype

versus dosage variation discussed by Coles *et al.* (2000). Additionally, the responses of young poults to diets varying in nutrient composition may be a factor. Recently, Suvarna *et al.* (2005) reported that the plasma thyroid hormone T_3, concentration for BUT turkey poults increased during the feeding of a high carbohydrate diet. Tovar *et al.* (2004) reported decreased circulating PYY levels in hyperthyroid rats. It is possible that a nutrient x genotype interaction may play a role in endogenous circulation PYY levels. This, in turn, may affect response to exogenous PYY administration. Further studies are needed to clarify the factors affecting PYY's effect on nutrient transport in turkey's before a consistent and efficacious protocol can be described for the use of PYY in poultry production systems.

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