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In ovo Peptide YY Administration and Jejunal Glucose Transport in Hatchling Turkey Poults: Effects of Dosage and Genotype[1]

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Abstract: Two trials were conducted to investigate the efficacy of *in ovo* administration of various dosages of peptide YY (PYY) on jejunal glucose absorption in hatchling poults from two strains of turkeys, Egg Line (EL) selected for egg production and British United Turkey (BUT) selected for growth. In both trials, either 100 µl saline (1.025% w/v) or saline plus PYY were injected into the air cell of fertile EL and BUT line eggs at day 25 of incubation. At hatch, poults were euthanized by cervical dislocation, the jejunum removed and its weight and unstretched length recorded. Two to 4 mg cross-sections of the mid-jejunum were used to estimate active and passive glucose absorption using the accumulation of 3-O-methy-D-glucose (3OMG) in the presence and absence of phlorizin. In Trial 1, EL and BUT eggs were administered saline or saline plus 600 µg/kg egg wt, while in Trial 2, EL and BUT eggs were administered either saline or saline plus 300, 600, or 900 µg/kg egg wt. No differences were observed in hatchling body weights of poults from saline and PYY treated eggs from either line in both trials. In Trial 1, poults from EL treated eggs (600 µg/kg egg wt) had greater active jejunal 3OMG uptake compared with saline treated controls (332 vs. 270 ρmol/min/mg tissue, $p < 0.05$, respectively). In Trial 2, poults from BUT eggs treated with 900 µg PYY/kg egg wt had greater ($p < 0.05$) jejunal glucose transport than by the control group or the 600 µg PYY/kg egg wt group. Poults from EL eggs treated with PYY had non-significant increases in 3OMG uptake at all levels of PYY administration. BUT poults from eggs treated with 900 µg PYY/kg had heavier jejunums adjusted for body weight. *In ovo* PYY administration at day 25 of incubation increases active glucose transport in the intestinal tract of turkey poults, however, response and dosage varies with turkey line.

Key Words: Genotype, glucose absorption, *in ovo*, intestinal, peptide, PYY, turkeys

Introduction

Peptide YY (PYY), a member of the pancreatic polypeptide family of hormones, which includes neuropeptide Y and pancreatic polypeptide, is produced in flask-like "L" cells of the lower small intestine as well as in neurons of the brain and central nervous system of mammals (Larhammar, 1996). Its receptors are also found in these organ systems (Dumont *et al.*, 1996). PYY has been isolated from chicken intestine and was found to contain 37 amino acid residues rather than the 36 found in all other vertebrate PP family members (Conlon and O'Harte, 1992). In mammals, PYY release is stimulated by the presence of free fatty acids in the lumen of the distal small intestine (Hallden and Aponte, 1997) and is the humoral agent believed responsible for the "ileal brake" phenomenon, a lipid-induced inhibition of gastric acid secretion and intestinal motility (Raybould *et al.*, 1999).

Digestive and absorptive processes are not fully developed in turkey poults and broiler chicks at hatch (Krogdahl and Sell, 1989; Nir *et al.*, 1993). The failure of the intestinal tract of hatchlings to fully digest and absorb feed may contribute to increased post-hatch mortality and subsequent decreases in performance (Lilburn, 1998; Noy and Sklan, 1998). Coles *et al.* (1999, 2001) has reported that *in ovo* administration of 600 µg PYY/kg egg wt to broiler chicken and turkey eggs resulted in increased body weight and feed efficiency of chicks and poults during the first week post-hatch. They postulated that PYY enhanced the absorptive capacity of hatchlings, which resulted in enhanced growth. Bird *et al.* (1996) reported that PYY administration increased jejunal uptake of glucose in mice. Croom *et al.* (1999) have reported that *in ovo* administration of 600 µg PYY/kg egg wt increased glucose absorption in 1-day Nicholas turkey poults. In addition, PYY is believed associated with increased apolipoprotein A-IV and may increase lipid absorption (Kalogeris *et al.*, 1997).

There has been no systematic study of the dose-response relationship between exogenous PYY administration and changes in intestinal nutrient absorption in any species. The present studies describe the effects of genotype and *in*

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ovo dosage of PYY on jejunal glucose uptake in 1-day turkey poults. Trial 1 was designed to repeat the first study of *in ovo* administration of 600 µg PYY/kg egg wt in Nicholas line (growth line) turkey eggs (Croom *et al.*, 1999), with lines bred for egg production, Egg Line (EL), and growth, British United Turkey (BUT). Trial 2 used EL and BUT turkey eggs administered with varying amounts of PYY in order to identify an optimal *in ovo* dosage of PYY.

Materials and Methods

In both Trials 1 and 2, fertile EL eggs (Nestor *et al.*, 1982) were obtained from flocks maintained at North Carolina State University. BUT eggs were obtained from a commercial hatchery (Prestage Farms, Clinton, NC). Eggs and hatched poults were treated in accordance with the guidelines of the North Carolina State University Institutional Animal Care and Use Committee. Eggs from both EL and BUT lines were incubated in a forced-air draft incubator equipped with an automatic egg turner (G. Q. F. Manufacturing Co., Savannah, GA, USA) at a temperature of 38-39 ℃." Settings of each egg line in both trials were separate and staggered due to the limited availability of eggs. In Trial 1, eggs from each line were randomly divided into the following two treatment groups; 100 µl 1.025% saline (control) or saline plus 600 µg human recombinant PYY (Quality Controlled Biochemicals, Hopkinson, MA) /kg egg wt. In Trial 2, eggs from each line were randomly divided into the following four treatment groups; 100 µl 1.025% saline (control) and saline plus 300, 600 or 900 µg human recombinant PYY/kg egg weight. Recombinant human PYY was employed because earlier studies had demonstrated that the 70% structural homology between human and chicken PYY (Larhammar *et al.*, 1993) was enough to evoke a biological response in turkey intestines (Croom *et al.*, 1999). In both trials, injections were made *in ovo* into the air cell on day 25 of incubation.

During both trials, on the day of hatch, poults were weighed and euthanized by cervical dislocation. The duodenum, jejunum, and ileum were identified and removed as previously described (Coles *et al.*, 1999, 2001), rinsed in saline, blotted, and their wet weights recorded. The jejunum was gently laid out, in such a manner as to keep proximal and distal ends distinct, and the lengths were recorded. The jejunum was then folded in half and then the proximal end was folded in half again to allow visual identification of the second quarter of the jejunum.

The second quarter of the jejunum, approximately 4 cm in length, was removed and placed in ice-cold normal saline prior to use for the estimation of glucose uptake. Eight 2mm rings, weighing 2-4 mg, were cut using a device designed to hold evenly spaced, single-edged razorblades (Bird *et al.*, 1994). Each section was inverted manually if the tissue did not automatically invert due to muscle contraction. The jejunal glucose assay was modified from the protocol as described by Fan *et al.* (1996, 1997). Active and passive glucose transport rates were estimated by measuring the accumulation of a non-metabolizable glucose analog, 3-O-methly-D-glucose (3OMG), in the presence and absence of phlorizin, a competitive inhibitor of glucose and 3OMG for binding on the SGLT1 Na^+-dependent glucose transporter. Glucose transport rates were expressed as ρmol/min/mg tissue.

Data were statistically analyzed using the General Linear Models procedure of SAS (1988) with treatment as the main effect. In Trial 2, dosage data from each line were tested for cubic or quadratic effects. Because eggs from each line were available at different times, no direct statistical comparisons could be made between egg lines. Means were separated using Least Square Means with comparisons made between saline controls and 600 µg PYY/kg egg wt in Trial 1 and between saline and 300, 600, or 900 µg PYY/kg egg wt in Trial 2. In both trials, differences were considered significant at $p < 0.05$.

Results

Trial 1: 600 µg PYY/kg egg wt: *In ovo* administration of 600 µg PYY/kg egg wt had no measurable effects on jejunal weight or length in either the EL or BUT turkey poults (Table 1). The mean body weights of EL and BUT poults did not differ at hatch (Table 1) between treatment groups. PYY administration resulted in an 18.6% increase ($p = 0.03$) in active 3OMG uptake in EL poults but had no effect on 3OMG uptake in BUT poults (Fig. 1).

Trial 2: Dosage/Response: The mean body weights of EL and BUT poults treated with either saline or PYY did not differ at hatch (Table 2). No linear, cubic, or quadratic effects were noted for any of the parameters measured. *In ovo* administration of PYY did not significantly affect any of the intestinal parameters measured in the EL poults. In contrast, BUT poults treated with 900 µg PYY/kg egg wt had significantly heavier ($p < 0.02$) jejunal wet weights as compared to eggs treated with 600 µg PYY/kg egg wt or saline (0.47g vs. 0.42g and 0.42g, respectively; Table 2). This relationship persisted when jejunal wet weights were adjusted for body weight (Table 2). BUT poults from eggs treated with 900 µg PYY/kg egg wt also had longer jejunal lengths than that of poults from eggs treated with 600 µg PYY/kg egg wt (13.64 vs. 12.52, $p = 0.006$, Table 2).

Although PYY had no significant effect on jejunal glucose transport in EL poults, BUT poults from eggs treated with 900 µg PYY/kg of egg wt had greater rates ($p < 0.01$; Fig. 2) of active 3OMG uptake as compared to poults from saline treated eggs or eggs treated with 300 and 600 µg/kg egg wt (405 vs. 367, 343 and 323 ρmol/min/mg tissue wt, respectively).

Discussion

To our knowledge this is the first investigation of the effects of *in ovo* PYY dosage on intestinal glucose

Table 1: Effect of *in ovo* administration of saline or 600 µg peptide YY (PYY)/kg egg weight on body weight and jejunal wet weights and lengths in hatchling British United Turkey (BUT) and Egg Line (EL) turkey poults

Parameter	British United Turkey (BUT)[a]			Egg Line (EL)[b]		
	Saline	PYY	Significance	Saline	PYY	Significance
Body weight (g)	62.0 ± 1.24	61.5 ± 1.32	0.37	45.2 ± 1.10	45.9 ± 0.73	0.79
Jejunum weight (g)	0.49 ± 0.02	0.49 ± 0.02	0.80	0.31 ± 0.01	0.31 ± 0.01	0.71
Jejunum weight/body weight	0.0079 ± 0.0003	0.008 ± 0.002	0.55	0.0069 ± 0.0002	0.0067 ± 0.0002	0.59
Jejunum length (cm)	13.48 ± 0.17	13.38 ± 0.28	0.99	11.33 ± 0.21	11.63 ± 0.25	0.46

[a]n = 12/treatment. Values = means ± SEM; [b]n = 19/treatment. Values = means ± SEM

Table 2: Effect of *in ovo* administration of saline, and 300, 600, 900 µg peptide YY/kg egg weight on body weight and jejunal wet weights and lengths in hatchling British United Turkey (BUT) and Egg Line (EL) turkey poults

Parameter	British United Turkey (BUT)[a]				Egg Line (EL)[b]			
	Saline	300	600	900	Saline	300	600	900
Body weight (g)	56.0 ± 1.7	59.4 ± 1.4	58.6 ± 1.5	57.4 ± 1.3	43.6 ± 1.46	47.1 ± 1.57	45.9 ± 1.51	45.8 ± 1.01
Jejunum weight (g)	0.42 ± 0.01	0.44 ± 0.01	0.42 ± 0.01	0.47 ± 0.01*	0.24 ± 0.02	0.29 ± 0.03	0.27 ± 0.02	0.28 ± 0.02
Jejunum weight/body weight	0.0075 ± 0.0003	0.0077 ± 0.0002	0.0073 ± 0.0003	0.0082 ± 0.0003*	0.0057 ± 0.0005	0.0062 ± 0.0006	0.0059 ± 0.0006	0.0062 ± 0.0004
Jejunum length (cm)	13.19 ± 0.29	12.86 ± 0.27	12.52 ± 0.32	13.64 ± 0.23*	10.71 ± 0.67	11.37 ± 0.75	11.09 ± 0.47	10.76 ± 0.43

[a]n = 12/treatment. Values = means ± SEM [b]n = 19/treatment. Values = means ± SEM *Significantly different from control; $P < 0.05$

transport. Two different lines of eggs were studied, those from turkeys selected for egg production (EL) and those turkeys selected for growth (BUT). Croom *et al.* (1999) reported that poults from Nicholas line turkey eggs administered 600 µg PYY/kg egg wt *in ovo*, on day 25 of incubation, exhibited a 300% increase in jejunal glucose transport at day-1 post-hatch. Since that time the only paper describing the effects of exogenous PYY administration on the gastrointestinal function of poultry has been that of Peebles *et al.* (2001) in which no effect of epidermal growth factor (EGF) or PYY on yolk sac or yolk stalk function was demonstrated.

In Trial 1, an 18.6% increase in the rate of active jejunal glucose absorption was observed in EL poults from eggs treated with 600 µg PYY/kg of egg wt, although this effect was not repeated in Trial 2. In Trial 2, BUT poults hatched from eggs treated with 900 µg/kg egg wt exhibited an 18% increase in active jejunal glucose transport as compared to poults hatched from saline treated eggs. Additionally, increases in jejunal wt and length were observed when BUT eggs were treated with 900 µg PYY/kg egg wt. In Trial 2, EL poults exhibited a non-significant increase in active jejunal glucose transport ranging from 11-16% across treatment groups.

These data illustrate that PYY does have an effect on active glucose transport and intestinal growth in the jejunum of hatchling turkey poults from eggs treated *in ovo* with PYY. An important departure from the findings reported by Croom *et al.* (1999) was the difference in the dosage of PYY that increased uptake and the variation in the effective dosage between turkey lines. In the present study, the magnitude of enhancement of active glucose transport was much smaller than previously reported by Croom *et al.* (1999).

The variables associated with differences in response to PYY are not understood. It is, however, very obvious that genotype affects the response of PYY on active glucose absorption in the intestinal tract. Croom *et al.* (1998) noted an increase in jejunal glucose absorption in Swiss Webster line mice administered 600 µg PYY/kg BW, subcutaneously; however, Berg *et al.* (2000) reported no response to subcutaneous injections of PYY in Ts65Dn, partially trisomic mice, or their diploid controls. Observations by Cefalu *et al.* (1998) have shown that the Ts65Dn mice have a lower efficiency of intestinal glucose transport than their diploid controls. It is possible that fundamental differences in the metabolism of

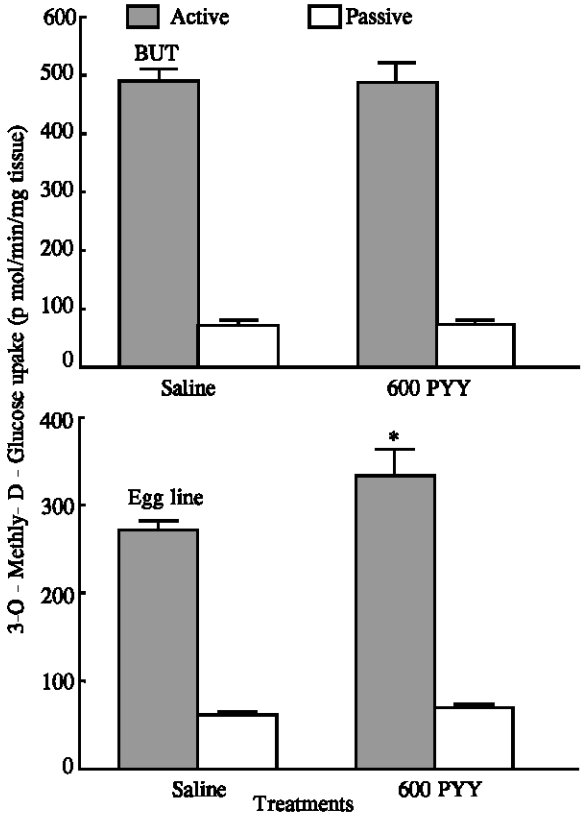

Fig. 1: Effect of administration of saline or 600 µg PYY/kg egg weight at day 25 of incubation on jejunal active and passive 3-O-methly-D-glucose uptake in British United Turkey (BUT) and Egg Line (EL) hatchling poults.
*different from saline controls ($P < 0.05$)

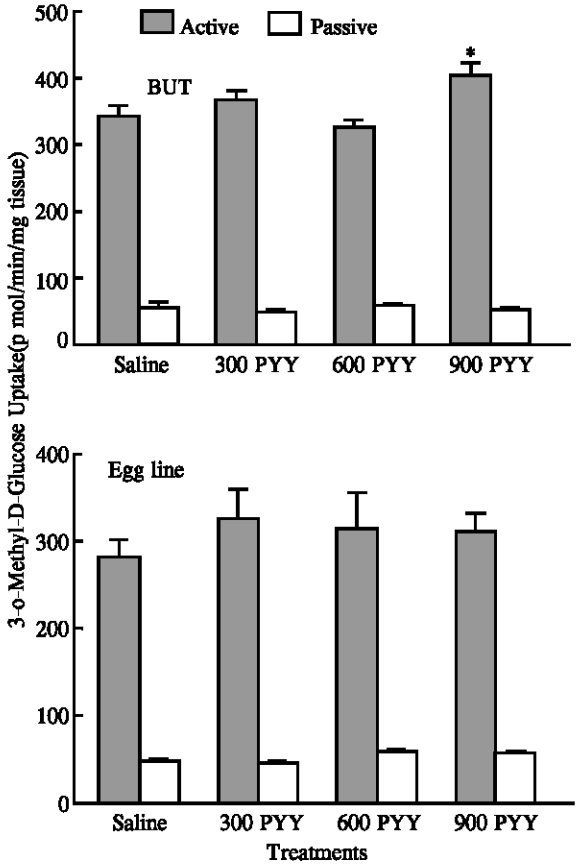

Fig. 2: Effects of administration of saline or 300, 600, or 900 µg PYY/kg egg weight at day 25 of incubation on jejunal active and passive 3-O-methy-D-glucose uptake in British United Turkey (BUT) and Egg Line (EL) hatchling poults.
*different from saline controls ($P < 0.05$)

jejunal enterocytes of the Ts65Dn mouse may have precluded any affect of PYY.

Croom *et al.* (1999) reported that *in ovo* injections of 600 µg PYY/kg egg wt in Nicholas line (growth selected) turkey eggs resulted in a dramatic increase in jejunal glucose transport (200-300%). The changes are much greater than that observed in the present study at the same treatment dosage with either EL (≈ 18%) or BUT line poults (no significant differences). It is interesting to note that BUT turkeys, like Nicholas turkeys, are selected for growth, yet display differences in magnitude of response and efficacious dosage levels compared to the Nicholas line. It is possible that lines of turkeys selected for growth, such as the BUT and the Nicholas strains, have responded to selection pressure in such a manner as to maximize the rate and efficiency of glucose absorption from the intestinal tract (Fan *et al.*, 1998). If intestinal absorption is optimized in these lines, one would expect less of an effect from PYY. This hypothesis explains the *in ovo* response to BUT but not the response of the Nicholas line. It is clear that, as yet, unknown variables are involved in the *in ovo* response to PYY.

Nothing is known of the physiological, genetic, or environmental effects on the ontogeny of PYY production and metabolism in avian embryos. In both Trials 1 and 2, eggs from EL and BUT lines were incubated at separate settings in a staggered fashion. It is possible that small variations in the incubation environment may have been responsible for differences in circulating concentrations of PYY in the embryos, thus affecting jejunal glucose transport of the hatchlings. The sporadic availability of eggs from the EL and BUT lines constrained our design and did not allow for statistical analyses of the variation associated with small differences in incubation conditions associated with the random fluctuations in the environment of the incubators.

Another source of variation that may have influenced the response to *in ovo* PYY is the kinetics of absorption from the air cell and the systemic distribution and metabolism

within the embryo. To enter the systematic circulation of the embryo, PYY introduced into the air cell has to transverse the inner layer of the shell membrane and the allantochorion (Burley and Vadehra, 1989). The inner shell membrane is approximately 20 µm thick and the allantochorion is highly vascularized. Human PYY is a small peptide of 36 amino acid residues (Larhammar *et al.*, 1993) and can likely traverse both structures and enter the systemic circulation of the embryo. Little is known, however, of the potential differences in the structure and function of these embryonic structures associated with genotype and incubation environment. Additionally, peptide YY is believed to have paracrine function in the intestinal tract (Taylor, 1989) and therefore may be active orally. It is currently unknown whether PYY administered *in ovo*, via the air cell, is available for oral ingestion by the embryo. Buchmiller *et al.* (1993) reported that administration of 300µg EGF/kg fetus wt/day, between days 19 and 21 of gestation, into the amniotic fluid of rabbits resulted in increased intestinal glucose and proline transport at day 24 of gestation. Goetzman *et al.* (1993) reported that injection of human recombinant EGF into the amniotic fluid of rhesus monkeys, concomitantly with intraperitoneal injection into the fetus, resulted in a 42% increase in the gut weight of neonates delivered by cesarean section after 78% of the gestation period. These data suggest that the action of gut peptides on intestinal function through prenatal or pre-hatch oral injection cannot be ruled out.

Despite the fact that we observed variation in glucose absorption and jejunal weight associated with *in ovo* PYY administration in turkey poults of different genotypes and different PYY dosages, the association between exogenous PYY administration and these effects in turkeys is compelling. PYY has been reported, in both poultry and mice, to increase intestinal glucose absorption rate and small and large intestinal growth (Chance *et al.*, 1998; Croom *et al.*, 1999; Berg *et al.*, 2000). In the present study, we observed increases in jejunal weight and in jejunal weight adjusted for body weight in BUT poults from eggs treated with 900 µg PYY/kg egg wt as well as increased glucose absorption rate. Recent studies by Coles *et al.* (1999, 2001) have demonstrated that *in ovo* administration of PYY to broiler and turkey eggs resulted in increased hatchling growth and feed efficiency during the first week post-hatch. These *in ovo* observations conform to current theory on the potential limiting effects of intestinal absorption on performance of modern hatchling poultry (Croom *et al.*, 1999) and serve as indirect evidence of the beneficial effect of PYY on intestinal nutrient uptake. Since the mechanisms and sources of variation of PYY's action in the intestinal mucosa are not yet fully described, the possible involvement of secondary PYY-induced physiological regulators of glucose absorption and hatchling performance cannot be ruled out. Additionally, it has been proposed that PYY may facilitate insertion of increased numbers of nascent nutrient transporters from vesicles within the terminal web into the luminal membrane of the enterocyte without a concomitant increase in intracellular energy expenditure (Croom *et al.*, 1999). Future studies will help establish the mechanisms of action of PYY on intestinal absorption and define the route of administration, dosage and incubation conditions under which it may be used most efficaciously in practical poultry productions systems.

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