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Effect of Dietary Selenium on Small Intestine Villus Integrity in Reovirus-Challenged Broilers†

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Abstract: Enteric Avian Reoviruses (ARV), associated with malabsorption, lower weight gains and increased mortality in broiler chickens, target enterocytes on intestinal villi causing villus dysfunction and decreased digestion and absorption of nutrients. This investigation examined whether enteric ARV infection with or without dietary Selenium (Se) (organic or inorganic) affected small intestinal integrity. Eggs from Cobb 500® broiler breeders fed low-Se semi-purified diets with no supplemental Se, or with 0.3 ppm supplemental Se provided by organic Se (Se-yeast, Sel-Plex®, Alltech, Inc., Nicholasville, KY, USA), or by sodium selenite, were hatched and the chicks were subjected to the same three dietary Se treatments as their respective parents. At hatch, 30 chicks per dietary Se treatment were placed into either control or ARV-infected groups in heated batteries in separate isolation rooms. ARV-infected chickens were given orally ARV-CU98 ($10^{4.2}$ pfu/chick in 0.5 mL) and control chickens received medium only. Intestinal tracts from 21-d-old chickens were examined histomorphometrically revealing longer and more narrow villi, greater surface perimeter, more shallow crypt depth and significantly greater height to crypt depth (H:D) ratios in Sel-Plex-fed control and infected birds, compared with respective values from birds fed no supplemental Se or sodium selenite. The differences in H:D ratios between Se treatments indicates that Sel-Plex is more effective than either no Se or sodium selenite supplementation in protecting the integrity of the small intestine villi.

Key words: Sel-Plex, histomorphometry, intestine, reovirus, broiler, selenium, selenium yeast

INTRODUCTION

A paucity of published investigations link Selenium (Se) with the responses of poultry species to viral infections. For chickens, one study (Panda and Rao, 1994) in which vitamin E and Se were supplemented to diets already adequate in these nutrients reported that, compared with unsupplemented groups, vitamin E and Se supplements enhanced immune function, including higher geometric mean titers of the tube agglutination test against Infectious Bursal Disease Virus (IBDV) and higher numbers of rosette-forming cells in the peripheral blood. Newcastle Disease (ND) viral infections also appear to be affected by vitamin E and Se supplementation. Bassiouni *et al.* (1990) observed improved resistance against ND, increased antibody titers and improved weight gain associated with Se and vitamin E supplementation. Swain *et al.* (2000) have shown that broiler chicken responses to ND vaccine were enhanced by Se and vitamin E supplements greater than NRC (1994) requirements. Additionally, Marin *et al.* (2003) reported increased antibody titers against IBDV and ND, increased serum protein levels, and prevention of bursal damage in aflatoxin-B-1-exposed chickens when the chickens were fed dietary supplements of Se and vitamin E. Singh *et al.* (2006) reported a synergistic effect between vitamin E and Se in chickens given a ND vaccine. ND-vaccinated chickens had higher hemagglutination inhibition titers when fed the combination of Se and vitamin E along with higher circulating levels of immunoglobulins and circulatory immune complexes.

There are numerous economically important RNA viruses that affect domestic poultry. Among these are the Avian Reoviruses (ARV), which are species-specific and have a broad range of virulence in chickens (Robertson *et al.*, 1984). ARV can cause infection in joints and tendons, respiratory tract and in the intestinal tract. Recently, there have been numerous field cases of enteric ARV infections leading to malabsorption syndrome, moderate to severe depression of weight gain and increased mortality. Malabsorption syndrome was investigated by Jensen *et al.* (1991) who found that both Se and vitamin E were effective in reducing mortality. In fact, Swick (1995) has recommended that Se and vitamin E be supplemented to the highest allowable dietary level to reduce ARV-related mortality associated with malabsorption syndrome.

The higher metabolic rates of the modern high meat-yielding broiler chicken might contribute to the severity of RNA viral infection, because the RNA viruses depend on high levels of oxidative stress to facilitate replication. Thus, it is important to investigate the role Se might play in RNA viral infection. This work was conducted to examine the influence of Se provided by both the

maternal carryover and the chick diet on intestinal morphology of broiler chickens challenged with an enteric ARV.

MATERIALS AND METHODS

Chicks: Cobb500® broiler chickens were hatched from eggs produced by breeder hens that had been maintained on a low-Se semi-purified diet (<0.02 ppm) with no supplemental Se, or with 0.3 ppm supplemental Se provided by organic Se (Se-yeast, Sel-Plex®, Alltech, Inc., Nicholasville, KY, USA), or by sodium selenite. Hatchling chickens were placed in pens in heated brooder batteries and were subjected to the same three dietary Se treatments as their respective parents (Table 1). Vitamin E was added to all the diets at 15 IU/kg to prevent the development of pancreatic fibrosis and exudative diathesis in the group given no supplemental Se. The chick diets used the same ingredients contained in the breeder diets. However, the proportions of the ingredients were adjusted to meet the nutrient requirements of young broiler chickens. Body weights were determined at 14 and 21 d of age and at those ages tissues were collected for other analyses related to Se and ARV infection.

Reovirus (ARV) challenge: On the day of hatch, 30 chickens from each of the three dietary treatments were placed into either control or ARV-infected groups (total of 180 chickens) in heated metal-growing batteries in separate isolation rooms. Each of the chickens in the ARV-infected groups was given an oral gavage of 0.5 mL of the reovirus ARV-CU98 ($10^{4.2}$ pfu/chick; Heggen-Peay *et al*., 2002); control chickens were given the medium only. At 21 d of age, the chickens were killed by carbon dioxide asphyxiation, intestinal tracts were dissected to measure total and segmental weight and length and tissues were collected for histomorphometry.

Histomorphometry: An individual bird was regarded as the experimental unit, and there were five birds per Se and virus group analyzed. A total of 30 randomly-selected chickens were used in this phase of the overall study. After dissection, the duodenum and ileal (divided into jejunum and ileum) segments were collected and fixed in Carnoy's solution. Tissues were processed for histology and stained with hematoxylin-eosin. A computerized microscope-based image analyzer (Southern Micro Instruments, Atlanta, GA) was used to determine the histomorphometric parameters for villus height, villus width at its mid-height, villus perimeter length, crypt depth, external muscle layer thickness and villus height to crypt depth (H:D) ratio (Fan *et al*., 1997). The criteria for selection of histological sections for examination were based on the presence of an intact lamina propria and villi that were perpendicularly sectioned through the midline axis.

Statistics: All data from this completely randomized experimental design were analyzed using the general linear models procedure of SAS (SAS Institute, 1996). For the histomorphometric calculations, ten measurements for each parameter from each bird per treatment, yielding 50 measurements per parameter mean, were analyzed as a one-way analysis of variance. Fisher's least significant difference was used to test differences between means only when the analysis of variance indicated significance at $p \leq 0.05$ (Motulsky, 2005).

RESULTS

Body weight was significantly depressed by reovirus infection at both 14 d (control = 514 g; infected = 362 g) and 21d (control = 962 g; infected = 724 g) post challenge, but there were no differences among body weights for the Se treatments within either control or infected groups. It was of interest, however, that weight gain from 14-21 d for Sel-Plex-fed chicks was somewhat greater than that of either the no Se- or sodium selenite-fed chicks in both control (no Se = 466 g, Sel-Plex = 476 g; selenite = 460 g) and infected (no Se = 355 g, Sel-Plex = 388 g, selenite = 342 g) groups.

Reovirus infection did not significantly affect small intestinal segment relative weights except that of the jejunum, which was increased by the infection (Table 2). A dietary Se effect was seen in the relative weights of the duodenum with Sel-Plex decreasing the relative weight in both control and virus-challenged birds and the selenite-fed diet causing an increase in relative weights in both control and infected birds compared with Sel-Plex. This dietary effect caused a significant diet x virus interaction for duodenum relative weight. The large intestine relative weights were decreased significantly by reovirus challenge, but Sel-Plex increased relative weight in both control and infected groups compared with no supplemental Se (Table 2). Reovirus infection did not affect length of the intestines (data not shown). However, there were statistically significant diet effects on average Gastrointestinal Tract (GIT) length. Feeding the diet with no Se supplement increased GIT length by almost 9% (data not shown) compared with the two Se diets ($p<0.05$).

Intestinal histomorphometric analysis of the duodenum (Table 3) revealed both diet and reovirus infection effects on the different parameters. The perimeter of the villus was significantly increased in the Sel-Plex-fed control and infected groups, compared with respective values for the no Se and selenite groups. There was no difference between control and selenite for villus perimeter. Villus height was significantly greater for birds fed Sel-Plex compared with the no Se-fed and selenite-fed birds in both control and reovirus-infected groups. Villus width was greater in selenite-fed birds in the control group compared with no Se-fed and Sel-Plex-fed

Table 1: Composition of torula-yeast diets, which were provided as a means to supply either no supplemental Se (<0.02 ppm), organic Se as Sel-Plex (0.3 ppm), or sodium selenite (0.3 ppm) to chickens hatched from eggs from parents who had consumed the same diets

	Ingredients of Diet		
Ingredients	No Se	Sel-Plex	Selenite
Corn (%)	38.12	38.02	38.02
Starch (%)	26	26	26
Torula yeast, (%)	12	12	12
Soybean meal, dehulled (%)	10	10	10
Wheat midds (%)	10	10	10
Dicalcium phosphate (22-18.5) (%)	1.7	1.7	1.7
Limestone, feed grade (38%) (%)	1.15	1.15	1.15
Iodized salt (%)	0.43	0.43	0.43
Trace mineral mix	0.25	0.25	0.25
Vitamin mix (+15 IU vitamin E/kg) (%)	0.25	0.25	0.25
DL-Methionine (99%) (%)	0.1	0.1	0.1
Sodium selenite mix* (%)	0	0	0.1
Sel-Plex mix* (%)	0	0.1	0
ME, kcal/kg	2913	2913	2913
Total P (%)	0.67	0.67	0.67
Available P (%)	0.4	0.4	0.4
Methionine (%)	0.41	0.41	0.41
Cysteine (%)	0.17	0.17	0.17
Ca (%)	3.3	3.3	3.3
Lysine (%)	1.04	1.04	1.04
Tryptophan (%)	0.18	0.18	0.18
DM (%)	92.37	92.37	92.37
CP (%)	16.37	16.37	16.37
Crude fat (%)	2.06	2.06	2.06
Crude fiber (%)	1.57	1.57	1.57
Moisture (%)	7.13	7.13	7.13
TSAA (%)	0.65	0.65	0.65

Table 2: Influence of reovirus infection and selenium source effects on Gastrointestinal Tract (GIT), duodenum, jejunum, ileum and large intestine relative weights in 21-d-old chickens

		Relative Weights (%)			
		[Section Wet Weight/GIT Wet Weight] x 100			
Dietary Treatments	Se	Duodenum	Jejunum	Ileum	Large Intestine
Control Basal	<0.02	23.41^{a}	27.80^{a}	21.86^{b}	23.63^{ab}
Control Sel-Plex	0.3	18.06^{c}	28.83^{a}	23.37^{a}	25.27^{a}
Control Na Selenite	0.3	20.50^{b}	29.90^{a}	21.96^{ab}	20.59^{bc}
Pooled Means ± SEM		20.66 ± 0.79^{A}	28.85 ± 1.03^{A}	22.40 ± 0.89^{A}	23.16 ± 1.05^{A}
Infected Basal	<0.02	23.11^{a}	31.42^{b}	22.53^{ab}	19.42^{c}
Infected Sel-Plex	0.3	18.07^{b}	31.40^{b}	22.35^{ab}	21.45^{b}
Infected Na Selenite	0.3	24.13^{a}	31.45^{b}	23.54^{ab}	21.32^{bc}
Pooled Means ± SEM		21.77 ± 0.79^{A}	31.42 ± 1.03^{B}	22.80 ± 0.89^{A}	20.73 ± 1.05^{B}

^{abc}In a column, means with unlike superscripts differ significantly ($p\leq0.05$)

^{AB}In a column, means with unlike superscripts differ significantly ($p\leq0.05$)

birds. However, in the infected groups, no Se-fed birds had the greatest villus width followed by selenite- and Sel-Plex-fed groups, respectively. There was a significant virus-related increase in crypt depth and there was a significant increase in crypt depth in the no Se-fed and selenite-fed birds compared with Sel-Plex-fed birds. Within both the control and infected groups, the villus height to crypt depth (H:D) ratio for chicks fed either no Se- or selenite-supplemented diets was increased compared with those fed Sel-Plex. Muscularis externa thickness was increased in virus-challenged chickens and was greatest in Sel-Plex-fed and no Se-fed birds. In the control groups, sodium selenite-fed birds had the greatest muscle thickness followed by no Se-fed and Sel-Plex-fed, respectively.

The results for histomorphometric analysis for the jejunum are shown in Table 4. The perimeter of the villus was increased significantly in the Sel-Plex control and infected groups compared with the perimeters of the no Se and selenite groups. There were no significant

Table 3: Selenium (Torula yeast with No Se, Sel-Plex (Sel-Plex), or sodium selenite) and reovirus influence on morphology (mean±SEM of villus perimeter, height, width, crypt depth, villus height to crypt depth [H:D] ratio and muscle [muscularis externa] thickness) of duodenum in 21–d-old chickens

Parameter	Control No Se	Control Sel-Plex	Control Selenite	Infected No Se	Infected Sel-Plex	Infected Selenite
Villus, µm Perimeter	2203±102^{b}	2581±92^{a}	2256±86^{b}	2168±165^{b}	2574±115^{a}	2035±149^{b}
Height	1058±52^{b}	1206±39^{a}	1010±62^{b}	1058±99^{b}	1231±48^{a}	983±70^{b}
Width	112±7.7^{c}	113±6.6^{c}	170±11.6^{ab}	201±21.7^{a}	132±9.5^{bc}	166±9.9^{b}
Crypt depth, µm	195±7.8^{b}	116±7.5^{c}	222±13.7^{b}	265±13.0^{a}	191±15.0^{b}	294±22.0^{a}
H:D ratio	5.43±0.46^{b}	10.37±0.88^{a}	4.54±0.39^{bc}	3.99±0.34^{bc}	6.46±0.45^{b}	3.34±0.28^{c}
Muscle, µm	292±9.3^{b}	230±3.3^{c}	317±22.1^{b}	356±18.9^{ab}	414±33.1^{a}	313±10.8^{b}

^{abcd}In a row, means±SEM with unlike superscripts differ significantly, $p \leq 0.05$, n = 50 for each mean presented

differences between no Se-fed and selenite-fed groups for villus perimeter. The infection-related depression in villus height was not significant in the Se-fed groups, but there was a significant depression in the no Se-fed group due to infection. Sel-Plex-fed chickens had significantly increased villus heights compared with the other dietary treatments. These responses did not induce a diet x virus interaction ($p>0.05$). Jejunum villus width was not affected by infection, but the feeding of Sel-Plex caused the development of a narrower villus width compared with those of the no Se and selenite-fed chickens. This dietary effect resulted in a significant diet x virus interaction for villus width. The crypt depth was less ($p<0.05$) in Sel-Plex-fed birds in both the control and virus-infected groups compared with no Se and sodium selenite treatments where an increased crypt depth was observed in both control and infected groups. There were significant Se and viral effects for height to crypt depth ratios. Sel-Plex-fed birds had the largest ratios in both control and infected groups and no Se- and sodium selenite-fed birds had significantly smaller H:D ratios. Diet and viral effects were evident for muscularis externa muscle thickness in the ileum, which showed that both Se treatments resulted in increased muscle thickness after infection. Birds fed no supplemental Se had no change in muscle thickness after infection.

The results for histomorphometric analysis for the ileum are shown in Table 5. In the control groups, the torula yeast-fed no Se treatment resulted in the longest perimeter followed by Sel-Plex and selenite treatments, respectively, but in the infected group, the no Se-fed treatment resulted in the shortest perimeter followed by the Se treatments that did not differ between themselves. These responses resulted in a significant diet X virus interaction. There was a significant depressive effect of viral infection on villus height in all dietary treatments. Villus height was negatively affected by viral infection in all Se treatments. In the control groups, the no Se- and Sel-Plex-fed birds had longer villi than selenite-fed birds and in the infected groups, Sel-Plex-fed birds had longer villi than either no Se- or selenite-fed birds. Villus width was increased in the infected birds fed either no Se or sodium selenite treatments, but villus width in the Sel-Plex-fed birds did not differ from controls. This difference resulted in a significant diet x virus interaction for villus width. The crypt depth was less in Sel-Plex-fed birds in both the control and virus-infected groups. No Se and sodium selenite treatments resulted in increased crypt depth in both the control and the infected groups. There were significant Se and viral effects for height to crypt depth (H:D) ratios. Sel-Plex-fed birds had the largest H:D ratios in both control and infected groups and no Se and sodium selenite fed birds had significantly smaller H:D ratios. Diet and viral effects were evident for muscularis externa muscle thickness in the ileum, which showed that both Se treatments resulted in increased muscle thickness after infection. Birds fed no Se had no change in muscle thickness after infection.

DISCUSSION

There is no doubt that ARV infection causes decreased weight gain in chickens. Part of the cause for this decreased weight gain after infection is attributed to the fact that the virus hijacks the protein synthesis mechanism in the cells early in the infection (Guy, 1998). Rebel *et al*. (2004) report that by 14 d post challenge, chickens exposed to an enteric ARV begin to recover from the initial infection as evidenced by less severity of lesions in the villus and crypt regions of the intestine. An external indication of the ARV infection can be observed as poor feathering, but as the infection begins to resolve, the protein synthesis mechanism regains its efficiency. In this study, weight gain was suppressed by reovirus infection through 21 d of age. However, of note, Sel-Plex-fed chickens were gaining weight at a slightly faster rate at 21 d than were chickens supplemented with no Se or selenite in both control and infected groups. This performance difference suggests that an improved antioxidant status in Sel-Plex-fed chickens facilitates a faster recovery after ARV infection. Mahmoud and Edens (2003) have demonstrated that organic Se as Sel-Plex has the ability to induce higher Se-dependent antioxidant enzyme activities than do sodium selenite or no Se supplements. The improved redox status then can be directly related to improved performance of broiler chickens fed Sel-Plex (Edens and Gowdy, 2004).

Surai (2006) reviewed extensively the health effects of dietary Se supplementation. He pointed out that Se does

not prevent virus-induced disease, but Se supplementation appears to play a pivotal role in reducing the severity of the infection and that observation is consistent with the results of this study with enteric ARV infection in broiler chickens. The improved health status imparted by the feeding of Se is attributed to enhanced immune responses and increased activity of antioxidant enzymes such as glutathione peroxidase (Surai, 2006; Mahmoud and Edens, 2003) and thioredoxin reductase (Edens and Gowdy, 2004). Even though there are improved redox status and enhanced immunity associated with the feeding of Sel-Plex, there are other physiological manifestations of the influence of Se on the overall heath status of the chicken.

Earlier studies have suggested that Se has a role in regulating virus replication. Balansky and Argirova (1981) concluded that Se depressed RNA-dependent RNA polymerase in an oncornavirus as an indicator of reverse transcriptase activity. Balansky and Argirova (1981) further hypothesized that Se prevents integration of the virus genome with the host's DNA. Similar observations were made earlier by both Billard and Peet (1974) and Oxford (1973) who reported that selenocystine and selenocystamine inhibited RNA-dependent RNA polymerase activity in influenza A_1, A_2 and B viruses. Schrauzer and Sacher (1994) also reported that Se causes symptomatic improvements and slows the course of the HIV disease possibly through the inhibition of reverse transcriptase activity in the HIV-virus-infected patients. Schrauzer and Sacher (1994) concluded that it is possible that Se might also prevent the replication of HIV and retard the development of AIDS in newly HIV-infected subjects. The depressive influence of Se on reverse transcriptase activity in RNA viruses is very important and suggests that Se is needed by the host to resist expression and possibly transmission of viruses.

In this study (data not shown in tables), Se increased the relative weight of the heart [Sel-Plex (0.512 g/100 g BW); sodium selenite (0.515 g/100 g BW); no Se (0.473 g/100 g BW),]. This positive effect on heart health is important because congestive heart disease is known to induce ascites, which can lead to mortality in modern, high-yielding broilers (Roch *et al*., 2000). Relative weights of the whole gastrointestinal tract were elevated in control and infected birds by feeding Sel-Plex (4.09 g/100 g BW; 4.25 g/100 g BW, respectively) compared with sodium selenite (3.58 g/100 g BW; 4.11 g/100 g BW, respectively) or no supplemental Se (3.80 g/100 g BW; 3.87 g/100 g BW, respectively). When the histomorphometry of the intestinal segments was examined, it was apparent that the muscle thickness of the ileum and duodenum, very large segments of the gastrointestinal tract, were not affected consistently by either of the dietary Se treatments. Selenium generally increased muscle thickness in the infected chickens. Whether this was a response leading to faster recovery from the ARV infection or for some other reason was not determined in this study. Nevertheless, it was shown in earlier work that selection for rapid growth caused an increase in mass of the small intestine (Mitchell and Smith 1991; O'Sullivan *et al*., 1992; Smith *et al*., 1990). Thus, in this study, our evidence suggested that Sel-Plex supplementation facilitated the expression of genetic potential in broiler chickens even when they were challenged by an enteric ARV infection.

One of the most impressive and consistent observations made in this histomorphometry study was that Sel-Plex generally maintained longer and more slender villi than either sodium selenite or no supplemental Se feeding. Histomorphometric analysis indicated that increased villus height and perimeter, as well as muscle thickness and crypt depth in the duodenum, jejunum and ileum were associated with Sel-Plex feeding compared with either sodium selenite or no Se (Table 3, 4 and 5). Muscle thickness generally was greater in the ileum than in the duodenum in both control and infected chickens, but muscle thickness was greatest in the jejunum segment compared with both duodenum and ileum.

A shortened villus height and a lower villus height to crypt depth ratio is directly correlated with increased enterocyte turnover (Fan *et al*., 1997). This condition was found in ARV-infected chickens and was most prominent in the birds fed sodium selenite or no supplemental Se in both control and ARV-infected groups. In the present study, analysis of those parameters strongly indicated that Sel-Plex feeding maintained the integrity of the intestinal tract as indicated by taller villi and an increased height to crypt depth ratio. Lower villus height to crypt depth ratios in both control and infected groups suggests that the increases in villus height and perimeter associated with Sel-Plex treatment are not associated with enterocyte turnover rates but instead with maintenance of the enterocyte population covering the villus. This maintenance effect may possibly be attributed to improved redox status in the gastrointestinal tract, which would result in less oxidative stress during normal and infective periods. An extension of this observation is that with an improved redox status, it is probably more difficult for the reovirus to replicate within the enterocytes of Sel-Plex-fed chickens.

Rebel *et al*. (2004) have described pathological changes associated with malabsorption syndrome in chickens as being characterized by degenerate epithelial cells in the crypt, villus atrophy and low villus height to crypt depth ratio similar to the condition associated with intestinal segments from no Se- and sodium selenite-fed chickens in this study. Thus, the improved weight gain after the peak infective state in the chicken fed Sel-Plex might be related to 1) greater surface area to volume ratio in the villi, 2) increased ability to assimilate

Table 4: Selenium (Torula yeast with No Se, Sel-Plex (Sel-Plex), or sodium selenite) and reovirus influence on morphology (mean± SEM of villus perimeter, height, width, crypt depth, villus height to crypt depth [H:D] ratio and muscle [muscularis externa] thickness) of jejunum in 21-d-old chickens

Parameter	Control No Se	Control Sel-Plex	Control Selenite	Infected No Se	Infected Sel-Plex	Infected Selenite
Villus, µm Perimeter	1889±54^{b}	2630±138^{a}	1649±82^{bc}	1658±86^{bc}	2293±176^{ab}	1589±45^{c}
Height	866±21^{a}	1264±67^{a}	740±29^{c}	782±28^{b}	1066±63^{a}	731±34^{c}
Width	135±11.1^{a}	93±5.2^{b}	134±10.2^{a}	137±13.4^{a}	108±8.6^{ab}	129±11.3^{a}
Crypt depth, µm	257±9.1^{a}	229±8.7^{ab}	256±10.2^{a}	264±10.3^{a}	224±8.4^{b}	279±12.6^{a}
H:D ratio	3.42±0.20^{c}	5.62±0.47^{a}	2.99±0.23^{c}	3.03±0.33^{cd}	4.75±0.44^{b}	2.64±0.27^{d}
Muscle, µm	390±12.8^{ab}	430±13.6^{a}	434±14.2^{a}	382±14.7^{b}	426±13.9^{a}	439±16.2^{a}

^{abc}In a row, means±SEM with unlike superscripts differ significantly, $p \leq 0.05$, n = 50 for each mean presented

Table 5: Selenium (Torula yeast with No Se, Sel-Plex (Sel-Plex), or sodium selenite) and reovirus influence on morphology (mean± SEM of villus perimeter, height, width, crypt depth, villus height to crypt depth [H:D] ratio and muscle [muscularis externa] thickness) of ileum in 21-d-old chickens

Parameter	Control No Se	Control Sel-Plex	Control Selenite	Infected No Se	Infected Sel-Plex	Infected Selenite
Villus, µm Perimeter	1872±43^{a}	1661±47^{b}	1434±54^{bc}	1340±74^{c}	1541±59^{b}	1451±37^{b}
Height	877±21^{a}	798±23^{a}	679±24^{b}	599±40^{c}	744±25^{ab}	602±53^{c}
Width	118±10.3^{b}	114±6.8^{b}	127±5.3^{b}	172±17.2^{ab}	135±17.4^{b}	217±19.1^{a}
Crypt depth, µm	235±8.8^{a}	130±8.6^{c}	182±12.3^{ab}	227±14.3^{a}	165±7.8^{b}	198±14.2^{a}
H:D ratio	3.73±0.32^{c}	6.11±0.52^{a}	3.73±0.32^{c}	2.64±0.22^{d}	4.51±0.38^{b}	3.04±0.26^{d}
Muscle, µm	391±13.7^{ab}	340±18.0^{b}	314±14.6^{b}	394±18.4^{ab}	426±13.8^{a}	416±18.2^{a}

^{abcd}In a row, means±SEM with unlike superscripts differ significantly, $p \leq 0.05$, n = 50 for each mean presented

nutrients compared with selenite- and no Se-fed birds, 3) decreased enterocyte cell death associated with oxidative stress, and 4) enhanced immunity during the infection.

Conclusion: In conclusion, it can be stated that dietary Se supplementation has improved performance of broiler chickens ever since it was introduced as an approved feed ingredient in 1974. Nevertheless, the advent of the modern broiler, which is a faster growing and significantly larger bird that the broiler of 1974, has placed it at risk for distress from increased oxidative stress due to its higher rate of muscle growth and metabolism. It has been demonstrated that it is possible to improve the performance of the modern broiler if a simple replacement of sodium selenite with Sel-Plex, which allows for development of an improved redox status (Edens, 2001). This study demonstrated that Sel-Plex also has a major influence on intestinal morphology, which facilitates the potential for improved nutrient assimilation. This potential is especially important for chickens that have been exposed to enteric viruses, such as the enteric reovirus, which has a limited time of residence but long-lasting effects on bird performance. As the bird begins to recover from the ARV infection about 14 d after viral challenge (Rebel *et al*., 2004), it is important to resume rapid and efficient assimilation of nutrients in the GIT. These results suggest that Sel-Plex, more so than sodium selenite, has the potential to improve nutrient assimilation in reovirus-infected chickens. These observations suggest that the improved performance of broiler chickens fed Sel-Plex can be partially explained by improved integrity of the intestinal tract and possibly by improved immune status.

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