The effect of hydrogen peroxide in the drinking water of newly weaned pigs

by

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ABSTRACT

Field reports indicate that a significant number of Iowa swine producers are using hydrogen peroxide, at the rate of 30 ppm, as a continuous treatment in the drinking water of newly weaned pigs. Producers are claiming benefits in the control and treatment of post-weaning diarrhea. To this date, there has not been research reported which either substantiates or disqualifies this claim, and addresses the safety of chronic administration of hydrogen peroxide to the newly weaned pig.

The purpose of this investigation was to determine the effects of hydrogen peroxide in the drinking water of newly weaned pigs. Immediately upon weaning, 351 pigs ranging in weight from 2.7 to 20.9 kg, were given either untreated water, or water treated with 58 ppm hydrogen peroxide, for a period of 35 days. At the end of the treatment period, 10 control pigs and 10 treatment pigs were examined at necropsy for evidence of pathologic changes.

The treatment had no significant effect on average daily feed intake, average daily gain, or on feed efficiency. In addition, there were no significant gastrointestinal findings revealed at necropsy.

INTRODUCTION

Hydrogen peroxide (H_2O_2) is a compound which is used in a variety of ways, including topical disinfection of wounds, oral cleansing, water purification, in the manufacture of organic chemicals, as a bleaching agent, and as an energy source (1). Currently, hydrogen peroxide is being used by a significant number of Iowa swine producers as a continuous water treatment in newly weaned pigs. Field reports indicate that most producers are purchasing 35 wt. % hydrogen peroxide and are using the hydrogen peroxide at a concentration of 30 ppm. Producers are reporting benefits in the control and treatment of post weaning diarrhea. To date, there has been no research performed which substantiates or disqualifies this claim, neither has there been research which addresses the safety of chronic hydrogen peroxide intake in the newly weaned pig. There are also certain safety concerns for the individual handling the hydrogen peroxide that need to be weighed against any perceived or real advantages of its use.

The purpose of this investigation was to determine the effects of hydrogen peroxide in the drinking water of the newly weaned pig. Parameters evaluated included average daily feed intake, average daily gain, feed efficiency, water quality, and pathologic changes in the pig.

LITERATURE REVIEW

Chemical Nature of Hydrogen Peroxide

Hydrogen peroxide (H_2O_2), a compound with a molecular weight of 34, is considered a weak acid. It is commercially available in a water base in various concentrations, ranging from 3 to 70 wt. percent. In dilute solutions, hydrogen peroxide has very little flavor or odor. In water, hydrogen peroxide exists as polymers and copolymers (H_2O_2, $(H_2O_2)_x$, $(H_2O)_y$, $(H_2O)_y*(H_2O_2)_x$) (1).

Hydrogen peroxide can be involved in a number of chemical reactions, including both oxidation and reduction reactions. In addition, spontaneous decomposition of hydrogen peroxide into water and oxygen can occur in the presence of an active catalyst, such as iron, copper, manganese, most other metals, dust, and alkaline substances as shown by the reaction below.

$$2H_2O_2 \dashrightarrow 2H_2O + O_2$$

This decomposition can be rapid and potentially destructive. Catalases, which are found in plants, nearly all animal cells and organs, and in almost all microorganisms except obligate anaerobes, can also serve as catalysts in the decomposition of hydrogen peroxide into water and oxygen. In the absence of a catalyst, hydrogen peroxide undergoes a slow rate of decomposition. The rate of decomposition is minimized at a pH of near 5 and increases as the pH moves in either direction. Temperature also has an effect on the rate of decomposition of

hydrogen peroxide, with decomposition occurring more rapidly with increasing temperatures (1). Peroxidases, which are found both in higher plants and in animal tissues and fluids, catalyze peroxidatic reactions involving a substrate and hydrogen peroxide. Some of the peroxidases found in animals include glutathione peroxidase, myeloperoxidase, lactoperoxidase, and salivary peroxidase (2).

Organic substances which are placed in concentrated hydrogen peroxide solutions may be set on fire, or the mixture may become detonatable. When concentrations of hydrogen peroxide approaching 50 wt. % are spilled on a combustible material, such as clothing, and allowed to dry, the hydrogen peroxide is concentrated and spontaneous inflammation can occur. It is recommended that goggles or a facemask be worn when working with concentrations of hydrogen peroxide above 3 to 5 wt. % (1).

Antimicrobial Mechanisms Involving Hydrogen Peroxide

To date, there has not been work published which involves the use of hydrogen peroxide as an antimicrobial in the drinking water of newly weaned pigs. However, there has been research concerning hydrogen peroxide which is applicable to the use of hydrogen peroxide in the pig. There are two potential mechanisms by which hydrogen peroxide may have a beneficial antimicrobial effect in the newly weaned pig, those being: 1. the peroxidase/thiocyanate/hydrogen peroxide

system, and 2. the direct inhibitory effect of hydrogen peroxide.

Peroxidase/thiocyanate/hydrogen peroxide system

The lactoperoxidase/thiocyanate/hydrogen peroxide system has been looked at extensively as a means of milk preservation, and has also received some attention as a potential mechanism to control enteric bacterial infections in calves. In addition, the salivary peroxidase/thiocyanate/hydrogen peroxide system has received interest as a means of promoting oral health in man.

All three components of the peroxidase/thiocyanate/hydrogen peroxide system can be found within the animal. There are several animal peroxidases, including lactoperoxidase, thyroid peroxidase, glutathione peroxidase, myeloperoxidase (found in phagocytic granules), and salivary peroxidases. Lactoperoxidase can be found in the milk of both swine and cattle (3). Peroxidase is also secreted by the salivary glands (4), and a peroxidase has been found in the mucosa of the pig intestine. This peroxidase appears to be bound within cell structures, and in all likelihood is an eosinophil peroxidase (5). Peroxidase is resistant to proteolytic activity (6) and low pH (7), but is inactivated by heating. It has been demonstrated that human salivary peroxidase is resistant to the action of gastric juices (7).

Milk has been found to contain thiocyanate, the levels of which depend upon the diet of the dam (8). Thiocyanate is also secreted in saliva (4), and it has been reported that thiocyanate is secreted in abomasal fluids (9).

Certain gram positive bacteria, including Lactobacillus lactis (10) and Streptococcus spp. (11), and animal host cells (phagocytic cells) (12) have been found to produce hydrogen peroxide in vivo.

At a neutral pH, and in the presence of peroxidase, thiocyanate is oxidized to hypothiocyanite ion by hydrogen peroxide as shown by the reaction below (4,13):

$$SCN^- + H_2O_2 \xrightarrow{\text{Peroxidase}} OSCN^- + H_2O$$

In the animal, the hypothiocyanite ion ($OSCN^-$), a highly reactive oxidizing agent, serves an antimicrobial function, and the reaction itself serves to prevent the accumulation of excess hydrogen peroxide which is potentially toxic to host cells (14). At a lower pH the reaction proceeds more readily with the major product being hypothiocyanous acid (HOSCN) (4). It is reported that hypothiocyanous acid may penetrate microbial cell membranes more readily than the hypothiocyanite ion (15), in which case the peroxidase/thiocyanate/hydrogen peroxide system would be expected to have a more potent antimicrobial effect at a lower pH.

It has been found that one of the first effects of the lactoperoxidase/thiocyanate/hydrogen peroxide system on

Escherichia coli is an inhibition of the energy-linked transport process of the organism (16). In further studies of the effects of the lactoperoxidase/thiocyanate/hydrogen peroxide system on Escherichia coli, it was found that the hypothiocyanite ion is involved in the oxidation of bacterial sulfhydryls to sulfenyl thiocyanate and sulfenic acid derivatives. This reaction results in the inhibition of cellular respiration. Following removal of the excess hypothiocyanite ion, the inhibition of cellular respiration is reversed, but with prolonged exposure to the hypothiocyanite ion, the inhibition of respiration becomes permanent. Antimicrobial action of the lactoperoxidase reaction is therefore not only determined by the amount of hypothiocyanite ion present but also by the time of exposure to the ion (17). In studies performed examining the effect of the lactoperoxidase/thiocyanate/hydrogen peroxide system on Salmonella typhimurium, it was found that the antimicrobial effects were dependent on the permeability of the bacterial cell envelope (18). This same relationship of cell permeability and bacterial inhibition by the lactoperoxidase/thiocyanate/hydrogen peroxide system has also been suggested to be involved in the killing of Escherichia coli (19). It has also been found that bacteria in the log phase of growth are more susceptible to the effects of the peroxidase system than are bacteria in the stationary phase (18).

The peroxidase/thiocyanate/hydrogen peroxide system has been found to be inhibitory to a number of bacteria, including Streptococcus spp. (20,21), Salmonella typhimurium (18,22,23), Salmonella dublin (23), Listeria monocytogenes (24), Pseudomonas fluorescens (25,26), Pseudomonas aeruginosa (22,27), and Escherichia coli (9,16,22,25,27). It has been observed that Escherichia coli possessing the K88 antigen lose the ability to attach to porcine brush border cells following exposure to the lactoperoxidase/thiocyanate/hydrogen peroxide system (3). The lactoperoxidase/thiocyanate/hydrogen peroxide system has been shown to be antimicrobial in bovine milk. In a study in which calves were fed milk containing glucose oxidase and glucose (a source of hydrogen peroxide in vivo), it was found that the abomasal fluid became bactericidal to Escherichia coli, however hydrogen peroxide forming lactic acid bacteria were not affected (9). Further studies demonstrated that the feeding of raw milk plus glucose and glucose oxidase to calves increased daily weight gain when compared to calves receiving only milk (28). However, in a separate study, calves being fed milk containing the lactoperoxidase system and experimentally infected with Salmonella typhimurium did not show any appreciable difference in clinical findings nor salmonella excretion patterns when compared to calves receiving milk only (23).

Direct Antimicrobial Effect of Hydrogen Peroxide

It has been determined that the direct antimicrobial effects of hydrogen peroxide are the result of the formation of superoxide ions and hydroxyl radicals (29,30). Hydrogen peroxide has been found to be bactericidal to Salmonella typhimurium in liquid whole egg (31), and in .1% peptone physiological saline at peroxide concentrations of .5% (5000 ppm) or higher. Poultry carcasses contaminated with Salmonella typhimurium also exhibited a decrease in bacterial numbers following treatment with .5% (5000 ppm) hydrogen peroxide. Hydrogen peroxide concentrations of .17% (1700 ppm) or less did not produce a lethal effect toward Salmonella typhimurium in .1% peptone physiological saline or on contaminated carcasses (32). It has been reported that .01% (100 ppm) hydrogen peroxide is effective in retarding the growth and gas production of Clostridium cultures (33). Studies have also shown that hydrogen peroxide added to poultry chiller water at concentrations of 5300 ppm or higher was effective in reducing the numbers of Escherichia coli by 97 to >99.9%, while hydrogen peroxide added at a concentration of 1100 ppm reduced counts by 20.7% (34).

Toxic Effects of Hydrogen Peroxide

The toxic effects of hydrogen peroxide range from the mild to the severe. "Hairy tongue", the development of hypertrophied filiform papillae of the tongue, has been seen

to occur following prolonged use of 1.5% (15000 ppm) hydrogen peroxide as a mouthwash. This condition has reversed itself following cessation of the use of hydrogen peroxide orally (35). More serious consequences of continued use of oral hydrogen peroxide have been reported in mice. Mice given .4% (4000 ppm) hydrogen peroxide as the sole source of water for up to 120 days developed multiple gastric and duodenal lesions. Gastric lesions were characterized by erosion and hyperplasia in the glandular portion of the stomach, and those in the duodenum by hyperplasia only. These lesions spontaneously regressed within 30 days following withdrawal of the hydrogen peroxide. Of the mice given .4 (4000 ppm) and .1% (1000 ppm) hydrogen peroxide for a period of 420 to 740 days, 5 and 1% respectively developed duodenal cancer (36).

The toxic effects of hydrogen peroxide on human fibroblasts at concentrations as low as 10 uM (.34 ppm) have been described. This toxicity is manifested as single strand breaks in cellular DNA with a subsequent loss of proliferating capacity of the cells. It is felt that the DNA damage is not the direct result of the hydrogen peroxide, but the result of a product of its reaction within the cell (14, 37).

The tumorigenicity of hydrogen peroxide may be the result of the formation of chemical linkages of various polycyclic hydrocarbons to DNA (38), or may be the result of a direct effect on DNA (39). Hydrogen peroxide has been shown to cause chain scission (cutting) of RNA and DNA both <u>in vivo</u> (cell

culture) and *in vitro* (40).

Decreased selenium dependent glutathione peroxidase activity in tissue may be seen following chronic intake of hydrogen peroxide. In a study in which rats were given .5% (5000 ppm) hydrogen peroxide as the source of drinking water for 8 weeks, decreased selenium dependent glutathione peroxidase activity was seen in skeletal muscle, kidney, and liver (41). In chicks given .45 mmole (15.3 ppm) hydrogen peroxide per day and increased by .1 mm (3.4 ppm) daily for 2 weeks growth rate was reduced as was glutathione peroxidase activity of the liver and plasma. These effects were related to a decreased selenium uptake and retention. Oral hydrogen peroxide may have an effect on dietary organic selenium compounds by oxidizing them to forms which are not as bioavailable, or there could be a detrimental effect on the intestinal mucosa resulting in a decreased rate of selenium absorption, thereby reducing the amount of selenium dependent glutathione peroxidase which is formed. This deficiency may lead to increased lipid peroxidation in tissues (42).

MATERIALS AND METHODS

Experimental Design and Animals

The research was conducted in a nursery on a local 200 sow farrow to finish operation near Ames, Iowa. The nursery consisted of 10 pens, providing 1.8 square feet per pig. There were five pens on a side, with solid dividers between adjacent pens, and wire flooring. Fresh air entered the nursery from the attic via one ceiling air inlet running the length of the center aisle. Two pit fans, one on each side of the building, served to exhaust air from the nursery. Water was provided by two nipple drinkers in each pen, and feed was provided by fenceline feeders which were split in order to measure feed disappearance by pen. Each pen was randomly assigned as either treatment or control, resulting in five treatment pens and five control pens. A total of 351 pigs (raised on site) ranging from 4 to 6 weeks of age were weaned, identified by ear tags, given ivermectin, and individually weighed (weights ranged from 2.7 to 20.9 kg). The pigs were ranked according to weight, and were assigned to either the treatment or control group in an alternating fashion. The first 35 treatment pigs were assigned to a treatment pen while the first 35 control pigs were assigned to a control pen. The next 35 treatment pigs were assigned to a treatment pen and so on. In this way the pig weights within a pen were similar, as were weights between the treatment and control pens. Nine

pens contained 35 pigs, while one pen contained 36 pigs. The pigs were fed a diet containing 20 percent protein.

Treatment

Immediately following weaning, the treatment pens received 58 ppm hydrogen peroxide continuously for 35 days, while the controls received untreated water. A water medicator, calibrated to deliver 1 gallon of stock solution to 128 gallons of water, was used to provide the treated water. Hydrogen peroxide (3%), diluted 1 part to 3 parts water, was used as the stock solution.

Measurements of Response

Water Quality

Both treated and control water samples were evaluated by the Iowa State Veterinary Diagnostic Laboratory for nitrates, sulfates, microtox (a test which evaluates the presence of substances inhibitory to microbial agents), iron (as atomic iron), coliforms, and total dissolved solids. In addition, the biochemical oxygen demand was determined for each sample by the City of Ames Water Pollution Control Plant using the procedure described in Standard Methods for the Examination of Water and Wastewater (43).

Oral and Fecal Bacterial Counts

Prior to the initiation of the treatment period, oral and fecal swabs were taken from twenty pigs. Two pigs were chosen at random from within each pen. At the termination of the treatment period, fecal and oral swabs were again taken from the same 20 pigs. Oral swabs were collected by inserting a cotton swab into the buccal cheek of the pig and rotating it 360 degrees. These swabs were then rinsed in 1 cc of sterile saline, after which 10 microliters of the saline was streaked onto Maconkey's agar. Rectal swabs were collected by inserting a cotton swab into the rectum of the pig and rotating it 360 degrees. These swabs were also rinsed in 1 cc of sterile saline. Ten microliters of the saline was then diluted with 1 cc of sterile saline. Ten microliters of this solution was then streaked onto Maconkey's agar. All plates were incubated for 24 hours, after which the number of bacterial colonies present was determined.

Post-mortem Examinations

At the conclusion of the treatment period, necropsies were performed on the 20 pigs selected for the oral and fecal bacterial counts.

Pig Performance

Pen feed disappearance was measured by subtracting the amount of feed left in the feeders at the end of the treatment period from the total amount of feed given to the pen over the 35 day period. The pigs were weighed at the beginning of the treatment period and again at the end of the treatment period. Average daily feed intake was calculated by dividing the total kilograms of feed attributed to pen feed disappearance by the number of pigs in the pen multiplied by the number of days of the treatment period. Feed efficiency was calculated as kilograms of feed consumed by the pen divided by the total kilograms of gain for the pen. Average daily gain was calculated by dividing the total kilograms of gain for the pen by the number of pigs in the pen multiplied by the number of days of the treatment period. In the event of pig death, the date of death, pig number, pen number, and weight of the pig were recorded. Performance figures were adjusted accordingly.

Statistical Analysis

The statistical analysis of the data was performed using the Statistical Analysis System (44). An analysis of variance procedure was performed on the data collected from the complete randomized block design. Probability values were calculated for feed efficiency, average daily gain, and average daily feed intake.

RESULTS

Clinical Observations

Clinical performance of the pigs did not appear to differ between the treatment and control groups. Two days after weaning, the smallest pigs began to exhibit diarrhea. Over the next few days, this diarrhea was seen in all of the pens except in the two pens containing the largest pigs (one control pen, and one treatment pen). Postmortem examinations and subsequent laboratory procedures revealed that the causative agent of the diarrhea was *Escherichia coli*. All pigs, except those in the two pens with the largest pigs, were treated with injectable antibiotics determined to be effective by *in vitro* sensitivity results. No difference in treatment response, duration of disease, nor severity of disease was seen between control and treatment pens. Twenty days into the treatment period, the two pens containing the largest pigs (one control, and one treatment pen) began to show signs of pneumonia. Pigs within these two pens were treated with injectable antibiotics found to be effective in similar previous episodes at this swine unit. Again, no difference in treatment response, duration of disease, nor severity of disease was seen between the treatment and control pens.

Subjective evaluation indicated that the level of activity, overall appearance, and thriftiness of the pigs did not differ between control and treatment pens.

Pathology Results

Twenty pigs, 10 control pigs and 10 treatment pigs, were necropsied 35 days following initiation of the treatment. Attention was directed towards the oral, esophageal, gastric, and duodenal mucosa. No gross lesions were discovered during the postmortem examinations of any of the pigs. Based upon these findings, it was determined that histopathological examinations were not indicated, and none were performed.

Pig Weights

The average beginning weight of pigs receiving the treated water was 7.9 kilograms with an average ending weight of 22.4 kg, while the average beginning weight of the pigs receiving the untreated water was also 7.9 kg with an average ending weight of 21.6 kg. The individual pen average beginning and ending weights and corresponding weight gains are shown in Table 1.

Table 1. Beginning and ending weights, and corresponding weight gains by pen number

Pen No.	Treatment/ Control	Beginning Wt. (kg)	Ending Wt. (kg)	Weight Gain (kg)
6	T	4.2	14.5	10.3
7	C	4.3	13.4	9.1
10	T	6.0	19.5	13.5
1	C	6.0	17.6	11.6
5	T	7.4	21.4	14.0
4	C	7.4	22.1	14.7

Table 1. (continued)

Pen No.	Treatment/ Control	Beginning Wt. (kg)	Ending Wt. (kg)	Weight Gain (kg)
9	T	8.9	25.1	16.2
3	C	8.9	23.9	15.0
8	T	13.0	31.4	18.4
2	C	12.9	31.2	18.3

Average Daily Gain

The average daily gain of the control pigs was .39 kg per pig per day, and that of the treatment pigs was .41 kg/hd/day. There was no significant difference in average daily gain between the treatment group and the control group (Pr>F=.31). The average daily gain for individual pens is shown in Table 2.

Average Daily Feed Intake

The average daily feed intake of the control pigs was .88 kg/hd/day, and that of the treatment pigs was .91 kg/hd/day. There was no significant difference in average daily feed intake between the control and treatment groups (Pr>F=.20). The average daily feed intake for individual pens is shown in Table 2.

Feed Efficiency

The mean feed efficiency of the control pigs was 2.26, and that of the treatment pigs was 2.20. There was no significant difference in feed efficiency between the control pigs and those receiving hydrogen peroxide (Pr>F=.28). The feed efficiency for individual pens is shown in Table 2.

Table 2. Average daily gain, feed efficiency, and daily feed intake by pen

Pen No.	Treatment/ Control	Average Daily Gain (kg)	Feed Efficiency	Average Daily Intake (kg)
6	T	.29	2.28	.66
7	C	.26	2.47	.65
10	T	.39	2.10	.82
1	C	.33	2.22	.73
5	T	.40	2.14	.86
4	C	.42	2.11	.89
9	T	.46	2.18	1.00
3	C	.43	2.25	.97
8	T	.52	2.30	1.20
2	C	.52	2.24	1.17

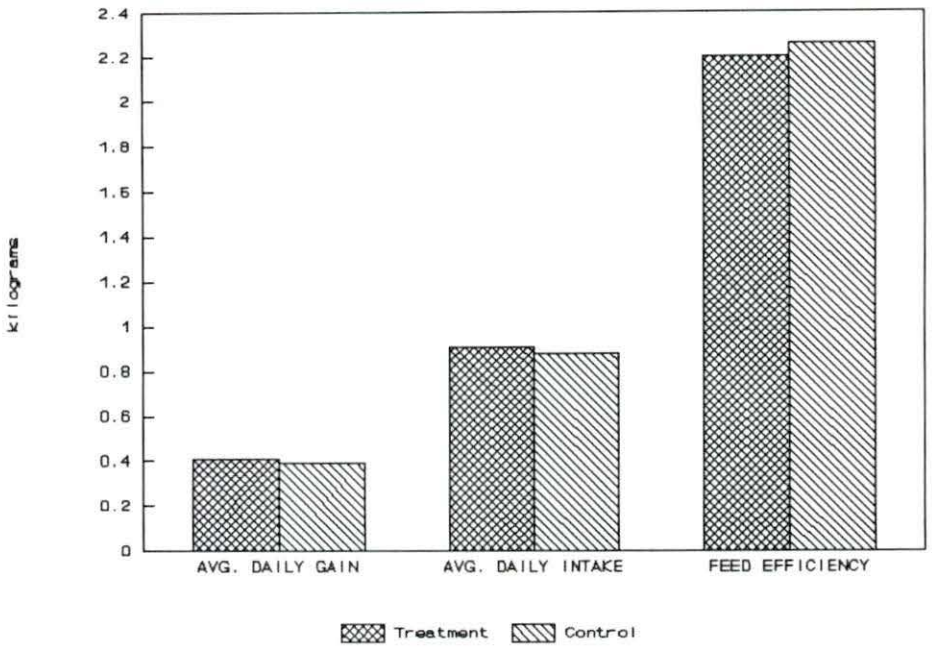

Figure 1. Average daily gain, average daily feed intake, and feed efficiency for treatment and control pigs

Water Analysis

Samples of both the treated and control water were evaluated for nitrates, sulfates, microtox, iron, total dissolved solids, coliforms, and biochemical oxygen demand. Hydrogen peroxide did not have a significant effect on any of the parameters evaluated. The results are listed in Table 3.

Table 3. Water analysis

	Control	Treatment
Nitrates (ppm of NO_2 & NO_3)	10	10
Sulfates (ppm of SO_4)	100	100
Microtox (% of control)	60	59
Iron (ppm)	2	1.8
Total Dissolved Solids (mg/L NaCl)	290	300
Coliforms (per 100 ml)	0	0
BOD (ppm)	<1.0	<1.0

Oral and Bacterial Fecal Counts

The bacterial counts are provided in the appendix. During collection of the swabs it became apparent that the technique used would not provide results which would be reliable, due to the inability to obtain standardized samples. Several factors contributed to the difficulty in collecting standardized samples, including: 1. the consistency of the fecal material varied from one pig to the next, 2. the amount of fecal material in the rectum was variable between pigs, and 3. the amount of saliva, feed, and water present in the mouth also varied among pigs.

Pig Deaths

During the treatment period 9 pigs died resulting in a mortality rate of 2.6%. The cause of death, days into the treatment period, and pen of origin are listed in Table 4.

Table 4. Pig deaths, beginning weight, days on trial, and postmortem diagnosis

Pen Number	Control/ Treatment	Beginning Wt. (kg)	Days on Trial	Diagnosis
9	T	8.6	1	colibacillosis
10	T	6.4	3	colibacillosis
10	T	6.4	4	colibacillosis
10	T	5.5	4	colibacillosis
3	C	9.1	5	colibacillosis
7	C	4.5	9	surgical complication
1	C	5.9	13	colibacillosis
10	T	6.4	22	colibacillosis
8	T	11.8	30	salmonellosis

DISCUSSION

The addition of hydrogen peroxide (58 ppm) to the drinking water of newly weaned pigs had no statistically significant effect on average daily feed intake, on average daily gain, or on feed efficiency. In addition, hydrogen peroxide induced no grossly observable pathologic changes in the pig digestive system. No attempts were made to draw conclusions from the oral and fecal bacterial counts due to the inability to obtain standardized samples. Of the 9 pigs which died during the treatment period, 6 were pigs receiving hydrogen peroxide. However, 4 of the 6 pigs were from the same pen. This indicates an environmental influence which contributed to the death loss in this particular pen.

The lack of effect of oral hydrogen peroxide on the newly weaned pig may be explained by several different theories. It may be that hydrogen peroxide has no effect whatsoever on the newly weaned pig. The literature review suggests potential mechanisms by which an effect is possible, but in vivo no effect was seen when hydrogen peroxide was administered in the drinking water at 58 ppm.

It should also be noted that this farm did not have coliforms in the drinking water (as shown by the water analysis). The lack of coliforms indicates that other pathogens were, in all likelihood, absent. Therefore, if the primary effect of the hydrogen peroxide was to be on bacterial contaminants in the water, this effect would have been

negated.

An important question that should be asked concerns the amount of hydrogen peroxide left in the water following delivery to the pig. Impurities in the water, such as iron and other metals, and the water lines themselves may have catalyzed the degradation of hydrogen peroxide into oxygen and water before the hydrogen peroxide ever reached the pig. It is quite conceivable that between the time the hydrogen peroxide was added to the water and the time it was consumed by the pig the hydrogen peroxide had undergone decomposition, and as such there was not enough present either to participate in the peroxidase/thiocyanate/hydrogen peroxide system, or to have a direct antimicrobial effect either in the oral cavity or the gastroduodenal area. In addition, pigs will tend to drink water immediately following the consumption of feed, and the presence of feed material in the mouth may contribute to the decomposition of hydrogen peroxide.

The hydrogen peroxide in the drinking water did not appear to have an effect on the water intake of the pig. Estimates of daily water usage, based on volume of hydrogen peroxide administered through the proportioner when compared to total body weight of pigs in treatment pens, indicated water consumption consistent with published expected levels. Further, production parameters (average daily gain, average daily feed intake, and feed efficiency) were not adversely affected by the treatment. These parameters would be

depressed if the treatment had limited water consumption.

Future research should be directed at quantifying the hydrogen peroxide delivered to the pig, as well as the peroxidase and thiocyanate present in the pig digestive tract. The activity of the peroxidase/thiocyanate/hydrogen peroxide system in the pig should also be studied. In addition, evaluating the effect of higher concentrations of hydrogen peroxide on the newly weaned pig, as well as the effects of various concentrations of hydrogen peroxide in water on swine enteric pathogens *in vitro*, would be of value. The absorption into the body of the products resulting from the chemical reactions involving hydrogen peroxide, occurring both in the pig and the water delivery systems, also needs to be studied.

In conclusion, hydrogen peroxide, at a concentration of 58 ppm in the drinking water, had neither a toxic or beneficial effect on the newly weaned pig as measured by production parameters and gross pathological examination.

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APPENDIX

Weaning, 35 day weights, and weight gain in kg.

Pen Number	Pig No.	Weaning Wt (kg)	35 day Wt (kg)	Weight Gain (kg)
1	87	5.5	15.5	10.0
1	90	6.8	16.8	10.0
1	93	5.9	12.3	6.4
1	96	5.9	16.4	10.5
1	108	5.9	17.7	11.8
1	110	5.9	13.6	7.7
1	114	5.5	12.7	7.3
1	115	5.5	14.1	8.6
1	133	5.0	16.4	11.4
1	149	5.5	17.7	12.3
1	178	5.9	19.1	13.2
1	182	5.9	17.7	11.8
1	190	5.9	died	
1	193	6.8	21.4	14.5
1	211	6.8	20.0	13.2
1	220	6.4	21.4	15.0
1	236	5.5	18.2	12.7
1	239	5.9	19.1	13.2
1	241	6.4	18.6	12.3
1	243	6.4	17.3	10.9
1	246	5.0	16.4	11.4
1	247	6.4	17.7	11.4
1	258	5.9	18.6	12.7
1	266	6.4	18.2	11.8
1	271	6.4	20.0	13.6
1	275	6.8	23.6	16.8
1	278	5.9	13.6	7.7
1	291	6.4	19.5	13.2
1	300	5.9	16.8	10.9
1	307	5.5	12.7	7.3
1	308	6.4	22.7	16.4
1	313	6.4	17.3	10.9
1	334	5.5	14.5	9.1
1	345	6.4	19.1	12.7
1	347	6.4	22.3	15.9
2	2	10.5	29.1	18.6
2	6	11.8	29.1	17.3
2	7	11.4	30.5	19.1
2	9	11.8	27.3	15.5
2	10	10.5	30.0	19.5
2	14	10.9	30.0	19.1

Pen Number	Pig No.	Weaning Wt (kg)	35 Day Wt (kg)	Weight Gain (kg)
2	15	12.7	31.8	19.1
2	16	13.6	32.7	19.1
2	19	11.8	31.8	20.0
2	21	10.9	32.7	21.8
2	22	18.2	40.9	22.7
2	26	12.7	30.0	17.3
2	31	13.6	30.9	17.3
2	34	12.7	31.8	19.1
2	36	19.5	40.9	21.4
2	38	14.5	32.3	17.7
2	39	12.7	30.0	17.3
2	42	15.5	35.5	20.0
2	44	10.9	30.9	20.0
2	55	11.8	33.2	21.4
2	56	15.5	34.1	18.6
2	59	13.6	32.7	19.1
2	64	14.5	37.3	22.7
2	68	12.7	30.9	18.2
2	71	11.8	30.9	19.1
2	80	14.5	32.7	18.2
2	81	10.9	30.0	19.1
2	82	13.6	32.7	19.1
2	322	15.0	25.5	10.5
2	329	10.5	26.4	15.9
2	333	11.8	25.5	13.6
2	340	11.4	26.4	15.0
2	342	12.3	30.0	17.7
2	343	13.2	28.2	15.0
2	350	10.5	27.3	16.8
3	4	9.1	23.6	14.5
3	58	8.2	23.6	15.5
3	61	10.0	33.6	23.6
3	150	9.1	18.2	9.1
3	156	10.0	25.5	15.5
3	159	8.2	23.2	15.0
3	161	8.2	23.6	15.5
3	163	8.2	20.9	12.7
3	168	9.1	22.7	13.6
3	205	9.1	21.4	12.3
3	255	8.6	24.5	15.9
3	268	8.2	23.2	15.0
3	274	8.6	22.7	14.1
3	276	9.5	25.5	15.9
3	281	8.2	17.3	9.1
3	287	8.6	22.7	14.1

Pen Number	Pig No.	Weaning Wt (kg)	35 Day Wt (kg)	Weight Gain (kg)
3	288	8.6	21.8	13.2
3	290	9.5	25.9	16.4
3	297	9.5	22.7	13.2
3	298	8.2	20.5	12.3
3	302	8.2	23.6	15.5
3	303	8.2	17.3	9.1
3	309	9.5	26.4	16.8
3	311	9.1	27.7	18.6
3	314	10.0	24.1	14.1
3	318	8.6	22.7	14.1
3	320	8.6	18.2	9.5
3	332	9.1	28.6	19.5
3	335	10.0	29.1	19.1
3	337	8.6	23.6	15.0
3	346	8.6	26.4	17.7
3	353	9.1	died	
3	355	9.1	27.3	18.2
3	358	10.0	25.5	15.5
3	359	8.6	28.2	19.5
4	11	6.8	24.5	17.7
4	129	7.3	21.4	14.1
4	147	6.8	21.8	15.0
4	152	7.3	18.2	10.9
4	153	8.2	23.6	15.5
4	165	7.3	23.2	15.9
4	172	7.7	22.7	15.0
4	174	7.3	26.4	19.1
4	181	7.3	25.0	17.7
4	184	7.3	26.8	19.5
4	186	7.3	22.3	15.0
4	189	6.8	23.2	16.4
4	194	7.7	22.7	15.0
4	196	6.8	19.5	12.7
4	198	6.8	20.0	13.2
4	199	7.7	18.6	10.9
4	201	6.8	17.3	10.5
4	204	6.8	20.5	13.6
4	208	6.8	20.5	13.6
4	209	6.8	20.0	13.2
4	217	7.7	25.5	17.7
4	223	7.7	21.4	13.6
4	226	7.7	25.5	17.7
4	228	6.8	21.4	14.5
4	232	7.3	22.7	15.5
4	245	7.3	23.2	15.9

Pen Number	Pig No.	Weaning Wt (kg)	35 Day Wt (kg)	Weight Gain (kg)
4	263	7.3	20.0	12.7
4	264	7.7	21.8	14.1
4	272	8.2	22.7	14.5
4	282	8.2	21.8	13.6
4	306	7.7	20.0	12.3
4	317	7.7	24.5	16.8
4	323	7.7	19.1	11.4
4	330	7.3	23.6	16.4
4	352	7.7	22.3	14.5
5	5	8.2	25.5	17.3
5	54	7.7	23.6	15.9
5	65	8.2	21.8	13.6
5	123	6.8	14.5	7.7
5	139	6.8	17.3	10.5
5	148	6.8	19.5	12.7
5	162	7.3	20.0	12.7
5	175	7.7	22.7	15.0
5	179	6.8	23.2	16.4
5	180	7.7	20.9	13.2
5	187	6.8	21.8	15.0
5	192	7.3	23.2	15.9
5	197	8.2	25.0	16.8
5	206	7.3	20.9	13.6
5	221	6.8	17.3	10.5
5	222	7.7	23.6	15.9
5	230	7.3	20.9	13.6
5	248	6.8	23.6	16.8
5	249	6.8	19.1	12.3
5	250	7.7	23.6	15.9
5	260	6.8	23.2	16.4
5	261	7.7	21.4	13.6
5	262	7.3	16.8	9.5
5	269	7.3	21.8	14.5
5	270	7.3	19.5	12.3
5	280	7.3	21.8	14.5
5	285	7.3	20.9	13.6
5	299	7.3	18.6	11.4
5	312	7.7	25.5	17.7
5	316	7.7	24.5	16.8
5	325	7.7	23.2	15.5
5	327	7.7	22.3	14.5
5	328	7.7	20.0	12.3
5	336	6.8	20.5	13.6
5	344	7.3	20.9	13.6
6	84	3.6	10.0	6.4

Pen Number	Pig No.	Weaning Wt (kg)	35 Day Wt (kg)	Weight Gain (kg)
6	88	3.2	6.4	3.2
6	95	2.7	9.1	6.4
6	97	4.1	10.9	6.8
6	98	5.0	15.9	10.9
6	102	4.1	10.9	6.8
6	103	4.5	15.0	10.5
6	104	4.5	15.5	10.9
6	107	4.1	14.5	10.5
6	111	3.6	9.5	5.9
6	113	5.0	15.9	10.9
6	116	3.2	12.3	9.1
6	118	5.0	15.9	10.9
6	125	4.1	13.2	9.1
6	132	4.5	17.3	12.7
6	138	4.1	11.8	7.7
6	142	4.1	12.7	8.6
6	143	4.5	13.6	9.1
6	145	4.5	13.2	8.6
6	151	4.5	15.9	11.4
6	173	4.5	18.2	13.6
6	176	5.0	17.3	12.3
6	188	4.1	18.2	14.1
6	191	4.5	16.8	12.3
6	200	4.5	19.1	14.5
6	231	4.5	16.8	12.3
6	233	4.5	17.3	12.7
6	235	5.0	17.3	12.3
6	240	5.0	20.0	15.0
6	251	5.0	17.3	12.3
6	252	3.2	13.2	10.0
6	257	3.2	11.4	8.2
6	277	4.1	14.5	10.5
6	304	4.1	12.7	8.6
6	315	5.0	21.8	16.8
7	83	4.5	13.6	9.1
7	85	4.5	14.5	10.0
7	86	4.5	12.7	8.2
7	89	4.1	11.8	7.7
7	91	4.5	died	
7	92	4.1	11.4	7.3
7	99	4.1	11.4	7.3
7	100	4.1	11.8	7.7
7	101	3.2	8.6	5.5
7	105	5.0	15.5	10.5
7	112	4.5	13.6	9.1

Pen Number	Pig No.	Weaning Wt (kg)	35 Day Wt (kg)	Weight Gain (kg)
7	117	4.1	16.4	12.3
7	124	3.2	7.3	4.1
7	126	5.0	13.6	8.6
7	127	4.5	17.3	12.7
7	130	4.5	14.5	10.0
7	131	4.5	10.9	6.4
7	134	5.0	15.0	10.0
7	135	4.5	10.9	6.4
7	136	5.0	16.4	11.4
7	140	4.1	14.5	10.5
7	141	3.2	9.1	5.9
7	144	4.5	17.3	12.7
7	146	4.1	12.3	8.2
7	183	3.6	17.3	13.6
7	185	5.0	18.2	13.2
7	215	4.1	13.6	9.5
7	242	3.6	16.4	12.7
7	253	5.0	14.1	9.1
7	254	5.0	13.2	8.2
7	256	2.7	10.0	7.3
7	259	4.5	16.4	11.8
7	294	4.5	11.4	6.8
7	295	3.2	10.5	7.3
7	321	5.0	14.5	9.5
8	1	12.3	35.0	22.7
8	8	10.5	20.9	10.5
8	12	11.8	30.9	19.1
8	23	14.1	29.1	15.0
8	25	11.8	25.9	14.1
8	27	10.9	29.5	18.6
8	29	14.5	36.4	21.8
8	30	11.4	33.6	22.3
8	32	13.6	27.3	13.6
8	33	11.8	32.3	20.5
8	35	18.2	35.5	17.3
8	40	20.9	39.5	18.6
8	41	14.5	36.4	21.8
8	43	11.8	32.3	20.5
8	46	10.5	30.9	20.5
8	47	11.8	33.2	21.4
8	48	13.6	40.9	27.3
8	50	15.5	37.3	21.8
8	52	13.6	20.0	6.4
8	53	11.8	33.6	21.8
8	62	12.7	20.9	8.2

Pen Number	Pig No.	Weaning Wt (kg)	35 Day Wt (kg)	Weight Gain (kg)
8	63	13.6	32.7	19.1
8	69	12.7	30.9	18.2
8	70	12.7	30.9	18.2
8	72	10.9	31.8	20.9
8	73	12.7	28.6	15.9
8	76	17.3	37.3	20.0
8	77	12.7	35.5	22.7
8	78	15.0	35.5	20.5
8	79	15.5	38.2	22.7
8	293	10.9	27.3	16.4
8	326	10.9	28.6	17.7
8	351	11.4	27.3	15.9
8	354	10.5	20.0	9.5
8	360	10.9	26.4	15.5
9	18	9.5	34.1	24.5
9	20	10.0	30.5	20.5
9	24	9.1	25.9	16.8
9	28	10.0	30.9	20.9
9	45	9.1	31.8	22.7
9	51	9.5	23.6	14.1
9	57	8.6	22.7	14.1
9	66	8.2	20.5	12.3
9	67	8.2	24.5	16.4
9	74	9.1	30.9	21.8
9	154	10.0	26.4	16.4
9	164	8.2	20.9	12.7
9	166	9.1	20.5	11.4
9	167	10.0	28.2	18.2
9	169	10.0	27.7	17.7
9	171	9.1	25.9	16.8
9	195	8.6	21.8	13.2
9	207	8.2	18.2	10.0
9	210	8.6	died	
9	212	8.2	21.8	13.6
9	213	8.2	20.9	12.7
9	218	8.6	27.3	18.6
9	229	8.6	24.1	15.5
9	265	8.6	18.6	10.0
9	267	9.5	23.6	14.1
9	283	9.5	26.4	16.8
9	284	8.2	23.6	15.5
9	310	8.6	24.1	15.5
9	319	9.1	25.0	15.9
9	324	9.5	24.5	15.0
9	331	8.6	25.5	16.8

Pen Number	Pig No.	Weaning Wt (kg)	35 Day Wt (kg)	Weight Gain (kg)
9	338	9.1	28.2	19.1
9	339	8.6	23.2	14.5
9	341	8.2	25.5	17.3
9	349	8.6	27.3	18.6
10	3	5.5	17.3	11.8
10	13	6.8	27.3	20.5
10	17	6.4	27.3	20.9
10	94	5.9	17.7	11.8
10	119	6.4	19.5	13.2
10	120	5.5	10.9	5.5
10	121	5.5	14.1	8.6
10	137	5.0	15.0	10.0
10	155	6.4	20.9	14.5
10	158	5.5	16.4	10.9
10	160	6.4	died	
10	170	5.5	19.1	13.6
10	177	6.4	22.7	16.4
10	202	6.4	died	
10	203	5.9	18.2	12.3
10	214	5.9	20.9	15.0
10	216	5.5	19.1	13.6
10	219	6.4	died	
10	224	6.8	25.0	18.2
10	225	6.8	24.1	17.3
10	227	5.9	17.7	11.8
10	234	6.8	19.1	12.3
10	237	5.5	20.5	15.0
10	238	6.4	20.9	14.5
10	244	5.0	18.2	13.2
10	273	5.5	died	
10	279	5.9	20.9	15.0
10	286	6.4	22.7	16.4
10	289	5.9	15.9	10.0
10	292	5.9	20.0	14.1
10	296	5.9	17.3	11.4
10	301	5.9	15.5	9.5
10	305	5.9	18.6	12.7
10	348	6.4	19.1	12.7
10	356	5.9	21.4	15.5

Per swab bacterial colony counts for control and treatment (58 ppm hydrogen peroxide) pigs at weaning and 35 days later

Pig Number	Control/ Treatment	Weaning Oral	35 Days Oral	Weaning Fecal	35 Days Fecal
10	C	883	462	10	4
117	C	11	0	119	0
204	C	9	628	29	20
245	C	31	369	10	0
246	C	83	264	14	219
259	C	299	42	857	0
276	C	0	314	210	71
313	C	5	701	65	11
322	C	39	28	443	1556
346	C	1	113	112	23
40	T	4	116	325	134
46	T	0	83	15	6
121	T	594	283	146	762
176	T	3	11	17	42
207	T	450	399	1091	570
231	T	344	2	134	22
289	T	438	1070	1004	21
327	T	120	54	NO COLL.	157
344	T	472	199	52	1
349	T	73	25	82	254