

EFFECT OF ADMINISTERING SODIUM CHLORATE IN DRINKING WATER ON *SALMONELLA* TYPHIMURIUM CONCENTRATIONS IN WEANED AND FINISHED PIGS

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Salmonella can cause disease and compromise food safety. Consequently, strategies are sought to reduce colonization of food animals by these pathogens. Because *Salmonella*, like most enterobacteria, possesses respiratory nitrate reductase activity, which coincidentally reduces chlorate to cytotoxic chlorite, we hypothesized that chlorate may selectively kill these pathogens but not beneficial gut anaerobes lacking this activity. Whereas we have since shown that oral gavage of sodium chlorate to weaned pigs does kill *Salmonella* in the gut, we now report results from experiments evaluating the efficacy of administering chlorate in drinking water, which is a more practical application of this technology. In our first experiment, weaned pigs orally infected with 10^8 colony forming units (CFU) of a novobiocin and nalidixic acid resistant *Salmonella* Typhimurium were allowed ad libitum access 24 h later to drinking water containing either 0 (0X treatment) or 15 mM (1X treatment) sodium chlorate. In order to promote expression of respiratory nitrate reductase activity by *Salmonella* and to provide at least a minimum amount of available reductant, the drinking water treatments also contained 2.5 mM sodium nitrate and 20 mM sodium lactate. The pigs were euthanized 24 and 36 h after being allowed access to the drinking water treatments and gut contents collected at necropsy were cultured quantitatively for the challenge *Salmonella* strain via direct plating of serial ten-fold dilutions to Brilliant Green Agar (BGA) supplemented with 25 and 20 μ g novobiocin and naladixic acid mL^{-1} , respectively. For enhanced sensitivity, the gut contents were also cultured qualitatively via initial enrichment in tetrathionate broth, further enrichment in Rappaport-Vassiliadis broth and selective differentiation on the antibiotic supplemented BGA. Incubation steps were performed at 37°C for 18 to 24 h. For the pigs provided 36 h access to the 1X chlorate treatment, cecal and rectal *Salmonella* concentrations were reduced ($P < 0.05$) compared to concentrations found in 0X-treated pigs. For instance, mean \pm SD ($n = 8$) cecal and rectal *Salmonella* concentrations were 3.3 ± 2.2 and 1.3 ± 1.4 log base 10 CFU/g, respectively, in 0X-treated pigs and were 0.6 ± 1.1 and 0.1 ± 0.4 log base 10 CFU/g, respectively, in contents from the 1X-treated pigs. Gut concentrations of *Salmonella* were also reduced ($P < 0.05$), but less so, in pigs provided 24 h access to water containing the 1X chlorate treatment. In the second experiment, similarly challenged finished pigs (6 pigs per treatment) were in this case provided ad libitum access 24 h post challenge to drinking water containing 0X, 1X or 2X sodium chlorate (0, 15 or 30 mM, respectfully) plus the nitrate and lactate. For these pigs, provision of the 1X or 2X drinking water treatments for 24 h resulted in reductions of 2.1 and 1.4 log units in cecal *Salmonella* concentrations, respectively, when compared to concentrations in 0X-treated pigs (3.8 ± 0.7 log base 10 CFU/g). *Salmonella* concentrations in rectal contents from pigs provided the 1X and 2X treatments were reduced 0.6 and 1.3 log units, respectfully, compared to concentrations found in 0X-treated pigs (1.9 ± 1.6 log base 10 CFU/g). No effect ($P > 0.05$) on water consumption (34 to 40 mL/kg body wt) was observed. These results demonstrate the practicality of administering sodium chlorate as a drinking water supplement to reduce gut concentrations of *Salmonella* in weaned and finished pigs.