Effects of a Quillaja saponaria extract on growth performance and immune function of weanling pigs challenged with Salmonella typhimurium

J. L. Turner2,3, S. S. Dritz4, J. J. Higgins5, K. L. Herkelman6, and J. E. Minton2,7

Kansas State University, Manhattan 66506

ABSTRACT: Ninety-six pigs (initially 8.9 kg and 24 d of age) were used in a 28-d experiment to determine the effects of Quillaja saponaria extract (QS) on weanling pig growth performance and immune function in response to enteric disease challenge with Salmonella typhimurium (ST). Experimental treatments were arranged in a 2 × 4 factorial with main effects of disease challenge (control vs ST-challenge) and dietary addition of QS (0, 125, 250, or 500 mg/kg). Pigs were fed QS diets for 14 d and then challenged orally with ST or sterile media. There were no differences in ADG or ADFI among dietary treatments, but gain/feed ratio (G/F) was depressed (P < 0.06) in pigs fed 250 mg/kg QS. ST-challenge reduced ADG (P < 0.05), ADFI (P < 0.05), and G/F (P < 0.05) 1 wk after challenge. Daily estimates revealed reductions in feed intake in ST-infected pigs on d 2 to 5 following infection (P < 0.05), and rectal temperature was increased maximally 2 d following infection (P < 0.05). There was a marked decline in serum IGF-I during the 6 d after ST-infection (P < 0.05). ST-challenge produced a rise (P < 0.05) in serum haptoglobin on d 7 after challenge, and serum α1-acid glycoprotein (AGP) in ST-challenged pigs also was elevated (P < 0.05) above controls on d 7 and 14 after challenge. Serum immunoglobulin (Ig) M increased (P < 0.05) over time in both groups, and serum IgM of ST-challenged pigs was greater than controls on d 7 after challenge (P < 0.05). Serum IgG was not affected by enteric disease challenge; however, on d 7 and 14 after disease challenge, serum IgG for both groups was greater (P < 0.05) than on d 0. Dietary QS had no significant influence on any of the end points used to characterize the acute phase response to ST-challenge. Phagocytic cell function was depressed in pigs fed 250 (P < 0.05) and 500 (P < 0.05) mg/kg as compared to pigs fed 125 mg/kg QS. Yet, there was no difference in phagocytic function among pigs fed 0, 250, or 500 mg/kg QS. We conclude that this model of enteric disease invokes an acute phase response accompanied by decreases in feed intake and serum IGF-I. Furthermore, dietary QS, at the levels fed in this study, appears to offer little benefit to growth performance or immune function in the presence or absence of ST-challenge.

Key Words: Disease Resistance, Piglets, Quillaja, Salmonella

Introduction

The popular press and empirical evidence have suggested that plant extracts may offer benefits in terms of boosting the immune system and preventing disease. Furthermore, there is growing sentiment among scientists and the general public to find alternatives to the use of feed-grade antibiotics to promote growth and prevent disease in food animal production systems.

An extract of the South American tree Quillaja saponaria (QS) has been widely used over the past three decades as a vaccine adjuvant (Kensil, 1996). The active ingredient appears to be the saponin fraction (Milgate and Roberts, 1995). Recent studies have shown that saponins can inhibit in vitro growth of Escherichia coli (Sen et al., 1998), and saponins alter the rumen microflora in vivo (Killeen et al., 1998). Cromwell et al. (1985) reported diets containing 62 or 125...
**Table 1. Diet composition**

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>% of diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn</td>
<td>51.72</td>
</tr>
<tr>
<td>Soybean meal (46.5% CP)</td>
<td>27.86</td>
</tr>
<tr>
<td>Spray-dried wheat</td>
<td>10.00</td>
</tr>
<tr>
<td>Select menhaden fish meal</td>
<td>4.50</td>
</tr>
<tr>
<td>Choice white grease</td>
<td>3.00</td>
</tr>
<tr>
<td>Monocalcium phosphate</td>
<td>1.20</td>
</tr>
<tr>
<td>Limestone</td>
<td>0.68</td>
</tr>
<tr>
<td>Salt</td>
<td>0.35</td>
</tr>
<tr>
<td>Vitamin premix</td>
<td>0.25</td>
</tr>
<tr>
<td>Trace mineral premix</td>
<td>0.15</td>
</tr>
<tr>
<td>L-Lysine HCl</td>
<td>0.15</td>
</tr>
<tr>
<td>Cornstarch</td>
<td>0.10</td>
</tr>
<tr>
<td>DL-Methionine</td>
<td>0.05</td>
</tr>
</tbody>
</table>

*a* Diet was formulated to contain 1.40% lysine, 0.90% Ca, and 0.79% P.

*b* Provided the following per kilogram of complete diet: vitamin A, 11,023 IU; vitamin D₃, 1,654 IU; vitamin E, 44 IU; menadione (menadione sodium bisulfate complex), 4.4 mg; riboflavin, 9.9 mg; pantothentic acid, 33 mg; niacin, 55 mg; vitamin B₁₂, 44 mcg.

*c* Provided the following per kilogram of complete diet: Mn, 40 mg; Fe, 165 mg; Zn, 165 mg; Cu, 17 mg; I, 298 mcg; Se, 298 mcg.

*d* Quillaja saponaria extract replaced cornstarch to provide the experimental treatments.

Samples were obtained and forwarded to the Kansas State University Veterinary Diagnostic Laboratory to ensure that all pigs were not shedding *Salmonella*. Pigs were weighed and feed disappearance was measured on d 0, 7, 14, 21, and 28 to determine ADG, ADFI, and gain/feed ratio (G/F). On d 14, each pig housed in the ST room (n = 48) received approximately $10^8$ cfu of *S. typhimurium* in 10 mL of tryptic soy broth (# T-8907 Sigma Chemical, St. Louis, MO). Each pig housed in the control room (n = 48) received a similar volume of sterile growth broth. Rectal temperature was measured on one pig per pen through 7 d after challenge. Daily feed intake also was monitored through 7 d after challenge. On d 0, 7, and 14 with respect to disease challenge, serum samples were obtained from one pig per pen and analyzed for haptoglobin, α₁-acid glycoprotein (AGP), immunoglobulin M (IgM), and immunoglobulin G (IgG) concentrations.

On d 7 and 14 after challenge, fecal samples were obtained from all pigs and cultured for *Salmonella* at the Kansas State University Veterinary Diagnostic Laboratory. On d 0, 2, 4, and 6 after challenge, serum samples were obtained from one pig per pen and analyzed for IGF-I.

**Experimental Procedures**

**Experimental Design.** The experimental protocol used in this study was approved by the KSU Institutional Animal Care and Use Committee. A total of 96 pigs (initially 8.9 kg and approximately 24 d of age) were blocked by initial weight, equalized for sex, and randomly allotted to one of eight treatments in a 28-d experiment. Each treatment had six replicates (pens) with two pigs/pen. The eight treatments were arranged in a $2^3$ factorial with main effects of disease challenge (control or ST) and dietary treatment (Table 1; 0, 125, 250, or 500 mg/kg). The QS extract used in this study was provided by Desert King International, Chula Vista, California. The range of dietary QS inclusion was based on unpublished preliminary data in poultry as recommended by the supplier.

All pigs were housed under constant illumination in two similar environmentally controlled rooms, according to disease challenge. Pens contained one self-feeder and one nipple waterer to provide ad libitum access to feed and water. Preceding the study, fecal samples were obtained and analyzed for IGF-I.

**Serum Analyses.** Blood was collected into glass tubes containing no anticoagulant. Blood was allowed to clot at room temperature and then was stored at 4°C overnight prior to harvest of serum by centrifugation. Serum was analyzed for haptoglobin using a colorimetric, enzymatic assay as described previously (Smith et al., 1998). Serum AGP was measured via radial immunodiffusion assay (Cardiotech Services, Louisville, KY). Serum IGF-I was determined via immunoradiometric assay as described previously for use in pigs (Balaji et al., 2000). Serum IgG and IgM concentrations were determined with a commercially available ELISA (Bethyl Labs, Montgomery, TX). Serum was diluted 1:100,000 and 1:10,000 with assay buffer for analysis of IgG and IgM, respectively.

**Phagocytosis Assay Reagents.** *Salmonella* typhimurium cultures that were used for inoculating pigs were formalin-killed, and the concentration (cfu/mL) was determined using a spectrophotometer. An approximate volume of ST-suspension to yield $3.3 	imes 10^9$ cfu was centrifuged at $16,600 \times g$ for 30 s to pellet bacteria. The supernate was aspirated and bacteria were resuspended in 0.1 M carbonate buffer (pH = 9.6) containing $100 \mu$g/mL of propidium iodide (Calbiochem-Novabiochem Corp., San Diego, CA). This suspension was vortexed and incubated overnight in the dark at 4°C. The suspension was centrifuged at $16,600 \times g$ for 30 s to pellet bacteria, and the pellet was washed three times with Hank's balanced salt solution (HBSS #14185052, Life Technologies, Rockville, MD). Propidium iodide-labeled salmonella (PILS) were opsonized with 1 mL of 40% pig serum in HBSS for 30 min in the dark at 37°C. Labeled bacteria were washed twice with HBSS and resuspended in sufficient volume to give $3.3 	imes 10^9$ cfu/mL. This concentation of ST gives an approximate
ratio of 20 ST:1 neutrophil, which was determined to be the most effective ratio in a preliminary trial. The opsonized PILS were stored at 4°C in the dark and used the following day in the phagocytosis assay.

Dihydrorhodamine-123 (DHR; Molecular Probes, Inc., Eugene, OR) was used to measure the oxidative burst of phagocytes. A stock solution of DHR (29 mM in HBSS) was prepared and stored in 25-μL aliquots at −20°C. Just before use, a working solution of DHR was prepared by diluting 25 μL of stock solution with 10 mL of HBSS, and this solution was kept in the dark until used.

The monoclonal antibody GM1 (VMRD, Inc., Pullman, WA) was used to label granulocytes and monocytes in two control samples each day. For a working solution of GM1, 10 μL of GM1 was diluted with 990 μL of PBS containing 2% BSA and 0.2% sodium azide. The secondary antibody, recognizing GM1, was an anti-mouse FITC-labeled antibody (#M32101, Caltag Laboratories, Burlingame, CA). The secondary antibody working solution was prepared by adding 25 μL FITC-labeled secondary antibody to 975 μL of PBS containing 2% BSA and 0.2% sodium azide. Once diluted, the antibody solutions were stored at 4°C in the dark until used.

Phagocytosis Assay Procedure. On d 6 and 13 after ST challenge, blood from one randomly chosen pig per pen of the ST-challenged pigs was collected into heparinized tubes and immediately placed on ice for transport to the laboratory. At the laboratory, 100 μL of whole blood was incubated with 20 μL of DHR-working solution at 37°C in a shaking water bath for 20 min, followed by addition of 10 μL of PILS, and incubation for another 60 min. Following incubation, red blood cells were lysed by addition of 1 mL of 0.2% NaCl. After 30 s, 1 mL of 1.6% NaCl was added, and cells were pelleted by centrifugation at 500 × g for 2 min. The lysis was repeated, and the pelleted cells were resuspended in 1 mL of HBSS. Twenty microliters of 0.4% trypan blue was added to quench the fluorescence of any extracellular bacteria attached to phagocytes. Cells were then washed and resuspended in 500 μL of HBSS. Samples were placed on ice and analyzed via flow cytometry within 1 h. The labeled leukocytes were analyzed by a FACScan flow cytometer (Becton Dickinson, San Jose, CA) with an argon laser at 488-nm excitation wavelength.

Whole blood samples from two pigs per day served as controls, which were incubated with the following: DHR only, PILS only, and GM1/FITC-labeled secondary antibody only. The control samples were used to adjust compensation between red fluorescence (FL2 channel) emitted by PILS and green fluorescence (FL1 channel) emitted by rhodamine generated from DHR after the oxidative burst. The GM1 monoclonal antibody control was used to establish a region gate around the phagocytic cell population. Ten thousand events in this gate were collected. Results are expressed as the percentage of cells that were positive for both ingestion of PILS (phagocytosis) and rhodamine production (oxidative burst).

Statistical Analyses. All animal data were analyzed with the PROC MIXED procedure of SAS (SAS Inst. Inc., Cary, NC; Littell et al., 1996) as a 2 × 4 factorial in a randomized complete block design with repeated measures over time on each experimental unit (individual pens). The model included terms for the fixed effects of disease challenge, dietary treatment, time, and the appropriate interactions, and block was considered a random effect. Unless otherwise indicated, comparisons of disease challenge, dietary treatments, and(or) time were made only when a significant F-test (P < 0.05) for the main effect or interaction was found using the LSD procedure. All means presented are least-square means. Chi squared analysis was used to detect differences in fecal shedding of Salmonella between disease challenge groups on d 7 and 14 (Snedecor and Cochran, 1989). Pen served as the experimental unit for the statistical analyses.

For the phagocytosis assay, the statistical model included terms for the fixed effects of dietary treatment, time, and the appropriate interactions, and block was considered a random effect. Comparisons among dietary treatments and(or) time were made only when a significant F-test (P < 0.05) for the main effect or interaction was found using the LSD procedure. All means presented are least-square means.

Results

No significant interaction was observed between the main effects of dietary treatments and disease challenge. Dietary treatment did not affect ADG or ADFI, but G/F was poorer (P < 0.05) for pigs fed 250 mg/kg QS (Table 2). No significant linear or quadratic effects of dietary treatment were observed for ADG, ADFI, or G/F. Before challenge, ADG, ADFI, and G/F were similar between control and ST-challenged pigs (Figure 1). However, ST-challenge resulted in reduced (P < 0.05) ADG, ADFI, and G/F as compared to controls during wk 3 of the study. The negative impact of ST-challenge on growth performance was quickly resolved, and ADG, ADFI, and G/F did not differ between control and ST-challenged pigs during wk 4. However, the end weights of ST-challenged pigs were less (P < 0.001) than controls (24.1 vs 25.8 kg, respectively; SEM ± 0.9).

To more accurately track the course of disease onset and recovery, feed intake and rectal temperature were also measured daily during the week following ST-challenge (Figure 2). Daily feed intake for the ST-challenged pigs dropped (P < 0.05) dramatically between 24 to 48 h after challenge, remained depressed (P < 0.05) through 5 d after challenge, and returned to levels comparable to controls by d 6 after challenge. Rectal temperature of control pigs remained constant during the 7 d after challenge, whereas ST-challenge resulted in a distinct and expected febrile response.
Rectal temperatures of ST-challenged pigs were higher ($P < 0.05$) on d 1 to 4 after challenge but returned to levels similar to controls by d 5 after challenge. Daily feed intake and rectal temperature were not affected by dietary QS treatment.

Concentrations of IGF-I in serum also were estimated in samples collected during the week following disease challenge (Figure 2). Insulin-like growth factor-I did not differ between control and ST-challenged pigs prior to challenge. *Salmonella typhimurium* challenge resulted in a reduction in circulating IGF-I on d 2 ($P < 0.05$) and 4 ($P < 0.05$) after challenge. Although IGF-I in ST-challenged pigs began to increase by d 6, it was still lower ($P < 0.05$) than controls. Serum IGF-I during this same period was not affected by dietary QS treatment.

Serum haptoglobin and AGP concentrations are illustrated in Figure 3. *Salmonella typhimurium* challenge produced a rise ($P < 0.05$) in serum haptoglobin on d 7 after challenge, but levels were comparable to controls by d 14 after challenge. Serum AGP did not differ between controls and ST-challenged pigs before challenge, but AGP in ST-challenged pigs was elevated above that in controls on d 7 ($P < 0.05$) and 14 ($P < 0.05$) after challenge. Serum IgM increased ($P < 0.05$) over time in both groups, and serum IgM of ST-challenged pigs was greater than controls on d 7 after challenge ($P < 0.05$; Figure 4). Serum IgG was not affected by enteric disease challenge. However, on d 7 and 14 after disease challenge, serum IgG for both groups was greater ($P < 0.05$) than on d 0 (Figure 4). Dietary QS treatment failed to affect serum acute phase proteins or immunoglobulins.

On d 7 after challenge, 91.7% (22/24) of pens in the ST treatment contained pigs that were shedding *Salmonella* compared to 4.2% (1/24) of the control pens ($P < 0.001$). The single positive pen in the control room resulted from one control pig that cultured positive for *Salmonella*. Retrospective evaluation revealed that the rectal temperature of this pig remained constant through d 7 after challenge, and serum haptoglobin and AGP levels on d 7 and 14 after challenge were actually lower than the prechallenge level. Therefore, we were satisfied that biosecurity was maintained and that this pig probably did not have an active subclinical infection. By d 14 after bacterial challenge, 42.7% (10/24) pens contained pigs shedding *Salmonella* in the challenged treatment, whereas no pens in the control room contained pigs shedding *Salmonella* ($P < 0.001$).

No day effect was observed for phagocytic function of peripheral white blood cells isolated from ST-challenged pigs. However, phagocytic function was depressed in pigs fed 250 ($P < 0.05$) or 500 ($P < 0.05$) mg/kg as compared to pigs fed 125 mg/kg QS. Yet, there was no difference in phagocytic function among pigs fed 0, 250, or 500 mg/kg QS (Figure 5).

### Discussion

**Growth Performance.** Cromwell et al. (1985) reported diets containing 62 or 125 ppm yucca plant extract as the saponin source, in conjunction with a dietary antimicrobial, improved growth rate and tended to improve feed efficiency of weanling pigs during a 30-d growth assay. In contrast, Yen and Pond (1993) found that growth rate and small intestinal mass of weanling pigs were not influenced by dietary inclusion of the same yucca plant extract when fed at 125 ppm for 56 d, in the presence or absence of an antimicrobial agent. Another study (Gipp et al., 1988) also found no beneficial effects of yucca plant extract, with or without the inclusion of antibiotics in the diet, on weanling pig growth performance when fed for 39 d. It is important to note that these studies made no inference regarding the health status of the pigs. In the absence of conventional antimicrobials in the current study, we were unable to detect an effect of dietary saponins from QS extract on ADG or ADFI, and only a minor difference was observed on G/F, in ST-challenged or unchallenged control pigs. Although saponins have a positive influence on ruminal fermentation (Cheeke, 2000), it would appear that saponins, at least from this source, offer little benefit to growth performance in pigs.

The negative impact of ST-challenge on ADG during the week following infection is similar to results obtained by Balaji et al. (2000). However, we did not observe a negative effect on ADG during the second week (wk 4) following infection in ST-challenged pigs. Pigs challenged with ST in the present study had reduced feed intake during d 2 to 5 following ST-chal-

### Table 2. Influence of dietary *Quillaja saponaria* (QS) extract on weanling pig growth performance from d 0 to 28

<table>
<thead>
<tr>
<th>Item</th>
<th>0a</th>
<th>125a</th>
<th>250a</th>
<th>500a</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADG, kg</td>
<td>0.56</td>
<td>0.58</td>
<td>0.57</td>
<td>0.58</td>
<td>0.02</td>
</tr>
<tr>
<td>ADFI, kg</td>
<td>0.86</td>
<td>0.86</td>
<td>0.92</td>
<td>0.88</td>
<td>0.03</td>
</tr>
<tr>
<td>Gain/feed</td>
<td>0.67b</td>
<td>0.70b</td>
<td>0.64c</td>
<td>0.68b</td>
<td>0.01</td>
</tr>
<tr>
<td>End wt, kg</td>
<td>24.5</td>
<td>25.1</td>
<td>25.0</td>
<td>25.1</td>
<td>0.97</td>
</tr>
</tbody>
</table>

*Individual means represent 12 pens per treatment.*

*Within a row, means without a common superscript differ ($P < 0.06$).*
Q. saponaria and enteritis in weaned pigs

Figure 1. Average daily gain (top panel), average daily feed intake (middle panel), and gain/feed ratio (G/F; bottom panel) of control and weanling pigs orally challenged (arrow) with $10.5 \times 10^9$ cfu of *Salmonella typhimurium*. Means without a common superscript differ ($P < 0.05$).

Figure 2. Daily feed intake (top panel), rectal temperature (middle panel), and serum insulin-like growth factor-I (bottom panel) of control and weanling pigs orally challenged with $10.5 \times 10^9$ cfu of *Salmonella typhimurium*. Means within day differ ($P < 0.05$). *Means without a common superscript differ ($P < 0.05$).

challenge, which was similar to results from Balaji et al. (2000). Because the present study used a greater number of ST ($10.5 \times 10^9$ cfu vs $3 \times 10^9$ cfu) for the challenge dose, we do not believe that the clinical symptoms observed in the present study were any less than those reported by Balaji et al. (2000). This difference in ADG and ADFI may be attributed to differences in experimental design. In the present study, we allotted two pigs per pen, whereas the previous study maintained pigs in individual pens. Perhaps the housing of pigs in pairs had a beneficial effect on feed intake and behavior (Herskin and Jensen, 2000) that prevented any sustained effect of disease-induced behavior. It should be noted that the end weights on d 28 of ST-challenged pigs were less than controls, suggesting that even a rapid and effective containment of the
Figure 3. Serum haptoglobin (top panel) and α\textsubscript{1}-acid glycoprotein (bottom panel) in control and weanling pigs orally challenged with 10.5 × 10\textsuperscript{9} cfu *Salmonella typhimurium*.\textsuperscript{a,b,c} Means without a common superscript differ (P < 0.05).

Enteric pathogen by the immune system did not entirely mitigate the disease-induced slowing of growth. 

Finally, relative to the effect of disease challenge on growth performance (and other measured end points), our design did not provide an optimal test for the effects of enteric disease challenge. That is, because of the limitation of suitable rooms in which to replicate the effect of disease challenge and biosecurity considerations, disease-challenged pigs and uninfected pigs were housed in separate rooms. Thus, strictly speaking, the effects of enteric disease challenge are confounded with room. Whereas the profound effects observed during wk 3 of the study (on growth, feed intake, and, as discussed below, rectal temperature, serum IGF-I, and acute phase proteins) are almost certainly due to ST-challenge, these effects are confounded with experimental housing conditions.

**Acute Phase Response and IGF-I.** Our previous work using this model of enteric disease (Balaji et al., 2000) revealed that feed intake was depressed, rectal temperature was elevated through 5 d after infection, and plasma IGF-I concentrations were reduced from 30 to 108 h after ST-challenge. In the present study, we report similar reductions in feed intake and IGF-I and increases in rectal temperature during the 7 d following ST-challenge. The observed changes in circulating IGF-I, in this study, also agree with values reported for fasted weanling pigs (White et al., 1991) and for growing pigs infected with the protozoan parasite *Sarcocystis miescheriana* (Prickett et al., 1992).

We observed a rise in serum haptoglobin on d 7 after infection for ST-challenged pigs, but this returned to prechallenge levels by d 14. Serum AGP also was elevated on d 7 for ST-challenged pigs and returned to basal levels by d 14. Unlike Eurell et al. (1992), we did not observe any fluctuations in serum haptoglobin of control pigs during the final 2 wk of the study, which would suggest that the pigs in the present study had a high health status and good potential for weight gain. On d 0 after challenge in the present study, pigs were approximately 38 d old; therefore, the decline in AGP over time in the controls may reflect the changes in normal serum concentrations of AGP associated
with age (Itoh et al., 1992). The fact that AGP and haptoglobin levels in ST-challenged pigs were higher on d 7 suggests that these acute phase proteins are appropriate indicators of the acute enteric disease experienced by infected pigs during the 7 d following ST-challenge.

Serum Immunoglobulins. The changes in IgM and IgG concentrations over time in both groups of pigs most likely reflect the active synthesis of antibodies by the pig’s own immune system. It has been reported that maternal IgM and IgG reach minimal levels in the piglet’s system by 2 and 4 wk, respectively (Hunter, 1986). Furthermore, active synthesis of IgM and IgG does not begin until 2 and 5 wk, respectively (Hunter, 1986). Thus, the observed increase in IgG and IgM in control pigs probably reflects age-associated differences in antibody synthesis. The elevated serum IgM of ST-challenged pigs as compared to controls on d 7 would suggest that the enteric infection was not contained within the gut, and that the ST-infection may have stimulated a primary antibody response in the systemic immune system.

Phagocytic Function of Peripheral Blood Leukocytes. Riber and Lind (1999) used a similar flow cytometric assay to investigate the ability of peripheral blood leukocytes to ingest heat-killed ST, and the values for three pigs ranged from 8 to 56%, which is greater than the variation among individual pigs observed in the present study. Our results suggest that higher inclusion levels of QS (250 and 500 mg/kg) may depress phagocytic function of peripheral white blood cells. Although the underlying cause of this effect is difficult to interpret, it is worthy to note that this depression in phagocytic function appears to be marginal.

Implications

The results of this study indicate that dietary Quillaja saponaria extract may affect phagocytic cell function in young pigs; however, from a physiological perspective, the impact of Quillaja saponaria on immune function seems marginal because no other measures of immune competence were negatively affected. Furthermore, Quillaja saponaria extract, at the levels fed to pigs in this study, offers little benefit to growth performance in the presence or absence of enteric disease in weaned pigs. The oral Salmonella typhimurium model of acute enteric disease challenge activates an acute phase response that is accompanied by a marked febrile response, decreases in feed intake and circulating serum IGF-I, and elevations in serum haptoglobin and AGP. This experimental model of enteric disease may be well suited for evaluation of alternatives to dietary antimicrobials in swine diets.

Literature Cited


Figure 5. Effect of dietary Quillaja saponaria (QS) extract on phagocytic function of peripheral blood phagocytic cells from weanling pigs orally challenged with $10^5$ cfu Salmonella typhimurium. a,bMeans without a common superscript differ ($P < 0.05$).