Feeding Sodium Arsanilate for Exciting Diarrhea and Identifying Carriers of Swine Dysentery

L.D. Olson and D.E. Rodabaugh*

ABSTRACT

Sodium arsanilate was fed to nondiarrhetic swine, previously exposed to and treated for swine dysentery, for the purpose of inducing them into developing a swine dysentery diarrhea. From 40 to 100% of these swine in each pen had previously had a swine dysentery diarrhea. The isolate of Treponema hyodysenteriae in the diced colon which was used to expose the swine was resistant to sodium arsanilate. After an interim of no treatment for swine dysentery, sodium arsanilate was fed at a level of 220 parts per million for 21 days. Of the 14 pens containing swine fed sodium arsanilate, ten pens had one or more swine that developed a swine dysentery diarrhea while being fed sodium arsanilate. This was significantly (P < 0.05) greater than the three pens that each had one pig that developed a swine dysentery diarrhea of 13 pens containing similar swine not fed sodium arsanilate during a comparable period. In the 14 pens containing swine fed sodium arsanilate, 14 swine were the first to develop a swine dysentery diarrhea since in four pens, two swine in each pen developed diarrhea within 24 hours of each other. This also was significantly (P < 0.01) greater than the three swine in the ten pens not fed sodium arsanilate. From these results, it was theorized that sodium arsanilate excited the nondiarrhetic carrier into developing a swine dysentery diarrhea and that this phenomenon may have potential in identifying the carrier state.

Key words: Swine dysentery, Treponema hyodysenteriae, sodium arsanilate, identifying carriers, diarrheal recurrences, exciting diarrhea.

RÉSUMÉ

Cette expérience portait sur des porcs qui ne manifestaient pas de diarrhée, après avoir été expérimenter à la dysentérie et traités contre cette condition; elle consistait à leur donner de l’arsanilate de sodium, dans l’espoir de leur faire développer la diarrhée caractéristique de la dysentérie. De 40 à 100% de ces porcs avaient déjà manifesté la diarrhée caractéristique de la dysentérie, dans chacun des parcs de sujets expérimentaux. L’isolat de Treponema hyodysenteriae que contenaient les morceaux de côlon utilisés pour infecter les porcs, était résistant à l’arsanilate de sodium. Après un arrêt du traitement des porcs, qui dura un certain temps, on leur donna de la moulée qui contenait 220 ppm d’arsanilate de sodium, pendant 21 jours. Dans dix des 14 parcs qui logeaient des porcs qui avaient reçu de l’arsanilate de sodium, on constata qu’un ou quelques sujets développèrent la diarrhée caractéristique de la dysentérie, au cours de la période durant laquelle ils recevaient de l’arsanilate de sodium. Un tel résultat se révélait significativement plus élevé (P < 0.05) que celui de trois des 13 parcs qui logeaient des porcs auxquels on n’avait pas donné d’arsanilate de sodium, où un seul sujet développait la diarrhée caractéristique de la dysentérie. Dans les 14 parcs qui logeaient des porcs auxquels on avait donné de l’arsanilate de sodium, 14 furent les premiers à développer la diarrhée caractéristique de la dysentérie parce que, dans sept de ces parcs, deux sujets développèrent de la diarrhée, à 24 heures d’intervalle. Ce résultat se révélait aussi plus élevé (P < 0.01) que celui d’après lequel trois porcs logés dans les dix parcs où on ne servait pas d’arsanilate de sodium développèrent de la diarrhée. À partir de ces résultats, les auteurs émirent l’hypothèse que l’arsanilate de sodium amena les porteurs qui ne manifestaient pas de diarrhée à en développer, et que ce phénomène aiderait à les détecter.

Mots clés: dysentérie porcine, Treponema hyodysenteriae, arsanilate de sodium, identification des porteurs, récidive de la diarrhée, provocation de la diarrhée.

INTRODUCTION

A major problem in the eradication of swine dysentery (SD) from a swine herd is the unavailability of feasible and accurate methods for identifying SD in nondiarrhetic infected carriers. In a recent study it was concluded that neither the staining of rectal swabs nor the culturing of rectal swabs was sufficient, either together or singly, for diagnosing SD without clinical signs and lesions (1). In an earlier study, it was also discovered, in addition to observing that sodium arsanilate at a concentration of 110 parts per million (ppm) was totally ineffectual against

*Department of Veterinary Pathology, College of Veterinary Medicine, University of Missouri, Columbia, Missouri 65211.
Dr. Rodabaugh’s present address since retirement: 2305 Melville Road, Springfield, Missouri 65803.
Contribution from the Missouri Agricultural Experiment Station. Journal Series No. 10.021.
SD, that swine fed this level had a shorter incubation period and developed a more severe form of SD with more days of hemorrhagic diarrhea than did swine exposed to SD and not fed sodium arsanilate (2). From these observations, it was decided that if sodium arsanilate could worsen the diarrhea of SD, then the compound must be making conditions more favorable for the disease. If it could do the latter, why could it not be used for inducing the nondiarrhetic infected carrier of SD into developing a SD diarrhea, particularly if the concentration in the feed were doubled to 220 ppm? With the latter concentration the worsening of the SD diarrhea was uniform and consistent (3). Reported in this paper are the results of feeding sodium arsanilate (3-nitro-4-hydroxyphenol arsonic acid) to nondiarrhetic swine, previously exposed to SD and treated, after an interim of no treatment and no remission of SD diarrhea.

MATERIALS AND METHODS

SOURCE OF SWINE

The 157 swine used in this study were the SD exposed principals of various treatment groups in experiments previously designed for evaluating drugs with potential therapeutic value for SD. The swine were either farrowed at the Veterinary Research Farm, University of Missouri, Columbia or purchased from a feeder pig producer. They were all maintained in enclosed pens with concrete walls and floors and perforated drainage covers in buildings with strict rodent control at the Veterinary Research Farm. The swine in these pens could be observed, fed and watered and their feces flushed down the drain without entering the pen. Whenever entering or leaving the pen, boots were disinfected.

INDUCING EXPERIMENTAL SWINE DYSENTERY

When between 8 and 12 weeks old, each pig was given orally approximately 20 g of diced colon from swine affected with SD, free of Salmonella spp. and euthanized on the first day of diarrhea (4). The swine in experiment II were reexposed to SD infected colon in the feed once each week for three weeks after the initial exposure. The isolate of Treponema hyodysenteriae in the inoculum and the causative agent of swine dysentery (5) had been previously found to be totally resistant to sodium arsanilate (2).

TREATMENT OF SWINE DYSENTERY

The six pens of swine in group 1 and the four pens in group 2 (experiment I) which all contained five swine each had been treated previously with ronidazole (Merck and Company, Inc, Rahway, New Jersey) in the drinking water (6,7). The dosage and number of seven-day-treatments for group 1 swine were as follows: two pens were each given two treatments of 120 ppm; two pens were each given two treatments of 60 ppm; one pen was given two treatments of 30 ppm; whereas another pen was given the same but in three treatments. In the group 2 swine, two pens were each given one treatment of 60 ppm for seven days and two pens were each given two seven-day-treatments of 30 ppm.

Each of the three pens in group 3 (experiment I) containing ten swine each had been split previously into two pens containing five swine each. They all had been treated previously with imironidazole (Hoffmann-LaRoche, Inc, Nutley, New Jersey) in the drinking water (8). The dosage and number of eight-day treatments for the group 3 swine were as follows: two pens were each given one treatment of 100 ppm; one pen was given one treatment of 50 ppm, whereas another pen was given the same but in two treatments; and two pens were each given three treatments of 25 ppm.

The three pens of swine in group 4 and the three pens in group 6 (experiment II) each contained five swine and had been fed previously carbadox (Pfizer, Inc, New York, New York) at 55 ppm. In each group there was one pen that had been fed carbadox for four weeks, one pen fed the same for five weeks and one pen fed the same for six weeks. Each of the pens in groups 8 and 9 contained seven swine that had been previously treated in the drinking water for five days with 60 ppm of tiamulin (ER Squibb and Sons, Inc, Princeton, New Jersey) and three swine that had been treated with 60 ppm of tylosin (Eli Lilly and Company, Indianapolis, Indiana).

FEEDING SODIUM ARSANILATE FOR IDENTIFYING CARRIERS

In each experiment the pens of swine previously exposed and treated were divided into those fed sodium arsanilate (Mayfield Laboratories, Charles City, Iowa) at a level of 220 ppm for 21 days, and those not fed sodium arsanilate during the same period. Each experiment had a similar number of pens as to the previous incidence of diarrhea, level of medication and days of treatment. The concentration of sodium arsanilate in each batch of feed was confirmed by analysis (9). Although most of the swine were from 16 to 20 weeks old when fed sodium arsanilate, many were small and only weighed between 35 and 60 kg because their growth had been retarded by the SD infection.

When the feeding of sodium arsanilate began in experiment I, medication had been withdrawn for at least two weeks previously and there had been no SD diarrhea in the pens for at least one week. When the feeding of sodium arsanilate began in experiment II, medication had been withdrawn for at least four weeks previously and there had been no SD diarrhea in the pens for at least two weeks. When the feeding of sodium arsanilate began in experiment III, medication had been withdrawn for at least six weeks previously and there had been no SD diarrhea in either pen for five weeks. After a pig had developed a SD diarrhea, it was removed from the pen and treated. Swine were observed four times a day.

VERIFICATION OF SWINE DYSENTERY

Criteria used to evaluate the presence or absence of an infected carrier were: 1) diarrhea, either nonhemorrhagic or hemorrhagic and either initial or a recurrence; 2) observation of large spirochetes in
TABLE 1. Comparison of Diarrheal Recurrence of Swine Dysentery (SD) during Feeding of 220 ppm of Sodium Arsanilate for 21 Days to Swine Previously Exposed to SD and Treated with that in Swine Previously Exposed and Treated but not Fed Sodium Arsanilate

<table>
<thead>
<tr>
<th>Experiment and Group No. (No. of pens)</th>
<th>Drug, Route, Range in Dose, No. of Treatments and Days of Treatment</th>
<th>No. of Swine that Previously Developed Diarrhea</th>
<th>No. of Swine Developing Diarrhea While Fed Sodium Arsanilate</th>
<th>Average No. and Range in Dose, (min.-max.) Days Fed Sodium Arsanilate or Observed Before Developing Diarrhea</th>
<th>No. and Type of Diarrhea in Individual Pigs: Recurrence or Initial</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Experiment I</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 1 (6 pens)</td>
<td>Fed sodium arsanilate</td>
<td>16/30</td>
<td>2/6</td>
<td>10.5(1-21)</td>
<td>1 - recurrence</td>
</tr>
<tr>
<td></td>
<td>Not fed sodium arsanilate</td>
<td></td>
<td></td>
<td></td>
<td>1 - initial</td>
</tr>
<tr>
<td>Group 2 (4 pens)</td>
<td>Fed sodium arsanilate</td>
<td>17/20</td>
<td>3/4</td>
<td>7(5-9)</td>
<td>4 - recurrence</td>
</tr>
<tr>
<td></td>
<td>Not fed sodium arsanilate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 3 (3 pens)</td>
<td>Fed sodium arsanilate</td>
<td>23/30</td>
<td>2/3</td>
<td>9(5-14)</td>
<td>3 - recurrence</td>
</tr>
<tr>
<td></td>
<td>Not fed sodium arsanilate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Experiment II</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 4 (3 pens)</td>
<td>Fed sodium arsanilate</td>
<td>14/14</td>
<td>1/3</td>
<td>21</td>
<td>1 - recurrence</td>
</tr>
<tr>
<td></td>
<td>Not fed sodium arsanilate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 5 (3 pens)</td>
<td>Fed sodium arsanilate</td>
<td>9/15</td>
<td>0/3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Not fed sodium arsanilate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 6 (3 pens)</td>
<td>Fed sodium arsanilate</td>
<td>9/13</td>
<td>1/3</td>
<td>18</td>
<td>1 - recurrence</td>
</tr>
<tr>
<td></td>
<td>Not fed sodium arsanilate</td>
<td></td>
<td></td>
<td></td>
<td>1 - initial</td>
</tr>
<tr>
<td>Group 7 (3 pens)</td>
<td>Fed sodium arsanilate</td>
<td>11/15</td>
<td>3/3</td>
<td>8(5-14)</td>
<td>3 - recurrence</td>
</tr>
<tr>
<td></td>
<td>Not fed sodium arsanilate</td>
<td></td>
<td></td>
<td></td>
<td>1 - initial</td>
</tr>
<tr>
<td><strong>Experiment III</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 8 (1 pen)</td>
<td>Fed sodium arsanilate</td>
<td>10/10</td>
<td>0/1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Not fed sodium arsanilate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 9 (1 pen)</td>
<td>Fed sodium arsanilate</td>
<td>10/10</td>
<td>1/1</td>
<td>14</td>
<td>1 - recurrence</td>
</tr>
<tr>
<td></td>
<td>Not fed sodium arsanilate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total or average, (min.-max.) for all swine</td>
<td></td>
<td>49/69</td>
<td>3/13</td>
<td>14(1-21)</td>
<td>3'/13</td>
</tr>
<tr>
<td>Total or average, (min.-max.) for all swine</td>
<td></td>
<td>70/88</td>
<td>10'/14</td>
<td>10(5-18)</td>
<td>14'/14</td>
</tr>
</tbody>
</table>

a Medication began in all experiments after the development of the first SD diarrhea in the pen

b Includes pigs that developed a SD diarrhea within 24 hours of the first pig in the pen

D'W = drinking water; ppm = parts per million

Significantly (P < 0.05, Chi square test) more pens of swine fed sodium arsanilate had SD diarrheal recurrences than in pens not fed sodium arsanilate

Significantly (P < 0.01, Chi square test) more swine fed sodium arsanilate were the first to develop SD diarrhea in their respective pens that the number not fed sodium arsanilate

RESULTS

EXPERIMENT I

In this experiment there was a recurrence of SD diarrhea while being fed 220 ppm of sodium arsanilate in five of seven pens (groups 2 and 3, Table 1) containing previously exposed and treated swine; whereas, during a comparable period after

smears of rectal and fecal swabs from swine with or without a diarrhea and stained with Victoria blue 4-R stain (10); and 3) in vitro cultivation of T. hyodysenteriae from the same sites. The latter was done by streaking rectal and fecal swabs on blood agar containing 400 μg of spectinomycin per mL, incubating anaerobically and observing for a strong beta hemolysis as previously described (11). Rectal swabs were also cultured in tetrathionate broth and subcultured on brilliant green agar for attempted recovery of Salmonella spp. The basic unit used in evaluating the effect of sodium arsanilate was the presence or absence of diarrheal recurrence in the pen of swine. These results were analyzed statistically using the Chi square test.
treatment in similar swine not fed sodium arsanilate (group 1) there was a recurrence of SD diarrhea in only two of six pens. Although this difference was not significant (P > 0.05), the percentage of pens of swine fed sodium arsanilate with a recurrence was more than twice that of the pens of swine not fed sodium arsanilate. In group 2 swine fed sodium arsanilate, SD diarrhea recurred in the two pens previously given one treatment of 60 ppm and one pen given two treatments of 30 ppm of ronidazole. In group 3 swine fed sodium arsanilate, SD diarrhea recurred in the pens previously given one treatment of 100 and three treatments of 25 ppm of ipronidazole.

In group 1 swine not fed sodium arsanilate, SD diarrhea recurred in one pen previously given two treatments of 60 ppm and one pen given three treatments of 30 ppm of ronidazole. In two pens (one in group 2 and one in group 3) while being fed sodium arsanilate there were two swine that developed a SD diarrhea within 24 hours of each other. Including these swine, a total of seven swine were the first to develop a SD diarrhea in the five pens being fed sodium arsanilate which was greater, although not significantly (P > 0.05), than the two pigs not fed sodium arsanilate that developed a SD diarrhea. The SD diarrhea for each of these seven swine fed sodium arsanilate was a recurrence and nonhemorrhagic, and first occurred from 5 to 14 days after starting to feed sodium arsanilate. In the swine not fed sodium arsanilate (group 1), the diarrhea was a recurrence and nonhemorrhagic for the pig in one pen and an initial diarrhea and hemorrhagic for the pig in the other pen. After the onset of the SD diarrhea in these swine, numerous large spirochetes were observed in their rectal and fecal smears stained with Victoria blue 4-R.

There was no apparent relationship in experiment I between the level or duration of the previous medication with either of the nitroimidazoles (ronidazole and ipronidazole) and the incidence of pens with SD diarrheal recurrences, whether the swine were fed sodium arsanilate or not, since SD diarrhea recurred in the pens of swine previously given high, moderate and low levels of medication and single and multiple treatments.

**EXPERIMENT II**

In this experiment there was a recurrence of SD diarrhea while being fed sodium arsanilate in six pens (groups 6 and 7) containing swine previously exposed and treated with either carbadox or lincomycin in the experimental period. In these pens fed sodium arsanilate, SD diarrhea recurred in one pen fed carbadox for four weeks which was one week after the last reexposure. In the group 7 swine fed the same, SD diarrhea recurred in pens fed lincomycin for four, five and six weeks which was one, two and three weeks, respectively, after the last reexposure. In the group 6 swine not fed sodium arsanilate, SD diarrhea recurred in one pen fed carbadox for five weeks which was two weeks after the last reexposure. In each of two pens (one in group 6 and one in group 7), while being fed sodium arsanilate there were again two swine that developed a SD diarrhea within 24 hours of each other. Including these swine, a total of six swine were the first swine to develop a SD diarrhea in the four pens with a recurrence being fed sodium arsanilate which was greater, although not significantly (P > 0.05), than the one pig with a SD diarrheal recurrence not fed sodium arsanilate. For the six swine individually, the diarrhea was a recurrence and nonhemorrhagic for four swine and an initial diarrhea and hemorrhagic for two swine. The recurrences of SD diarrhea occurred from 5 to 18 days after the start of feeding sodium arsanilate. The diarrhea in the one pig in the one pen not fed sodium arsanilate was a recurrence and nonhemorrhagic. In addition to seeing numerous large spirochetes in rectal and fecal stained smears in experiment II, *T. hyodysenteriae* were cultured from rectal swabs of the swine that developed a diarrhea. *Treponema hyodysenteriae* was also cultured from the rectum of one pig in group 4 not fed sodium arsanilate immediately prior to the 21-day-posttreatment observational period; however, this pig did not develop a diarrhea during this period.

In addition, *T. hyodysenteriae* was cultured from the rectum of one pig in each of two pens in group 7 immediately prior to the start of feeding sodium arsanilate although large spirochetes were not seen in stained rectal smears. However, both of these pigs were the first to develop a diarrhea in their respective pens while being fed sodium arsanilate.

Again, as in experiment I, there was no apparent relationship in experiment II between the previous medication used for treatment or the duration of treatment and the incidence of pens with a SD diarrheal recurrence, whether fed sodium arsanilate or not, since diarrhea recurred in pens of swine previously given carbadox for 28 and 35 days and given lincomycin for 28, 35 and 42 days.

**EXPERIMENT III**

In this experiment there was a SD diarrheal recurrence in the pen of swine fed sodium arsanilate which occurred 14 days after the start of the feeding. It was also a recurrence for the individual pig and nonhemorrhagic. From rectal swabs of this pig, numerous large spirochetes were observed in stained smears and *T. hyodysenteriae* were cultured. *Treponema hyodysenteriae* was cultured from the rectum of one pig in group 8 not fed sodium arsanilate immediately prior to the 21-day-posttreatment observational period; however, neither this pig nor any other swine in the pen developed a diarrhea during this period.

**TOTAL FOR ALL EXPERIMENTS**

Combining the results of the three experiments, a total of 10 of the 14 pens containing swine fed sodium arsanilate had a recurrence of SD diarrhea while receiving the compound which was significantly (P < 0.05) greater than the 3 of 13 pens containing swine that developed SD diarrhea during a comparable period after treatment but did not receive sodium arsanilate. A total of 14 swine were the first to develop a SD diarrhea in the 14 pens of swine fed
sodium arsanilate, which also was significantly (P < 0.01) greater than the three swine that were the first to develop a SD diarrhea in the 13 pens not fed sodium arsanilate. The SD diarrhea was more severe in the swine in which it was an initial diarrhea than in those in which it was a recurrence.

None of the swine fed 220 ppm of sodium arsanilate for 21 days in this study developed any lameness or altered gait during or after withdrawal of the compound (12).

**DISCUSSION**

Unknown at the start of feeding sodium arsanilate or the comparable observational period for the swine not fed sodium arsanilate was the number of nondiarrhetic carriers of SD in each pen; however, it was presumed that all pens had carriers since: 1) all swine had been exposed to SD inoculum one or more times; 2) SD diarrhea had been evident in at least two of the four or five swine in all pens; 3) large spirochetes were observed in rectal swabs and diarrhetic feces and *T. hyodysenteriae* were cultured from the same; and 4) previous medication had not been given either at a sufficient level or for a long enough duration for the possible elimination of the disease.

Although in four pens, two swine developed a SD diarrhea within 24 hours of each other, too short a period to incubate the disease and induce a SD diarrhea in the other swine in the pen, the basic unit for evaluating the effectiveness of sodium arsanilate in exciting the nondiarrhetic carrier into developing a SD diarrhea was the pen and not the individual pig since after the first diarrheal recurrence, the pen was considered to have been recontaminated and the remaining swine were considered to have been reexposed. The shortest incubation period for SD that we have observed is three days. Since in all pens one or more swine had previously had SD diarrhea, the development of SD diarrhea in one or more swine in the pen during the feeding of sodium arsanilate was a recurrence for the pen; however, for the individual pigs it could have been either a recurrence or an initial SD diarrhea.

The SD diarrheas in the swine which were a recurrence with the feeding of sodium arsanilate, were not as severe as those observed in the swine which developed an initial diarrhea, probably because the former swine had become partially immune (13).

The problem of diagnosing SD becomes complicated when attempting to evaluate the carrier status of swine from herds with a clinical history of SD but which do not have diarrhea at the time of collecting rectal swabs. Nondiarrhetic swine which are shedders of *T. hyodysenteriae* do exist, since in a previous study (1) 11.9% of the specimens producing strong beta-hemolytic reactions when cultured and 15.1% of the stained smears with large spirochetes came from swine with no diarrhea at the time of swabbing. The use of culturing and the isolation of *T. hyodysenteriae* may be of assistance in identifying endemic, but nondiarrhetic herds of swine suspected of being infected with SD, but not relied on exclusively without clinical signs or lesions. Likewise, the staining of fecal smears should not be used alone in nondiarrhetic herds because of the possibility that the presence of nonpathogenic large spirochetes, *T. innocens* (14) could be mistaken for pathogenic large spirochetes. With the use of culturing in swine herds without clinical signs, the possibility exists of isolating *T. innocens* when attempting to recover *T. hyodysenteriae* and falsely diagnosing SD, or of not getting any growth and reporting an infected herd as being free of SD because the nondiarrhetic carriers were not shedding *T. hyodysenteriae* at the time of swabbing.

It is postulated by one of us (Olson) that the transmission of SD most often occurs via diarrhetic feces and that carriers transmit the disease principally when they have a recurrence of diarrhea. In a previous unpublished study he observed that SD did not develop in comingling sentinel swine introduced during the drug withdrawal period until there was a diarrheal recurrence in the pen. Diarrhetic feces contain, particularly at the onset of diarrhea, a high number of large spirochetes, and swine are more apt to eat diarrhetic feces. It is also the author’s observation that when using diced colon as inoculum and unlike that reported when using cultures of *T. hyodysenteriae* as inoculum (15) he has been unable to culture *T. hyodysent-

It is paradoxical that a compound such as sodium arsanilate which was previously used to treat SD should now be used to induce SD diarrhea in nondiarrhetic carriers. Apparently many isolates of *T. hyodysenteriae* have become resistant to sodium arsanilate since the compound was first introduced over 30 years ago (2). When first used, sodium arsanilate was partially effective for the treatment of SD; however, diarrheal recurrences were common after the withdrawal of the compound (16). It was recently reported that 69% of 214 isolates of *T. hyodysenteriae* grown *in vitro* were resistant to sodium arsanilate; however, 30% of the 214 isolates were inhibited by 50 μg of sodium arsanilate per mL of media which can be interpreted to mean that there probably still are isolates of *T. hyodysenteriae* which are sensitive to sodium arsanilate (17).

The mechanism by which the feeding of sodium arsanilate augments SD and induces the carrier swine into developing a SD diarrhea is yet unknown. It may be that sodium arsanilate alters the intestinal environment by modifying the intestinal flora in the colon and thereby making conditions more favorable for the *T. hyodysenteriae* to multiply, or it may stimulate the growth of *T. hyodysente-

The arsenic in sodium arsanilate is in the pentavalent form and does not react readily with sulfhydryl groups (18). It has been proposed that the beneficial growth-promoting and disease reducing effects of arsenicals as feed additives may be in part through the increased interferon activity that they stimulate since it has been observed in cell cultures that low concentrations of arsenicals increased the antiviral activity of low levels of interferon (19).
There probably are other compounds which have no inhibitory effect on all isolates of *T. hyodysenteriae* and which will induce the nondiarrheic carriers of SD into developing a SD diarrhea. Most epizootics of SD probably occur because nondiarrheic carrier swine are introduced into a herd. The feeding of a compound which would induce a SD diarrhea in prospective nondiarrheic breeding stock confined to isolation could be useful in detecting carriers before they were introduced into a herd. A compound with this property could also be used in evaluating vaccines and drugs for their efficacy in eliminating SD.

REFERENCES


17. **KINYON JM, HARRIS DL.** In vitro susceptibility of *Treponema hyodysenteriae* and *Treponema innocens* by the agar dilution method. Proc Intl Symp Vet Lab Diagnost, Lucerne, Switzerland, 1980: 125-128.
