REPRODUCTIVE CRITERIA OF BEEF BULLS DURING AND AFTER EXPOSURE TO INCREASED AMBIENT TEMPERATURE

D. C. Meyerhoeffer, R. P. Wettemann, S. W. Coleman, and M. E. Wells

US Department of Agriculture, El Reno, OK 73036 and Oklahoma Agricultural Experiment Station, Stillwater 74078

Summary

Sixteen yearling Angus bulls were randomly assigned to one of two temperature-controlled chambers to determine the effects of elevated ambient temperature on body functions and semen characteristics. After 8 wk adjustment at 23 C, eight heat-stressed bulls were exposed to 35 ± 1 C for 8 h and 31 ± 1 C for 16 h during each 24-h period, and eight control bulls were maintained at 23 ± 1 C for 8 wk. Then all bulls were exposed to 23 C for 8 wk. Bulls were fed so that both control and stressed bulls gained at similar rates (.58 kg/d). Semen was collected with an artificial vagina twice weekly before, during and after heat stress. During treatment, the respiratory rate of stressed bulls was greater (P<.001) than that of control bulls (54.2 ± 1.5, 29.9 ± 1.5 breaths/min, respectively). Rectal temperatures were increased (P<.01) from 38.2 ± .1 to 38.7 ± .1 C and water consumption was increased by 35% in stressed bulls when compared with controls. Semen volume was not altered by treatment, but percentage of motile sperm decreased (P<.01) in stressed bulls by 2 wk after the start of heat treatment. Sperm motility of stressed bulls returned to normal values 8 wk after the end of heat treatment. Similarly, the percentage of aged acrosomes on sperm from stressed bulls increased (P<.01) by the second week of treatment and remained greater than that of controls throughout the stress period. Heat stress also resulted in more abnormal cells from the second week of treatment until 7 to 8 wk after heat stress. These data indicate that exposure of bulls to elevated ambient temperatures results in decreased semen quality as evidenced by a reduced percentage of motile sperm, reduced sperm output and an increased percentage of abnormal and aged sperm. Approximately 8 wk is required before semen quality returns to normal after heat stress of bulls.

(Key Words: Heat Stress, Reproduction, Bulls, Semen Quality, Sperm Morphology.)

Introduction

Reproductive performance is reduced in males exposed to a hot environment. Seasonal climatic changes are related to fertility and semen quality of bulls (Johnson and Branton, 1953). Exposure of bulls to increased ambient temperature resulted in impaired spermatogenesis (Casady et al., 1953; Rhynes and Ewing, 1973). Local heating of the scrotum of bulls for 1 (Austin et al., 1961) or 3 d (Gerona and Sikes, 1970) caused a reduction in the number of live sperm ejaculated. Exposure of bulls to whole body heat stress for as little as 12 h can reduce spermatogenesis (Skinner and Louw, 1966). Rhynes and Ewing (1975) observed a 43% decrease in plasma testosterone during the first 2 wk of heat stress, but the concentration returned to near normal afterwards. Minton et al. (1981) observed no differences in plasma testosterone of heat stressed and normal bulls.

Semen quality deteriorated in boars and bulls about 2 wk after whole body exposure to increased ambient temperature (McNitt and
First, 1970; Zaremba, 1975; Wettemann et al., 1976). Daurte Irala (1973) observed that motility of bovine sperm did not return to normal until 8 to 10 wk after exposure to heat stress.

This study was designed to evaluate semen characteristics and physiological functions of beef bulls during long-term exposure to increased ambient temperature and to determine semen quality during the first 8 wk after heat stress.

Materials and Methods

In each of two replicates, eight yearling Angus bulls were randomly allotted to either a hot or a control temperature chamber. Each 14.6 × 3.1 m chamber had four stanchions with tie chains and the concrete floor was covered with a rubber mat. Gravity flow water buckets were available to each animal and water entered from calibrated drums outside the chambers.

All bulls were trained and semen was collected with an artificial vagina. After bulls had been ejaculated for at least 3 wk and had been exposed to ambient temperatures less than 23 C for 8 wk, treatments were initiated. The temperature in one chamber (heat stressed) was maintained at 35 ± 1 C from 0850 to 1650 h and 31 ± 1 C for the remaining 16 h during each 24-h period for 8 wk. They were subsequently exposed to 23 ± 1 C for 8 wk. Control bulls were maintained at 23 ± 1 C throughout the trial. Humidity was not controlled in these chambers but ranged from 46 to 80% in the hot chambers and from 60 to 85% in the control chambers. Bulls in both treatments were exposed to 12 h of light each day throughout the trial. Water was supplied ad libitum and feed intake was controlled so that bulls gained about .58 kg/d. Respiratory rates and rectal temperatures were determined daily for all bulls at 0800 and 1300 h. Semen was collected with an artificial vagina twice weekly during and after treatment (one ejaculate every 3 or 4 d). Semen volume, number of sperm, rate of sperm progression (1 = slow and 5 = fast), percentage motile sperm and sperm morphology were evaluated at each collection. Sperm morphology was quantified by observation of 200 sperm/stained smear for each ejaculate in replicate (rep) 1 as described by Wells et al. (1971). Aged acrosomes are alterations in the acrosome caps caused by cell injury or aging that may reflect detrimental changes in the sperm cell.

These data were analyzed by split-plot analysis of variance with two between-block treatments (replicate and temperature) and one within-block treatment (week of treatment) as described by Gill and Hafs (1971). When week × treatment interactions were significant, orthogonal comparisons were used to separate treatment effects in 2-wk periods.

Results and Discussion

Respiratory rates of bulls exposed to elevated ambient temperature were greater (P<.001) than those of control bulls throughout the 8-wk treatment period (54.2 ± 1.5 vs 29.9 ± 1.5 breaths/min, respectively). For stressed bulls, maximum rates averaged 60 breaths/min and occurred 4 wk after the beginning of exposure to heat stress. Respiratory rates were also greater (P<.01) at 1300 h (61.0 ± 2.7 breaths/min) than at 0800 h (48.2 ± 2.5 breaths/min) for stressed bulls (after they had been exposed to 35 C for the previous 4 h). However, time of day did not influence respiratory rates of control bulls. Within 24 h after the heat-stressed bulls had returned to the control environment (23 C), respiratory rates had nearly returned (27.5 ± .3 breaths/min) to pretreatment values (25.6 ± .2 breaths/min). Similar effects of heat stress on respiratory rates have been observed in boars (Wettemann et al., 1976). Rectal temperatures of bulls subjected to increased ambient temperatures were increased (P<.01) compared with those for controls (38.7 ± .1 vs 38.2 ± .1 C).

Water consumption by heat-stressed bulls was 35% greater than that of control bulls (27 ± 1 vs 17 ± 1 liters/d, P<.01). The increases in respiratory rates, rectal temperatures and water intake indicate that these bulls were heat stressed by exposure to ambient temperatures of 31 to 35 C.

The volume of semen per ejaculum was decreased (P<.05) during the first 6 wk of treatment (table 1; figure 1). Heat stress did not influence semen volume in dairy bulls (Johnson and Branton, 1953) or boars (Wettemann et al., 1976) subjected to increased ambient temperatures. In addition, testosterone concentration in plasma of bulls was not influenced by heat stress (Minton et al., 1981).

The rate of progressive movement and the percentage of motile sperm were similar (P>.10) for control and heat-stressed bulls before treatment (figures 2 and 3). However, percentage
TABLE 1. MEAN SQUARES FOR ORTHOGONAL COMPARISONS OF TREATMENT X WEEK INTERACTION ON GENERAL SEMEN CHARACTERISTICS OF BULLS

<table>
<thead>
<tr>
<th>Time</th>
<th>df</th>
<th>Volume</th>
<th>Sperm/ ejaculum</th>
<th>Rate of progressive movement</th>
<th>Percentage motile sperm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment period</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 1 to 2</td>
<td>1</td>
<td>8.0*</td>
<td>5.6*</td>
<td>.4</td>
<td>278.4**</td>
</tr>
<tr>
<td>Week 3 to 4</td>
<td>1</td>
<td>25.1**</td>
<td>1.3</td>
<td>.5</td>
<td>1,130.3**</td>
</tr>
<tr>
<td>Week 5 to 6</td>
<td>1</td>
<td>23.5**</td>
<td>13.3**</td>
<td>4.4**</td>
<td>3,012.3**</td>
</tr>
<tr>
<td>Week 7 to 8</td>
<td>1</td>
<td>.1</td>
<td>3.0</td>
<td>4.2**</td>
<td>10,252.6**</td>
</tr>
<tr>
<td>Error</td>
<td>79</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recovery period</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 1 to 2</td>
<td>1</td>
<td>.4</td>
<td>11.7**</td>
<td>8.4**</td>
<td>7,195.9**</td>
</tr>
<tr>
<td>Week 3 to 4</td>
<td>1</td>
<td>8.2*</td>
<td>.2</td>
<td>7.9**</td>
<td>3,984.8**</td>
</tr>
<tr>
<td>Week 5 to 6</td>
<td>1</td>
<td>1.8</td>
<td>.1</td>
<td>5.5**</td>
<td>5,166.0**</td>
</tr>
<tr>
<td>Week 7 to 8</td>
<td>1</td>
<td>4.9</td>
<td>1.6</td>
<td>.0</td>
<td>825.5**</td>
</tr>
<tr>
<td>Error</td>
<td>84</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*P<.05.  
**P<.01.

of motile sperm was decreased (P<.05) by 2 wk and rate of sperm progression was decreased (P<.05) after 4 wk of exposure to increased ambient temperature (table 1). The percentage of motile sperm from stressed bulls decreased from 76 ± 1.9 (pretreatment) to 48 ± 1.0% after bulls were stressed for 8 wk. A lag time of about 2 wk from initial exposure to heat until percentage of motile sperm decreased was observed and is consistent with results from bulls (Zaremba, 1975) and boars (McNitt and First, 1970; Wettemann et al., 1976, 1979). Because epididymal transport takes about 2 wk, this lag time suggests that epididymal sperm may not be affected by heat stress, or that it takes several weeks for any changes to occur in the epididymis that result in a reduction in motile sperm. The rate of sperm movement and percentage of motile sperm increased gradually during the post-treatment period (figures 2 and 3) and were similar for both treatments by 8 wk after the end of heat stress. The 8-wk interval from the end of treatment until ejaculation of sperm with normal motility is in agreement with studies with dairy bulls exposed to whole body heat (Duarte Irala, 1973) or elevated scrotal temperatures (Gerona and Sikes, 1970).

Total sperm per ejaculate in general was not affected by heat treatment, even though sperm number from heat-stressed bulls were lower (P<.01) during wk 1 to 2 and 5 to 6 of the treatment period and wk 1 to 2 of the recovery period (figure 4; table 1). However, pretreatment means were also different, which probably indicates an inherent difference in the animals between the two treatments.

Morphological characteristics of sperm in the ejaculates were similar for both groups before treatment, but were altered by exposure of bulls to increased temperatures. The percentage

![Figure 1. Volume of semen from bulls before, during and after exposure to control or elevated ambient temperatures. Vertical bars represent standard error of the means.](image-url)
of sperm with aged acrosomes was increased from the initial week of treatment (table 2). The reduction in the percentage of normal nonaged sperm was accompanied by significant increases in the percentages of normal aged sperm (figure 5). The percentage of abnormal cells were variable for both treatments but were higher for stressed bulls from 3 to 4 wk after treatment until the first 2 wk into the recovery period (figure 6). At 6 to 8 wk of treatment, a total of about 30% of the sperm from heat-stressed bulls had aged acrosomes compared with 10% for control bulls. In general, sperm morphology of stressed bulls returned to pre-treatment values less than 8 wk after heat-stressed bulls were returned to a control environment. Apparently, recovery of normal morphology occurs sooner after the end of heat
## Table 2. Mean Squares for Orthogonal Comparisons of Treatment x Week Interaction on Sperm Morphology

<table>
<thead>
<tr>
<th>Time</th>
<th>df</th>
<th>Aged(^a)</th>
<th>Normal</th>
<th>Nonaged(^b)</th>
<th>Aged(^a)</th>
<th>Abnormal</th>
<th>Nonaged(^b)</th>
<th>Aged(^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment period</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 1 to 2</td>
<td>1</td>
<td>82.6*</td>
<td>62.6</td>
<td>409.6**</td>
<td>120.4**</td>
<td>18.5</td>
<td>2.3</td>
<td></td>
</tr>
<tr>
<td>Week 3 to 4</td>
<td>1</td>
<td>846.5**</td>
<td>286.1*</td>
<td>744.3**</td>
<td>297.7**</td>
<td>1.6</td>
<td>172.3**</td>
<td></td>
</tr>
<tr>
<td>Week 5 to 6</td>
<td>1</td>
<td>813.4**</td>
<td>1,010.6**</td>
<td>2,282.0**</td>
<td>260.8**</td>
<td>275.5**</td>
<td>161.6**</td>
<td></td>
</tr>
<tr>
<td>Week 7 to 8</td>
<td>1</td>
<td>1,220.3**</td>
<td>638.9**</td>
<td>2,726.6**</td>
<td>620.4**</td>
<td>323.8*</td>
<td>74.4**</td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>37</td>
<td>15.9</td>
<td>57.6</td>
<td>53.5</td>
<td>16.1</td>
<td>34.6</td>
<td>6.9</td>
<td></td>
</tr>
<tr>
<td>Recovery period</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 1 to 2</td>
<td>1</td>
<td>493.7**</td>
<td>1,344.4**</td>
<td>2,052.2**</td>
<td>71.0**</td>
<td>848.8**</td>
<td>58.8**</td>
<td></td>
</tr>
<tr>
<td>Week 3 to 4</td>
<td>1</td>
<td>175.8**</td>
<td>66.1</td>
<td>11.3</td>
<td>116.3**</td>
<td>84.5</td>
<td>4.0</td>
<td></td>
</tr>
<tr>
<td>Week 5 to 6</td>
<td>1</td>
<td>57.8*</td>
<td>270.3*</td>
<td>472.8**</td>
<td>22.8</td>
<td>215.3*</td>
<td>4.4</td>
<td></td>
</tr>
<tr>
<td>Week 7 to 8</td>
<td>1</td>
<td>77.0**</td>
<td>66.6</td>
<td>247.0*</td>
<td>51.0**</td>
<td>48.2</td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>42</td>
<td>10.9</td>
<td>38.7</td>
<td>37.1</td>
<td>5.9</td>
<td>34.1</td>
<td>3.0</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)Aged acrosomes.  
\(^b\)Nonaged acrosomes.  
*P<.05.  
**P<.01.
stress than does the ejaculation of a normal percentage of motile sperm (3 vs 8 wk). Similarly, Wettemann et al. (1979) observed that the percentage of sperm with aged acrosomes was similar for heat-stressed and control boars within 1 wk after the end of heat stress, but the percentage of motile sperm from boars did not return to normal until 5 wk after the end of heat stress.

Exposure of bulls to increased ambient temperature (31 and 35 C for 8 wk) results in reductions of the percentage of motile sperm and percentage of normal sperm with nonaged acrosomes. The motile sperm and percentage of abnormal sperm in ejaculates from stressed bulls did not return to the levels of control bulls until 8 wk after the end of heat stress. Reduction in fertility with summer and early fall breedings may be associated with the decreased semen quality during and after heat stress of bulls.

**Literature Cited**


