Relationship between mean body surface temperature measured by use of infrared thermography and ambient temperature in clinically normal pigs and pigs inoculated with Actinobacillus pleuropneumoniae

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Objective—To determine the relationship between ambient temperature and mean body surface temperature (MBST) measured by use of infrared thermography (IRT) and to evaluate the ability of IRT to detect febrile responses in pigs following inoculation with Actinobacillus pleuropneumoniae.

Animals—28 crossbred barrows.

Procedures—Pigs (n = 4) were subjected to ambient temperatures ranging from 10 to 32 °C in an environmental chamber. Infrared thermographs were obtained, and regression analysis was used to determine the relationship between ambient temperature and MBST. The remaining pigs were assigned to groups in an unbalanced randomized complete block design (6 A pleuropneumoniae-inoculated febrile pigs [increase in rectal temperature ≥ 1.67 °C], 6 A pleuropneumoniae-inoculated nonfebrile pigs [increase in rectal temperature < 1.67 °C], and 12 noninoculated pigs). Infrared thermographs and rectal temperatures were obtained for the period from 2 hours before to 18 hours after inoculation, and results were analyzed by use of repeated-measures ANOVA.

Results—A significant linear relationship was observed between ambient temperature and MBST (slopes, 0.40°C). For inoculated febrile pigs, a treatment × method interaction was evident for rectal temperature and MBST, whereas inoculated nonfebrile pigs only had increased rectal temperatures, compared with noninoculated pigs. A method × time interaction resulted from the longer interval after inoculation until detection of an increase in MBST by use of IRT.

Conclusions and Clinical Relevance—Infrared thermography can be adjusted to account for ambient temperature and used to detect changes in MBST and radiant heat production attributable to a febrile response in pigs. (Am J Vet Res 2001;62:676-681)

I nfrared thermography (IRT) can be used to measure changes in mean body surface temperature (MBST) caused by variations of heat flow to the skin surface as a result of alterations in tissue conduction rates and convection via blood flow underlying the skin surface. These processes are affected by intrinsic and extrinsic factors such as location of blood vessels, biorhythms, disease, stress, and environment. Therefore, adjustments for environmental factors must be made to the observed MBST to accurately reflect variations of heat flow in MBST.

The environmental condition that typically affects MBST the most is ambient temperature. Changes in ambient temperature affect the subcutaneous vasodilatory response, thus altering the rate of heat flow to the skin surface. Limited research has been performed to relate ambient temperature to MBST under controlled conditions. Results of studies indicate that when subjects are exposed to ambient temperatures at or below thermoneutral conditions, MBST in humans and pigs changes at a constant rate relative to ambient temperature. However, when ambient temperatures are above thermoneutral conditions, MBST responds curvilinearly in humans as latent heat losses increases to maintain homeothermy, but MBST remains constant in pigs. Researchers who used an infrared spot meter found that MBST in pigs changed at a constant rate of 0.4°C for every change of 1°C in ambient temperature. However, these values were derived from temperatures ranging from 11 to 26°C, which is primarily within the thermoneutral zone of pigs. Additional research is needed to determine the rate and pattern of change in MBST in a range of ambient temperatures below, within, and above the thermoneutral zone.

Infrared thermography (IRT) has been used in veterinary medicine to detect peripheral thermal alterations associated with musculoskeletal lesions. It also has been evaluated as a tool to detect febrile swine. In 2 of those studies, researchers had limited success when attempting to detect a febrile condition by calculating changes in rectal temperature on the basis of MBST measured with an infrared spot meter. Other researchers were more successful, because they reported a detectable febrile response on the basis of the increase in MBST measured with an infrared camera. Because of the inconsistent results, it is unclear whether IRT is a viable diagnostic tool for detection of febrile swine.

Infrared thermography has the advantage of being a passive noninvasive remote temperature-sensing method. More traditional temperature-sensing
devices require restraint of animals. In many commercial swine production systems, the required proximity to pigs for measurement purposes precludes the use of most devices such as rectal or aural probes before the visible onset of somnolence and lethargy associated with the acute phase of an immune response to a pathogen. Because body temperature increases rapidly during an acute stress response, the use of IRT would be preferable for pigs that are not conditioned to contact with humans and would enhance the diagnostic capabilities of veterinarians and producers for assessment of an animal's clinical condition.

Therefore, the study reported here had 2 objectives. Because ambient temperature accounts for a large proportion of the variation in MBST, our first objective was to determine the appropriate adjustment factor to account for this variation. Second, we wanted to evaluate the use of IRT as a method for detecting febrile pigs following exposure to *Actinobacillus pleuropneumoniae*. Materials and Methods

Animals—Twenty-eight crossbred pigs were obtained from our institutional Swine Teaching and Research Farm. Pigs were housed separately in portable stainless-steel stalls (0.8 X 1.8 m) elevated 0.7 m above the floor. Pigs had ad libitum access to a formulated diet (Appendix) and water. Experimental protocols were approved by the Kansas State University Institutional Animal Care and Use Committee.

Collection of thermal images—A high-resolution short-wave (3 to 5 µm) radiometric infrared thermal imaging camera was used. The camera is equipped with a 16º FOV lens with the images displayed in a focal plane array. Images were obtained while pigs were standing unrestrained in their crate. Images were obtained from a distance of 2 m perpendicular to the left side of each pig and collected on a 10-megabyte card for data processing. Digital images were analyzed by a certified technician, using analytical software. The MBST was calculated from an approximately 3,500-pixel image of a range of an animal's clinical condition.

Experimental design—Four castrated male pigs (mean ± SD initial body weight, 30 ± 5 kg) were used to determine the relationship between environmental temperature and MBST. Pigs were allowed 3 days prior to the start of the experiment to acclimate to the stalls. During the adjustment period, ambient temperature was maintained at 21 ± 2 C. Pigs and stalls were moved to an environmental chamber on day 4 and allowed 24 hours to acclimate to the new environment. To minimize heat-producing effects associated with digestion, feed but not water was removed 12 hours prior to obtaining the initial IRT image. On day 5, ambient temperatures were adjusted to 10, 13, 16, 18, 21, 24, 27, 29, and 32 C, using a computer-controlled closed-loop environmental chamber with a controlled air speed of ≤0.15 m/s. Each temperature was maintained constant for a period of 60 minutes at the aforementioned values (±0.3 C), as determined on the basis of the average chamber temperature measured by copper wire thermistors located 0.7 m above the floor at the front and rear of each crate. Infrared thermography images were obtained for each pig 30 minutes after each environmental test temperature was reached. This 30-minute period ensured that heat flow between a pig and its environment had stabilized at each new ambient temperature.

Twenty-four castrated male pigs (initial body weight, 30 ± 1 kg) were allotted in an unbalanced randomized complete block design to inoculated or noninoculated groups. All pigs were seronegative for *A. pleuropneumoniae* prior to inoculation in this experiment. Pigs were housed separately in stainless-steel metabolism stalls in an environmentally controlled room set at 21 ± 2 C with constant lighting. Pigs were given a 3-day acclimation period in the stainless-steel stalls. Feed was removed 6 hours before inoculation to minimize differences in generation of body heat associated with differences in feed intake among pigs.

*Actinobacillus pleuropneumoniae* serotype 1 reference strain 4074 was thawed and subcultured on chocolate agar and blood agar to confirm purity. After a 24-hour incubation at 37 C in 6% CO2, an isolated colony was selected from the chocolate agar and inoculated into 25 ml of brain heart infusion broth in accordance with manufacturers' instructions and supplemented with 2.5% fetal bovine serum containing 0.25 mg of NAD/ml. This culture was incubated to stationary phase at 37 C with shaking. Ten milliliters of stationary-phase culture was inoculated into 100 ml of warm RPMI+ and incubated for 4 hours at 37 C. Each inoculated pig received 4 ml of this culture (5 X 10^10 colony-forming units [CFU]) via intranasal inoculation. Negative-control pigs received 4 ml of sterile RPMI+.

Within the inoculated pigs, there were 2 preplanned subgroups that were classified on the basis of febrile response. Pigs that had an increase in rectal temperature of ≥1.67 C after inoculation were categorized as the inoculated-febrile group (n = 6), and the remainder were categorized as the inoculated-nonfebrile group (6). Reportedly, the acute febrile response to inoculation with *A. pleuropneumoniae* includes rectal temperatures of 40.5 to 41 C, consistent with a temperature increase of ≥1.67 C above the reference range. Mean rectal temperatures of inoculated-febrile pigs in our experiment exceeded this limit (ie, ≥1.67 C) by 6 hours after inoculation, increasing from 38.8 C before inoculation to 40.6 C 6 hours after inoculation.

Measurement of body temperature—Infrared thermographs and rectal temperatures were obtained simultaneously 2 and 1 hour before inoculation, immediately before inoculation (time 0), at 15-minute intervals for 6 hours after inoculation, and then at 3-hour intervals from 9 to 18 hours after inoculation. Rectal temperatures were obtained by use of a handheld digital thermometer with an attached thermistor probe inserted in the rectum to a depth of approximately 10 cm. The thermometer had a precision of 0.1 C.

Using IRT, mean ambient temperature was measured at each sample collection time by evaluation of 3 high-emissivity targets (emissivity, 0.97) positioned at the level of the pigs and equally spaced throughout the room. Mean ambient temperatures were used to correct MBST, using the adjustment factor determined from results of the first experiment; they were used as average absolute radiant heat exchange. Radiant heat exchange between the body surface and environment was calculated from the following equation:

\[ Q_r = A \phi (T_e - T_s) \]

where \( Q_r \) is radiant heat exchange, \( A \) is effective radiant surface area, \( \phi \) is emissivity (assumed to be 1.0), and \( T_e \) and \( T_s \) are the Stefan-Boltzmann constant, Te is average absolute radiant environmental temperature, and Ts is average absolute radiant surface temperature.

Statistical analysis—Using a linear regression model,
data obtained from the 4 pigs in the initial experiment were analyzed to determine the effects of increasing ambient temperature on MBST. Each pig was an experimental unit. Observations at each temperature were used to determine the linear relationship between ambient temperature and MBST.

For the inoculation experiment, data were analyzed as an unbalanced randomized complete block design, using a mixed model with repeated measures. Pigs were blocked by group, with each pig as an experimental unit. Time period was used for the repeated measures. Except for the heat exchange analysis, data were analyzed for main effects of treatment, time, and method (rectal or IRT) as well as associated 2- and 3-way interactions. Heat exchange was analyzed similarly by use of repeated measures, but because the heat exchange calculation did not use rectal temperature, only the factors of treatment, time, and the treatment × time interaction were analyzed. Combined MBST and rectal temperature were the mean of the 2 values for each pig within each time period. Spearman correlation analysis was used to measure the strength of the relationship between rectal temperature and MBST. Values of \( P < 0.05 \) were considered significant.

**Results**

A positive linear relationship \( (r^2, 0.97) \) was observed between ambient temperature and MBST. For ambient temperatures ranging from 10 to 32 C, each increase of 1 C caused an increase of 0.40 C in MBST of pigs (Fig 1). This linear relationship was expressed as follows: \( y_{MBST} = 0.40x + 24.82 \). A significant treatment × time interaction \( (P < 0.001) \) was observed as a result of increased combined MBST and rectal temperatures for the inoculated-febrile pigs, compared with values for noninoculated \( (P < 0.001) \) or inoculated-nonfebrile \( (P < 0.05) \) pigs from 4.25 to 18 hours after inoculation (Fig 2). In addition, inoculated-nonfebrile pigs had significantly \( (P < 0.001) \) increased combined MBST and rectal temperatures 9 and 12 hours after inoculation, compared with values for noninoculated pigs.

A significant \( (P < 0.001) \) treatment × method interaction was observed as a result of differences in rectal temperatures versus differences in MBST on the basis of treatment (Table 1; Fig 3 and 4). Rectal temperatures were significantly increased for inoculated-nonfebrile pigs, compared with noninoculated pigs, whereas MBST did not differ significantly \( (P = 0.55) \) between these groups. In contrast, rectal temperature was significantly \( (P < 0.001) \) higher, and MBST was higher, but not significantly \( (P = 0.07) \), for inoculated-febrile pigs, compared with values for those same variables in noninoculated pigs.

A significant \( (P < 0.001) \) method × time interaction was observed as a result of the differences in observed temperature increases in MBST, compared with rectal temperature (Fig 5). Compared with values at time 0, rectal temperature increased significantly \( (P < 0.001) \) 1.5 hours after inoculation, and MBST was significantly \( (P < 0.001) \) increased 0.25 to 1.5 and 4.25 to 18 hours after inoculation.

Although a significant interaction was not observed, radiant heat loss was significantly \( (P < 0.001) \) affected by treatment (Table 1). Inoculated-febrile pigs had significantly \( (P < 0.001) \) greater radiant heat loss than noninoculated or inoculated-nonfebrile pigs. In contrast,

![Figure 1—Changes in mean body surface temperature (MBST) of 4 pigs (mean ± SD initial body weight, 30.0 ± 0.5 kg) relative to changes in ambient temperatures ranging from 10 to 32 C. There was a significant linear correlation \( (r^2, 0.97; P < 0.001) \) between values. Equation of the linear equation was as follows: \( y = 0.40x + 24.82 \). The SEM for ambient temperature was 0.279, whereas SEM for the intercept was 0.013.](image)

![Figure 2—Effects of inoculation of Actinobacillus pleuropneumoniae and febrile condition on mean values for combined rectal temperature and MBST in pigs (mean ± SD initial body weight, 30 ± 1 kg). Inoculations were administered at time 0. Pigs were allocated to groups as follows: noninoculated (control); n = 12, solid circle), inoculated febrile (6, open square), and inoculated nonfebrile (6, asterisk). The SEM was 0.30, 0.33, and 0.33 for the noninoculated, inoculated-nonfebrile, and inoculated-febrile group, respectively. A significant \( (P < 0.001) \) time × treatment interaction was detected.](image)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Febrile (n = 6)</th>
<th>Nonfebrile (n = 6)</th>
<th>Noninoculated (n = 12)</th>
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</thead>
<tbody>
<tr>
<td>Temperature (C)*</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Rectal</td>
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<td>39.4 ± 0.3</td>
<td>39.1 ± 0.3</td>
</tr>
<tr>
<td>Surface</td>
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<td>32.2 ± 0.3</td>
<td>32.3 ± 0.3</td>
</tr>
<tr>
<td>Radiant heat loss (kcal/h)*</td>
<td>-33.5 ± 0.4</td>
<td>-31.8 ± 0.4</td>
<td>-32.4 ± 0.4</td>
</tr>
</tbody>
</table>

Values reported mean ± SD. *Rectal temperature was obtained by use of a digital thermometer with a thermistor probe, and surface temperature was obtained by use of infrared thermography. For temperature, there was a significant \( (P < 0.001) \) group × method interaction. **Significant \( (P < 0.001) \) effect of group. a,b,c = Within a row, values differ significantly \( (P < 0.001) \). c,d = Within a row, values differ significantly \( (P < 0.05) \).
inoculated-nonfebrile pigs had significantly lower (P = 0.04) radiant heat loss than noninoculated pigs. Rectal temperature and MBST were significantly correlated (r, 0.52; P < 0.001). Using the conditions reported here, a moderate relationship existed between rectal temperature and MBST.

Discussion

Analysis of our results indicated that for pigs in closely controlled environmental conditions, MBST increases at a constant rate as ambient temperature increases from 10 to 32 C. Although we recognize that ambient temperatures > 32 C are common for pigs in commercial production facilities, our intent was to establish the relationship within temperatures frequently encountered at our research facilities. This relationship allows adjustment of MBST obtained at various ambient temperatures within the range of 10 to 32 C to a common ambient temperature, which enables more accurate comparisons of actual treatment differences. Further research is necessary to determine the upper limit at which the linear relationship between MBST and ambient temperature changes when IRT techniques are used. Our results are consistent with results of other studies3-5,14 in which investigators observed that MBST increased at a constant rate in humans and pigs subjected to temperatures that were less than the upper thermoneutral limit.

For humans exposed to temperatures higher than the upper thermoneutral limit, the rate of increase changed because of increased latent heat losses.3 In our study, MBST continued to increase at a constant rate. This likely was a result of the pigs increasing the rate of sensible heat loss, thus maintaining internal homeothermy, because swine do not increase their rate of nonrespiratory evaporative heat loss in response to increased ambient temperature.1,15 Although cutaneous blood flow was not measured, our results are consistent with the expected physiologic responses by mammals to changes in ambient temperature. Mean body surface temperature is less at low ambient temperatures than MBST at neutral and warm ambient temperatures because of subcutaneous vasoconstriction to minimize heat loss to the environment. At neutral and warm temperatures, heat loss increases as subcutaneous vasodilation increases in proportion to increased ambient temperature. The vasodilatory response to increased ambient temperature maintains the heat loss rate, thus maintaining internal homeothermy as the thermal gradient between a pig and its environment narrows.1

Results for the initial experiment reported here can be used in future studies of IRT to adjust MBST obtained at ambient temperatures varying between 10 and 32 C to a common ambient temperature. This adjustment factor will allow more accurate and representative comparisons among specific animals or groups of animals. Although this is a wider temperature range than previously evaluated,5 additional research is needed to clarify the relationship for ambient temperatures greater than 32 C.

Analysis of results for the second experiment reported here indicated that IRT was an effective method to detect a febrile response in MBST associated with A pleuropneumoniae inoculation. The difference in the interval until an increase in rectal temperature is detected, compared with an increase in MBST, is consistent with physiologic responses as a febrile response progresses. In a nonfebrile healthy state, excess internal body heat is transferred to the surface by tissue
conduction and blood-flow convection routes, then to the environment via sensible and latent heat-loss routes to maintain internal homeothermy. At the onset of febrile conditions, proinflammatory cytokines stimulate decreased blood flow to the skin surface and increased cellular heat production, leading to increased internal heat loads. Over time, a new temperature threshold is reached, and excess body heat then is released via increased blood flow to the body surface and increased respiration rates.

We observed an increase in MBST attributable to stress of handling pigs during the inoculation procedure, which subsided by 1.5 hours after inoculation (Fig 5). This stress response probably was the result of catecholamine release during restraint, which initially causes vasoconstriction that is followed by vasodilation as internal heat builds above threshold temperatures. Furthermore, anecdotal evidence from our laboratory suggests that rectal temperatures in clinically normal pigs (body weight, 13.6 kg) typically exceeds 40°C within 1 minute after onset of restraint. Because of this variability, rectal temperatures may be relatively insensitive for detecting febrile pigs that are still active and avoiding contact with humans.

After the temperature increase associated with restraint subsided, rectal temperatures increased approximately 2 hours before MBST increased, presumably in response to infection attributable to *A. pleuropneumoniae*. This differential probably was the result of the increased heat production and retention necessary for a fever to develop. After the increase in internal heat load reached the new threshold, the excess heat was dissipated to the environment. This heat loss was evident in the increased MBST and radiant heat loss of inoculated-febrile pigs, compared with noninoculated pigs. Analysis of our results indicated that mean radiant heat loss per hour for febrile pigs averaged over the entire 20 hours of the experimental period increased by almost 4% more than that for noninoculated pigs. This increase presumably was attributable to the increased rate of heat loss to maintain the new thermal setpoint in the hypothalamus.

Although the combined value for MBST and rectal temperatures was numerically higher for the inoculated-febrile pigs, lower MBST and radiant heat loss indicated that this numeric increase probably was the result of increased heat retention. This increase was insufficient to cause a detectable febrile response at the surface before the immune system effectively controlled the pathogenic challenge.

Although our data were consistent with an expected physiologic response, results of other studies in which IRT was used to detect a febrile response or condition have been inconsistent. This inconsistency was probably attributable to differences in experimental design and IRT methods. Investigators who used IRT with a camera to measure MBST for an area 10 cm in diameter obtained results consistent with ours, indicating that IRT can detect increased MBST of specific pigs. In that study, MBST of individual pigs was compared with the group mean by measuring MBST on the back or hind quarters. In addition, it was observed in that report that an MBST ≥ 2°C higher than the group mean suggested a febrile condition and was associated with rectal temperatures ≥ 1.5°C higher than the group mean. This is consistent with our preplanned increase in rectal temperature of 1.67°C to segregate febrile and nonfebrile responses. Thus, use of an infrared camera with a relatively large area for surface detection apparently is important to accurately detect differences in MBST resulting from changes in internal heat production. Although differences were detected in MBST in 1 report, investigator failed to differentiate between increased body temperatures resulting from feed intake versus those resulting from challenge-exposure with a pathogen. Because feed intake is a metabolically exothermic process, we chose to withhold feed throughout the experimental period to clearly identify changes in MBST associated with a febrile response. Confounding effects for increased MBST in healthy pigs receiving feed versus the increased MBST of febrile pigs undergoing stress attributable to infection with a pathogen remain to be addressed.

In another study, investigators used an infrared spot meter to measure temperature changes at the base of the ear in growing pigs administered noninfectious endotoxin; however, they were unable to detect changes in the febrile condition by use of body surface temperature to predict changes in rectal temperature. This lack of response was partly attributable to the fact that they intended to detect a febrile response by attempting to predict rectal temperature on the basis of changes in a point estimate of body surface temperature. Multiple factors influence the inability of investigators to use MBST changes to predict changes in rectal temperature. Use of an infrared spot meter derives an estimate for mean surface temperature for a small area approximately 0.64 cm in diameter. Surface temperature can vary widely within small areas on an animal's head, limbs, or trunk as a result of changes in vasodilation, biorhythm of the animal, density of veins and arteries immediately beneath the site, and environmental conditions. Thus, estimates obtained by use of a spot meter may not accurately reflect MBST, even within a local region. In addition to difficulties associated with use of infrared spot meters, the febrile response to administration of noninfectious endotoxin is quite rapid, with the febrile state lasting only a few hours in pigs. This rapid flux in body temperature may not have provided a sufficient internal heat load to cause a detectable change in mean surface temperature at the base of the ear. Also, failure to segregate pigs administered endotoxin on the basis of the amount of actual change in rectal temperature may have masked their ability to use their method to detect a febrile response. In contrast, our results indicated that a febrile condition from inoculation of an infectious pathogen in growing pigs is detectable as a change in MBST when an infrared camera is used for a broad surface area.

In addition to the increase in MBST, we observed a significant correlation (r = 0.52) between MBST and rectal temperature. Wendt et al used an infrared spot meter and reported moderate correlations between MBST and rectal temperature of growing pigs (r = 0.70) and sows (r = 0.64) after accounting for body weight and environmental conditions. Zinn et al also used an
infrared spot meter in their attempt to predict changes in rectal temperature of sows before and after farrowing, using a predictive equation that combined temperatures from several points on the body surface. They were able to detect only a weak correlation between temperature for any single point or the combined MBST and rectal temperature, and the strongest correlation was between MBST and rectal temperature (r = 0.061). After correction for environmental conditions, the correlation improved (r = 0.273).

Analysis of results of the study reported here indicated that use of an infrared camera to obtain measurements of MBST for a broad area (> 10 cm) in pigs can accurately detect changes in MBST associated with a febrile response. Because IRT is a remote noninvasive method, it may have potential for use in most reliably identifying febrile pigs in situations where handling and restraint is impractical.

References