Effect of High Temperature and Humidity in the Periovulatory Phase on Swine Ovarian Function

Akira OGASA, Hiroshi MIYAJIMA1), Shokichi IWAMURA1), Ikuo DOMEKI1), Eiichi KAWAKAMI, and Toshihiko TSUTSU1
Department of Reproduction, Nippon Veterinary and Zootechnical College, 1-7-1 Kyonan-cho, Musashino, Tokyo 180, 1)Ueda Health Center, Ueda, Nagano 386, 2)National Institute of Animal Health, Tsukuba, Ibaraki 305, 3)National Institute of Animal Industry, Tsukuba Norin-danchi, Ibaraki 305, Japan
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It is suspected that several factors such as hot, humid climates, restraint and malnutrition may be involved in the increased incidence of swine ovarian diseases such as anestrus, silent estrus and ovulation failure in late summer and early fall.

The purpose of this investigation is to determine whether heat and humid stress have any effects on estrous behavior or ovarian function in the sow. The possible effect of thermal stress on the ovarian function was evaluated on the basis of abnormal estrous behavior, ovarian structural changes detected by rectal palpation and irregularity in plasma hormonal patterns of estrogens, progesterone and luteinizing hormone (LH).

This investigation consisted of two experiments. One Landrace sow was used repeatedly in both experiments. The sow was 5 times parous and weighed 215 kg. In experiment 1 the sow was kept under natural environmental temperature to serve as controls of experiment 2. In experiment 2 the sow was subjected to an elevated environmental temperature of 33°C and 80% humidity in a climatic chamber for 3 hr (9:00–12:00) every day for 1 week beginning on day 4 before ovulation. In addition to the above-mentioned treatment, the sow was exposed to a high temperature of 35°C and 80% humidity for 3 hr, 5 times during 41 hr between the onset of estrus and ovulation. The rest of the time the climatic chamber was maintained at a temperature of 26°C and 60% humidity and was illuminated from 5:00 to 17:00.

Blood samples were collected via an indwelling jugular catheter at 1 to 3 hr intervals. Plasma concentrations of estrone (E1), estradiol-17β (E2), progesterone (P) and LH were measured using radioimmunoassay methods described by Niwender et al. [3] and Domeki et al. [2].

Experiment 1: Under natural environmental conditions the sow exhibited obvious estrous behavior. Plasma E2 elevated to the level of 43–59 pg/ml and maintained the high level with two distinct peaks during 48 hr prior to ovulation then decreased sharply. It was still low on the day of ovulation. The peripheral plasma P concentration remained low until ovulation, tending to increase gradually after ovulation (Fig. 1). Plasma LH levels began to increase gradually 7–8 hr after the onset of estrus, and showed two sharp peaks at 21 and 27 hr, respectively, then dropped to low levels (Fig. 2).

Experiment 2: When the sow was exposed to high temperature and humidity, she showed feeble estrus and the estrous period was a little shorter than that in experiment 1. Furthermore, this sow exhibited ovarian cysts associated with corpora lutea. This fact suggests that the thermal and humid stress given may inhibit ovulation in some of mature follicles and it may lead to produce cystic follicles. Thague et al. [7] and Scholten and Liptrap [6] also reported that high temperature significantly decreased the ovulation rate in the swine species.

In this sow, however, a distinctly sharp peak of plasma E2 was not observed, and also the duration of high levels of E2 was shorter than in experiment 1 (Fig. 3). These results were in accord with the result in our other experiment in which E2 concentrations during the follicular phase in sows given daily adrenocorticotropic hormone (ACTH) did not show any clear peak and the sows displayed feeble estrus (unpublished data). In experiment 2, plasma P values remained low during the period of daily exposure to the high temperature, but they rose slowly after day 3 of the subsequent estrous cycle (Fig. 3).

Plasma LH concentrations showed a smaller peak for short duration than those in experiment 1 (Fig. 2). Barb et al. [1] described that treatment
Fig. 1. Peripheral blood concentrations of progesterone and estrogens in the periovulatory phase in a sow kept under natural environment (Exp. 1).

Fig. 2. Concentrations of LH in plasma samples collected during two periovulatory phases under natural environment (Exp. 1) and exposed to high temperature and high humidity (Exp. 2) in a sow.
with hydrocortisone or ACTH blocked the preovulatory surge of LH and ovulation in all giltts used. In addition, Olster and Ferin [4] and Rivier and Vale [5] described that exogenous corticotropin releasing hormone administration resulted in inhibition of LH and follicle stimulating hormone secretion.

These results suggest that stressful conditions such as high ambient temperature and humidity during the summer season cause to decrease gonadotrophin secretion and lead to reproductive disfunctions such as anestrus, silent estrus, ovulation failure and ovarian quiescence.

### References