Ovarian Response to hCG in the Postweaning Sow

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ABSTRACT. Five non-cyclic postweaning sows were given intramuscularly 1,000 IU of hCG on day 2 (4 sows) and day 36 (1 sow) after weaning. Single or multiple follicular cysts were developed in ovaries in all cases. Plasma E_2 fluctuated at low levels in 4 of 5 sows resulting in silent heat. The sows which exhibited neither estrus nor ovulation after hCG administration did not display any preovulatory LH surge, and plasma progesterone levels remained low (≤ 1 ng/ml). These results suggest that abnormal follicular growth or impairment of the ovulation process as a consequence of unsatisfactory LH release after hCG treatment in swine with ovarian inactivity may promote the formation of ovarian cysts.—**KEY WORDS:** hCG, LH, swine cystic follicle.

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Hormone preparations have been widely used to treat porcine ovarian subfunction. At the present time, among hormone preparations available to treat ovarian inactivity in Japan, pregnant mare serum gonadotropin (PMSG) is the most frequently administered alone or in combination with estradiol benzoate (E_2B), human chorionic gonadotropin (hCG) or gonadotropin releasing hormone (GnRH) [13].

hCG has also been used alone for the treatment of persistently anestrous sows. Treatment with hCG caused an increase in circulating concentrations of estradiol-17 β (E_2) and ovulation. However, no surge in luteinizing hormone (LH) was observed in half of the persistently anestrous sows [1]. Ovulatory doses of hCG (500 to 1,500 IU/head) caused attenuated secretion of E_2 and blocked a preovulatory LH surge in some treated gilts which were synchronized for estrus by feeding Altrenogest and following PMSG injection [14].

Johmen *et al.* [7] reported that hCG was capable of inducing ovarian cysts in swine. The ovarian response to hCG in non-cyclic sows was more drastic than in cyclic females and typical multiple cysts were induced by administering a large dose of hCG.

The mode by which hCG causes ovulation or induces cystic ovaries in swine, especially in non-cyclic females, is therefore still obscure.

The purpose of the present study was to assess ovarian responsiveness to hCG in non-cyclic postweaning sows by means of rectal palpation of ovaries and measurement of circulating plasma concentrations of LH, E_2 and progesterone.

Five Large White multiparous sows farrowed in May, 1987 and April, 1988 were used in this study. Indwelling cannulas were inserted into the jugular veins of these sows 2 days before hCG administration [6]. Four of the sows were given intramuscularly 1,000 IU of hCG on 2 days postweaning, 28–32 days postpartum. One sow (No. 122) with quiescent ovaries for 36 days after weaning was also given 1,000 IU of hCG.

Plasma concentrations of LH [12] and E_2 [2] were measured by RIA and progesterone by EIA [11] in a series of blood samples collected at 3–6 hours intervals for 3–4 days after hCG administration and once daily for at least 9–15 days thereafter. After hCG administration the

animals were checked for estrus at least twice daily in the morning and evening. Rectal palpation was performed daily for 10–15 days after treatment to examine changes in the ovaries. Sow No. 122 was slaughtered 10 days after hCG administration.

The growth of follicles was monitored by palpation in all sows 2–3 days after the administration of hCG. Excepting sow No. 122, clinical examination of the sows failed to reveal any noticeable signs of estrus, including vulvar swelling and hyperemia, increases in diameter or firmness of the cervix. In two (Nos. 9 and 121) of the 5 sows ovulation did not occur and single or multiple follicular cysts were developed after treatment. In two other sows (Nos. 13 and 15) ovulation was induced without estrus on day 3 after treatment, and one or two cystic follicles, 1.5–2.0 cm in diameter, were found on day 8–13 after treatment. In the remaining sow (No. 122) many follicles ovulated with weak signs of estrus and 3–4 non-ovulated follicles became large follicular cysts in both ovaries on day 6 after treatment.

Plasma E_2 levels fluctuated within the range 18–40 pg/ml without any marked increases in one sow with multiple cysts (No. 9), any in another sow with single cysts (No. 121) the levels peaked to 40 pg/ml on day 3 after treatment. Progesterone levels remained low (≤ 1 ng/ml) until day 7–8 after hCG injection in sows Nos. 9 and 121, and thereafter the levels increased more than 1 ng/ml (Figs. 1 & 2). This may be caused by luteinization of unovulated atretic follicles and partial luteinization of the walls of large cystic follicles. In two other sows with single cysts during the early luteal phase (Nos. 13 and 15), plasma progesterone levels started to increase on day 3 after treatment.

The sows which exhibited neither estrus nor ovulation after hCG administration (Nos. 9 and 121) did not display any preovulatory LH surge. One sow with multiple large cysts (No. 122) and two other sows with single cysts displayed a preovulatory LH surge or a slight increase in LH. When sow No. 122 was autopsied 10 days after treatment, the right ovary was found to contain 4 cystic follicles (CF), 1.5–2.3 cm in diameter, and 16 corpora lutea (CL), while the left ovary exhibited 3 CF, 1.8–2.2 cm in diameter, and 22 CL.

The ovaries of anestrous and postweaning sows did not

Fig. 1. Plasma concentrations of LH, estradiol-17 β and progesterone in sow No. 9 intramuscularly treated with hCG 1,000 IU on day 2 after weaning. Notes: - ~ +++: Degree of follicular growth after hCG administration. ++++: Formation of cystic follicles.

Fig. 2. Plasma concentrations of LH, estradiol-17 β and progesterone in sow No-121 intramuscularly treated with hCG 1,000 IU on day 2 after weaning. Notes: - ~ +++: The same as in Fig. 1.

undergo adequate follicular development and maturation in response to hCG treatment, estrogen production tended to be low, and these sows exhibited silent heat in the follicular phase as a result [4]. It has been postulated that the development of multiple cysts with a number of corpora lutea followed by an LH surge may be due to a decrease in the number of LH receptor [8].

Miyake *et al.* [9, 10] and Ziecik *et al.* [14] observed the absence of any preovulatory LH surge in some hCG-

treated gilts. They also suggested that hCG acts by inhibiting GnRH release from the hypothalamus [3, 5, 15].

The results of this study suggest that abnormal follicular growth or impairment of the ovulation process as a consequence of inappropriate LH release after hCG treatment in swine with ovarian inactivity may promote the formation of ovarian cysts.

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