

Human chorionic gonadotrophin induces ovarian follicle growth in pregnant sows

Fertility of female pigs is often significantly depressed during the summer and early autumn, an effect referred to as seasonal infertility. Manifestations of seasonal infertility can include reduced pregnancy rates and, less often, smaller litter sizes. These problems are most likely to occur with younger sows. The underlying cause of this infertility likely involves many factors but ultimately it is a failure of the sows to adequately maintain the corpora lutea of pregnancy. Maintenance of corpora lutea requires a signal for maternal recognition of pregnancy (MRP) which, if not received results in the sow thinking she is not pregnant so she returns to estrus. In the pig, the signal for MRP is the embryonic production of estrogen which results in the redirection of the luteolytic signal and enhancement of the luteotrophic signal. If the corpora lutea do not get the luteolytic signal pregnancy is continued. The signal for maternal recognition of pregnancy occurs at about day 11 or 12 of pregnancy which is also the time from which corpora luteal progesterone is released in response to luteinizing hormone (LH). If the pregnancy recognition signal is weak then the corpora lutea become more susceptible to environmental stresses and so more prone to failure. If an enhanced estrogenic signal was produced it could support susceptible corpora lutea and result in the maintenance of fertility. A potential source of estrogen is medium to large growing ovarian follicles and, in the pig, the primary endogenous driver of the growth of this sized follicle is LH. Therefore, we hypothesised that injection of the LH analogue human chorionic gonadotropin (hCG) would induce growth of estrogen producing follicles and, by mimicking the signal for MRP and stimulating progesterone secretion, improve the fertility of young sows.

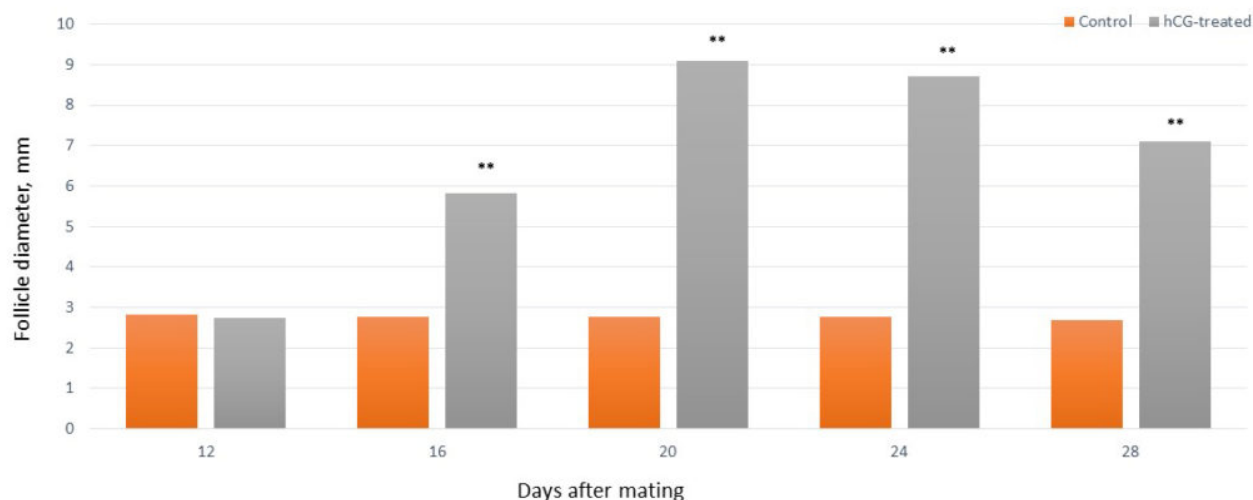


Fig. 1. Effect of injection of 1,000 IU hCG at 12 d after mating (n=17) or non-treatment (n=19) on follicle diameters between days 12 and 28 after mating.

In our first experiment, we injected 1,000 international units of hCG into mature sows at 12 days after mating while others were non-injected controls. We then followed follicle growth by ultrasound on days 12, 16, 20, 24, and 28 plus obtained blood samples on days 12, 14, and 15 for estradiol measurement. The hCG did indeed induce follicles to grow for about a week, these follicle did produce estradiol, and the corpora lutea also produced more progesterone. Having shown that the hCG did have the desired effect on the ovaries we tested the ability of hCG to maintain fertility of young sows during the period of seasonal infertility. Again, we injected the hCG at 12 days after mating while others were non-injected controls. Pregnancy status was determined at 28 days using ultrasound and sows were allowed to go to term to determine the number of sows producing a litter and the average number of piglets in the litters. Our hypothesis was confirmed in that hCG injection was associated with a higher pregnancy rate (P less than 0.05) and more sows going on to produce a litter. However, there was no effect on the number of piglets in the litters. These data confirm that hCG stimulates growth of estrogenic follicles and corpora luteal function, and improves the fertility of young sows during the summer months.

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Publication

[Human chorionic gonadotrophin in early gestation induces growth of estrogenic ovarian follicles and improves primiparous sow fertility during summer.](#)

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Anim Reprod Sci. 2016 Sep