AGE/WEIGHT - RELATED SEXUAL MATURATIONAL CHANGES IN PREPUBERAL GILTS

by

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A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfilment of the requirements for the degree of Master of Science.

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Montreal, Quebec, Canada

February, 1987

Suggested short title:

PUBERTY AND PREGNANCY IN THE GILT

Karalus

DEDICATION

This thesis is humbly dedicated to:

Emmi Braecker
Martha & John Karalus

and

my gilts

in respect and affection.

ACKNOWLEDGEMENTS

He that does good for good's sake, seeks neither praise nor reward, but he is sure of both in the end.

- William Penn

The Surgical/Bleed Team

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MY GILTS-ALL 54 OF THEM!

This research was supported by Agriculture Canada, Grant #84008, and CRSAQ, Grant #MCA-86-B-2020.

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GENERAL INTRODUCTION

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Gilts represent up to 40% of the breeding herd on many farms today(Tomes and Nielsen, 1982). Thus, their reproductive performance will have a strong influence on total piglet production during the farming year. Unfortunately, reproduction is the least efficient facet of the pork production system. Gilts add to this inefficiency in two ways: there is a time lag before they become part of the breeding herd(age at puberty) and they farrow a smaller litter than brood sows.

A single injection of pregnant mare's serum gonadotropin(PMSG) followed 72 h later by another of human chorionic gonadotropin(hCG) has been used experimentally in immature gilts to supply a source of materials for follicular and cytological studies *in vitro* and to study follicular development and ovulation *in vivo*. The capacity of this hormonal combination to induce estrus and continued cyclicity and/or pregnancy in prepuberal gilts has been studied but many questions remain unanswered. There might be a potential to increase reproductive performance through their use. Two experiments were undertaken: the first to assess maintenance of cyclicity and pregnancy in three age/weight groups of prepuberal gilts following treatment with PMSG/hCG and the second to study LH scretion patterns before and after GnRH administration in an attempt to

explain the variation among groups that was anticipated in the first study.

ABSTRACT

M.Sc. I. Ursula Karalus Animal Science (Physiology)

AGE/WEIGHT - RELATED SEXUAL MATURATIONAL CHANGES IN PREPUBERAL GILTS

The induction of puberty with PMSG(750 i.u.) and hCG(500 i.u. 72 h later) and the maintenance of pregnancy after artificial insemination at second estrus was assessed in 48 prepuberal gilts each representing one of three age/weight groups. Signs of estrus and ovulation were induced in 46/48 animals but based on plasma concentrations of progesterone and estrone sulphate and reproductive tract measurements taken at slaughter(34 d post-A.I.), 54% of gilts reverted to the prepuberal state after one estrous cycle. Of the cyclic gilts, 52% were pregnant with 3-10 live embryos. Older, heavier gilts($\geq$ 160 d, $\geq$ 75 kg) maintained cyclicity and pregnancy more reliably than their younger counterparts. Overall, 89% of treated animals reached puberty by 200 d. In a second experiment, 8-h pulsatility patterns of plasma LH were determined in six catheterized prepuberal gilts at 110, 140 and 170 d(mean ages of the three groups in Experiment 1). As a measure of anterior pituitary responsiveness, a single i.v. dose of GnRH(0.5 μg/kg) was administered at the end of the 8 h and blood samples were collected for an additional 4 h. Pre-GnRH LH means, amplitudes and frequencies of release, and post-GnRH LH surge heights tended to be higher at 110 d.

RÉSUMÉ

M.Sc. I. Ursula Karalus Science Animale (Physiologie)

CHANGEMENTS DE LA MATURITÉ SEXUELLE EN RELATION A L'AGE ET AU POIDS CHEZ LES TRUIES PRÉPUBÈRES

L'induction de la puberté avec PMSG(750 u.i.) et 500 u.i. de gonadotrophine chorionique humaine 72 h plus tard de meme que le maitien de la gestation après insemination artificielle au second oestrus a été determine chez 48 truies prépubères chacune représentant un des trois groupes age/poids. Les manifestations d'oestrus et d'ovulation ont été induites chez 46 des 48 animaux cependant 54% sont retournés à un statut prépubère après un cycle. Cette dernière observation est basée sur les concentrations plasmatiques de progestérone et d'oestrone sulphate et sur les dimensions du tractus génital après l'abbattage(34 jours post-insemination). 52% des truies démonstrant un cycle etaient gestantes avec 3 a 10 embryons vivant. Les cycles sexuels et la gestation étaient maintenus plus fortement par les truies plus agées et de plus grand poids($\geq$ 160 jours, $\geq$ 75 kg) que par les truies plus jeune. Au total, 89% des animaux traités araient atteint la puberté au 200e jour. Dans une seconde expérience, la sécrétion pulsative de LH a été determiné pendant 8 h chez six truies prépubères cathétèrizée au jours 110, 140 et 170(age moyen des trois groupes dans l'expérience 1). A la fin de la période de 8 h une simple dose i.v. de GnRH(0.5 µg/kg) a été administré et

des échantillons sanguins ont été recueillis pendant 1 h additionnelle afin de mesurer la sensibilité de l'hypophyse antérieure. Les moyennes, amplitudes et frequences de relâchement de LH pré-GnRH de même que la hauteur des courbes de relâchement de LH post-GnRH tendaient à être plus élevées au 110e jour.

EXPERIMENT 1
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LITERATURE REVIEW

THE NORMAL REPRODUCTIVE PROCESS: Domestic pigs are born in large litters (9-12 piglets) after a gestation of about 115 d (Hughes and Varley, 1980). From then on, they grow rapidly until the state of sexual maturity or puberty is reached. Puberty can be defined as the exhibition of first estrus followed by continued cyclicity. There is a large variation in puberal age and weight. Onset of puberty is reported to range from 102 to over 350 d of age (Brooks and Smith , 1980) with less than 10% reaching puberty prior to 160 d (Callaghan and King, 1978) and within a weight range of 55 to over 120 kg (Hughes and Cole, 1975).

Externally, the approach of estrus is often initially noticeable 2-6 d earlier by the reddening and swelling of the female's vulva(Burger, 1952), a reaction to increasing levels of estrogen (E_2) produced by developing follicles. At this time, the gilt will be active, have reduced appetite and search for the male. At the peak of sexual receptivity, the female will arch her back, cock her ears, 'stand' rigid and wait for the male to mount and breed her(Signoret, 1980c). Estrous behaviour, which lasts 40-70 h (in the sow) can be induced artificially by E_2 injection or implant(Signoret, 1967; Stephens and Challis, 1974).

Ovarian follicles grow under the influence of follicle stimulating hormone (FSH) and when mature, ovulate in response to a surge of luteinizing hormone (LH). Total time for ovulation averages 3.8 h which occurs 38-42 h after the onset of estrus (Hughes and Varley, 1980). The number of eggs released at the puberal estrus by Landrace gilts was found to be 11.5 by Squiers et al. (1952) regardless of an animal's age or weight. A corpus luteum (CL) forms at each ovulation point, and progesterone (P_4) is synthesized and released in increasing amounts as the CL's develop. Estrous cycle length varies from 18-24 d but averages 21 d. The first day of estrus is designated as Day 0. The puberal estrus is nearly always fertile with mating occurring from the day before estrus to the third day of estrus (Burger, 1952; Hancock and Hovell,1962). Conception rates over 80% are normal (Burger, 1952; Brooks and Cole, 1973; Pay and Davies, 1973; MacPherson et al., 1977).

REPRODUCTIVE TRACT DEVELOPMENT: Development of the reproductive tract appears to be age dependent. Tertiary follicles first appear around 10 weeks of age (Casida, 1935; Hadek and Getty, 1959; Kather and Smidt, 1974; Oxender et al., 1979). From 70-112 d of age, there is a rapid increase in ovarian weight followed by a period of slower steady growth until puberty. Between 126 and 168 d, Dyck (1972) reported a change in follicle numbers greater than 3 mm, from two to a population of 12. The secretion of low levels of E_2 accompanies follicular growth.

The uterine wall increases in thickness and gland development to 60 d (Schnurrbusch et al., 1980). Upon completion of uterine gland development(70-84 d), the uterus begins a period of elongation and further weight increases until puberty (Dyck, 1972). Hadek and Getty (1959) postulated that from three months onward, gonadotropins begin to influence the growth of the uterus. Fallopian tube maturation is complete by 112 d (Dyck, 1972). Between 12-24 weeks, the uterine glands begin secretion (Erices and Schnurrbusch, 1979). The development of mucosal clefts and folds accounts for further weight increases up to 168 d (Hadek and Getty, 1959; Bal and Getty, 1970; Dyck, 1972). The endometrium which was 126 μ thick in the newborn expands to 5520 μ within six months (Hadek and Getty, 1959). Schnurrbusch et al.(1980) and Erices and Schnurrbusch(1979) reported no further tract growth from 170 d until puberty and hypothesized that this resulted from the lack of appropriate stimulation.

At puberty, there is a twofold increase in uterine and Fallopian tube weight (Shaw et al., 1970; Dyck, 1972; Schnurrbusch and Erices, 1979) which has been attributed to the hormonal influence of E_2 (Raeside, 1963). The first cycles are characterized by more uterine growth and stabilization of its function. Schnurrbusch and Erices (1979) noted that the myometrium and endometrium continued to thicken from cycle one to three and thus advised breeding at the second estrus.

With pregnancy, additional changes can be expected in the reproductive tract. Dhindsa et al. (1967) suggested a pattern of development in the accommodation of fetuses by the uterus: firstly, a preparatory stage (6 to 15 d) after conception with no increase in the weight or volume; secondly, a stage of enlargement; and finally, a period of stretching. Bilaterally ovariectomized (OVX) gilts had shorter uteri than normal gilts which suggested that ovarian hormone levels were likely responsible for differences in uterine size.

FACTORS AFFECTING ONSET OF PUBERTY: The onset of puberty is influenced by various internal and external factors which can be either stimulatory or inhibitory in varying degrees. These factors include age, weight, breed, season, social environment, housing, stress, nutrition, and exogenous hormones. Many of these variables can be manipulated by the stockman.

Several studies involving breed comparisons have found the Landrace to be one of the earliest maturing breeds(Christenson and Young, 1978; Hutchens et al., 1982; Allrich et al., 1985). Regardless of breed, heritability of age at puberty is considered to be low so that genetic selection for this trait is slow. Unless crossbreds were desired instead of purebred animals, heterosis could not be exploited in most cases (Reutzel and Sumption, 1968). However, even within the breeds, maternal (Reutzel and Sumption, 1968), family (Burger, 1952) and sire effects

(Hughes and Cole, 1975) have been reported. Burger (1952) found a strong tendency for littermates to reach sexual maturity within a brief span of time, 54% of sisters attaining puberty within 5 d of each other.

Gilt age seems to be of equal importance to weight in regard to when puberty is reached. Individual variation is much greater than variation between breeds or in different environments (Brooks and Cole, 1973).

If growth is delayed, puberty will be delayed. Levasseur (1977) felt this was a safety measure by the body to prevent reproduction before a certain stage of body development or metabolism was reached. Foxcroft (1980) regarded the stage of growth as 'permissive', compatible with but not the trigger for puberty.

Burger (1952) tested a group of sisters of the Large White and Large Black breeds. Growth in one group was limited to half that of the normally fed gilts in the control group. Puberty was delayed 46 d in the limit-fed gilts. The oldest gilt in the ad libitum group was still younger than the youngest animal in the retarded group.

Undernutrition delays sexual development. When starved animals are allowed rehabilitation, growth of the rest of the body restored sex organs to their proper proportions. It is thought that undernutrition accentuates body priorities, reproduction not being high priority. Luteinizing hormone is only produced in significant quantities when the animal approaches the size where puberty

normally occurs. Possibly, the production of hormone or the response to it, may be linked with the attainment of a definite body size (Dickerson et al., 1964). Likewise, Ershoff (1952) in his review of the effect of 'malnuriture' on the synthesis and secretion of pituitary hormones, commented that when there was a weight loss of 25-40%, the formation and release of gonadotropins was depressed. Ovarian hypofunction, typified by uterine atrophy and anestrus, appeared to result from the absence or lowering of circulating gonadotropins, not to a refractory state of the ovaries. Upon injection of supplemental gonadotropin, starved animals re-established ovarian and uterine function.

Tassel (1967), in surveying literature on the effects of diet, found the experiments difficult to evaluate in that they were based on varying protein levels, caloric levels, breeds and seasons. He concluded that underfeeding causes a marked delay in the age at first estrus. Studies by Robertson et al.(1951), Haines et al.(1959), Brooks and Cole (1973, 1974) and MacPherson et al.(1977) show that ovulation rate is maximized in gilts fed ad libitum.

In trial after trial, a significantly lower percentage of gilts reached puberty from June to October than November to May (Wiggins et al.,1950b; Scanlon and Krishnamurthy, 1974; Bane et al, 1976; Christenson, 1981). A combination of photoperiod and temperature seems to be responsible. Yet Hughes (1982), in his overview of photoperiod alone, concedes that 17-18 h light should be sufficient for good

reproductive performance as most research isolating this factor shows puberty to be advanced with increasing day length.

A substantial number of gilts(25% or more) close to the age of natural puberty, when relocated and mixed with strange animals, react physiologically by coming into heat in a few days (du Mesnil du Buisson and Signoret, 1962; Signoret, 1970). The 'transport' or stress effect was later found to be complementary to another stimulus - that of introducing a boar to the gilt. When 165 d old gilts were transported and a proportion of them exposed to a boar soon after, the percentage of females in estrus increased from 53% to 71% (Bourn et al., 1974). Similar results were obtained by du Mesnil du Buisson and Signoret (1962), Sheimann et al.(1976), Zimmerman et al.(1976), Kinsey and Zimmerman (1977). By boar exposure alone, gilts reached puberty earlier than those not exposed to a male (Thompson and Savage, 1978).

The optimum age for gilt receptivity to the boar appears to be 160-165 d (Brooks and Cole, 1970; Bourn et al., 1974; Hughes and Cole, 1976; Brooks and Smith, 1980). Sixty to 90% of gilts attained puberty in 7-10 d. In animals stimulated at a younger age, estrus synchronization was reduced and many had not reached puberty by 260 d. Brooks and Cole (1973) considered that very young gilts introduced to a male are not able to respond to this stimulus but become conditioned to it thereby delaying puberty. However,

Hughes and Cole(1976) and Kirkwood and Hughes(1979) felt that continued physiological development leads to a growing ability to respond to the boar even at young ages. Gilts exposed to the boar later than 160 d showed no reduction in the percentage of animals reaching puberty but the degree of estrus synchronization decreased with advancing age (Brooks and Cole, 1970; George and England, 1974; Hughes and Cole, 1976; Kirkwood and Hughes, 1979; Brooks and Smith, 1980; Cronin, 1983). The effect of the male in hastening and synchronizing puberty has also been reported in ewe lambs (Dyrmundsson and Lees, 1972) and mice (Vandenbergh, 1973; Bronson and Desjardins, 1974).

In regard to postpuberal effects, age of the gilt at exposure has no effect on ovulation rate, percentage embryo survival or the number of embryos at 20 d gestation (Hughes and Cole, 1975; Kirkwood and Hughes, 1979; Paterson and Lindsay, 1980; Eastham et al., 1984).

Although the extent to which exposure to the boar stimulates onset of puberty in gilts is still being debated, one fact is very clear - the mature odoriferous male is the most potent stimulator (Kirkwood and Hughes, 1981). Olfaction is of utmost importance to the porcine female. Signoret (1980b), in his remarks on the boar effect, supposed that information from the environment must be processed at a fairly high level in the brain rather than acting directly on neural mechanisms controlling anterior pituitary secretion. He maintained that the female must be

in the appropriate physiological condition for her to respond, thus making the success of boar stimulation highly dependent on her body development and age. Boars were ineffective as puberty stimulators when tried with anosmic gilts (lacking sense of smell) (Kirkwood et al., 1981). A pheromone produced by the male is the likely mediator. Such pheromones are produced by preputial and submaxillary salivary glands. Some doubt is cast on the preputial scent since its use as 'Boarmate' did not alter age at puberty from that of a control group (Close et al., 1982). In another experiment, Perry et al. (1972) found that boars from which submaxillary salivary glands had been removed were unable to elicit a full standing response in estrous gilts. The important pheromone is likely one generated by the salivary gland (Kirkwood and Hughes, 1980; Kirkwood et al., 1981). Pheromones are present in low amounts in the male until approximately one year of age(Booth, 1975). Prepuberal males (castrate or entire) seem to have no effect on their female littermates when raised together (Kirkwood and Hughes, 1981; Nathan and Cole, 1981) and the gilts react as expected when exposed to a mature boar at 160 d (Paterson and Lindsay, 1980). However, Paterson and Lindsay (1981) have shown that the presence of a mature boar enhances the maintenance of cyclicity in gilts induced by exogenous hormones.

HORMONAL INDUCTION OF PUBERTY: It has been known since Casida's published report in 1935 that injections of

pituitary extracts could successfully induce estrus in prepuberal gilts. Various agents have been tested for their induction efficacy from the 1950s to the present day. These include estrogens such as estradiol benzoate (EB) or ethinyl estradiol (Hughes and Cole, 1978; Foxcroft et al., 1984), gonadotropin releasing hormone (GnRH) (Baker et al., 1973; Baker and Downey, 1975; Guthrie, 1977), and 400 i.u. (PMSG) with 200 i.u.(hCG) in a single injection (Schilling and Cerne, 1972; Baker and Rajamahendran, 1973; Cerne and Nikolić, 1976; Bielański, 1977; Foxcroft et al., 1984).

One of the most effective combinations has been PMSG followed by hCG several days later (Dziuk and Gehlbach, 1966; Shaw et al., 1970; Baker and Rajamahendran, 1973; Kather and Smidt, 1974). Under 56 d of age, the ovary is insensitive to gonadotropin stimulation (Guthrie, 1977). However, by the ninth week of life, the ovaries will respond to PMSG and ovulate by the twelfth week when hCG is also given (Kather and Smidt, 1974; Oxender et al., 1979). The responsiveness at this time is probably due to the functioning of appropriate receptors for the hormones. The majority of gilts over 100 d respond to this preparation(Dziuk and Gehlbach, 1966; Shaw et al., 1970). Unfortunately, not all gilts continue to cycle regularly but revert back to the prepuberal state(Dziuk and Gehlbach, 1966).

The use of PMSG and hCG in separate injections has often been put forward as a practical means of inducing early

puberty(Schilling and Cerne, 1972; Baker and Downey, 1975; Guthrie, 1977). In Wrathall's review (1971), he cautioned that PMSG is least dangerous in reasonable doses (up to 800 i.u.) with 500 i.u. hCG given 3-4 d later. Excessive doses (including some products in doses at recommended levels) cause superovulation.

There is a linear relationship between dose of PMSG and ovulation rate(Baker and Coggins, 1968; Phillippo, 1968). A mean ovulation rate of 10.4 eggs was shown to be comparable to a dose of 250-500 i.u. PMSG. Above 500 i.u., superovulation occurred. Nonetheless, percentage of eggs fertilized was not affected by the dosage used (Baker and Coggins, 1968).

Effects of PMSG/hCG are thought to mimic normal physiologic events in the gilt. External estrous signs are often present with most gilts ovulating and a high percentage of their eggs being fertilized at artificial insemination (A.I.) (Dziuk and Gehlbach, 1966). Blood E_2 levels rise 50-60 h after PMSG(Esbenshade et al., 1982) and gilts already reflect these increasing levels by reddening and swelling of the vulva within 48 h of injection (Dziuk and Gehlbach, 1966; Baker and Coggins, 1968). After hCG or LH surge, E_2 production decreases and remains low while P_4 levels begin to rise after ovulation(Esbenshade et al., 1982). Number of follicles and their pattern of maturation are similar to that of untreated gilts (Baker and Coggins, 1968; Ainsworth et al., 1980). Egg development follows

previously reported patterns seen in the sow and steroid production by follicles agrees with what has been reported in females of other species (Daguet, 1978; Ainsworth et al., 1980). A study by Esbenshade et al. (1982) concluded that blood serum patterns of E_2, LH and glucocorticoid were similar whether or not the gilt was subjected to PMSG, boar exposure or relocation and boar exposure. Gross changes in the reproductive tract are the same as those observed following spontaneous estrus (Shaw et al., 1970; Schnurrbusch at al., 1980). Gilts which were subjected to PMSG/hCG and subsequently reverted to the prepuberal state did not show any ill effects from the treatment. Such animals underwent puberty later and conceived (Dziuk and Gehlbach, 1966; Ellicott et al., 1973). McMenamin and King (1974) injected gilts with PMSG (1000 i.u.) and hCG (800 i.u.) 72 h later at age 100 d, and again at 121 and 142 d. At slaughter, no difference was found in the ratio of endometrium to total uterus between the test and control groups.

A major problem with hormonal induction is the failure to maintain pregnancy when bred on the induced heat under 160 d of age (Dziuk and Gehlbach, 1966; Guthrie, 1977) or 175 d (Rampacek et al., 1976; Callaghan and King, 1978) or, if not bred at induced estrus, their failure to continue cycling (Dziuk and Gehlbach, 1966). This failure has been attributed to insufficient uterine development, and/or insufficient steroidal or luteotrophic support (McMenamin

and King, 1974). It has been shown that CL's of immature gilts are more sensitive to luteolysis (Puglisi et al., 1979). Researchers had a higher success rate when heavier, older gilts, closer to the age and weight of natural puberty, were used (Schilling and Cerne, 1972; Guthrie, 1977; George and England, 1974; Callaghan and King, 1978).

PREGNANCY AND ESTRONE SULPHATE: After ovulation and fertilization, the ova take 24-48 h to traverse the oviduct and reach the uterus in the 4-cell stage (Pomeroy, 1955). On average the number of eggs tends to be equal in the two uterine horns resulting from migration of the eggs from one horn to another before 11 d post-breeding(Corner, 1921; Warwick, 1926; Burger, 1952; Dhindsa et al., 1967).

At 10.5-11 d, while still a 10 mm sphere, estrogen synthesis is initiated (Heap et al., 1975). The commencement of attachment of blastocyst(expanded egg) to uterine wall (implantation) can be seen by 13 d, is well advanced by 18 d and considered complete by 24 d (Crombie, 1970; Heap et al., 1975). The estrogen of embryonic origin is conjugated in the uterine wall and transported in the maternal blood in sulphated form as estrone sulphate (E_1SO_4) (Heap et al., 1975).

In 1974, Robertson and King found the metabolite E_1SO_4 in maternal blood and confirmed that it was of embryonic origin. Estrogenic compounds had previously been detected in urine of pregnant sows (Lunaas,1973). Velle (1960) reported the use of a single 100 ml urine sample collected

between 24-32 d post-breeding for pregnancy diagnosis. At that time the chemical processing was too complex and expensive for field application.

Blood E_1SO_4 levels are elevated in pregnant females between 16 and 30 d of gestation (Robertson and King, 1974) and peak between 27 and 29 d (Robertson et al., 1978; Horne et al., 1983). A classification system was set up based on serum levels (Hattersley et al., 1980; Saba and Hattersley, 1981; Cunningham et al., 1983): pregnant, >0.5 ng/ml; non-pregnant, <0.4 ng/ml; inconclusive, 0.4-0.5 ng/ml. Based on this designation, with samples taken 25-30 d after mating, accuracy was rated 98% for pregnant females, 100% for non-pregnant with less than 1% being inconclusive. These results were not affected by the collection day or breed of pig (Cunningham et al., 1983).

The level of E_1SO_4 is influenced by day of gestation and the numbers of live embryos. At 20, 22, 24, 26 and 30 d E_1SO_4 increased with increasing litter size (Stoner et al., 1981; Cunningham et al., 1983; Horne et al., 1983). When litter sizes were grouped 1-6 and 9-16, significant differences were found between 24 and 27 d. Small litters of 1-5 were easily distinguished from those of 8-15 embryos (Horne et al., 1983). However, under field conditions, E_1SO_4 levels are not accurate in predicting litter size for a particular sow since it is unlikely that a single sample will be taken at the time when E_1SO_4 is at its peak (Hattersley et al., 1980; Cunningham et al., 1983).

In the pig, a litter loss of up to 40% of eggs ovulated can be expected during the first trimester of pregnancy (Wrathall, 1971). Approximately 95% of eggs are fertilized (Squiers et al., 1952; Perry and Rowlands, 1962) and by 25 d of gestation, 30% of embryos will have been lost (Burger, 1952; Squiers et al., 1952; Perry, 1954). Most of this loss occurs during the period of implantation (Perry and Rowlands, 1962; Wrathall,1971). McFeely (1967) attributed one third of early embryonic mortality to chromosome abnormalities. Bishop (1964) considered that this was nature's way to remove unfit genotypes, resulting from spontaneous mutations, at a low biologic cost.

From 25-40 d, embryo numbers are further reduced by 5-7% (Gossett and Sorenson, 1959; Perry and Rowlands, 1962). It has generally been accepted that embryo crowding is not a factor in prenatal mortality up to 35 d of pregnancy (Dziuk, 1968; Fenton et al., 1970; Webel and Dziuk, 1974; Knight et al., 1977). Because embryonic growth is positively correlated with placental development, Knight et al. (1977) suggested that placental insufficiency was the primary cause of increased embryonic death and reduced embryonic growth. Bazer (1975) postulated the availability of an essential biochemical factor that limits litter size. Another factor which becomes operative in certain environments is temperature. Decreased conception rates, reduced survival rates and a reduction in numbers of viable embryos all occur when the dam is exposed to temperatures

above 32°C within 15 d of mating (Tompkins et al., 1967; Edwards et al., 1968; Omtvedt et al., 1971).

INTRODUCTION(EXPERIMENT_1)

Many attempts have been made in the past to induce puberty earlier with a variety of hormones. Valid comparison of the results between published studies is hampered by the large range of factors which affect age at puberty and were not standardized or reported.

The response of gilts to hormonal treatment with PMSG/hCG was followed in three age/weight groups. These groupings were selected on the hypothesis that the youngest animals would be too immature to become puberal. The oldest gilts, close to the age of spontaneous puberty, would be expected to exhibit continued cyclicity, be able to maintain pregnancy and have puberty hastened. The third group, being intermediate, was expected to give varying outcomes to the treatment. The occurrence of puberty was assessed by P_4 profiles and reproductive tract data. Maintenance of pregnancy was evaluated up to 34 d of gestation by monitoring plasma P_4 and E_1SO_4 levels and from post mortem details. In order to minimize any effects they might have, factors known to affect attainment of puberty were kept constant throughout the duration of the study.

MATERIALS_AND_METHODS(EXPERIMENT_1)

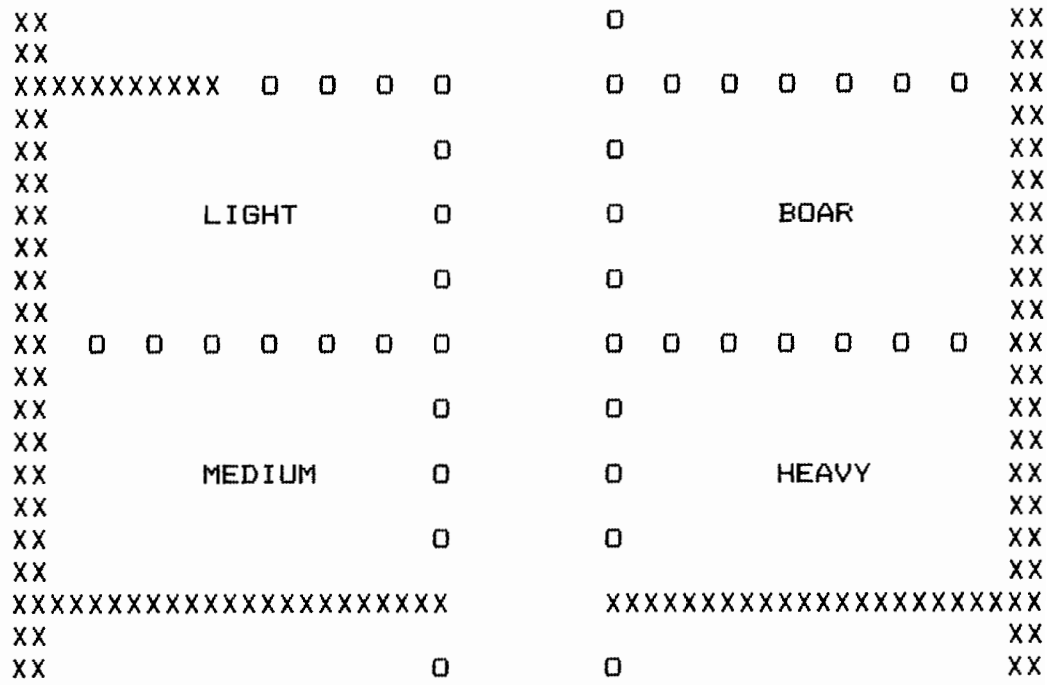
ANIMALS: Forty-eight prepuberal Landrace gilts from the Macdonald College herd were used. The gilts were selected at

random within three age/weight groups (16 per group): Light (L), Medium (M), Heavy (H) (Table 1). Animals were subjected to a pen change and mixed with strange gilts. They were penned at the Macdonald College Swine Unit in groups of four animals similar for age and weight. Pens (3.7 X 2.0 m) were concrete floored and bedded with wood shavings. The same mature boar was present in an adjacent pen throughout the study (Figure 1). All gilts were supplied with a commercially pelletted 16% crude protein ration and water ad libitum. Gilts were maintained under a regime of 16 h light and 8 h dark.

TABLE 1. AGE/WEIGHT GROUPINGS OF PREPUBERAL GILTS.

NO. OF GILTS	GROUP	WEIGHT (kg)	AGE (d)
16	Light(L)	45-55	up to 120
16	Medium(M)	60-70	130-150
16	Heavy(H)	75-85	160 and over

FIGURE 1. PEN ARRANGEMENT FOR EXPERIMENT 1.



EXPERIMENTAL PROCEDURE: The experiment consisted of four replicates of 12 gilts each with trials beginning on October 19, 1984; January 11, March 22 and May 31, 1985. Every replicate involved four gilts from each of the three treatment groups. With one exception (Group H, Replicate 3), the use of littermates was avoided. The animals were treated with an intramuscular injection of 750 i.u. PMSG (Equinex®; Ayerst Laboratories, Montreal, P.Q.) on the first day of the experiment followed 72 h later by 500 i.u. hCG(APL®;Ayerst). This treatment has been shown to induce ovulation over the age range of animals used (Dziuk and Gehlbach, 1966; Shaw et al., 1970).

A 10 ml blood sample was obtained by jugular venipuncture into a syringe containing 0.5 ml of heparin solution (200 i.u./ml saline)(Hepalean®; Organon Canada Ltd., Toronto, Ont.) beginning between 10.00 and 11.00 h every Monday, Wednesday and Friday throughout the duration of the experiment(Table 2). The samples were held on ice and centrifuged as soon as possible at 1000 X g for 10 min. The supernatant was split into two fractions and stored separately at -20°C until assayed.

All gilts were observed for signs of approaching estrus and checked for estrus with the same boar. The following scale was used to assess the proximity of estrus and estrus:

0- no outward physical or behavioural signs

1- some redness and swelling of the vulva

2- vulva red and swollen but failure to stand

3- vulva red and swollen, standing estrus or other signs such as riding or vaginal discharge.

Estrus checking was done in the morning and afternoon. Gilts which rated 2 or 3 on the estrus scale were bred by A.I. at the time of their second estrus approximately 21 d after the induced estrus) with at least 75 ml of fresh, mixed, undiluted semen from two boars of proven fertility. Each gilt was inseminated at least twice.

Animals were weighed weekly. After 63 d on the experiment(approximately 34 d post-A.I.), all gilts were slaughtered at a commercial abattoir and their reproductive tracts were recovered and examined within 6 h. The uterus,

TABLE 2. SCHEDULE FOR EXPERIMENT 1*.

Sun.	Mon.	Tues.	Wed.	Thurs.	Fri.	Sat.
					*PMSG B1	
				WO	d0	d1
	*hCG B2	DO W1	D1 B3		B4	
d2	d3	d4	d5	d6	d7	d8
	B5	W2	B6		B7	
d9	d10	d11	d12	d13	d14	d15
						D
	B8	W3	B9		B10	
d16	d17	d18	d19	d20	d21	d22
D	D	D	D	D	D	
	B11	W4	B12		B13	
d23	d24	d25	d26	d27	d28	d29
	B14	W5	B15		B16	
d30	d31	d32	d33	d34	d35	d36
						D
	B17	W6	B18		B19	
d37	d38	d39	d40	d41	d42	d43
D	D	D	D	D	D	
	B20	W7	B21		B22	
d44	d45	d46	d47	d48	d49	d50
	B23	W8	B24		B25	
d51	d52	d53	d54	d55	d56	d57
	B26	W9			S	
d58	d59	d60	d61	d62	d63	

* Where: W -weigh in
* -injection
B -blood sample taken
D -estrus checking and/or A.I.
d -day of the experiment
S -slaughter

oviducts and ovaries were separated from the connective tissue. The tract was severed through the middle of the cervix and the total tract weight was recorded. Ovaries were trimmed at the hilus and weighed separately. The number of

CL's on each ovary were counted . Uterine horns were measured from the body of the uterus to the tubo-uterine junction taking care not to stretch the organ. The uterus was sliced open longitudinally, embryos and/or reabsorbing tissue located and their condition noted. Each embryo was removed from its placental membranes and the wet weight taken. Finally ,the weight of the tract minus uterine contents and ovaries was recorded . Any apparent abnormalities were also noted.

HORMONE ASSAYS: Plasma progesterone concentrations were measured by the direct radioimmunoassay (RIA) procedure described by Schanbacher (1979) with the following modifications. Twenty µl aliquots of plasma were assayed in duplicate.Progesterone anti-serum, raised in rabbits against progesterone-11∝-hemisuccinate-HSA (lot 147-A; Immunotech Diagnostic, Montreal, P.Q.), was used in an initial dilution of 1:10,000 at a volume of 100 µl. Anti-rabbit gamma globulin was obtained from ovariectomized ewes. After addition of cold phosphate buffer, vials were centrifuged for 40 min at 1250 X g at 15°C. Subsequent to aspiration of the supernatant, 3 ml of scintillation fluid (Universol®; ICN Radiochemicals, Irvine, CA.) were added along with 200 µl of double distilled water. Following capping, shaking and overnight incubation, all samples were counted for 5 min in a liquid scintillation counter (Model LS-235; Beckman Instruments Inc., Toronto, Ont.) using the internal standard at 2% accuracy. Results are reported in ng 4-pregnen-3,20-

dione/ml plasma (Batch 1974; Steraloids Inc., Wilton, N.H.). Standard curves covered the range of 10-1000 pg per tube. Serum from charcoal stripped mature boar and barrow pools was included in all assays for quality control. Intra-assay coefficients of variation were between 2.1% and 8.1% while the inter-assay coefficient of variation for eight assays was 16.8%. The average lower limit of detection was 0.66 ng/ml.

The assay for E_1SO_4 as done at the Animal Research Centre (A.R.C.), Ottawa under the supervision of Dr. L. Ainsworth. Estrone sulphate determination followed the method of Tsang (1964) and the RIA procedure of Dorrington and Armstrong (1975). The estrone anti-serum used was raised in ewes at A.R.C. and has characteristics which were detailed by Robertson et al. (1985). This anti-serum was diluted to 1:100,000 before addition to vials. Unknowns were assayed in duplicate and their values reported as ng/ml plasma. The minimum amount of E_1SO_4 detectable was 6.8 pg/tube. Standard curves covered the range of 5-640 pg/tube (estrone-3-sulphate ; Sigma Chemical Co., St. Louis , MO.). Estradiol-17β cross-reaction with estrone antibody was 1.27%. The mean recovery of E_1SO_4 following extraction, solvolysis and column chromatography was 61.8$\pm$2.89%. The calculated mean of E_1SO_4 after RIA of estrone and correction for procedural losses was 0.977$\pm$.035 ng. Intra-assay coefficients of variation were 1.6% for the high standard and 4.5% for the low standard. Inter-assay coefficients of

variation were 9.6% and 5.3% for high and low standards, respectively.

STATISTICAL ANALYSIS: The nonparametric Kruskal-Wallis test (Siegel,1976) was used for one way analysis of variance of variables measured. Proportional data were analyzed by the Chi-square test. Correlation coefficients were used to determine relationships between variables of interest. Duncan's New Multiple Range test(Steel and Torrie, 1960) was used to test differences among means.

RESULTS(EXPERIMENT 1)

AGE AND WEIGHT AT PUBERTY: The occurrence of puberty was established after the fact by studying blood P_4 levels(Appendix Table 3). In gilts becoming puberal directly after PMSG/hCG treatment, age at puberty was taken to be the day of ovulation(40 h after hCG injection)(Baker and Coggins, 1968). Age at puberty for gilts becoming puberal spontaneously, was assigned based on the first P_4 reading over 3.0 ng/ml followed by increasing levels for a further three collections. Only data on 46 gilts could be used in the final analysis. One gilt was eliminated due to the absence of both ovaries(OVX previously) and the second because she failed to respond to the PMSG/hCG injections.

Reddening and swelling of the vulva(the most obvious symptom of estrus)was present in all responding gilts(46/46) within 72 h of PMSG treatment and in 45/46 animals at the expected time of second estrus, although only 21/46 females

FIGURE 2. GILTS WITH A RED AND SWOLLEN VULVA AT SECOND ESTRUS.

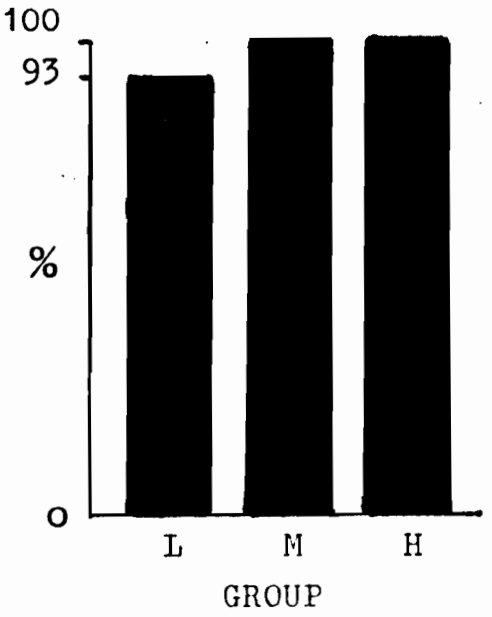
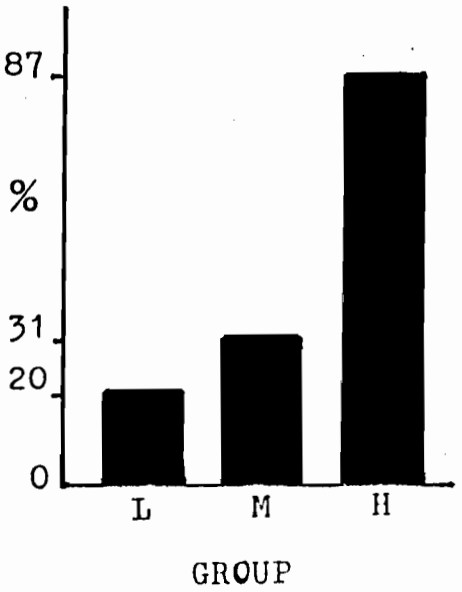


FIGURE 3. GILTS OVULATING AT SECOND ESTRUS.

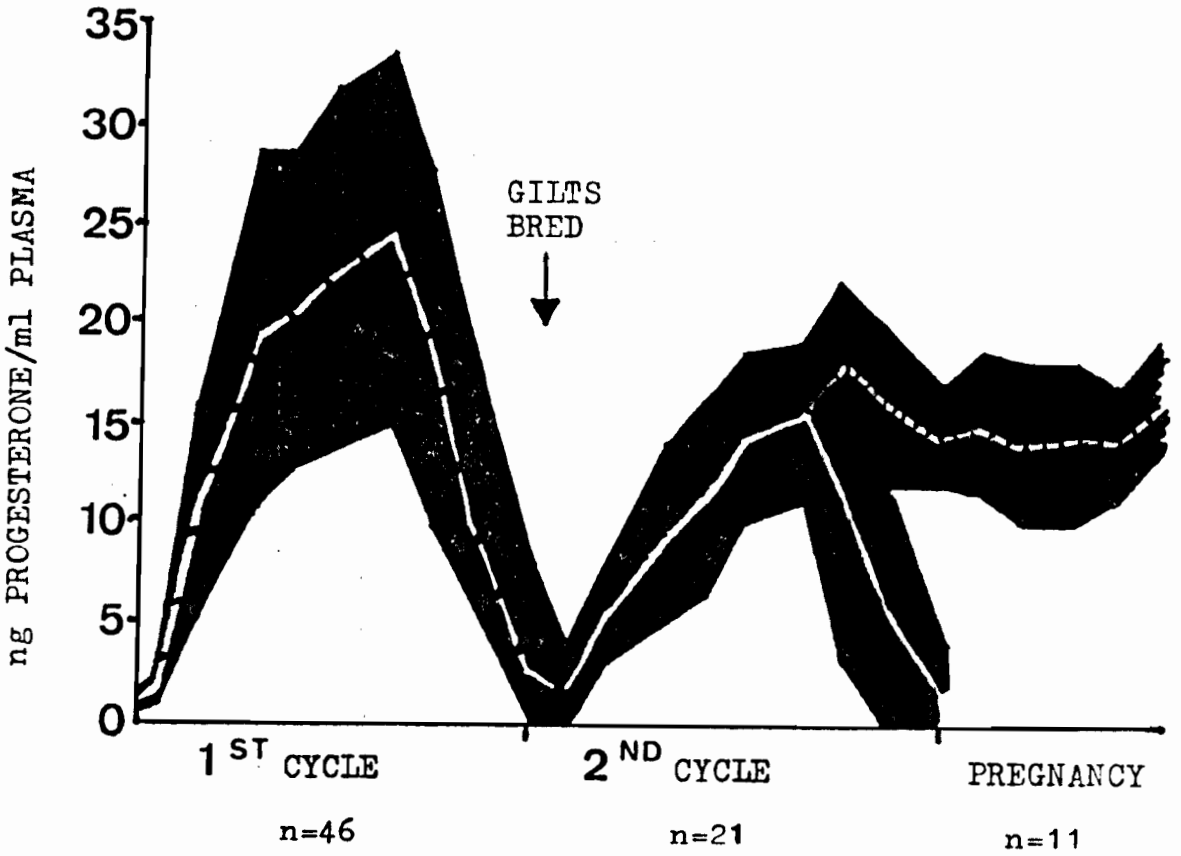


actually ovulated at second estrus(Figures 2 and 3). Standing estrus and/or riding were only observed in 11 instances during the first two estrous cycles(evenly distributed among the three Groups) Three of the very light and young gilts attained puberty as a direct result of induction with PMSG/hCG. Those animals had the following ages and weights: 109 d, 53.6 kg ; 112 d , 49.1 kg and 114 d ,50.9 kg (Appendix Table 5). In total, 21 gilts became cyclic subsequent to hormonal treatment(L,3/15;M,5/16; H,13/15). Sixty-two percent were puberal before 170 d and 95% by 200 d.

The difference in weight at puberty among the Groups was not significant. Conversely, age at puberty was different between L and H Groups ($0.02<p<0.05$) and between M and H Groups ($p<0.001$)(Appendix Table 14). There was a marked increase in puberty maintenance in H Group over L or M Groups ($p<0.001$) (Appendix Table 15).

By the end of the experiment, an additional five gilts had achieved puberty(L,4/15; M,8/16; H,14/15)(Appendix Table 5). Collectively, of the 26 gilts, 89% (23/26) experienced puberty prior to 200 d, 58% before 170 d. The actual age ranges of gilts at the end of the experiment were; L,148-183 d; M,191-214 d; H,224-273 d. Therefore, L gilts were still below the average age when they would express puberty. Of M Group gilts, half had their puberal estrus before slaughter, and the oldest prepuberal gilt was 200 d of age. In the H Group, all but three gilts reached puberty before

FIGURE 4. PROGESTERONE PROFILES($\bar{X}\pm$SEM) ILLUSTRATING OVARIAN RESPONSE TO PMSG/hCG TREATMENT AT FIRST CYCLE(DASHED LINE), OVARIAN ACTIVITY AT SECOND CYCLE(SOLID LINE), AND OVARIAN RESPONSE TO PREGNANCY(DOTTED LINE).*



* Values were arranged such that initial rises in titre occurred on the same day.

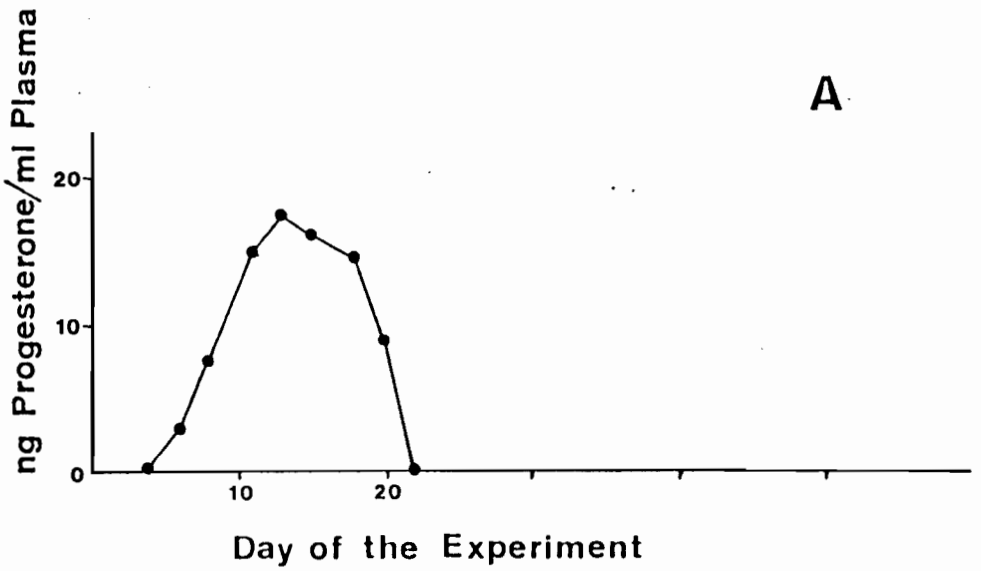
200 d ($\bar{x}\pm$SEM, 173.1$\pm$6.8 d). Puberty was attained at 214 d in one gilt but she was already 209 d of age when the experiment began. She responded directly to the induction.

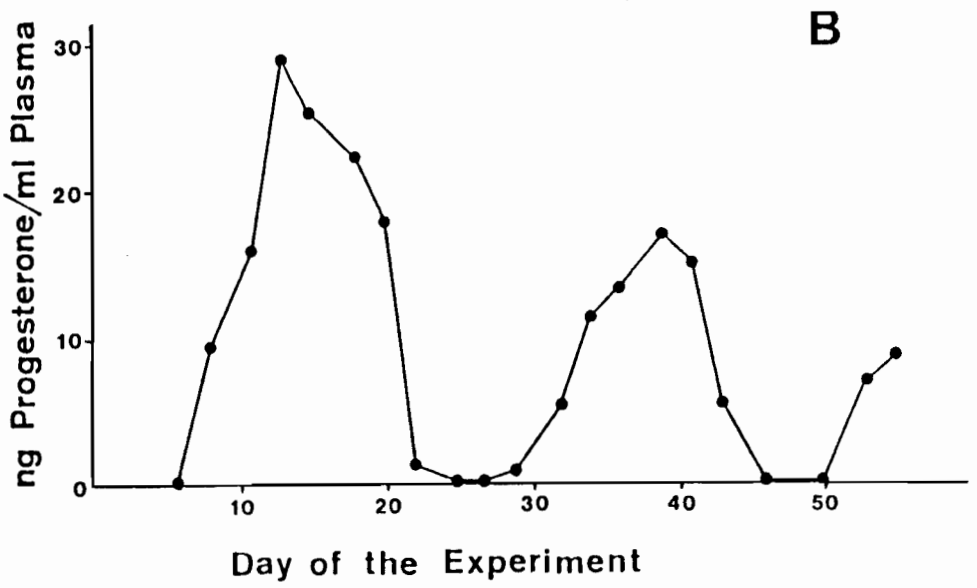
PROGESTERONE PROFILES: Progesterone levels were used as a measure of ovarian function and to assess the treatment response to PMSG/hCG injections. In total 46 gilts reacted to the gonadotropins by ovulating, forming CL's and increasing their P_4 production. Neither gilt age nor weight (Group) had any effect on the P_4 output.

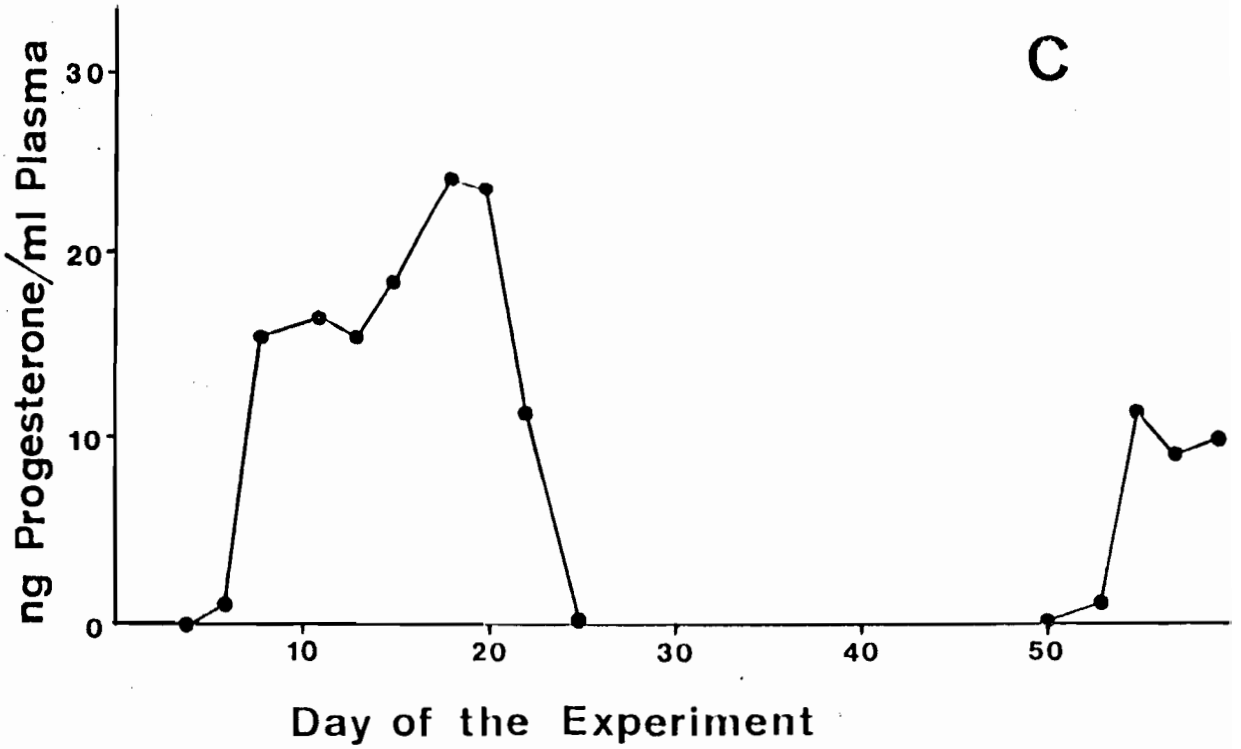
Progesterone increased steadily from undetectable or near undetectable levels (Day -4 to Day 1) to peak between Days 6-17. Peak levels ranged from 7.5-46.0 ng/ml. Thereafter levels dropped quickly (within 3 d) to 3.0 ng/ml or less by Days 17-20(Figure 4). The next cycle, when experienced, showed increased P_4 production beginning Days 6-8. Peak levels in the second cycle (8.5-26.0 ng/ml) were rarely as high as at the induced cycle (Figures 4 and 5B). Most gilts appeared to follow a P_4 pattern consistent with an average 21 d estrous cycle. Exact cycle length could not be determined as sampling was not done daily. Some individuals seemed to have a longer than average second cycle; their Day 20 value(first cycle) was under 3.0 ng/ml, yet the Day 20 titre(second cycle) was above 10.0 ng/ml.

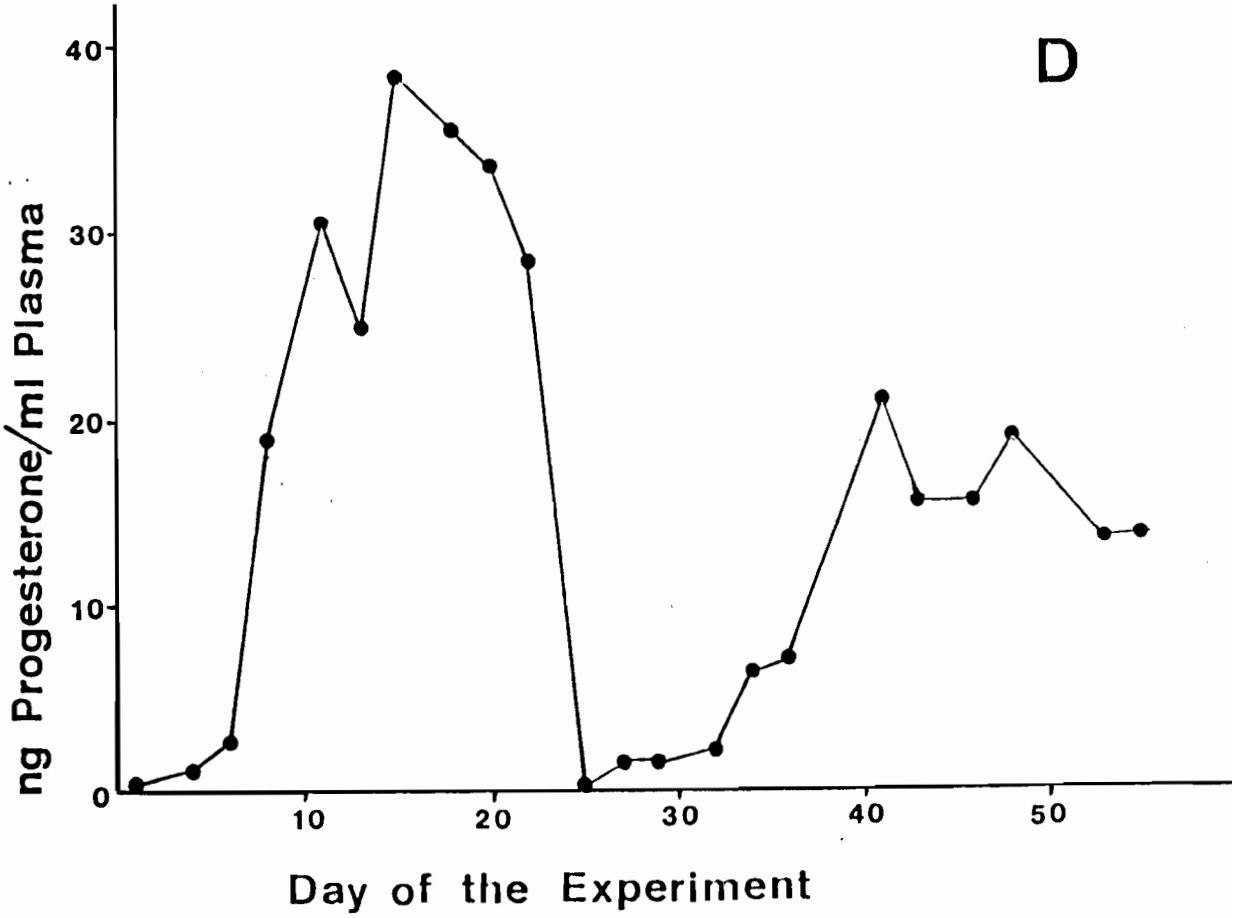
The area under the curve (AUC) of P_4 release for the first estrus was greater ($p<0.01$) than AUC for second estrus (Appendix Table 14). No difference was found in P_4 profile at induced estrus between gilts that continued to

FIGURE 5. PROGESTERONE PROFILES OF PREPUBERAL GILTS GIVEN PMSG ON THE FIRST d (0) OF THE EXPERIMENT, A/ A GILT WHICH REVERTED TO THE PREPUBERAL STATE AFTER TREATMENT(2/L/4), B/ A GILT WHICH BECAME CYCLIC SUBSEQUENT TO TREATMENT(2/H/2), C/ A GILT WHICH REVERTED TO THE PREPUBERAL STATE AFTER PMSG/hCG TREATMENT AND LATER EXPERIENCED SPONTANEOUS PUBERTY(1/L/3), D/ A GILT WHICH BECAME PREGNANT WHEN BRED AT SECOND ESTRUS (4/M/2).






C
ng Progesterone/ml Plasma
0
10
20
30
10
20
30
40
50
Day of the Experiment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ng Progesterone/ml Plasma
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Day of the Experiment

cycle and those that reverted to the prepuberal state. In spite of the fact that there was no difference in P_4 AUC between L, M and H Groups, there was a large individual variation in P_4 response within the same Group whose development might be thought to be more uniform.

In gilts which continued to cycle, the first estrus P_4 response was highest in replicate 3 ($0.02<p<0.01$ between replicates 3 and 4) (Appendix Table 14). Replicate 3 gilts (regardless of Group) had greater P_4 output at the induced cycle than all other replicates($p<0.05$). The difference in CL numbers was greatest between the second and and third replicate (winter and spring) ($0.01<p<0.001$) (Appendix Table 14).

During the second cycle, P_4 in pregnant gilts peaked at Days 13-17 (14.0-24.5 ng/ml) and then dropped somewhat, no gilt being below 7.0 ng/ml when the final samplings were taken at 34 d of gestation (Appendix Table 3). There was a definite relationship between P_4 production and number of CL's present. This was apparent whether maximum P_4 levels or AUC of P_4 were measured and compared to the CL numbers. The correlations were $r=0.76$, $p=0.0071$ and $r=0.80$, $p=0.003$ when using AUC of P_4 and P_4 maximum values, respectively. (P_4 AUC and P_4 maxima were correlated;$r=0.77$, $p=0.0055$) (Appendix Table 16).

Gilts which reverted to the prepuberal state showed two types of profiles during the remainder of the experiment. The majority had P_4 levels continuously just above or below

the limit of detection. A smaller group (five) experienced true puberal estrus out of sequence from the gilts who had responded with puberty as a direct result of PMSG/hCG stimulation. In total, four different P_4 profiles were seen in this experiment (Figures 5:A,B,C,D).

THE REPRODUCTIVE TRACT: Physical differences in prepuberal, puberal and pregnant tracts were very obvious on gross examination. Prepuberal ovaries were smooth surfaced to slightly undulating with many having small (<3 mm) follicles within the ovarian cortex. The uterine horns were thin, translucent and pale pink in colour.

Cyclic animals showed a progression of stages ; developing antral follicles of various sizes, corpora hemorrhagica and CL's. The uterus was bright pink with the increased vascularization and the criss-cross of numerous capillaries was easily seen. The uterine horn walls were noticeably thicker . The entire tract appeared massive when compared to the prepuberal tract. In pregnant uteri, the positions of the embryos were readily located without opening the uterus. The weight increase was considerable over that of the non-pregnant tract principally due to the large volume of fluid. Only one major abnormality was seen. Gilt 3/M/1 had the condition uterus unicornis(missing uterine horn) as well as the Fallopian tube on the same side being absent. Her uterine weight was not used in calculations.

Puberty caused increases in ovarian weight(76%), uterine

horn length(84%), and uterine weight(215%),($p<0.001$)(Table 3). Pregnancy resulted in a further increase only in uterine weight(218%) up to 34 d gestation over gilts which were open(non-pregnant cyclic)($p<0.001$)(Table 3). Differences in horn length between pregnant and non-pregnant tracts approached the 5% significance level. For prepuberal gilts, horn length and uterine weight were positively correlated($r=0.60$, $p=0.0049$). Likewise, the same relationship was found in non-pregnant cyclic gilts($r=0.52$, $p=0.0469$) (Appendix Table 16).

The number of CL's in cycles 2 and 3 were not different(12.4±1.9 vs. 12.5±1.1) nor for pregnant as opposed to non-pregnant females at second estrus (12.2±1.9 vs. 12.1±2.1), nor among the three Groups.

TABLE 3. REPRODUCTIVE TRACT MEASUREMENTS($\bar{X}$±SEM) TAKEN AT SLAUGHTER 34 D POST-BREEDING.

	n	Ovarian Weight(g)	Uterine Horn Length(cm)	Uterine Weight(g)
PREPUBERAL	20	7.2±1.7[a]	51±9[a]	168±84[a,t]
CYCLIC(NOT PREGNANT)	15	12.7±3.6[b]	94±18[b]	528±159[b]
PREGNANT	11	13.8±1.6[b]	106±15[b]	1682±463[c]

Means within a column bearing a different superscript are significantly different($p<0.001$).
t n=19.

PREGNANCY: Based on estrous signs(red and swollen vulva), it appeared that 45/46 gilts experienced second estrus approximately 21 d after the induced estrus. Subsequent plasma P_4 levels indicated that only 21/46 were actually cycling and could potentially conceive(Figure 3).

At slaughter, 11/21 gilts(which were cyclic subsequent to PMSG/hCG treatment) were pregnant (L,2/3; M,3/5; H,6/13). There was no statistical difference in the number of gilts pregnant between Groups. Live litter size ranged from 3-10 with $\bar{x}\pm$SEM of 6.5$\pm$2.8. The $\bar{x}\pm$SEM of H Group, and M, L Groups combined were 7.7$\pm$2.7 and 5.2$\pm$2.5, respectively.

The assessment of living versus dead embryos was made according to the system published earlier by King and Young(1957). Total litter loss was calculated by comparing the number of live embryos to the number of CL's. The resulting litter loss was 39.5% up to 34 d of pregnancy, 16% of which could be accounted for by reabsorbing or dead embryos. There was no litter loss in one gilt (CL number equal to the number of embryos). Five gilts had dead or degenerating embryos in their uteri. One gilt had a horn containing only a single dead embryo. Dead embryos varied from amorphous masses or empty embryonic sacs to specimens which had died recently. Location varied within the horns and neither horn was more likely to contain embryonic remains. Healthy live embryos were found beside dead ones.

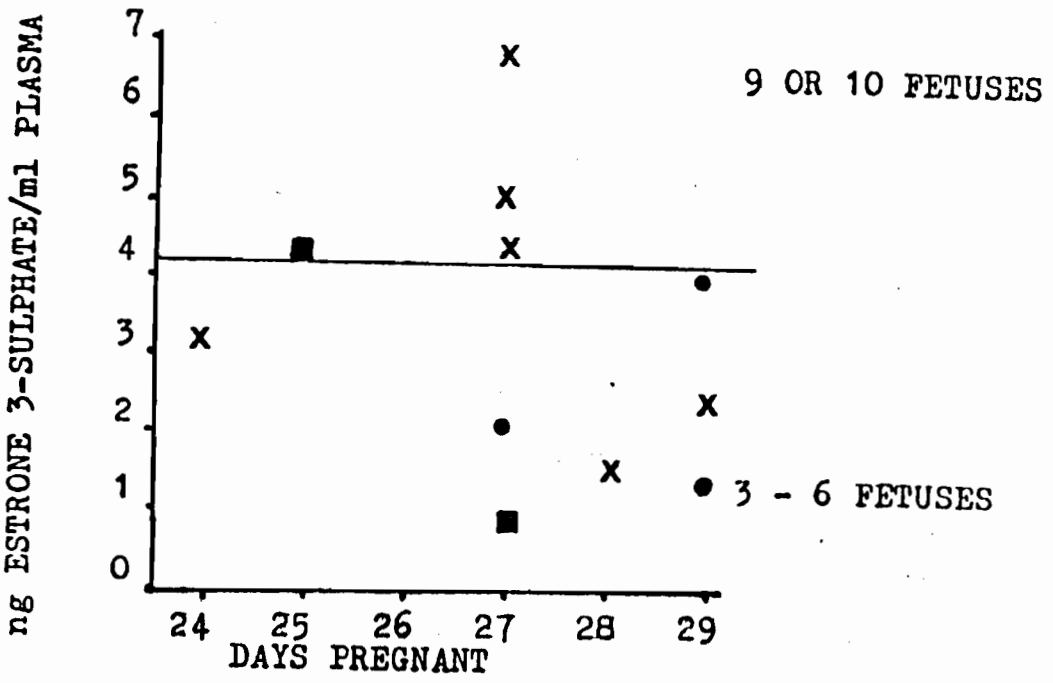
Numbers of live embryos tended to be equal in the two horns:four gilts had the same number of live embryos, three

differed by one embryo and three by two embryos. This was despite the fact that the numbers of ovulations were unequal in all pairs of ovaries.

At 34 d post-insemination, the following characteristics were positively correlated: uterine horn length and the number of live embryos(r=0.61, p=0.0447); embryonic live weight and live embryo numbers (r=0.62, p=.0423); the number of live embryos and the number of CL's(r=0.67, p=0.0229)(Appendix Table 16).

ESTRONE SULPHATE: Peripheral plasma concentrations of E_1SO_4 were 100% accurate in confirming pregnancy based on

FIGURE 6. MAXIMUM E_1SO_4 VALUES OF PREGNANT GILTS IN RELATION TO LITTER SIZE(GROUP L, ■ ; GROUP M, ● ; GROUP H, X).



samples taken between 20-29 d after breeding when the reading was > 0.5 ng/ml.

The number of live embryos and E_1SO_4 were very strongly correlated at 22-27 d($r>0.72$, $p<0.01$)(Appendix Table 16). Higher maximum E_1SO_4 values were recorded at 23-29 d of gestation in large litters(9-10 embryos) versus small litters(3-6 embryos)($0.01<p<0.001$)(Appendix Table 14, Figure 6). Means for maximum E_1SO_4 determinations were different for H Group than M, or L Groups($p<0.05$).

DISCUSSION(EXPERIMENT 1)

In the general domestic pig population, 50% of gilts will attain puberty by 200 d of age(Brooks and Cole, 1973; Hughes and Varley, 1980). Herdsmen at this Swine Unit(Hatcher and Wilson, personal communication) observed that puberal estrus is not commonly seen prior to 90 kg body weight(approximately 200 d of age). From the results of this experiment PMSG/hCG treatment appeared to hasten puberty(87% of H Group gilts became puberal subsequent to treatment at 173.1 ± 6.8 d of age. The weight at puberty between Groups was not statistically different but age at puberty was especially significant between M and H Groups indicating that age is probably the more important factor. Similarly, Robertson et al.(1951) and Hughes and Cole(1976) found more variability at constant live weight than age when gilts were exposed to external stimuli.

At induced estrus, few gilts showed any signs of riding

other females, being ridden or 'standing' to a back pressure test(total of 11 instances). The same tendency was repeated at the time of the second estrus. Winfield(1980) noted that 26% of gilts showed some deficiency of estrous behaviour at first cycle. Also, there are individual differences in frequency and intensity of sexual behaviour patterns(Signoret,1980a). 'Standing' for the boar is much more common in multiparous sows than gilts(Signoret, 1980c). Another factor unique to the present experiment was that the gilts expected to be caught and bled when the writer entered their pen. This initiated avoidance behaviour and may have lessened the likelihood of 'showing' for the boar particularly in shy or fearful gilts. The classic estrous symptoms of red and swollen vulva were apparent at first estrus and at the time of expected second estrus.It appeared that induction was successful as evidenced by outward signs. However, in this experiment, these were not reliable indicators of estrus except for the H Group. The vulval reaction occurred whereas ovulation did not always follow.

A number of factors could bring about vulval reactions. Andersson and Einarsson(1980) noted a synchronization of maturity within pens of gilts. It was felt that pheromones, which the female is considered to secrete during proestrus and estrus, could affect the sexual function of others in the same pen. Paterson(1982) and Esbenshade et al.(1982) mentioned that vulval development was often observed in young gilts when mixed and relocated. Secondly, the presence

of the boar has been shown to enhance gilt cyclicity(Paterson and Lindsay, 1981; Huehn et al., 1978). Thirdly, Dyck et al.(1980) suggested that the E_2 rise associated with this false estrus was caused by an abnormal pattern of follicular development. Ovulatory control is more complex and regulated by different factors. Reasons for lack of ovulation include: maturational changes influencing circulating E_2 levels so that the threshold is not reached, E_2 levels may not be present for a sufficient duration or the gilt may not be at the correct physiological age for the response to occur. Further, Esbenshade et al.(1982) put forth the notion that the ability to initiate follicular growth and the ability to respond to the higher E_2 levels with ovulation, do not develop simultaneously.

Some very young and light gilts became puberal after PMSG/hCG treatment but it is believed by the author that this is the exception rather than the rule. These individuals were physiologically mature sooner than average and could respond to the stimuli when provided. The majority of the gilts did not reach this maturational stage until near the age and weight ranges of the H Group ($\geq$160 d age, $\geq$75 kg body weight). There was a sizeable increase in the number of responders at that point which suggests a critical change in maturity just previous to this. The age 150-160 d(between M and H Groups) may be a 'cross-over' period in the maturation process. This hypothesis is further supported by two other research groups. Camous et al.(1985)

recognized a series of four phases in sexual maturation based on estrone levels. Phase four, the highest level of urinary estrone excretion, began around age 150 d and signalled the beginning of a waiting period, the duration of which is dependent on the age at puberty. Schnurrbusch and Erices(1979) found that reproductive tract development ceased after 160 d. They suggested that this cessation of growth could be prevented if gilts were stimulated at 160 d either by exogenous hormones, boar exposure, relocation or mixing with strange gilts.

Tract dimensions are dependent on the sexual age of the animal, not its chronological age or weight. This is consistent with the results of other experiments(Teige, 1957; Dziuk and Gehlbach, 1966; Shaw et al., 1970; Dyck, 1972; Guthrie, 1977). A prepuberal gilt could be easily distinguished by her tract, or measurements thereof, from a puberal gilt regardless of whether she was from the L, M, or H Group.

That puberty leads to great increases in reproductive tract measurements agrees with previous reports in the literature(Dziuk and Gehlbach,1966; Shaw et al., 1970; Dyck, 1972; Huehn et al., 1978). The probable cause seems to be endogenous hormonal stimulation. Various gradations of hormonal influence are apparent in this study.

Prepuberal gilt ovaries continually produce low levels of E_2(Dyck, 1972) which influences growth of the uterus(Hadek and Getty, 1959). Gilts showed a positive

correlation between uterine weight and uterine horn length as noted previously by Shaw et al.(1970). In spite of the fact that the gilts were PMSG/hCG treated, the tracts in animals failing to continue cycling reverted to the prepuberal state. As in other investigations(Shaw et al., 1970; McMenamin and King, 1974), the ability of the uterus and ovaries to respond to endogenous signals later is not hampered by the use of PMSG/hCG. Some gilts became puberal spontaneously after reverting to the prepuberal state for a short time.

The puberal gilt represents the next stage in the progression. Now both E_2 and P_4 are actively and cyclicly secreted. These individuals exhibit a positive correlation of uterine horn length with uterine weight as also reported by Shaw et al.(1970). It appears to be the combination or synergism of the two hormones(E_2 and P_4) which results in this uterine growth(length and weight). Tract measurements recorded for these puberal gilts are similar to those having spontaneous puberty(Shaw et al., 1970; Hughes and Cole, 1978).

The pregnant females represent an extension of this hormonal influence. Stretching of the tissues, thickening of uterine walls, increasing amounts of fluid and growing embryos account for the additional uterine weight and horn length in this experiment. This is borne out by the correlation between live embryo numbers and uterine horn length. The relationship between uterine horn length and

uterine weight approaches significant levels at d 34 post-breeding, a finding also reported by Perry and Rowlands(1962).

Two animals(4/H/2, 4/M/3), aged 239 and 198 d, respectively, at slaughter, were more than one SEM above the mean of the prepuberal gilts for ovarian weight, uterine weight, and uterine horn length. Thus, they were considered by the writer to be in the late prepuberal stage since marked increases in all these variables are typical of puberal gilts.

One gilt(3/M/1) had a genital abnormality, uterus unicornis(missing uterine horn) as well as the Fallopian tube on the same side being absent. Previous literature reveals that this condition has a prevalence up to 11.4% but that fertility is not affected though half the normal litter size is expected(Warnick et al., 1949; Wiggins et al, 1950a; Nalbandov, 1952; Teige, 1957; Einarsson and Gustafsson, 1970; Bashforth et al., 1972). Thus, this individual was expected to be as representative as the rest of her Group regarding reaction to the experimental treatment.

Overall patterns of P_4 release were similar to statistics revealed by other authors for cyclic gilts(Stabenfeldt et al., 1969; Henricks et al., 1972; Parvizi et al., 1976; Shille et al., 1979; Van de Wiel et al., 1981). Pregnant gilts followed established patterns including the 30-70% drop in circulating P_4 after 20 d of gestation(Tillson and Erb, 1967; Guthrie et al., 1972;

Shearer et al., 1972; Robertson and King, 1974). Hughes and Varley(1980) remarked that P_4 could be highly variable in pattern and level (7.5-56.1 ng/ml at its peak Days 8-16) without affecting normal cyclicity.

The basic P_4 profile remained essentially the same from cycle to cycle as has been reported by Andersson and Einarsson(1980) and Karlbom et al.(1981/1982). Normally, the number of ova liberated increase from the first to the fifth cycle(Robertson et al., 1951; Warnick et al., 1951; Burger, 1952; George and England,1974; Andersson and Einarsson, 1980). There is a strong positive correlation between P_4 output and the number of CL's present(Brinkley and Young, 1970; Dziuk et al., 1972; Wettemann et al., 1974; Webel et al., 1975; Paterson and Martin, 1981). Therefore, it would be expected that the maximum amplitude of the P_4 curve would rise for each successive cycle plotted. Such was not the case. At the second cycle, AUC of P_4 did not surpass that of the induced cycle and individually gilts rarely exceeded maximum P_4 values reached during the induced cycle. This strongly suggests that the PMSG preparation used stimulated the gilts to the point of at least mild superovulation. Ovulation rates at heats 2 and 3 were not statistically different. Wettemann et al.(1974) took blood samples at the 27th d of pregnancy and killed the gilts the following day. Progesterone and CL numbers were found to be positively correlated in that study just as they were in the present research.

An effect of season on P_4 levels was apparent during the induced estrus in gilts which became cyclic, being significantly higher in spring. Replicates were run such that they closely followed the seasons: replicate 1(October 19- December 21), fall; replicate 2(January 11- March 15), winter; replicate 3(March 22- May 24), spring; replicate 4(May 31- August 2), summer. Feral pigs are seasonal breeders and as such the domestic pig might be expected to display characteristics reflecting that of its ancestors(Signoret,1980c). It is difficult to find a reason for this difference in P_4 profiles since all the gilts were in indoor confinement.The gilts had been raised from weaners in the same barn. This barn has patterned glass windows at regular intervals down both sides. Hence, one could postulate that the natural light augmented that from the artificial sources indoors and was perceived by the pig's sensory organs with the resultant physiological response. Foxcroft(1980) also reported a seasonal effect but after injection of constant doses of EB. The lowest number of gilts experiencing puberty was in the summer. He proposed that photoperiod had a direct effect on the activity of the hypothalamic-anterior pituitary-ovarian axis.

Treatment with PMSG/hCG produced three kinds of gilts based on P_4 profiles and estrous symptoms(red and swollen vulva) at the time of second estrus 1/ estrous symptoms and ovulation followed by cyclicity or pregnancy(21/46), 2/ estrus symptoms and no ovulation(24/46), 3/ no estrus

symptoms and no ovulation(1/46). Paterson and Lindsay(1981), Paterson and Martin(1981), and Esbenshade et al.(1982) also obtained comparable results using boar and/or hormonal stimulation. Paterson and Martin(1981) supposed that there was not enough E_2 present for an LH surge, not enough LH to provoke ovulation or that the ovary was not sensitive to LH. The appearance of the three types of gilts is probably due to differences in maturity of the hypothalamic-anterior pituitary-ovarian axis. Puberty was hastened in 'mature' gilts, those sufficiently developed that treatment with exogenous hormones triggered continued cyclicity, but failed to do so in 'immature' gilts which reverted back to the prepuberal state. Unfortunately, there seems to be no way to predict the likelihood of puberty, as a result of PMSG/hCG treatment from P_4 profiles.

Full reproductive potential in the pig is rarely achieved. Losses occur at conception, egg, blastocyst and embryo stages(Wrathall, 1971). The potential number of offspring is considered to equal the number of CL's(Hammond, 1921; Corner, 1923; King and Young, 1957; Perry and Rowlands, 1962; Longenecker et al., 1968).

Conception rate among these experimental gilts was poor(52%) and far below the average of 80% or more. The writer suspects that the stress of frequent blood sampling lowered the conception rate as blood collections were three times weekly throughout the duration of the experiment and gilts were often sampled 1 h after insemination. Temperature

was not likely a factor as the mean daily maximum inside the barn was 26.5°C up to 21 d post-mating during the summer, not high enough to affect conception rate according to previous experimental results(Tompkins et al., 1967; Edwards et al., 1968; Omtvedt et al., 1971).

The total litter loss of 39.5% falls within the range reported in the literature(Wrathall, 1971). Reabsorbing or dead embryos made up 16% of this loss. It is likely that this embryonic loss occurred after d 14 since Dhindsa and Dziuk(1968) demonstrated that after d 14 of gestation the number of live embryos present had no effect on pregnancy maintenance. The remaining 23.5% litter loss was likely due to the lack of fertilization or embryonic death prior to 14d of gestation as egg or blastocyst resorption would have been complete by the time of slaughter. The fact that one gilt had no litter loss at all is not uncommon. It is more likely to happen than entire litter loss(Wrathall, 1971).

Using King and Young's(1957) classification system, it was fairly simple to assign live or dead status for each embryo. The bulk of embryos were normal and healthy macroscopically and this included a number of embryos which were smaller than average but apparently healthy. A certain percentage were degenerating or in the process of reabsorption. As in Warwick's study(1928), they were found throughout the uterus, healthy beside dead specimens. Hammond(1921) had recounted that the membranes could be living while the embryo had died some time before. This

seemed to be the case when an embryonic sac was found empty, others contained partially reabsorbed embryos and some had small anemic individuals contained therein. Burger(1952) and King and Young(1957) classified anemic embryos as dead. Embryos dying after 17 d of growth should have been identifiable by some remains and would be classed as embryonic, not egg loss(Burger, 1952). In one case the remains were simply a brown slimy mass similar to what Corner(1923) described. Dead embryos were of varying sizes indicating that mortality took place at different ages.

A curious phenomenon was observed by this author and others(Robertson et al., 1951; Squiers et al., 1952; King and Young, 1957; Gossett and Sorenson, 1959). Some outwardly healthy embryos were found in amniotic sacs containing blood. These were assumed to be alive until the dam's death and the blood due to trauma during the slaughter process. The reproductive tracts in this experiment were not recovered until after the scalding, dehairing and evisceration processes were completed.

The number of embryos tended to be equalized horn to horn yet the number of CL's were very different side to side in most gilts. According to the literature(Corner, 1921; Warwick, 1926; Burger, 1952; Dhindsa et al., 1967), migration of eggs must have taken place and these pigs were guite normal in the extent of egg migration displayed. RUnlike Sanada's(1986) findings, embryos were concentrated in the centre part(39%) and oviduct end(38%) of the uterus

rather than just the central portion.

Pork producers accept a first litter size of eight for gilts(Hatcher and Wilson, personal communication). Groups L and M carried an average of 5.2 embryos and H Group, 7.7 on average(litter sizes: 10, 10, 10, 6, 6, 4). Good size litters can be carried by gilts and the chances are increased if the females are of the age and weight of the H Group.

This particular experiment displayed the accuracy of using E_1SO_4 to diagnose pregnancy. Because this metabolite is produced by each embryo, their cumulative production raises the E_1SO_4 levels. It was a good indicator of litter size especially since multiple blood sampling was done unlike field trials where only one sample would be collected and not likely when E_1SO_4 was maximal. Large litters of 9-10 were easy to identify(over 4.0 ng E_1SO_4/ml) on peak days. In this experiment, P_4 could also have been used for pregnancy diagnosis. In comparison, other studies with high embryonic mortality and the presence of cystic follicles in sows have only been 60% accurate in this regard(Williamson et al.,1980). However, unlike E_1SO_4, P_4 will give no indication of litter size as P_4 production is independent of the number of embryos(Webel et al.,1975).

SUMMARY(EXPERIMENT_1)

1. There was no difference in response to a constant dose of PMSG regardless of gilt age or weight(Group) for the characteristics measured.

2. Plasma P_4 levels were within normal ranges and patterns in cyclic and pregnant gilts.

3. Puberty(cyclicity and/or pregnancy) was maintained more reliably in H Group($p<0.001$).

4. Puberty appeared to be hastened by PMSG/hCG treatment as 87% of H Group gilts became puberal subsequent to treatment at 173.1±6.8 d of age.

5. Vulval reactions at the time of second estrus were associated with estrus for H Group.

6. The display of standing estrus was poor and conception rate was low(52%).

7. AUC of P_4 at the induced estrus was greater than AUC at second estrus($p<0.01$). This suggests that PMSG was superovulatory.

8. There was a seasonal difference in P_4 levels in gilts which became cyclic subsequent to PMSG/hCG, replicate 3(spring) having the highest output at the induced estrus($p<0.05$).

9. The amount of P_4 was positively correlated with CL numbers in cyclic($r=0.76$, $p=0.0071$) and pregnant gilts($r=0.75$, $p<0.007$).

10. There was no significant difference in ovulation rate at second(12.4±1.9) and third estrus(12.5±1.1).

11. Ovarian and uterine weight, and uterine horn length were greater after puberty($p<0.001$).

12. Estrone sulphate was 100% accurate in determining pregnancy 22-27 d post-A.I..

13. Maximum E_1SO_4 levels were higher in large litters(9-10 embryos) versus small litters(3-6 embryos)($0.01<p<0.001$).

14. There was no difference in pregnancy rate among Groups.

15. Total litter loss was 39.5% up to 34 d of gestation; 23.5% as eggs or blastocysts(prior to 14 d) and 16% as embryos(after 14 d).

16. Uterine weight was greater in pregnant than non-pregnant gilts at 34 d post-breeding($p<0.001$).

EXPERIMENT 2
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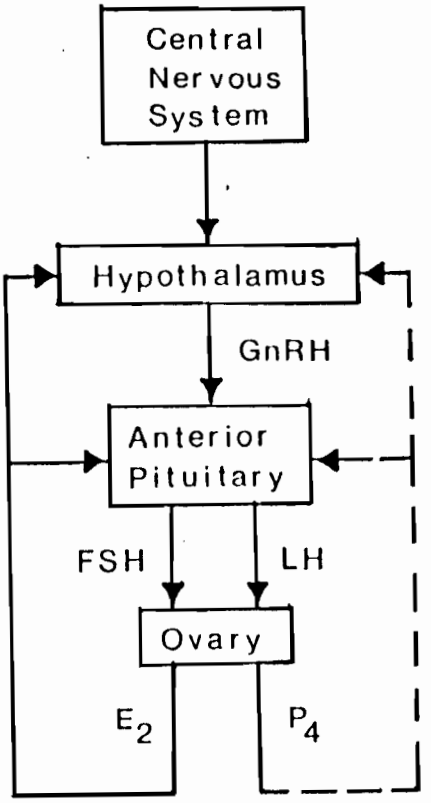
LITERATURE REVIEW

NEUROENDOCRINE CONTROL MECHANISMS INVOLVED IN THE ONSET OF PUBERTY: Reproduction in the female mammal is controlled by several neural and endocrine components in intricate communication with each other. Collectively, they are termed the hypothalamic-anterior pituitary-ovarian axis (H-AP-O axis). These units are functionally integrated with communication between them being mainly by chemical signals in the form of hormones. The hormones can have a stimulatory or inhibitory effect on each other or target organs. The basic functions of the H-AP-O axis are as follows: the hypothalamus(which is part of the central nervous system or CNS) responds to the CNS signal by releasing GnRH in the appropriate amounts, the anterior pituitary which discharges FSH and/or LH in the correct ratio when stimulated by GnRH and the ovary whose specialized cells synthesize and release P_4 and/or E_2 into the general blood circulation. In turn, plasma steroid hormone concentrations have a modifying effect on GnRH and FSH/LH release through regulation by a network of positive and negative feedback particularly by E_2(Hughes and Varley, 1980). Variations in steroid levels are a result, not a cause, of gonadotropic function patterns.(Figure 7).

There are currently two theories to explain prepuberal restraint of gonadotropic secretion during sexual development until puberty, 1/ intrinsic CNS inhibition(sex steroid independent), 2/ the gonadostat theory(sex steroid dependent). Levasseur (1977) suggested that control of

FIGURE 7 . LEVELS OF ORGANIZATION IN THE HYPOTHALAMIC-ANTERIOR PITUITARY-OVARIAN AXIS.



puberty might be under the influence of the CNS. The CNS perceives and monitors physical and chemical body changes constantly. These inputs, both stimulatory and inhibitory, may alter CNS relations with the hypothalamus and in turn affect the rest of the H-AP-O axis. Gonadotropic function in

the fetus is complete but is suddenly damped at or shortly after birth. This damping process appears to be basic to the evolution of the young mammal. The inhibition is completely independent of steroid control. In the late juvenile stage, there is a gradual suppression of the neural inhibitory mechanism which leads to the activation of gonadotropic secretion. Gonadotropic levels rise with increases in pulse amplitude and frequency. Finally, there is a maturation of the system regulating FSH and LH such that secretion is coordinated for the two gonadotropins. The CNS inhibition is accompanied by a change in steroid sensitivity of the hypothalamic-anterior pituitary units.

The 'Sexualzentrum' was first postulated by Hohlweg and Junkman in 1932. Low serum gonadotropin concentrations characteristic of prepuberty result from hypersensitivity of the hypothalamic-anterior pituitary system to the negative feedback action of small quantities of ovarian steroids(Fonda et al.,1983). In 1963 Ramirez and McCann reintroduced the same concept under the name of the gonadostat theory. Puberty will occur when the gonadostat (which is very sensitive to negative steroid feedback) gradually desensitizes allowing gonadotropic secretion to increase. The threshold for negative feedback increases with age while the sensitivity to inhibitory effects of sex steroids decreases. There is always some secretion of gonadotropins but at a very low tonic level. The negative feedback loop is dependent on the development of E_2

receptors in the hypothalamus and pituitary which appear midway through the prepuberal period. The level of inhibition by a given dose of E_2 varies with the maturation of the female(Hughes and Varley, 1980). The parts of the H-AP-O axis mature at different rates.

Gonadotropins cannot be secreted unless GnRH is released by the hypothalamus and the pituitary, which synthesizes and stores the gonadotropins, is able to respond with a release of FSH and LH. Pulsatile release of LH and FSH is due to the pulsatile release of GnRH. In pigs, the pituitary can respond to GnRH at 60 d of age(Foxcroft et al., 1975 ; Pomerantz et al., 1975). Thus, puberty is not prevented or delayed by the lack of release or synthesis of GnRH nor by the ability of the pituitary to respond. Spontaneous episodic release of LH is evident by the tenth week of life(Foxcroft et al., 1975; Kanematsu et al., 1984) with a frequency of 1.3 peaks/h. Follicles appear at 8-10 weeks and are then sensitive to gonadotropic stimulation(Kather and Smidt, 1974; Oxender et al., 1979). At this time, the follicles begin the production of low levels of E_2. This follicular appearance is entirely age dependent. Early ovarian development is independent of the pituitary(Colenbrander et al. 1982b).

The feedback effects of E_2 have been studied by OVX and administration of E_2, usually in the form of EB. In the mature cyclic animal, OVX causes an increase in LH release(Parvizi et al., 1976; Berardinelli et al., 1984).

Doses of EB will suppress LH for a period of h, partly due to suppression of GnRH at the hypothalamus(Cox and Britt, 1982a). In the one week old miniature pig, OVX leaves LH secretion unchanged(Elsaesser et al., 1978). Up to 80 d old, OVX does not affect LH levels(Wise et al., 1981; Wise and Zimmerman, 1982). At 120, 150, 180, 210 d old when OVX is performed, increased LH levels result though peak frequency before and after OVX are similar in all age groups(Berardinelli and Anderson, 1981). Fonda et al.(1983) noted, however, that frequency and amplitude increase when 170 d gilts were OVX. When OVX gilts were implanted with E_2 at 120 d , both lowered frequency and amplitude decreased mean LH. The same procedure at 150 d initially suppressed only LH frequency and at 14 d(post-OVX-E_2) LH levels began to rise. This delay suggests a change in sensitivity to negative feedback(Berardinelli and Anderson, 1981). Gilts OVX at birth show different responses. Between 80 and 135 d, LH rises two times above its pre-80 d mean(Wise et al., 1981; Wise and Zimmerman, 1982). Doses of EB(20 ug/kg body weight) were given at 7, 32, 56, 112, 140 d of age. Luteinizing hormone was unchanged at 7 d, decreased at 32 d and was suppressed at all other ages. Follicle stimulating hormone was suppressed at all ages. Intact gilts showed increased LH and FSH 2 d after EB challenge at 112 and 140 d but OVX gilts show no positive response at any age. For the same dosage of EB, E_2 levels differed markedly with age. Gilts aged 7, 32 and 56 d averaged 23.9 pg E_2/ml serum while

112 d gilts had levels of 39.5 pg/ml and 140 d gilts 85.2 pg/ml. Wise and Zimmerman(1982) concluded that 1/ ovarian inhibition of LH/FSH started between 112 and 140 d of age, 2/ negative feedback of FSH began prior to 7 d but LH not until after 32 d , 3/ no positive feedback develops in female pigs OVX at birth , 4/ there is a change in E_2 metabolism between 112 and 140 d.

Chronic estrogenic environments in the intact gilt lead to depressed LH basal secretion and responses to exogenous GnRH while pituitary responsiveness remains unchanged (Foxcroft et al., 1975). Acute doses of EB give no LH response at 6 d old and the response is weak at 60 d(Elsaesser and Foxcroft, 1978; Elsaesser and Parvizi, 1979). Low(60 μg) versus high doses(600 μg) of EB modified the response pattern but LH only increased to 5.0-7.5 ng/ml. In 160 d gilts, either dose produced a strong, well-defined surge to near 15.0 ng/ml. It appears that positive feedback maturation is gradual and may be mature prior to puberty(Foxcroft, 1977). Dial et al.(1984) found LH amplitude to differ when gilts aged 100, 135, 175 and 190 d were treated but time from injection to LH surge was constant(50-54 h). Surges occurred at 06.00 or 24.00 h in 33/34 gilts. When EB dose was increased, multiple surges appeared at 24 h intervals in 100, 135, 150 and 175 d old gilts but only one surge at 70 or 190 d(Dial et al., 1983,1984). Using PMSG to raise E_2 levels, Dial et al.(1984) found that surges took 66-72 h to appear but LH amplitudes

were not different for the ages tested. Foxcroft(1977) concluded from studies on suppression of episodic LH in gilts 40-180 d of age that there was no characteristic change in sensitivity to E_2 during these stages of development at physiological E_2 levels.

Levels of hormones in the blood are determined by secretion rate, interconversion from the precursor and metabolic clearance rate(MCR). The metabolism of E_2 changes with age(Elsaesser and Foxcroft, 1978;Elsaesser and Parvizi, 1979; Wise and Zimmerman, 1982). Elsaesser et al.(1982) calculated a higher MCR for E_2 in 60 d gilts than those 160 d old. The rate of inactivation rose more slowly with age than body weight and, therefore, blood volume. Constant levels of E_2 production, combined with a lower MCR, could raise the steroid level and provide an effective ovarian E_2 feedback signal which would lead to the preovulatory LH surge. It could be the lack of an ovarian signal, rather than the lack of capability by the rest of the H-AP-O axis, which prevents the onset of the puberal estrus.

Low E_2 levels during the prepuberal period suggest that increasing E_2 titres are not necessary for positive E_2 feedback maturation in swine(Foxcroft, 1977). It seems that, for puberty to occur, E_2 levels must be raised over a certain period of time and over a certain threshold(Elsaesser, 1982). Prior to this, certain conditions must be present: FSH-primed follicles must be secreting enough E_2 to reach and maintain threshold levels

in the blood, sufficient LH must be synthesized to produce a surge and the hypothalamus must have adequate GnRH available to respond with acute release(Reiter and Grumbach, 1982).

Trout et al.(1984b) determined the pituitary concentration of LH and the hypothalamic concentration of GnRH in 210 d old gilts. Regardless of the gilt's reaction to relocation and boar exposure, hormonal concentrations did not change. Before puberty very little FSH and LH are released into the circulation. Yet Parlow et al.(1964) found that FSH and LH concentrations in the pituitary were two to four times greater during this time than during estrous cycles. Foxcroft(1977) concluded that the size of the releasable pool of LH was likely constant and thus would limit the maximum response provoked by endogenous or exogenous means. The number of receptors for E_2 in the pituitary and hypothalamus were studied by Diekman and Anderson(1982b). At age 120 and 165 d, no changes in their number were found as puberty neared. When they treated 150 d gilts with PMSG/hCG, the same conclusion was reached(Diekman and Anderson, 1982a).

HORMONE PATTERNS: Several different patterns of LH secretion are evident during maturation in the gilt:

1/ tonic or basal- always present and characteristic of the physiological state of the animal(usually <1.0 ng LH/ml); it is regulated by negative feedback

2/ pulses- which are small deviations(1.5-3.0 ng LH/ml) from basal levels varying in frequency and amplitude

3/ surges- sudden threefold(or more) increases over basal levels with durations much longer than a pulse.

Ovarian steroid levels are low throughout development. Progesterone remains below 50 pg/ml blood until puberty. It is not likely that P_4 priming is necessary for puberty in gilts(Esbenshade et al., 1982;Andersson et al., 1983). Likewise, E_2 levels remain low(<20 pg/ml) with a sharper rise only within 10 d of estrus(Elsaesser and Foxcroft, 1978; Lutz et al.,1984). Urine is the primary route for estrogen excretion in the pig and increases in its excretion(as estrone) have been observed during the period when antral follicles become active and FSH/LH activity is the highest(Prunier et al., 1982; Camous et al., 1985).Around 125 d when gonadotropin levels decrease, there is a substantial increase in the estrone excretion rate(Camous et al., 1985).

Follicle stimulating hormone secretion tends to be greater and less variable than LH. It is maximal prior to 10 weeks of age(average 10 ng/ml)(Diekman et al., 1983; Camous et al., 1985). The titre decreases steadily to near 7.0 ng/ml by 126 d(Prunier et al., 1982; Diekman et al., 1983) and then follows a slower decline to about 3.0 ng/ml prior to puberty(Prunier et al., 1982; Camous et al., 1985; Grieger et al., 1986). Ponzilius et al.(1984) found no definite pattern until 5 d before estrus. When pulsatility has been studied, pulses of FSH and LH were synchronous from 70 to 126 d which coincided with the time of maximum levels

for both these gonadotropins(Diekman et al., 1983).

During sexual maturation, LH is the most active hormone not only in mean levels but in pulse activity. Mean levels start high being 1.7 ng/ml at 40 d(Fleming and Dailey, 1982; Kanematsu et al., 1984). After this LH falls below 1.0 ng/ml (Karlbom et al., 1981/1982; Lutz et al., 1984). Once at this low level, some researchers have reported a continuous decreasing trend; 60 d, 0.38 ng/ml; 120 d, 0.16 ng/ml; 160 d, 0.13 ng/ml(Guthrie et al., 1984). Mean levels are a reflection of the amplitude and particularly the frequency (Pelletier et al., 1981). Amplitude is constant and highest at 54 to 125 d(approximately 4.0 ng/ml)(Kanematsu et al., 1984) falling between 137 and 196 d to near 2.0 ng/ml(Prunier et al., 1982; Grieger et al., 1986). Frequency is highest at 83 to 126 d(2.0$\pm$1.6 peaks/6 h)(Pelletier et al., 1981; Prunier et al., 1982; Camous et al., 1985) with a drop to 1.0$\pm$1.2 peaks/ 6 h by 140 to 196 d(Prunier et al., 1982; Andersson et al., 1983). Pelletier et al.(1981) felt this decrease was due to an increase in E_2 or a change in pituitary or hypothalamic sensitivity. Two other groups reported no change in frequency from 120 to 210 d(Diekman et al., 1983; Grieger et al., 1986). During some bleeding windows, experimenters saw no pulses in some gilts(Kanematsu et al., 1984; Camous et al., 1985). It appears to be age related. Camous et al.(1985) found 12.5% of gilts aged 83-125 d with no pulses in 6 h and this proportion increased to 35% when gilts 137-192 d of age were bled. A

significantly greater proportion of episodic LH pulses were found during the night in 160 d but not 60 d old gilts by Elsaesser and Foxcroft(1978). There seems to be a link with the light/dark cycle. During the late prepuberal period in the human, the number of LH pulses increase and occur synchronously with sleep(dark portion of photoperiod)(Boyar et al., 1972). Involvement with the sleep-wake cycle implies control by the CNS. Amplitude, frequency and means change dramatically 3-12 d before estrus(Pelletier et al., 1981; Lutz et al., 1984; Diekman and Trout, 1984). This is in contrast to the mature cyclic female. Her basal LH is fairly constant and peaks are suppressed in the follicular stage (Van de Wiel et al.,1981). During the luteal phase pulses rise to approximately 3.0 ng LH/ml from undetectable levels(Parvizi et al., 1976; Van de Wiel et al., 1981).

GnRH AND ANTERIOR PITUITARY RESPONSIVENESS: Under normal circumstances, every pulse of GnRH released by the hypothalamus is followed by an LH increase in the blood due to LH release by the pituitary. The importance of this relationship was demonstrated when gilts were immunized against GnRH(Esbenshade et al., 1984; Esbenshade and Britt, 1985). Reproductive function was impaired. Serum levels of gonadotropins and steroids were reduced and gonadal dysfunction resulted. All immunized gilts eventually became acyclic and their ovaries were immature in appearance. There was no response to GnRH injection and following OVX, LH became undetectable(Esbenshade and Britt, 1985).

Beginning with puberty, the cyclic gilt displays a single preovulatory LH surge ranging in amplitude from 1.5-9.6 ng/ml due to endogenous secretion(Wilfinger et al., 1973; Van de Wiel et al., 1981; Esbenshade et al., 1982). This surge has a duration of approximately 28 h from initial rise until return to baseline(Wilfinger et al., 1973; Van de Wiel et al., 1981; Karlbom et al., 1981/1982). The surge peak is most often reached between 24.00 h of Day 0 and 06.00 h of Day 1 of the estrous cycle(Wilfinger et al., 1973; Van de Wiel et al., 1981). Cyclic females have the LH surge triggered by a sustained increase in E_2 above a threshold level(Van de Wiel et al., 1981).

Researchers have sought to test the H-AP-O axis by using exogenous GnRH and relating the subsequent LH release to anterior pituitary responsiveness at various points throughout the maturation process. The doses used have varied widely, either kept constant or adjusted to body weight. The response depends on the physiological state of the animal. Fetuses immediately prior to parturition will respond to GnRH with an average sized surge(approximately 4.0 ng LH/ml)(Colenbrander et al., 1982a). At the same time, their dams will give a very low response(<3.0 ng LH/ml). After birth and as lactation proceeds, the sow's response becomes very fast and strong(>6.0 ng LH/ml)(Bevers et al., 1981). A cyclic gilt in either the follicular or luteal phase will give an average response(2.0-4.0 ng LH/ml) similar to a prepuberal female but less prolonged(Vandalem

et al., 1979). Gilts with delayed puberty were given large doses of GnRH(1 mg) resulting in low to medium surges(1.45-6.1 ng LH/ml) which peaked after 1-1.5 h and took approximately 6 h to return to baseline(Edqvist et al., 1978).

Young weaners(13 kg) produced surges of 16.2 ng/ml in response to 25 μg of GnRH(Chakraborty et al., 1973). Most other prepuberal gilts in the literature(40-210 d age) responded with surges in the normal range for LH(Foxcroft et al., 1975; Feming and Dailey, 1982; Trout et al., 1984a; Guthrie et al., 1984). Significant rises in LH could be observed within 5 or 10 min of injection(Chakraborty et al., 1973; Foxcroft et al., 1975; Pomerantz et al., 1975). A linear relationship was found between GnRH dose and LH response(Foxcroft et al.,1975; Trout et al., 1984b). There was no difference found in the amount of LH released between ages at a constant dose when a correction was made for increased blood volume. Also, when area under the LH release curve(AUC) was considered at a specific dose, there was no difference between gilts of various ages(Vandalem et al., 1979; Guthrie et al., 1984; Trout et al., 1984a/b). Guthrie et al.(1984) discovered that as the gilt ages, the time to reach peak LH appears to increase. Interestingly, Trout et al.(1984b) found gilts responding to boar exposure and relocation with puberty to have a lower pituitary responsiveness(4.6±1.3 ng LH/ml) than gilts remaining prepuberal(9.8±0.8 ng LH/ml). After the LH surge, LH

gradually returned to pre-injection levels. Foxcroft et al.(1975) noted that LH pulse peaks were superimposed on the downhill side of the surge. Fleming and Dailey(1982) noted that these pulses appeared with decreasing frequency in older animals(40 d, 2.1 peaks/3 h; 80 d, 2.4 peaks/3 h; 120 d, 2.2 peaks/3 h; 160 d, 1.0 peaks/3 h)). Since LH release is so much higher in younger gilts('hyper-responsive'), this appears to amplify later LH release.

INTRODUCTION(EXPERIMENT_2)

From the results of Experiment 1 it was very clear that there was a distinct difference in puberty maintenance between the Groups of gilts, particularly between H Group and M or L Group. The response of all the gilts to PMSG and hCG indicated that the ovarian part of the H-AP-O axis was operational and that perhaps the block to puberty was at the hypothalamus or the anterior pituitary. Since LH is the more active gonadotropin and the one which triggers the first ovulation, its concentration in the blood was of particular interest if the anterior pituitary was to be evaluated. The objectives of this second experiment were: 1/ to chart the pulsatility of LH at three points in time during the maturation process 2/ to test the responsiveness of the anterior pituitary, as a measure of sexual maturation, after the administration of exogenous GnRH. Prepuberal females were tested at ages 110, 140 and 170 d which were the average ages of Groups L, M and H from Experiment 1.

MATERIALS AND METHODS (EXPERIMENT 2)

ANIMALS: Six prepuberal Landrace gilts, approximately 105 d of age, were randomly selected from the Macdonald College herd. Gilts were transported to another barn away from breeding stock and housed three animals per pen. Feed, water and lighting were supplied as described previously (Expt.1, Materials and Methods). No littermates were used in this experiment. After catheterization, each animal was penned separately and allowed unrestricted movement until the catheter was removed.

EXPERIMENTAL PROCEDURE: Three blood collection dates were scheduled at 29 or 30 d apart when the group of six gilts reached ages of approximately 110, 140 and 170 d (Bleeds A, B and C were carried out on June 18, July 18, and August 16, 1985, respectively). To facilitate frequent blood sampling, all gilts were surgically fitted with an indwelling jugular catheter at least 2 d prior to the blood collection dates. Catheters were checked daily for patency and filled with heparinized saline (150 i.u./ml).

Beginning at 09.00 h, a four ml blood sample was removed via the catheter every 10 min for eight h. After the 17.00 h sample was collected, synthetic GnRH (chloride form, Batch 2; NIAMDD-NIH, Bethesda, MD.) was injected intravenously 0.5 μg/kg body weight in 1.0 ml of physiological saline. The 10 min sampling regime continued for a further four hours until 21.00 h. To obtain

a sample, the heparinized saline along with some blood was drawn into a syringe and discarded. Then the blood sample was taken up into a heparinized syringe, and the catheter was refilled with heparinized saline (25 i.u./ml) and the stopper reinserted. Samples were refrigerated overnight at 5°C, then centrifuged at 2500 X g for 10 min. The plasma was decanted and stored at -20°C until assayed for LH and FSH. Catheters were removed nonsurgically 2 d after the blood collection date and new ones reinserted again prior to the next bleeding date.

Before each of the three blood sampling dates, every gilt was weighed in order to calculate the dose of GnRH. In addition, a weekly 10 ml blood sample was taken from all gilts via the catheter or jugular venipuncture for P_4 determination. These samples were handled as outlined previously (Expt.1, Materials and Methods). Just prior to being assayed, 100 µl aliquots were removed from each of the pre-GnRH samples for each blood collection day (A, B and C) to form pools which were used for FSH determinations. All of the serial samples were quantified separately for LH.

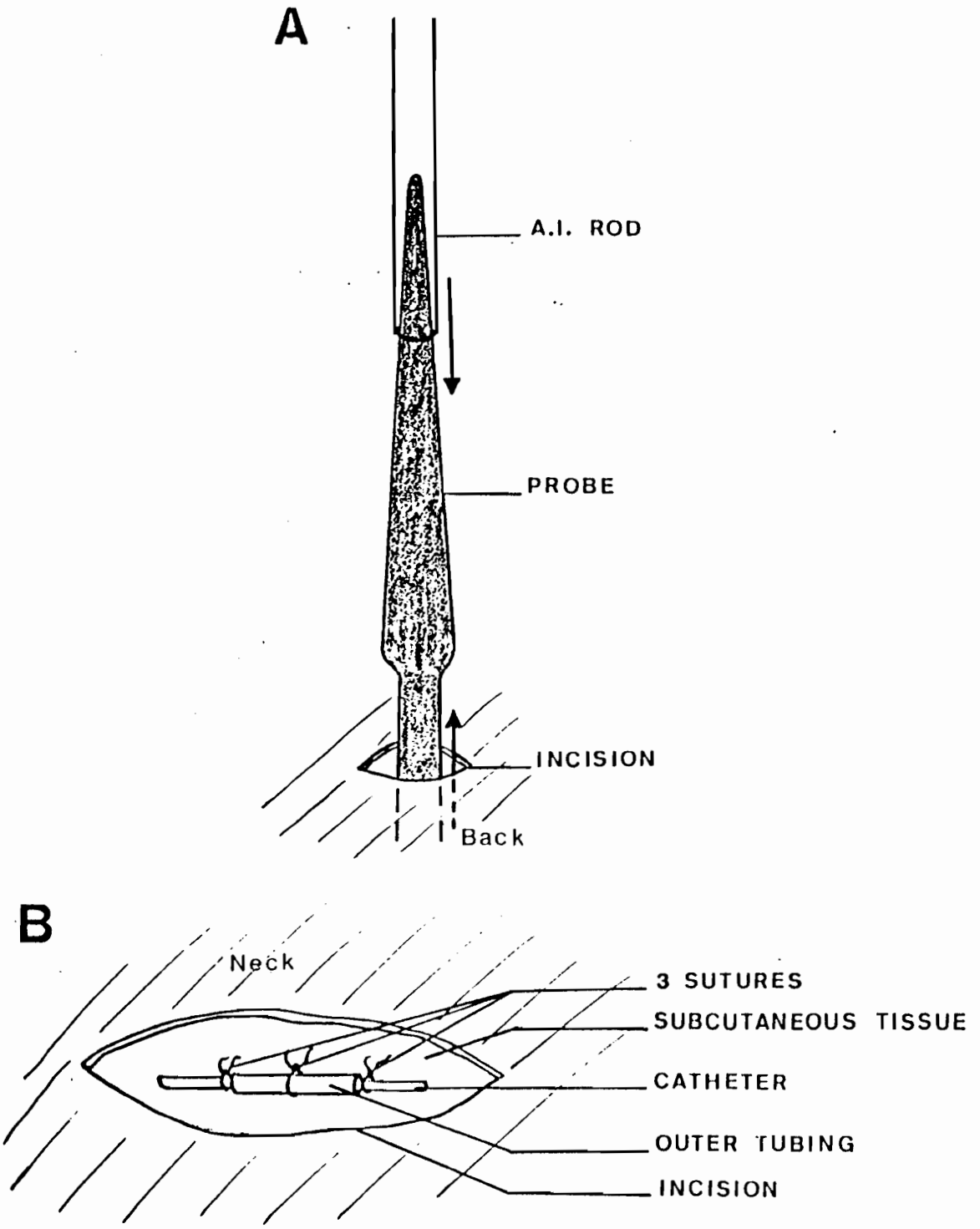
Gilts were slaughtered at least three weeks after the 170 d blood collection to allow time for antibiotic clearance from the tissues. Reproductive tracts were collected and examined as described previously under Expt.1, Materials and Methods.

SURGICAL CATHETERIZATION: The basic technique used was described by Ford and Maurer (1978). Differences in weights

of animals and non-restraint housing necessitated some changes in the procedure. Animals were sedated with 1.5-2.0 ml of Innovar®-Vet (Pitman-Moore, Scarborough, Ont.) and given 1.0 ml atropine (1/30 g/ml atropine sulphate ; Vetcom Inc., St. Liboire, P.Q.) at the same time to reduce salivary secretion. Each ml of Innovar®-Vet contains 0.628 mg fentanyl citrate and 20 mg droperidol. After the sedation, the gilt was placed under halothane inhalation anaesthesia (Somnothane® ; Hoechst, Montreal , P.Q.) and incision sites were disinfected. With the gilt on her side, a 12 gauge needle (Delvo® 2.80; CDMV Inc. , St. Hyacinthe, P.Q.) , with a 20 ml syringe attached, was used to puncture the jugular vein. At 110 d of age, a 51 mm needle was of sufficient length (gilt mean weight 47.4±2.8 kg). By 140 and 170 d of age, when mean weights were 66.7±8.2 and 90.4±5.9 kg, respectively, a 102 mm needle was required. The catheter (V/7; id 1.14 mm, od 1.63 mm; Bolab, Lake Havasu City, AZ.) was threaded into the vein via the needle, a distance of 12, 20 and 25 cm from the skin surface at Bleeds A, B and C, respectively.

A 50 cm long stainless steel probe with an 8 cm tapered head was introduced into the incision and forced dorsally under the skin to a point anterior to the scapula and just to the side of the cervical vertebrae. A 2 cm incision was made over the head of the probe. Next, the end of an A.I. rod was placed over the exposed head of the probe(Figure 8A).Both the probe and the A.I. rod were pushed back through

FIGURE 8. THE SURGICAL CATHETERIZATION PROCEDURE: A -PROBE MANIPULATION ON THE DORSAL SIDE OF THE NECK, B - ANCHORING THE CATHETER TO THE SUBCUTANEOUS TISSUE, C -PREPARATION OF PVC TUBING, D - ATTACHMENT OF THE CATHETER AT THE POINT OF EXTERIORIZATION, E -POSITION OF ADJUSTABLE CLOTH JACKET.



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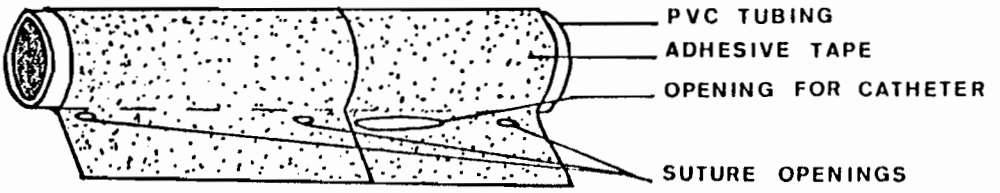
PVC TUBING
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OPENING FOR CATHETER
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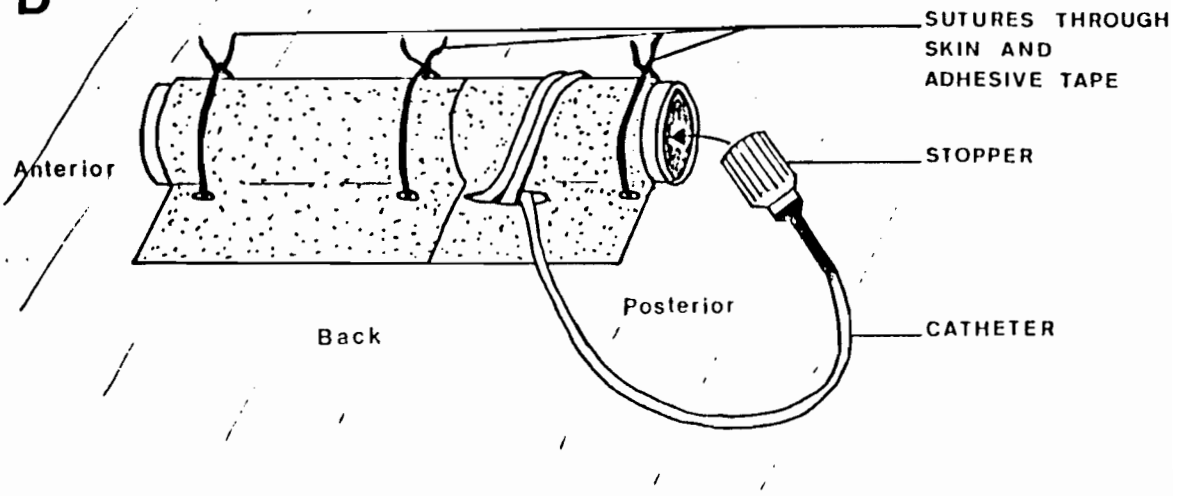
SUTURES THROUGH SKIN AND ADHESIVE TAPE
STOPPER
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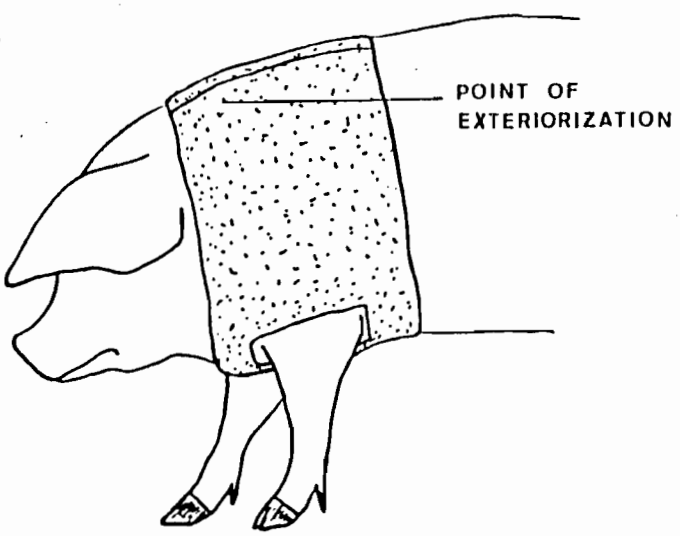
CATHETER

E


POINT OF EXTERIORIZATION

the subcutaneous tissue until the A.I. rod was positioned with both ends protruding - one ventrally, one dorsally. The catheter was then threaded through the A.I. rod leaving some slack remaining in the ventral neck incision, afterwhich the A.I. rod was removed.

To prevent slippage out of the vein, the catheter was anchored to the subcutaneous tissue in the ventral neck incision by putting a 2 cm section of large diameter vinyl tubing (V/12; id 1.78 mm, od 2.79 mm; Bolab) around the catheter within the incision and securing the two pieces of tubing together with glue(527 Cement®; Bond Adhesives Co., Jersey City, N.J.). Three stitches, one directly anterior, one directly posterior and one central to the outer tubing, were inserted as portrayed in Figure 8B and the remaining slack in the catheter was pulled up through the dorsal incision.

At the point of exteriorization, the catheter was wrapped twice around the middle of a 9 cm length of PVC tubing (id 12 mm, od 15 mm; Canlab, Mt. Royal, P.Q.) which had been covered with 5.0 cm wide adhesive tape (Zonas Porous Tape®; Johnson & Johnson Ltd., Montreal, P.Q.)(Figure 8C). The catheter and tubing were secured to the skin with three sutures . The length of exposed catheter was reduced to 10 cm, stoppered and pushed inside the PVC tubing(Figure 8D).An adjustable cloth jacket (Figure 8E) was put on the gilt to protect the catheter ,keep the wounds clean and allow the animal unrestricted movement within her pen.

Each gilt was injected with antibiotic (Liquamycin-LP®; 100 mg oxytetracycline hydrochloride per ml, Rogar/STB Inc., Montreal,P.Q.) after surgery (6.6 ml/100 kg body weight).

HORMONE ASSAYS: Progesterone assays were carried out as discussed previously (Expt.1, Materials and Methods).

Concentrations of LH were determined by radioimmunoassay according to the method of Niswender et al. (1969). Intra-assay coefficients of variation ranged from 3.0% to 10.2% for four assays while the inter-assay coefficient of variation was 3.9%. The assay had a sensitivity of 0.05 or 0.1 ng/ml (95% of B/B_0). Anti-ovine LH serum (GDN #15) was used at a dilution of 1:75,000. Two hundred µl aliquots of unknown sera were assayed in duplicate and the concentration expressed as ng LH(LER-778-4) per ml.

The assay for FSH was done at the Reproductive Endocrinolgy Laboratory, Macdonald College, under the supervision of Dr. L.M. Sanford. The procedure of Ponzilius et al.(1984) was followed. The pooled serum samples were assayed at 100 and 200 µl volumes in triplicate and the concentrations reported in ng FSH(NIH-FSH-P2) per ml. Purified rat FSH (NIH-FSH-I-5) was labelled for this assay and anti-human FSH serum (GP-202-B4) was used at a dilution of 1:8,000. Assay sensitivity was 270 ng/ml. This assay gives hormone readings approximately 80 to 100 times higher than homologous FSH assays reported in the literature. Intra and inter-assay coefficients of variation were 8.2% and 2.6%, respectively.

STATISTICAL ANALYSIS: For the purpose of this experiment, LH characteristics measured were defined as follows:

mean- arithmetic mean calculated from 49 separate samples/pig before GnRH injection on each bleed day

pulse- when any values was one SD or greater above the mean(Diekman et al., 1983; Lutz et al., 1984)

pulse duration- first rise above the mean until the mean was reached again

pulse amplitude- mean total height of all the identified pulses during the pre-GnRH period

pulse frequency- number of pulses identified during the 8 h prior to GnRH

surge- a sudden increase (more than threefold) in the mean LH level which was sustained longer than 1 h following GnRH injection

surge height- maximum total LH concentration observed during the surge

Δheight- surge height minus the LH mean

AUC- area under the GnRH induced LH release curve above the LH mean line

MTM- min to return to mean LH levels following GnRH injection

Analysis of variance was done with GLM(General Linear Model) procedure of Statistical Analysis Systems (SAS) (Helwig and Council,1979). Differences among treatment means

were tested by Duncan's New Multiple Range test(Steel and Torrie,1960). Correlation coefficients were used to evaluate interrelationships of interest.

RESULTS(EXPERIMENT 2)

HORMONE PATTERNS: Based on P_4 concentrations(low or undetectable), all gilts were prepuberal until after 170 d of age. Gilts 1 and 3 had attained puberty by slaughter at ages 205 and 192 d, respectively, as assessed by P_4 determinations and the appearance of the reproductive tract. The other gilts were still prepuberal at ages 182, 189 and 199 d. Gilt 6 died after surgery at 140 d and was confirmed to be prepuberal from P_4 and post mortem findings(Appendix Table 19).

TABLE 4. PRE-GnRH GONADOTROPIN MEASUREMENTS($\bar{X}\pm$SEM).

	110 d n=6	140 d n=5	170 d n=5
FSH-Mean (ng/ml)	573±155[a]	593±136[a]	688±152[a]
LH-Mean (ng/ml)	.43±.20[a]	.35±.13[a]	.38±.11[a]
Frequency (pulses/8h)	2.2±1.6[a]	1.6±1.1[a]	1.6±.05[a]
Amplitude (ng/ml)	2.4±1.1[a]	1.3±.47[a]	1.7±.68[a]

Means within a row bearing a different superscript are signicantly different($p<0.05$).

Mean FSH levels throughout this study were not significantly different among sampling dates(Table 4). Frequency, amplitude and mean LH values were highest at 110 d but the differences between collection dates were not significant for any of these measurements(Figure 9, Table 4). Mean and frequency of LH before GnRH were positively correlated(r=0.8316, p=0.0001)(Appendix Table 16). Pulse duration averaged 20-30 min.

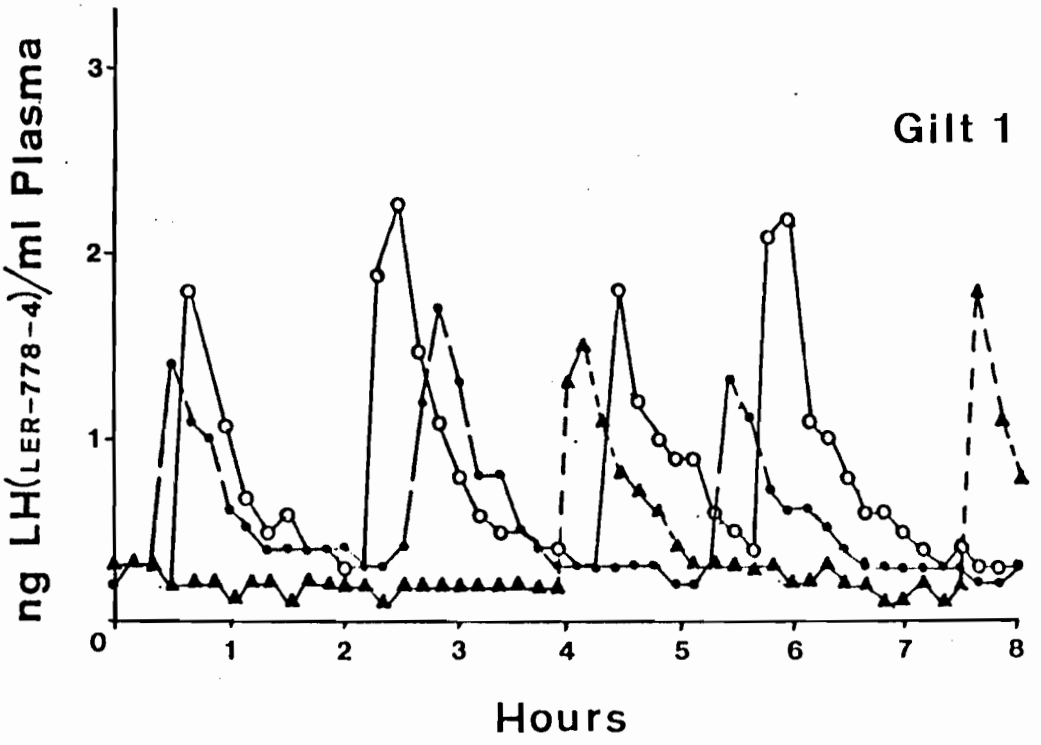
ANTERIOR PITUITARY RESPONSIVENESS: After GnRH injection, LH levels increased above the mean within ten min in all cases. Surge height was achieved by 20 min in 11/16(69%) of collection periods and by all gilts within 50 min. Increases up to and decreases from surge height were faster at 110 d than at other ages. Peak levels appeared to be sustained longer at ages 140 d and 170 d(Figure 10). The LH release

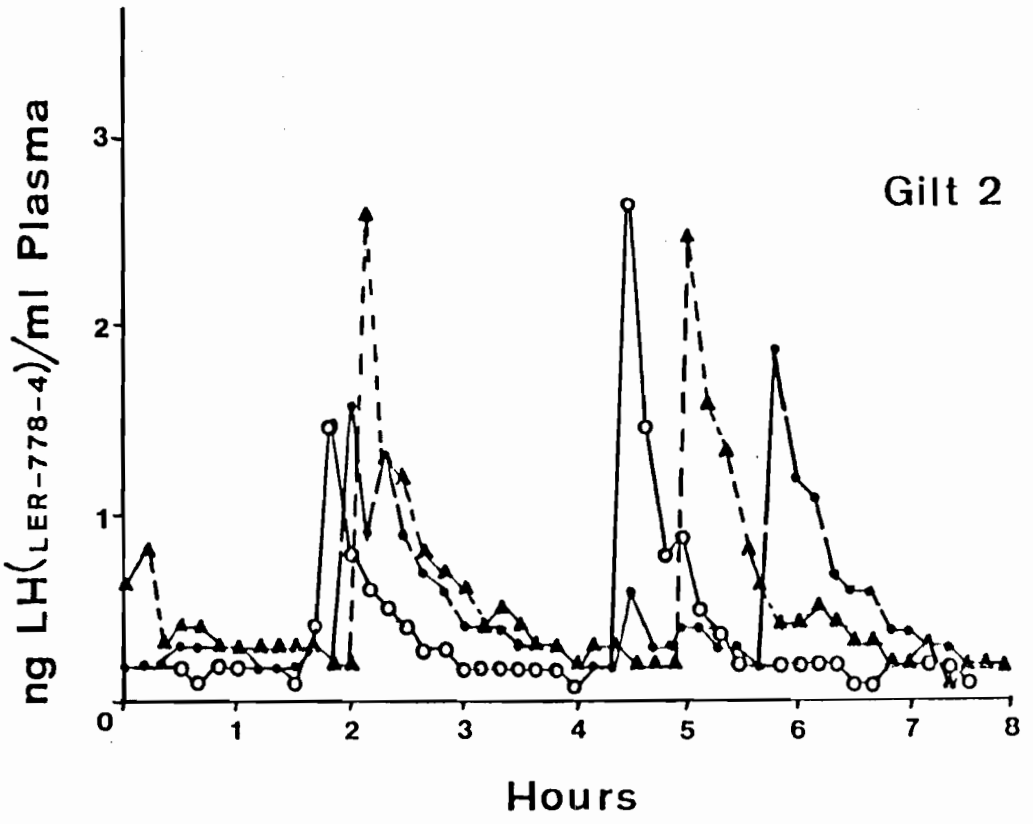
TABLE 5. POST-GnRH LH MEASUREMENTS($\bar{X}\pm$SEM).

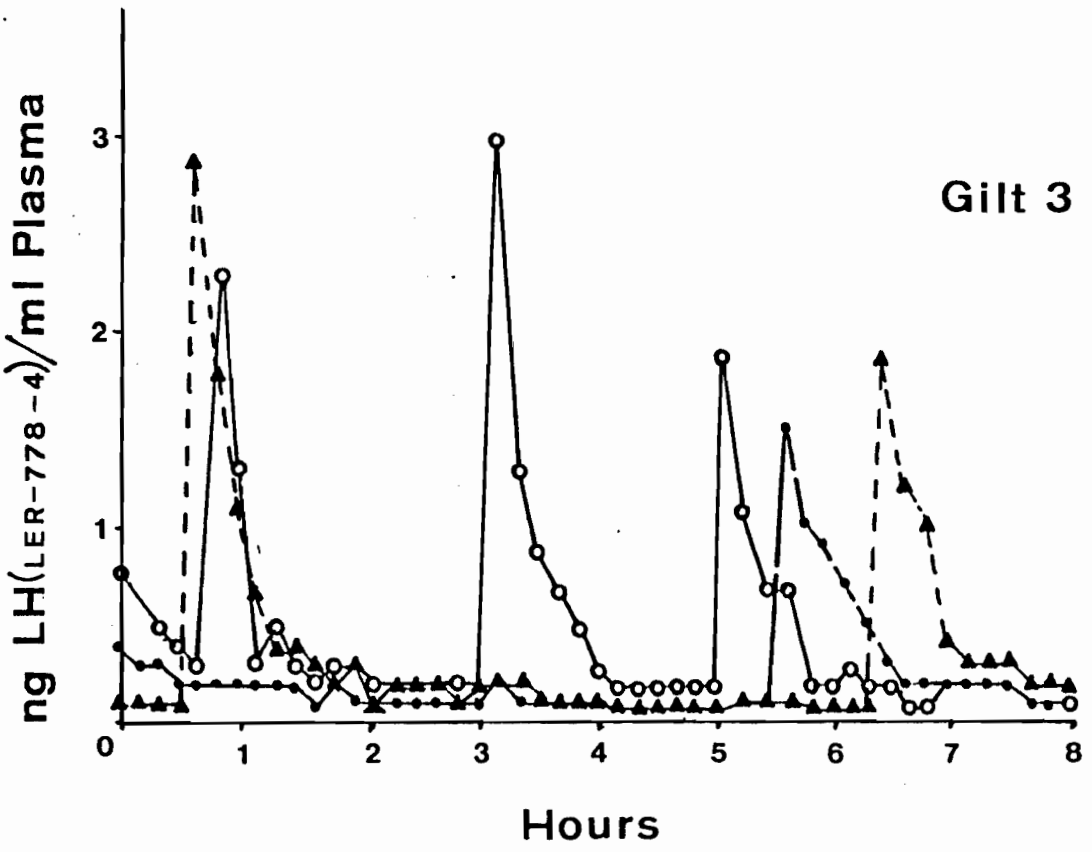
	110 d n=6	140 d n=5	170 d n=5
Surge Height (ng/ml)	12.1±5.8[a]	7.6±1.7[a]	9.3±3.5[a]
ΔHeight (ng/ml)	11.6±5.9[a]	7.3±1.8[a]	8.9±3.5[a]
AUC(arbitrary units)	4968±1918[a]	4773±1443[a]	4923±1868[a]
MTM(min to return to baseline)	152±46.2[b]	210±37.4[a]	174±43.4[a,b]

Means within a row bearing a different superscript are significantly different($p<0.05$).

FIGURE 9. LUTEINIZING HORMONE CONCENTRATIONS FOR 8 h AT 110 d(O—O), 140 d(●—●) AND 170 d(▲---▲).



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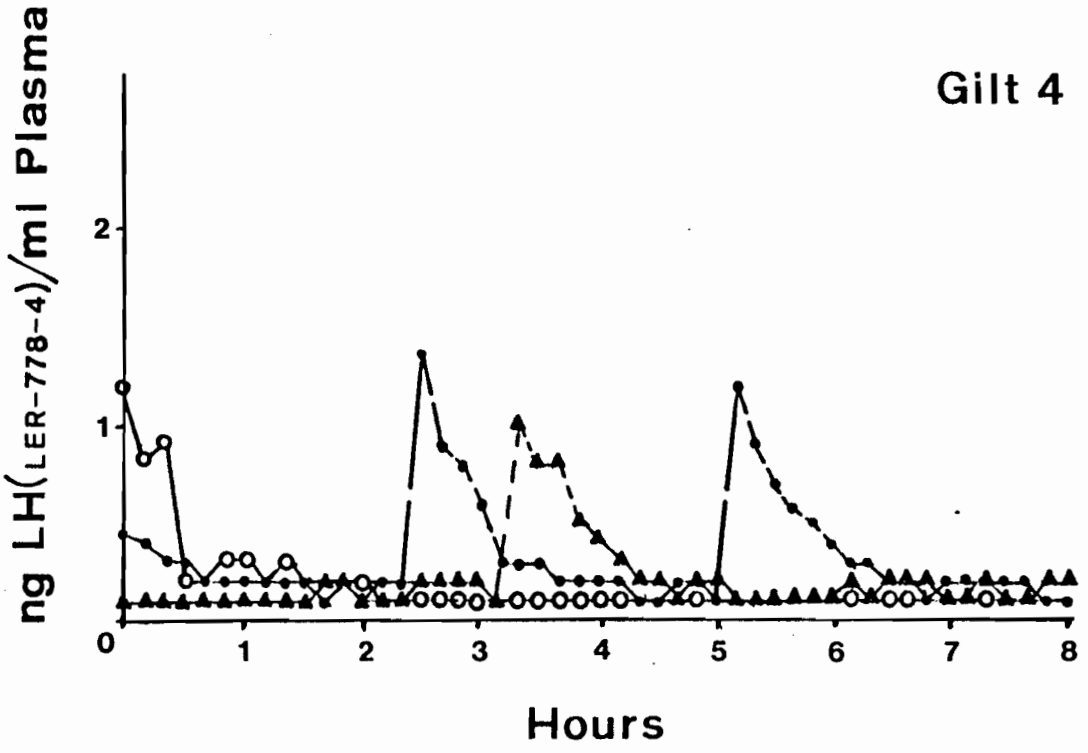
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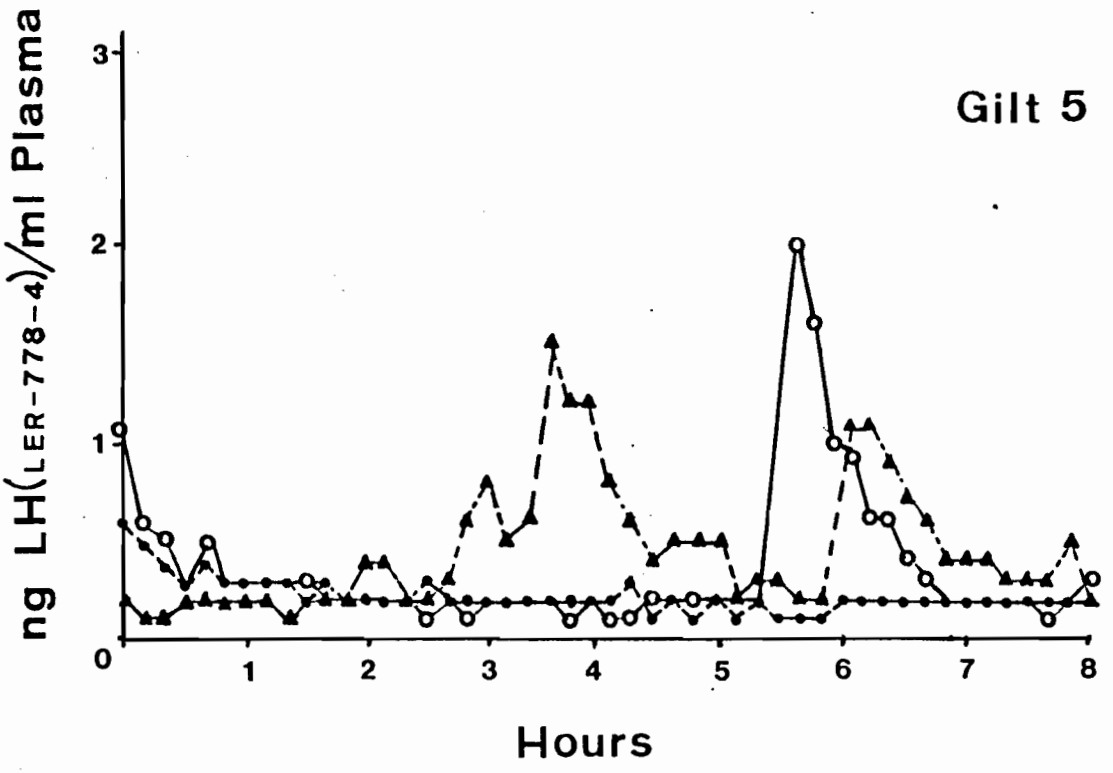
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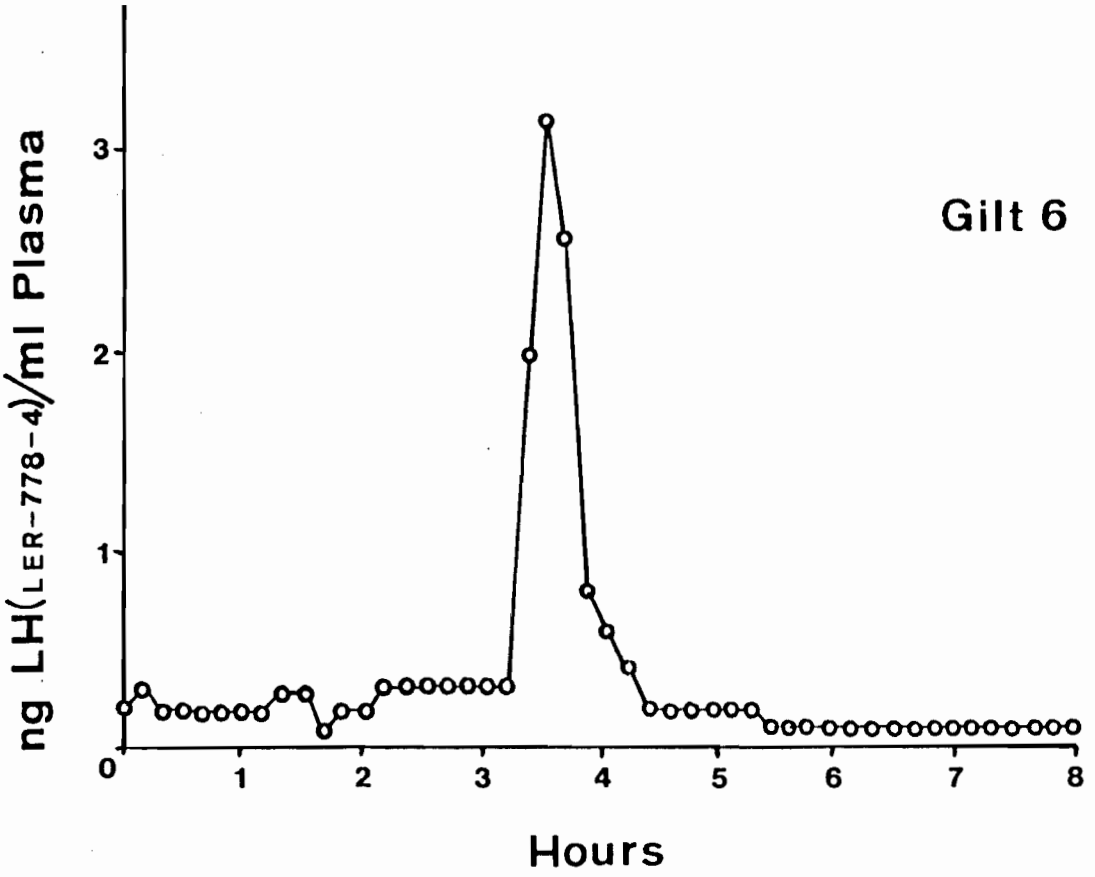
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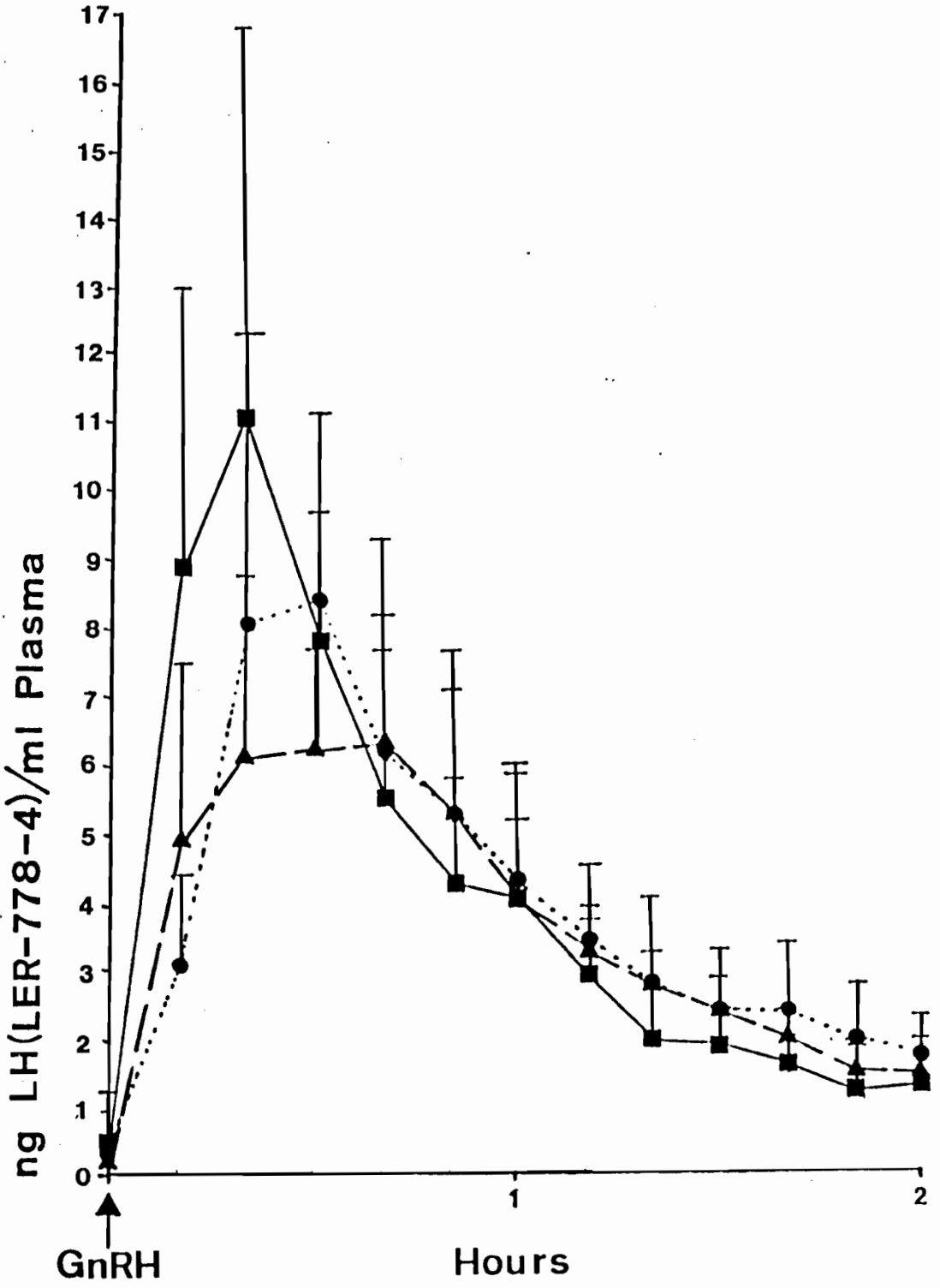
was highest at 110 d in terms of surge height and Δheight. Neither of these measurements were significantly different among any of the three ages tested. Likewise, AUC were similar. The only LH characteristic which changed significantly was MTM(α=0.04)(Table 5). Many of the surges had episodic pulses superimposed on their downhill side(average 1.5 ng LH/ml amplitude) but these never appeared until 2 h post-GnRH.

DISCUSSION(EXPERIMENT 2)

As expected, P_4 levels stayed low throughout the study and this small sampling of pigs seemed to follow the general Landrace population for age at puberty (average 200 d). Levels of FSH remained relatively constant. This experiment did not display the decrease in FSH seen by other authors(Prunier et al., 1981; Camous et al., 1985; Grieger et al., 1986).

Lack of significant differences between collection dates for the various characteristics measured suggest that 'maturational changes' from 110-170 d, when they occur, are very subtle. This study, as well as others (Pelletier et al., 1981; Prunier et al., 1982; Andersson et al., 1983; Grieger et al., 1986) do reveal a trend in decreasing LH mean, frequency and amplitude from 110 d onward. There is general agreement that LH means are below 1.0 ng/ml throughout the prepuberal period. Camous et al.(1985) reported a difference in percent of samples 1.0 ng LH/ml or

FIGURE 10. MEAN(±SEM) LH CONCENTRATIONS AFTER INJECTION OF GnRH AT 110 d(n=6)(■—■), 140 d(n=5)(▲—▲), AND 170 d(n=5)(●·····●).



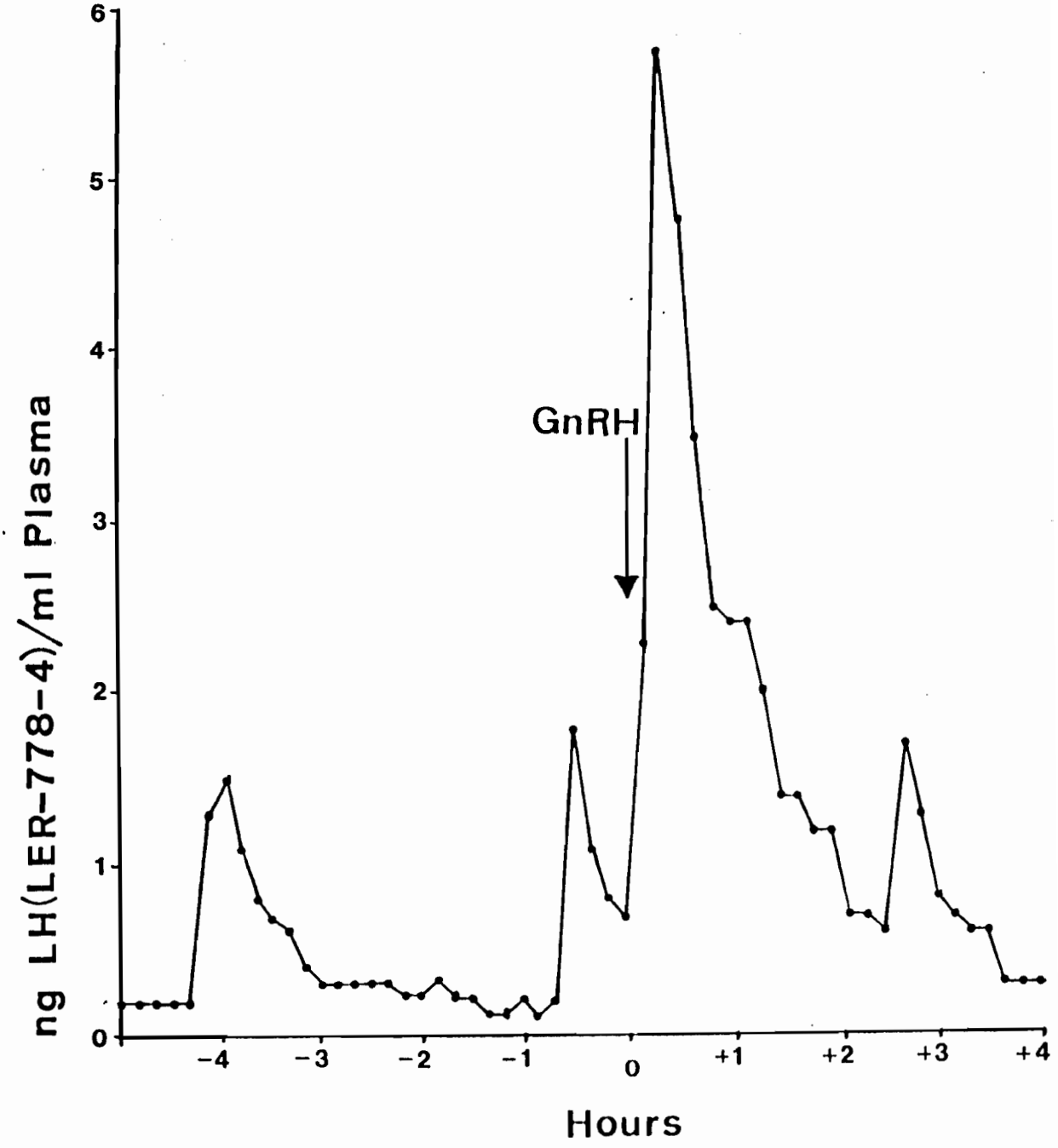
more. No such pattern was found in this experiment at that level or at 0.5 ng/ml among the ages tested. The correlation between LH mean and frequency seems to indicate that frequency has more effect in establishing mean levels than does amplitude. This concurs with the observation of Pelletier et al.(1981). The animals in Experiment 2 were too far from natural puberty(>14 d) to see any of the striking pulse changes observed within one week of estrus(Diekman and Trout, 1984). A recent report by Diekman et al.(1986) indicates that the sampling frequency of 10 min used in the present experiment was sufficient to evaluate accurately the LH characteristics studied with the exception of pulse height. They recommended sampling at 2 min intervals as maximum LH concentrations were maintained for only 2-4 min before decreasing.

The LH surge mechanism necessary for ovulation seems to be intact before the onset of puberty. As in other studies, GnRH injection caused LH titres to increase very rapidly, rising above the LH mean within ten min(Chakraborty et al., 1973; Foxcroft et al.,1975; Pomerantz et al., 1975). There is a large range in the individual response to the same dose at the same chronological age which has also been noted by other researchers(Foxcroft et al., 1975; Bevers et al., 1981), possibly due to inherent variation or different physiological ages. Gilts produced surges which were in the upper physiological range(>7.0 ng LH/ml).Other studies using higher doses have given smaller responses(Foxcroft et al.,

1975; Edqvist et al., 1978; Trout et al., 1984b). It is not known what dose, if any, gives a 'normal' physiological response. Dosage in the present experiment was based on the minimum shown to produce a 4.0-8.0 ng LH/ml surge and was given according to body weight to take into account varying body size at the three ages tested.

The anterior pituitary appears to be the most responsive at 110 d. The surge at 110 d was steeper than at other ages, probably because surge height was greatest at this age(Figure 9). Also, response was not lessened when an LH pulse occurred just before GnRH injection(Figure 10). Parlow et al.(1964) showed that during the prepuberal period, anterior pituiary LH concentration was two to four times greater than during the estrous cycle. They concluded that most of the gonadotropins must be stored with little released into the circulation. This certainly would account for the ability of the pituitary to release such large quantities of LH during the surge. Vandalem et al.(1979) found no difference in response to exogenous GnRH during prepuberal development. Trout et al.(1984a) reported that LH release was not significantly affected by age(105-195 d), yet their 105 d old gilts(in confinement as in this study) produced the highest LH response to exogenous GnRH. Age had a very significant effect on LH release in the investigation done by Fleming and Dailey(1982). Magnitude of LH release decreased from 40-160 d of age. Trout et al.(1984b) found that, during the very late prepuberal period, gilts which

FIGURE 11. PRE AND POST-GnRH LH CONCENTRATIONS IN GILT 1 AT 170 d. (NOTE THE PULSE IMMEDIATELY PRIOR TO GnRH ADMINISTRATION).



did not respond to puberty induction had a higher pituitary responsiveness to GnRH than those which did respond. They could not attribute this observation to changing E_2 serum levels, pituitary LH or hypothalamic GnRH concentrations.

Episodic pulses were superimposed on the surges (Figure 11),visibly prolonging the time for LH to reach mean levels. They were not present in all gilts at all ages. The reappearance of episodic pulses and the fact that other LH characteristics showed no significant differences among ages, makes the importance of MTM questionable. Foxcroft et al.(1975) found these episodic pulses 60 min after GnRH injection but their appearance showed no relation to dose or time after surge. In the present trial, no pulses were seen for the first two h by which time LH levels were below 2.0 ng/ml. Sampling was not done often enough to detect pulses in two other studies (Vandalem et al.,1979; Guthrie et al., 1984). In the earlier work, levels dropped below 2.0 ng/ml between 2 to 3 h after GnRH. Guthrie et al.(1984) observed that LH levels took longer to fall to 2.0 ng/ml in 160 versus 120 d old gilts.

SUMMARY(EXPERIMENT_2)

1. Mean plasma FSH levels did not differ significantly over the age range studied.

2. Luteinizing hormone levels and patterns were within normal ranges reported in the literature.

3. Mean LH concentration, frequency and amplitude of LH pulses and magnitude of GnRH-induced LH surge were non-significantly higher at 110 than 140 or 170 d.

4. The anterior pituitary is capable of responding to a GnRH stimulus resulting in a surge resembling a preovulatory one.

GENERAL DISCUSSION
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Results of Experiment 1 indicate that puberty can be reliably induced in H Group gilts(≥160 d age, ≥75 kg body weight) by treatment with PMSG/hCG. Furthermore, these gilts can be bred at second estrus and produce acceptable litter size up to 34 d of gestation(half of these gilts were carrying ten embryos).

Producers have long had a reluctance to mate gilts early for fear of affecting later performance. Research over the last decade has shown that these suppositions are groundless. Brooks and Smith(1980) mated two groups of gilts at second estrus, one of which was induced into early puberty by boar exposure. At the end of five parities there was no difference in: the number of piglets, the piglets were just as viable, the gilts were as good at feeding and rearing their young, the same number of gilts on test finished the experiment as did control gilts, weight differences disappeared by the end of the second pregnancy, feed efficiency was increased(6.2% less food consumed/unit weaner live weight over five litters) and the test gilts consumed 159±38 kg less feed than the control gilts.

As a result of the present work, no firm evidence emerged as to what is happening to the gilts in the 160 d age range. No overt changes were evident in characteristics measured in Experiment 2, and no P_4 profile differences

between 'mature' and 'immature' gilts were apparent in Experiment 1 at the induced estrous cycle. It was impossible to predict which gilts would respond to the PMSG/hCG treatment with puberty.

The ovary is capable of reacting to hormonal stimuli with E_2 production and ovulation. Likewise, the anterior pituitary responds to GnRH with LH surges that mimic the preovulatory surge. Thus , it would seem that final maturation before puberty takes place in the hypothalamus. Cox and Britt(1982b) pulsed GnRH into lactating anestrous sows and succeeded in provoking estrus and ovulation. In 1985, Carpenter and Anderson, and Lutz et al. used similar techniques to obtain estrus and ovulation in prepuberal gilts. Frequency and dose of GnRH infusion were found to be important factors in deciding the proportion of gilts which responded to the treatments. Researchers achieved precocious ovulation, as in Experiment 1, but not precocious puberty. The pituitary and the ovaries were found to be competent and researchers concluded that final maturation must take place at the hypothalamic level. In a similar fashion, Reiter and Grumbach(1982) concluded that both experimental and clinical studies support the hypothesis that the CNS, not the pituitary or the ovary, restrains the activation of the rest of the H-AP-O axis in prepuberal children. This restraint is apparently mediated through the suppression on GnRH synthesis and its pulsatile secretion.

CONCLUSIONS AND AGRICULTURAL IMPLICATIONS

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Puberty induction has the potential to increase reproductive and economic efficiency by: reducing the length of the unproductive period, hastening puberty, allowing early breeding, reducing the generation interval, and increasing the number of piglets/sow/year. When used as a management tool, PMSG/hCG could provide the producer with alternatives when: sufficient boar power is unavailable, facilities are inadequate to allow relocation and/or boar exposure, there is not enough manpower and because of its ease of application. Ovulation occurs at a predicted time after hCG injection allowing synchronization of gilts for A.I. use or batch farrowing. Both drugs are available through a veterinarian at an economical cost(total for both injections approximately $6.00 Cdn.).

Future research should be centred on gilts approximately 160 d of age since the majority respond to hormonal stimulation at this time. It would be very useful to do an experimental comparison of PMSG/hCG stimulation versus boar exposure versus the combination of the two stimuli. The use of PMSG/hCG might also prove to be effective in overcoming the seasonal influence on reproductive function.

Pulsatility studies on prepuberal gilts are hampered by the inability to predict when puberty will occur in the absence of stimuli. One way to overcome this problem is the

use of littermates which have been shown to reach puberty within a few days of each other when raised together. They are much more likely to be the same physiological age than a random selection of gilts.

Proper maturity of the entire H-AP-O axis, stimuli and receptivity must all be in place before puberty can occur. From the present research, it appears that though gilts can respond to induction stimuli at very young ages, the majority will only attain full sexual function reliably when stimulated near 160 d of age. It would appear that animals stimulated younger than 160 d give less satisfactory results to PMSG/hCG stimulation(poor maintenance of cyclicity). However, waiting for natural puberty around 200 d of age without using the stimulatory techniques available to any producer, seems a waste of good opportunity and poor economics.

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APPENDIX TABLES
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APPENDIX TABLE 1. EXPERIMENT 1 TIMETABLE.

REPLICATE	STARTING DATE	SLAUGHTER DATE
1	Oct. 19, 1984	Dec. 21, 1984
2	Jan. 11, 1985	Mar. 15, 1985
3	Mar. 22, 1985	May 24, 1985
4	May 31, 1985	Aug. 2, 1985

APPENDIX TABLE 2. GILT AGE AND WEIGHT DATA ON d 0 OF EXPERIMENT 1.

GILT	GROUP	REPLICATE	BIRTHDATE	AGE(d)	WEIGHT(kg)
1	L	1	June 28, 1984	113	52.7
2	L	1	June 27, 1984	114	49.1
3	L	1	July 4, 1984	107	51.3
4	L	1	July 9, 1984	102	52.7
1	M	1	June 8, 1984	133	64.3
2	M	1	June 6, 1984	135	62.5
3	M	1	June 7, 1984	134	65.2
4	M	1	May 22, 1984	150	67.0
1	H	1	May 10, 1984	162	82.1
2	H	1	May 10, 1984	162	77.7
3	H	1	May 3, 1984	169	78.6
4	H	1	Mar. 24, 1984	209	80.4
1	L	2	Oct. 10, 1984	93	46.4
3	L	2	Oct. 3, 1984	100	49.6
4	L	2	Sept.19, 1984	114	50.0
1	M	2	Sept. 6, 1984	127	58.0
2	M	2	Sept. 5, 1984	128	58.9
3	M	2	Aug. 28, 1984	136	58.9
4	M	2	Aug. 23, 1984	141	64.3
1	H	2	July 11, 1984	184	75.9
2	H	2	July 18, 1984	177	79.5
3	H	2	July 11, 1984	184	79.5
4	H	2	July 20, 1984	175	76.8
1	L	3	Nov. 23, 1984	119	53.1
2	L	3	Nov. 26, 1984	116	51.8
3	L	3	Dec. 7, 1984	105	46.4
4	L	3	Dec. 5, 1984	107	52.2
1	M	3	Oct. 31, 1984	142	63.4
2	M	3	Nov. 11, 1984	131	68.8
3	M	3	Oct. 23, 1984	150	64.3
4	M	3	Nov. 8, 1984	134	64.3
1*	H	3	Oct. 13, 1984	160	82.1
2	H	3	Oct. 10, 1984	163	76.8
3	H	3	Oct. 11, 1984	162	71.4
4*	H	3	Oct. 13, 1984	160	83.0
1	L	4	Mar. 8, 1985	84	51.8
2	L	4	Feb. 22, 1985	98	50.0
3	L	4	Feb. 13, 1985	107	45.5
4	L	4	Feb. 4, 1985	116	47.3
1	M	4	Jan. 7, 1985	144	65.2
2	M	4	Jan. 16, 1985	135	65.2
3	M	4	Jan. 19, 1985	132	59.8
4	M	4	Jan. 6, 1985	145	67.9
1	H	4	Dec. 19, 1984	163	83.0
2	H	4	Dec. 12, 1984	170	75.0
4	H	4	Dec. 15, 1984	167	75.0

* Littermates.

APPENDIX TABLE 3. SERUM PROGESTERONE LEVELS (ng/ml) OF GILTS IN EXPERIMENT 1.

BLEED	DAY OF CYCLE*	GILT (REPLICATE 1, GROUP L) 1	2	3	4
1	-4	-†	-	-	1.0
2	-1	.4	.4	-	.75
3	1	.6	16.0	1.5	1.1
4	3	6.0	30.5	16.5	16.0
5	6	13.75	30.75	20.0	16.75
6	8	18.25	28.75	25.5	18.0
7	10	24.0	36.5	26.5	21.5
8	13	23.0	31.5	28.0	32.0
9	15	5.5	7.25	25.0	19.0
10	17	-	-	14.0	1.75
11	20	-	-	-	-
12	1	-	-	-	-
13	3	-	-	1.5	-
14	6	-	-	-	4.5
15	8	-	-	5.0	11.0
16	10	-	-	6.5	15.5
17	13	-	-	10.75	16.5
18	15	-	-	15.25	24.5
19	17	-	-	19.75	23.5
20	20	-	-	13.5	20.75
21	1	-	-	16.5	16.0
22	3	-	-	20.0	14.0
23	6	-	-	15.0	20.0
24	8	-	-	18.0	23.5
25	10	-	-	16.0	15.0
26	13			12.5	18.25

* Based on an estrous cycle length of 21 d where D 0 is the first d of estrus.

† Below the limit of detection for this assay.

TABLE 3 (cont'd).

		GILT (REPLICATE 1, GROUP M)			
BLEED	DAY OF CYCLE	1	2	3	4
1	-4	.8	-	.4	-
2	-1	.55	-	-	-
3	1	2.0	.2	2.0	1.25
4	3	12.0	6.0	12.25	10.5
5	6	24.5	4.3	20.0	29.5
6	8	23.5	7.5	19.0	20.0
7	10	21.5	5.35	22.5	20.0
8	13	21.0	6.0	27.25	29.5
9	15	18.25	7.25	21.0	23.0
10	17	1.3	4.75	22.25	17.0
11	20	-	-	1.4	-
12	1	-	-	-	-
13	3	-	-	1.0	.15
14	6	-	3.0	-	4.4
15	8	-	4.75	-	6.25
16	10	.5	3.5	.4	12.0
17	13	.4	10.0	-	13.25
18	15	-	7.5	-	18.25
19	17	-	6.5	-	16.25
20	20	-	-	-	14.75
21	1	-	-	-	12.0
22	3	.5	-	-	11.0
23	6	-	1.5	.4	13.5
24	8	-	5.75	-	15.0
25	10		7.5	.4	13.25

TABLE 3 (cont'd).

		GILT (REPLICATE 1, GROUP H)			
BLEED	DAY OF CYCLE	1	2	3	4
1	-4	2.0	-	-	-
2	-1	.5	-	-	-
3	1	1.3	.4	2.75	4.25
4	3	3.5	17.0	10.25	13.5
5	6	10.5	32.0	19.0	17.5
6	8	13.0	27.0	19.0	17.5
7	10			22.0	19.0
8	13	16.25	37.0	16.0	25.0
9	15	16.5	29.25	12.0	29.5
10	17	5.0	6.25	6.5	4.75
11	20	-	-	1.0	-
12	1	-	-	1.5	-
13	3	2.0	1.75	-	3.5
14	6	11.5	9.5	6.25	8.5
15	8	13.0	13.0	7.75	12.5
16	10	15.75	13.75	9.25	8.0
17	13		20.75	10.0	12.5
18	15	16.25	17.75	21.0	15.5
19	17		21.75	8.25	15.0
20	20	19.0	20.75	12.0	14.25
21	1	16.5	14.0	2.25	16.5
22	3	19.25	20.0	-	9.25
23	6	18.0	15.5	1.0	17.0
24	8	14.0	20.25	1.0	7.0
25	10	20.0	16.0	1.0	

TABLE 3 (cont'd).

BLEED	DAY OF CYCLE	GILT (REPLICATE 2, GROUP L)		
		1	3	4
1	-4	-	-	-
2	-1	-	3.0	3.25
3	1	2.5	-	3.25
4	3	8.5	2.5	16.5
5	6	23.0	9.0	22.5
6	8	21.0	3.0	10.5
7	10	19.5	19.5	19.0
8	13	22.5	18.5	22.5
9	15	6.75	14.5	17.5
10	17	2.25	10.0	-
11	20	2.25	-	-
12	1	1.75	-	-
13	3	2.5	-	-
14	6	1.25	-	-
15	9	-	-	-
16	10	-	-	-
17	13	-	-	-
18	15	-	-	-
19	17	-	-	-
20	20	2.5	2.5	-
21	1	-	-	-
22	3	-	-	-
23	6	-	-	-
24	8	3.5	3.0	-

TABLE 3 (cont'd).

		GILT (REPLICATE 2, GROUP M)			
BLEED	DAY OF CYCLE	1	2	3	4
1	-4	-	1.0	-	-
2	-1	-	2.0	-	1.25
3	1	-	5.5	-	2.75
4	3	5.0	9.5	1.25	19.0
5	6	5.0	9.5	2.25	30.5
6	8	2.75	9.5	5.0	25.0
7	10	7.0	17.5	7.0	38.5
8	13	6.5	9.5	12.5	35.5
9	15	7.0	8.25	8.0	33.5
10	17	7.0	1.0	11.0	28.5
11	20	-	-	-	-
12	1	-	-	-	1.25
13	3	-	-	-	1.25
14	6	-	-	2.1	2.0
15	8	-	-	-	6.25
16	10	-	-	3.75	7.0
17	13	-	-	-	
18	15	-	-	-	21.0
19	17	-	-	2.0	15.5
20	20	-	-	1.0	15.5
21	1	-	-	2.0	19.0
22	3	-	1.5	1.5	
23	6	-	1.0	-	13.5
24	8	-	1.0	-	13.75
25	10		1.75		

TABLE 3 (cont'd).

BLEED	DAY OF CYCLE	GILT (REPLICATE 2, GROUP H) 1	2	3	4
1	-4	-	-	-	-
2	-1	8.0	-	-	-
3	1	8.75	-	1.25	2.25
4	3	14.75	9.5	13.25	18.0
5	6	18.75	16.0	17.5	31.5
6	8	18.75	29.0	26.0	30.5
7	10	21.0	25.5	17.5	34.5
8	13	1.5	22.5	23.75	30.5
9	15	-	18.0	24.0	17.5
10	17	-	1.5	20.5	-
11	20	1.25	-	-	-
12	1	6.0	-	-	1.0
13	3	12.0	1.0	-	1.0
14	6	10.75	5.5	1.0	-
15	8	19.0	11.5	1.25	-
16	10	18.5	13.25	1.25	1.5
17	13	24.5	17.0	1.0	-
18	15	16.5	15.0	7.75	-
19	17	15.0	5.5	12.25	-
20	20	13.0	-	13.25	1.0
21	1	11.0	-	-	-
22	3	11.5	-	-	-
23	6	13.0	7.0	-	1.75
24	8	14.25	8.75	3.75	1.0

TABLE 3 (cont'd).

BLEED	DAY OF CYCLE	GILT (REPLICATE 3, GROUP L) 1	2	3	4
1	-4	-	1.25	3.75	1.25
2	-1	.75	-	-	2.0
3	1	1.0	1.5	1.0	6.0
4	3	15.5	5.0	14.0	25.5
5	6	16.5	11.0	26.5	39.5
6	8	15.5	8.5	24.0	42.5
7	10	18.5	13.75	27.5	42.0
8	13	24.0	15.5	30.0	45.5
9	15	23.5	12.5	28.0	30.0
10	17	11.25	13.25	12.5	39.5
11	20	-	-	-	2.25
12	1	-	-	-	2.0
13	3	-	.9	-	1.25
14	6	-	-	3.25	-
15	8	-	-	6.5	1.0
16	10	-	-	8.5	1.75
17	13	-	-	18.5	-
18	15	-	-	21.0	1.0
19	17	-	-	19.5	1.25
20	20	-	.9	5.75	-
21	1	-	-	-	2.25
22	3	-	.9	-	1.5
23	6	1.0	-	1.6	1.5
24	8	11.5	-	7.5	-
25	10	9.0	.75	20.5	-
26	13	10.0		20.5	

TABLE 3 (cont'd).

BLEED	DAY OF CYCLE	GILT (REPLICATE 3, GROUP M) 1	2	3	4
1	-4	3.0	.9	1.25	1.0
2	-1	2.25	1.0	-	1.25
3	1	3.25	-	1.75	2.0
4	3	14.5	2.25	8.25	2.0
5	6	37.0	10.0	10.25	28.0
6	8	29.0	25.0	10.0	39.5
7	10	41.5	33.5	11.25	18.5
8	13	33.5	25.0	13.25	23.0
9	15	7.5	30.0	16.5	30.0
10	17	1.0	19.75	11.5	18.0
11	20	2.0	14.5	1.0	2.5
12	1	-	2.0	-	2.75
13	3	.95	-	-	1.25
14	6	.95	-	-	1.25
15	8	-	2.1	-	2.75
16	10	-	1.5	-	1.75
17	13	-	1.5	-	1.5
18	15	-	-	-	1.5
19	17	1.75	-	-	1.25
20	20	1.25	1.0	-	1.25
21	1	-	-	-	1.0
22	3	-	1.0	-	1.75
23	6	-	1.0	-	1.0
24	8	-	-	-	1.0
25	10	-	-	-	1.0

TABLE 3 (cont'd).

BLEED	DAY OF CYCLE	GILT (REPLICATE 3, GROUP H) 1	2	3	4
1	-4	-	-	-	-
2	-1	3.5	-	-	-
3	1	1.0	1.75	2.25	5.0
4	3	4.0	21.0	13.0	11.25
5	6	16.5	30.0	19.5	26.0
6	8	33.5	34.0	20.5	22.0
7	10	31.5	46.0	29.0	43.0
8	13	31.5	44.0	27.5	39.0
9	15	20.0	36.5	22.0	21.0
10	17	25.0	16.5	22.5	23.0
11	20	7.25	-	2.0	1.25
12	1	-	-	1.5	-
13	3	-	-	2.0	-
14	6	3.75	5.0	3.5	4.75
15	8	6.5	9.5	8.75	9.25
16	10	3.75	14.75	11.5	10.75
17	13		14.5	11.75	14.5
18	15	8.5	12.25	14.0	15.0
19	17	4.0	14.5	19.0	14.5
20	20	5.25	-	13.25	8.75
21	1	1.25	-	1.75	13.25
22	3	5.0	-	1.25	16.5
23	6	-	5.75	2.0	11.5
24	8	-	11.5	10.75	9.5
25	10	-			13.0
26	13				12.0

TABLE 3 (cont'd).

		GILT (REPLICATE 4, GROUP L)			
BLEED	DAY OF CYCLE	1	2	3	4
1	-4	-	1.0	1.25	1.25
2	-1	-	-	-	-
3	1	-	3.0	2.5	3.25
4	3	7.0	7.5	12.0	12.0
5	6	9.5	15.0	21.5	18.5
6	8	15.5	17.5	23.5	23.0
7	10	18.0	16.0	29.0	16.0
8	13	14.75	14.75	20.5	23.5
9	15	10.5	9.0	15.25	22.0
10	17	3.55	-	17.5	16.0
11	20	-	-	-	3.25
12	1	-	-	2.0	2.0
13	3	-	-	1.0	3.25
14	6	-		2.0	3.75
15	8	-	1.25	1.0	2.5
16	10	1.0	-	1.0	3.0
17	13	1.0	1.5	-	-
18	15	1.25	-	-	1.25
19	17	-	1.25	1.0	1.25
20	20	-	.9	1.25	2.75
21	1	1.5	1.0	-	2.5
22	3	-	-	1.25	2.25
23	6	-	-	-	-
24	8	-	-	-	2.0
25	10		-	1.0	

TABLE 3 (cont'd).

		GILT (REPLICATE 4, GROUP M)			
BLEED	DAY OF CYCLE	1	2	3	4
1	-4	2.25	-	-	-
2	-1	1.25	-	-	.35
3	1	2.75	-	.35	.35
4	3	16.5	6.0	3.5	7.5
5	6	25.0	20.5	9.5	18.0
6	8	27.0	16.0	9.0	20.5
7	10	42.0	8.75	13.0	16.5
8	13	44.5	17.0	13.5	15.5
9	15	12.0	17.0	15.5	15.0
10	17	6.25	17.0	1.25	2.25
11	20	2.25	3.25	-	-
12	1	3.75	1.25	-	.35
13	3	3.0	2.75	-	.5
14	6	-	2.0	7.0	9.5
15	8	1.5	2.0	22.5	18.5
16	10	-	2.5	11.75	24.5
17	13	-	-	16.75	16.5
18	15	-	-	11.5	16.5
19	17	-	1.25	20.5	3.0
20	20	-	1.0	20.0	.35
21	1	-	1.25	13.0	.35
22	3	-	11.75	14.5	3.25
23	6	-	11.75	13.75	11.25
24	8	3.0	11.75	16.5	15.25
25	10	1.5	11.75	17.5	10.25

TABLE 3 (cont'd).

		GILT (REPLICATE 4, GROUP H)		
BLEED	DAY OF CYCLE	1	2	4
1	-4	.5	.35	.4
2	-1	1.5	.5	.9
3	1	2.0	.6	1.0
4	3	22.0	4.25	2.0
5	6	22.0	11.0	12.5
6	8	15.75	12.5	14.5
7	10	24.0	13.5	20.0
8	13	24.0	17.0	23.0
9	15	19.0	12.5	28.5
10	17	11.25	11.0	23.0
11	20	1.25	1.0	3.75
12	1	–	1.0	–
13	3	.35	.35	1.0
14	6	6.5	.5	.25
15	8	8.5	5.0	3.5
16	10	5.5	4.0	5.5
17	13	6.25	7.25	7.0
18	15	12.0	8.5	10.5
19	17	14.0	13.0	14.5
20	20	11.0	7.5	26.0
21	1	12.0	2.75	16.5
22	3	11.25	.5	9.5
23	6	9.0	1.0	1.0
24	8	11.5	2.75	.6

APPENDIX TABLE 4. PROGESTERONE OUTPUT OF GILTS AT FIRST AND SECOND ESTROUS CYCLE IN EXPERIMENT 1.

GILT	GROUP	REPLICATE	PROGESTERONE[b] FIRST ESTRUS	SECOND ESTRUS
1	L	1	1059	
2	L	1	2096	
3	L	1	1812	
4	L	1	1494	
1	L	2	1230	
3	L	2	929	
4	L	2	1369	
1	L	3	1409	
2	L	3	951	
3	L	3	1926	976
4	L	3	3236	
1	L	4	938	
2	L	4	952	
3	L	4	1538	
4	L	4	1588	
1	M	1	1414	
2	M	1	469	412
3	M	1	1760	
4	M	1	1751	
1	M	2	478	
2	M	2	818	
3	M	2	569	
4	M	2	2458	
1	M	3	2022	
2	M	3	992	
3	M	3	1933	
4	M	3	1800	
1	M	4	2122	
2	M	4	1216	
3	M	4	744	
4	M	4	1062	972
1	H	1	898	
2	H	1	2085	
3	H	1	1271	890
4	H	1	1571	
1	H	2	1049	
2	H	2	1375	778
3	H	2	1628	388
4	H	2	1851	
1	H	3	1598	504
2	H	3	2666	798
3	H	3	1835	990
4	H	3	2203	
1	H	4	1675	
2	H	4	975	534
4	H	4	1507	1099

[b] Area under the curve measured in arbitrary units.

APPENDIX TABLE 5. PUBERTY AND BREEDING DATA FOR PUBERAL GILTS IN EXPERIMENT 1.

GILT/GROUP /REPLICATE	AGE AT PUBERTY(d)	WEIGHT AT PUBERTY(kg)	AGE AT BREEDING(d)	WEIGHT AT BREEDING(kg)
3 L 1	114	50.9	136	72.7
4 L 1	109	56.3	131	71.8
1 L 3	173	83.9		
3 L 3	112	49.1	133	61.8
1 M 1	189	102.7		
2 M 1	142	66.1	162	84.5
4 M 1	157	67.9	177	85.5
4 M 2	143	67.9	166	91.8
1 M 3	201	100.0		
2 M 4	184	95.5		
3 M 4	139	65.2	158	80.0
4 M 4	152	73.2	170	94.5
1 H 1	169	83.0	188	103.6
2 H 1	169	78.6	189	96.4
3 H 1	176	78.6	196	90.9
4 H 1	214	74.1	236	88.2
1 H 2	187	81.3	209	95.5
2 H 2	184	80.4	202	103.2
3 H 2	224	108.9		
1 H 3	167	81.3	185	105.5
2 H 3	170	77.7	188	104.5
3 H 3	169	75.9	187	100.0
4 H 3	165	83.9	187	104.5
1 H 4	170	85.7	188	102.7
2 H 4	177	76.8	196	94.5
4 H 4	174	76.8	192	95.5

APPENDIX TABLE 6. GILT REPRODUCTIVE TRACT MEASUREMENTS FOR EXPERIMENT 1.

GILT/GROUP /REPLICATE	TOTAL OVARIAN WEIGHT(g)	TOTAL UTERINE WEIGHT(g)	UTERINE HORN LENGTH(cm)	NUMBER of CL's
1 L 1	6.6	116	33	0
2 L 1	5.8	119	42	0
3 L 1	17.3	1342	91	11
4 L 1	16.2	1754	118	14
1 L 2	7.1	128	38	0
3 L 2	7.2	200	51	0
4 L 2	6.5	144	44	0
1 L 3	19.3	630	133	15
2 L 3	5.8	278	68	0
3 L 3	17.9	544	87	12
4 L 3	6.6	116	40	0
1 L 4	4.7	136	55	0
2 L 4	11.3	96	48	0
3 L 4	5.7	117	49	0
4 L 4	7.4	147	47	0
1 M 1	12.2	153	62	6
2 M 1	11.4	701	87	12
3 M 1	8.1	95[a]	59	0
4 M 1	12.1	1298	94	12
1 M 2	7.1	122	49	0
2 M 2	6.8	212	67	0
3 M 2	8.2	124	47	0
4 M 2	13.7	1744	89	13
1 M 3	8.2	316	78	13
2 M 3	8.0	145	48	0
3 M 3	6.4	149	57	0
4 M 3	11.0	437	61	0
1 M 4	5.3	116	52	0
2 M 4	10.4	634	81	11
3 M 4	13.5	1304	105	11
4 M 4	10.2	738	75	12
1 H 1	13.3	2333	110	15
2 H 1	13.4	2599	102	13
3 H 1	5.5	308	64	0
4 H 1	13.4	1330	110	10
1 H 2	12.3	2061	100	15
2 H 2	12.8	490	80	12
3 H 2	13.0	556	105	12
4 H 2	9.1	283	62	0
1 H 3	11.3	545	120	15
2 H 3	16.5	604	105	12
3 H 3	13.1	510	102	14
4 H 3	13.2	1375	141	10
1 H 4	13.3	1358	110	10
2 H 4	14.2	648	118	12
4 H 4	15.0	544	90	12

[a] One uterine horn missing.

APPENDIX TABLE 7. PLASMA ESTRONE SULPHATE LEVELS (ng/ml) OF GILTS IN EXPERIMENT 1.

GILT/GROUP /REPLICATE	DAY OF GESTATION[a]										
	18	19	20	21	22	23	24	25	26	27	28	29	30
1 L 1			.04		.06			.11		.07		.14	
3 L 1	.09		.18			.37		.56		.85			.65
4 L 1	.27		.46			2.06		4.29		2.44			.87
1 M 1								.13			.09		.13
2 M 1			.14					.12		.09		.13	
3 M 1										.13		.17	
4 M 1								.92		2.07			
1 H 1			.15		.95			3.93		4.34		3.71	
2 H 1			.99		2.93			4.89		5.15		2.85	
3 H 1			.04		.02			.12		.17		.15	
4 H 1			.27		.23			1.32				2.43	
1 L 2			.13		.11		.11			.11		.13	
3 L 2			.12		.13		.15			.10		.17	
4 L 2		.19		.12			.12		.12		.14		
1 M 2			.06		.04		.08			.07		.07	
2 M 2	.13		.06		.09			.09		.06		.05	
3 M 2			.15		.21		.15			.17		.14	
4 M 2			.13		.31		.55			2.67		3.99	
1 H 2			1.73		2.78		3.18			2.00		.60	
2 H 2			.09		.18		.08			.17		.14	
3 H 2			.19		.19		.19			.17		.17	
4 H 2			.15		.15		.17			.15		.19	

[a] Based on the last d of insemination.

TABLE 7 (cont'd)

GILT/GROUP /REPLICATE	DAY OF GESTATION											
	18	19	20	21	22	23	24	25	26	27	28	29	30
1_L_3	.08		.10		.14			.14		.11		.08	
2_L_3		.17		.12			.23		.15		.20		
3_L_3		.12		.12			.16		.14		.12		
4_L_3		.13		.11			.11		.15		.14		
1_M_3		.15		.17			.12		.11		.12		
2_M_3		.19		.09			.14		.13		.16		
3_M_3		.19		.20			.15		.21		.21		.13
4_M_3	.14		.21		.16			.16		.13		.20	
1_H_3			.22		.22		.29			.18		.28	
2_H_3			.25		.32		.40			.24		.26	
3_H_3			.27		.35		.25			.31		.27	
4_H_3	.30		.39		.93		4.15			6.85		5.78	
1_L_4		.30		.21		.17			.18		.21		
2_L_4		.23		.22		.21			.27		.28		
3_L_4		.10		.09		.10			.15		.10		
4_L_4	.07		.08		.10		.10			.14		.13	
1_M_4	.06		.06		.04		.04			.04		.04	
2_M_4		.05		.05		.06			.04		.04		
3_M_4	.10		.17		.42			1.23		1.26		1.38	
4_M_4		.17		.13		.12			.11		.15		
1_H_4		.15		.31		.43			1.46		1.47		
2_H_4	.12		.06		.12			.19		.16		.18	
4_H_4		.03		.07		.09			.08		.09		.06

APPENDIX TABLE 8. DAY OF LAST BREEDING FOR PREGNANT GILTS IN EXPERIMENT 1.

GILT	GROUP	REPLICATE	DAY OF LAST BREEDING[a]
3	1	1	25
4	1	1	25
4	2	1	27
4	2	2	25
3	2	4	26
1	3	1	27
2	3	1	27
4	3	1	27
1	3	2	25
4	3	3	27
1	3	4	25

[a] Day of the experiment.

APPENDIX TABLE 9. REPRODUCTIVE DATA OF PREGNANT GILTS IN EXPERIMENT 1.

GILT/GROUP /REPLICATE	NUMBER OF CL'S	NUMBER_OF_EMBRYOS[h] TOTAL	DEAD	ALIVE	MAXIMUM E_1SO_4 (ng/ml)	P_4[j]
3 L 1	11	5	2	3	.85	1884
4 L 1	14	14	5	9	4.29	2520
4 M 1	12	4	1	3	2.07	1600
4 M 2	13	12	6	6	3.99	1573
3 M 4	11	5	0	5	1.38	1946
1 H 1	15	10	0	10	4.34	2203
2 H 1	13	10	0	10	5.15	2262
4 H 1	10	4	0	4	2.43	1539
1 H 2	15	11	5	6	3.18	2096
4 H 3	10	10	0	10	6.85	1384
1 H 4	10	6	0	6	1.47	1156

[h] Determined at least 34 d after the last breeding.

[j] Area under the curve measured in arbitrary units.

APPENDIX TABLE 10. EMBRYONIC DATA.

GILT/GROUP REPLICATE	LIVE EMBRYO WEIGHT(g) HORN I	HORN II	TOTAL	PARTICULARS
3 L 1	3.1	2.7 3.5	9.3	HORN I - 2 REABSORBING
4 L 1	3.8 3.2 4.4 4.6 2.7	4.5 4.0 4.4 4.5	36.1	HORN I - 3 REABSORBING HORN II - 1 DEAD (2.7g) - 1 REABSORBING
4 L 1	4.5 3.4 4.0		11.9	HORN II - 1 DEAD (4.1g)
1 L 1	4.8 5.6 5.9 5.2 4.9	6.0 5.6 5.6 6.2 5.9	56.2	
2 H 1	6.7 7.2 6.8 5.6 7.0	6.8 6.7 5.8 6.6 7.2	66.4	
4 H 1	5.7 6.0	5.3 5.4	22.4	
4 M 2	3.9 2.9	3.6 3.7 3.4 4.2	21.7	HORN I - 2 REABSORBING HORN II - 4 REABSORBING
1 H 2	11.1 12.9 12.3	11.8 10.3 12.4	61.8	HORN I - 1 REABSORBING HORN II - 4 REABSORBING
4 H 3	5.0 5.1 4.6 4.7	4.7 3.5 5.1 3.8 5.0 5.2	46.7	

TABLE 10 (cont'd).

GILT/GROUP REPLICATE		REPLICATE	LIVE EMBRYO WEIGHT(g) HORN I	HORN II	TOTAL	PARTICULARS
3	M	4	8.0 8.5 9.2	9.6 8.9	44.2	
1	H	4	11.8 10.9 11.5 10.5	11.0 11.7	67.4	

APPENDIX TABLE 11. GILT WEIGHT (kg) THROUGHOUT EXPERIMENT 1.

	REPLICATE 1							
	GROUP							
	L				M			
WEIGH IN	GILT							
	1	2	3	4	1	2	3	4
W0[k]	52.7	49.1	51.3	52.7	64.3	62.5	65.2	67.0
W1	50.0	49.1	50.9	56.3	65.2	66.1	65.2	67.9
W2	58.0	51.8	59.8	59.8	74.1	73.2	73.2	75.0
W3	66.1	56.3	65.2	67.9	80.4	78.6	80.4	81.3
W4	68.8	60.7	71.4	70.5	83.0	83.0	84.8	83.9
W5	74.1	63.4	75.0	75.9	87.5	87.5	90.2	86.6
W6	75.9	65.2	82.1	82.1	92.9	92.0	91.1	92.9
W7	81.3	67.9	87.5	87.5	95.5	94.6	98.2	99.1
W8	85.7	73.2	92.9	92.0	102.7	97.3	105.4	101.8
W9	91.1	75.9	99.1	100.0	106.3	106.3	112.5	107.1
WG[m]	38.4	26.8	47.8	47.3	42.0	43.8	47.3	40.2

[k] Pretest weight (1 d before PMSG).

[m] Total weight gain during the experiment.

TABLE 11 (cont'd).

	REPLICATE						
	1				2		
	GROUP						
	H				L		
WEIGH IN	GILT 1	2	3	4	1	3	4
W0	82.1	77.7	78.6	80.4	46.4	49.6	50.0
W1	83.0	78.6	78.6	74.1	47.3	51.8	51.8
W2	90.2	87.5	82.1	77.7	51.8	56.3	55.4
W3	97.3	90.2	86.6	81.3	58.0	63.4	63.4
W4	101.8	94.6	89.3	86.6	61.6	63.4	69.6
W5	104.5	98.2	92.0	86.6	67.9	69.6	75.0
W6	113.4	104.5	97.3	92.0	72.3	72.3	78.6
W7	112.5	105.4	102.7	92.9	80.4	78.6	84.8
W8	121.4	110.7	99.1	99.1	85.7	83.9	91.1
W9	126.8	113.4	100.0	104.5	91.1	91.1	95.5
WG	44.6	35.7	21.4	24.1	44.6	41.5	45.5

TABLE 11 (cont'd).

	REPLICATE 2							
	GROUP							
	M				H			
WEIGH IN	GILT							
	1	2	3	4	1	2	3	4
WO	58.0	58.9	58.9	64.3	75.9	79.5	79.5	76.8
W1	60.7	58.9	59.8	67.9	81.3	80.4	81.3	80.4
W2	63.4	64.3	64.3	74.1	85.7	89.3	89.3	86.6
W3	71.4	71.4	72.3	83.0	89.3	100.0	97.3	95.5
W4	73.2	75.9	77.7	90.2	93.8	101.8	100.0	97.3
W5	77.7	74.1	80.4	92.9	97.3	107.1	100.0	100.9
W6	83.0	81.3	85.7	102.7	100.0	117.0	108.9	104.5
W7	92.0	87.5	92.0	110.7	105.4	118.8	117.0	108.9
W8	97.3	91.1	98.2	111.6	110.7	125.9	117.9	117.0
W9	102.7	99.1	102.7	121.4	111.6	133.9	124.1	121.4
WG	44.6	40.2	43.8	52.1	35.7	54.5	44.6	44.6

TABLE 11 (cont'd).

	REPLICATE 3							
	GROUP							
	L				M			
WEIGH IN	GILT							
	1	2	3	4	1	2	3	4
W0	53.1	51.8	46.4	52.2	63.4	68.8	64.3	64.3
W1	54.5	52.7	49.1	56.3	65.2	71.4	65.2	65.2
W2	58.9	59.8	54.5	60.7	72.3	78.6	73.2	74.1
W3	64.3	63.4	58.0	65.2	77.7	85.7	86.6	77.7
W4	69.6	68.8	60.7	70.5	81.3	91.1	84.8	83.9
W5	70.5	71.4	64.3	72.3	84.8	94.6	88.4	86.8
W6	73.2	73.2	68.8	75.0	88.4	100.0	91.1	89.3
W7	78.6	78.6	75.0	79.5	94.6	103.6	94.6	92.9
W8	83.9	84.8	77.7	83.9	100.0	108.9	101.8	97.3
W9	89.3	87.5	83.9	79.5	100.0	113.4	102.7	100.9
WG	36.2	35.7	37.5	27.2	36.6	44.6	38.4	36.6

TABLE 11 (cont'd).

	REPLICATE							
	3				4			
	GROUP							
	H				L			
WEIGH IN	GILT							
	1	2	3	4	1	2	3	4
W0	82.1	76.8	71.4	83.0	51.8	50.0	45.5	47.3
W1	81.3	77.7	75.9	83.9	58.0	57.1	53.6	52.7
W2	92.9	87.5	83.9	92.0	65.2	61.6	61.6	58.0
W3	100.0	94.6	92.9	100.0	70.5	67.9	67.9	64.3
W4	103.6	102.7	98.2	102.7	74.1	69.6	70.5	67.0
W5	106.3	99.1	100.9	104.5	76.8	74.1	73.2	69.6
W6	112.5	108.0	108.0	113.4	82.1	78.6	79.5	76.8
W7	118.8	110.7	115.2	119.6	85.7	83.0	83.9	79.5
W8	120.5	113.4	108.9	123.2	91.1	87.5	88.4	83.9
W9	121.4	120.5	118.8	128.6	97.3	94.6	96.4	91.1
WG	39.3	43.8	47.3	45.5	45.5	44.6	50.9	43.8

TABLE 11 (cont'd).

	REPLICATE 4						
	GROUP						
	M				H		
WEIGH IN	GILT 1	2	3	4	1	2	4
W0	65.2	65.2	59.8	67.9	83.0	75.0	75.0
W1	72.3	71.4	65.2	73.2	85.7	76.8	76.8
W2	80.4	79.5	73.2	83.0	93.8	85.7	85.7
W3	88.4	88.4	97.5	90.2	99.1	94.6	92.9
W4	91.1	88.4	78.6	92.9	100.9	92.9	93.8
W5	90.2	87.5	81.3	84.6	105.4	96.4	95.5
W6	98.2	94.6	90.2	104.5	115.2	106.3	103.6
W7	99.1	95.5	91.1	102.7	119.6	108.9	108.9
W8	100.0	102.7	98.2	111.6	128.6	104.5	103.6
W9	116.1	112.5	105.4	120.5	134.8	114.3	112.5
WG	50.9	47.3	45.5	52.7	51.8	39.3	37.5

APPENDIX TABLE 12. MEANS (±SEM) BASED ON GROUP FOR EXPERIMENT 1.

VARIABLE	GROUP L	M	H
n	15	16	15
AGE AT d 0 (d)	106.3±9.7	137.0±7.1	171.1±13.3
AGE AT PUBERTY(d)	127.0±30.7[p] 111.7±2.5[pp]	163.4±24.3[q] 146.6±7.6[qq]	179.6±17.9[r] 176.2±13.1[rr]
AGE AT BREEDING(d)	133.5±10.4	163.6±7.3	196.8±13.4
BODY WEIGHT AT d 0 (kg)	50.9±2.3	64.8±3.3	79.9±3.5
BODY WEIGHT AT PUBERTY(kg)	60.1±16.2[p] 52.1±3.7[pp]	79.9±16.5[q] 68.1±3.1[qq]	81.6±8.5[r] 79.5±3.4[rr]
TOTAL BODY WEIGHT GAIN(kg)	41.7±8.0	44.9±6.1	41.1±9.4
BODY WEIGHT AT BREEDING(kg)	69.0±4.3	85.5±5.9	99.1±5.3
PROGESTERONE*	1501±609	1350±641	1612±479

[p] n=4 [q] n=8 [r] n=14(at the end of the experiment).

[pp] n=3 [qq] n=5 [rr] n=13(within 10 d of PMSG injection).

* Area under the curve measured in arbitrary units at the induced estrous cycle.

APPENDIX TABLE 13. MEANS (±SEM) BASED ON REPRODUCTIVE STATUS FOR EXPERIMENT 1.

	REPRODUCTIVE STATUS			
VARIABLE	PREGNANT	CYCLIC AND/OR PREGNANT	CYCLIC AND NOT PREGNANT	PREPUBERAL
n	11	26	15	20
TOTAL OVARIAN WEIGHT(g)	13.8±1.6	13.2±2.9	12.7±3.6	7.2±1.7
UTERINE WEIGHT(g)	1681±463		528±159	168±84*
HORN LENGTH(cm)	106±15		94±18	51±9
PROGESTERONE[t]	1612±543	1542±517	1483±572	1411±658
AGE AT PUBERTY(d)		166.5±28.2		
WEIGHT AT PUBERTY(kg)		77.8±14.3		

* n=19

[t] Area under the curve measured in arbitrary units at the induced estrous cycle.

APPENDIX TABLE 14. KRUSKAL-WALLIS ANALYSES OF VARIANCE.

POPULATIONS	VARIABLE	DF	H	PROBABILITY
PREGNANT & CYCLIC NON-PREGNANT	UTERINE WEIGHT	1	18.37***	p<.001
	UTERINE HORN LENGTH	1	3.62	.1<p<.05
PREPUBERAL & CYCLIC NON-PREGNANT	UTERINE HORN LENGTH	1	23.20**	.001
	OVARIAN WEIGHT	1	16.78**	.001
	UTERINE WEIGHT	1	21.60	.001
CYCLIC NON-PREGNANT	AUC FOR P_4: 1 1st & 2nd CYCLE		9.68**	.01
ALL CYCLIC GILTS: REPLICATES 3 & 4	AUC FOR P_4: 1st CYCLE	1	6.58*	.02<p<.01
LITTER SIZE: 3-6 & 9-10	MAXIMUM E_1SO_4	1	7.04**	.01<p<.001
L & M	AGE AT PUBERTY	1	3.48	.1<p<.05
L & H		1	4.98	.05<p<.02
M & H		1	11.54**	p<.001
L & M	WEIGHT AT PUBERTY	1	3.48	.1<p<.05
L & H		1	3.08	.1<p<.05
M & H		1	1.30	.5<p<.3
ALL GILTS: REPLICATES	AUC FOR P_4: 1st CYCLE			
1 & 3		1	5.05*	.05<p<.02
2 & 3		1	9.43**	.01<p<.001
3 & 4		1	5.33*	.05<p<.02

* Significant at .05 level.
** Significant at .01 level.
*** Significant at .001 level.

APPENDIX TABLE 15. CHI-SQUARE TEST RESULTS.

GROUPS	CHARACTERISTIC	DF	X^2	PROBABILITY
L & M & H	INDUCED PUBERTY	2	19.16***	p<.001
M & H	"	1	13.12***	p<.001
L & H	"	1	19.55***	p<.001
L & M	"	1	.46	.7<p<.5
L & M & H	PUBERTY PRIOR TO SLAUGHTER	2	15.07***	p<.001
M & H	CYCLIC OR PREGNANT AT SLAUGHTER	1	9.32**	p<.01
L & H	"	1	14.75***	p<.001
L & M	"	1	1.95	.2<p<.1

** Significant at .01 level.

*** Significant at .001 level.

APPENDIX TABLE 16. CORRELATIONS OF VARIOUS OBSERVATIONS IN EXPERIMENTS 1 AND 2.

POPULATION	VARIABLES	r	p<R
PREGNANT	NO. LIVE EMBRYOS & E_1SO_4 DAY OF PREGNANCY): 22	.72	.0120
	23	.81	.0025
	24	.85	.0010
	25	.86	.0007
	26	.88	.0004
	27	.86	.0006
	NO. LIVE EMBRYOS & UTERINE HORN LENGTH	.61	.0447
	NO. LIVE EMBRYOS & TOTAL EMBRYONIC WEIGHT	.62	.0423
	NO. OF CL's & AUC OF P_4 (2nd CYCLE)	.76	.0071
	NO. OF CL's & MAX P_4 LEVEL (2nd CYCLE)	.80	.003
	MAX P_4 LEVEL & AUC OF P_4 (2nd CYCLE)	.77	.0055
	NO. OF CL's & NO. OF EMBRYOS	.67	.0229
NON-PREGNANT CYCLIC	UTERINE HORN LENGTH & UTERINE WEIGHT	.52	.0469
PREPUBERAL	UTERINE HORN LENGTH & UTERINE WEIGHT	.60	.0049
n=16 GILT SAMPLINGS	PRE-GnRH LH MEAN AND FREQUENCY	.8316	.0001

APPENDIX TABLE 17. PRE-GnRH SERUM LH LEVELS (ng/ml).

GILT 1 TIME (h)	BLEED A	B	C
09.00	.29	.21	.25
09.10	.27	.30	.27
09.20	.26		.22
09.30	.22	1.36	.21
09.40	1.79	1.05	.20
09.50		.95	.14
10.00	1.15	.58	.21
10.10	.67	.40	.18
10.20	.53	.33	.14
10.30	.58	.30	.16
10.40	.42	.30	.16
10.50	.38	.32	.16
11.00	.33	.29	.16
11.10	.29	.21	.14
11.20	1.90	.19	.19
11.30	2.31	.27	.16
11.40	1.54	1.24	.16
11.50	1.07	1.74	.18
12.00	.84	1.28	.19
12.10	.60	.83	.16
12.20	.55	.78	.17
12.30	.51	.54	.19
12.40	.38	.35	.18
12.50	.37	.30	1.30
13.00	.33	.30	1.54
13.10	.26	.30	1.13
13.20	1.78	.32	.75
13.30	1.22	.28	.70
13.40	.99	.30	.64
13.50	.91	.23	.44
14.00	.85	.24	.26
14.10	.64	.27	.30
14.20	.46	1.34	.30
14.30	.40	1.07	.30
14.40	2.17	.68	.26
14.50	2.20	.59	.21
15.00	1.08	.57	.25
15.10	1.02	.49	.26
15.20	.81	.41	.24
15.30	.58	.31	.18
15.40	.56	.33	.14
15.50	.50	.26	.12
16.00	.43	.27	.16
16.10	.34	.27	.15
16.20	.42	.26	.17
16.30	.31	.24	1.75
16.40	.28	.17	1.14
16.50	.31	.28	.80
17.00	2.03	.22	.69

TABLE 17 (cont'd).

GILT 2 TIME (h)	BLEED A	B	C
09.00	.18	.17	.59
09.10	.16	.19	.69
09.20	.17	.23	.34
09.30	.14	.29	.39
09.40	.17	.33	.41
09.50	.22	.26	.32
10.00	.23	.27	.32
10.10	.21	.21	.29
10.20	.14	.23	.26
10.30	.44	.16	.27
10.40	1.51	.26	.28
10.50	.82	.20	.21
11.00	.61	1.65	.21
11.10	.47	.94	2.56
11.20	.36	1.35	1.28
11.30	.33	.94	1.19
11.40	.31	.71	.78
11.50	.24	.64	.70
12.00	.21	.39	.59
12.10	.22	.41	.44
12.20	.23	.36	.46
12.30	.22	.34	.37
12.40	.20	.32	.31
12.50	.14	.29	.32
13.00	.24	.24	.23
13.10	.18	.24	.28
13.20	2.68	.22	.25
13.30	1.48	.59	.20
13.40	1.80	.30	.21
13.50	.89	.28	.18
14.00	.55	.35	2.53
14.10	.39	.38	1.60
14.20	.27	.33	1.27
14.30	.22	.29	.77
14.40	.19	.23	.55
14.50	.18	1.91	.42
15.00	.22	1.24	.37
15.10	.20	1.12	.49
15.20	.17	.67	.38
15.30	.13	.54	.26
15.40	.13	.55	.26
15.50	.20	.40	.25
16.00	.16	.39	.23
16.10	.17	.29	.27
16.20	.16	.29	.14
16.30	.12	.18	.19
16.40	.15	.19	.17
16.50	.15	.20	.17
17.00	.23	.21	.19

TABLE 17 (cont'd).

GILT 3 TIME (h)	BLEED A	B	C
09.00	.78	.37	.10
09.10	.54	.34	.13
09.20		.26	.12
09.30	.30	.21	.12
09.40	2.26	.20	.14
09.50	1.34	.17	2.90
10.00	.31	.16	1.80
10.10	.51	.20	1.15
10.20	.32	.17	.73
10.30	.18	.17	.43
10.40	.29	.13	.45
10.50	.26	.19	.31
11.00	.18	.13	.23
11.10	.21	.13	.26
11.20	.17	.11	.12
11.30	.20	.10	.19
11.40	.20	.12	.15
11.50	.18	.12	.18
12.00	2.99	.12	.14
12.10	1.30	.16	.17
12.20	.92	.11	.15
12.30	.72	.11	.15
12.40	.49	.13	.11
12.50	.34	.12	.13
13.00	.19	.12	.13
13.10	.18	.12	.14
13.20	.18	.13	.11
13.30	.18	.12	.15
13.40	.17	.08	.13
13.50	.19	.12	.10
14.00	1.89	.12	.10
14.10	1.14	.12	.12
14.20	.71	.11	.09
14.30	.68	1.54	.08
14.40	.21	1.02	.09
14.50	.16	.87	.09
15.00	.26	.65	.09
15.10	.18	.50	.12
15.20	.21	.33	1.85
15.30	.10	.25	1.16
15.40	.14	.24	.98
15.50	.18	.22	.39
16.00	.19	.21	.32
16.10	.18	.20	.31
16.20	.18	.20	.28
16.30	.14	.14	.17
16.40	.13	.14	.21
16.50	.15	.21	.20
17.00	.16	.14	.17

TABLE 17 (cont'd).

GILT 4 TIME (h)	BLEED A	B	C
09.00	1.18	.37	.12
09.10	.84	.27	.09
09.20	.87	.28	.09
09.30	.19	.23	.15
09.40	.34	.21	.11
09.50	.27	.17	.13
10.00	.20	.19	.13
10.10	.26	.16	.10
10.20	.19	.20	.14
10.30	.19	.14	.13
10.40	.19	.24	.15
10.50	.17	.13	.15
11.00	.13	.16	.13
11.10	.12	.19	.14
11.20	.12	1.36	.21
11.30	.10	.88	.20
11.40	.11	.82	.21
11.50	.11	.56	.23
12.00	.12	.30	.15
12.10	.09	.30	.99
12.20	.13	.27	.83
12.30	.10	.22	.75
12.40	.07	.21	.55
12.50	.09	.17	.36
13.00	.11	.20	.27
13.10	.09	.12	.23
13.20	.08	.12	.24
13.30	.10	.16	.15
13.40	.09	.17	.16
13.50	.08	.14	.16
14.00	.09	1.15	.14
14.10	.08	.88	.13
14.20	.08	.73	.13
14.30	.09	.57	.14
14.40	.09	.51	.14
14.50	.09	.38	.12
15.00	.10	.30	.17
15.10	.08	.26	.13
15.20	.12	.23	.17
15.30		.23	.16
15.40	.08	.14	.15
15.50	.10	.18	.13
16.00	.10		.12
16.10	.09	.18	.15
16.20	.08	.15	.14
16.30	.07	.16	.10
16.40	.08	.13	.18
16.50	.09	.14	.15
17.00	.08	.13	.18

TABLE 17 (cont'd).

GILT 5 TIME (h)	BLEED A	B	C
09.00	1.05	.55	.17
09.10	.59	.50	.10
09.20	.47	.41	.14
09.30	.33	.32	.22
09.40	.48	.37	.21
09.50	.24	.32	.19
10.00	.33	.28	.17
10.10	.29	.27	.16
10.20	.31	.27	.12
10.30	.27	.22	.16
10.40	.30	.30	.18
10.50	.22	.21	.23
11.00	.18	.19	.44
11.10	.17	.20	.44
11.20	.19	.19	.17
11.30	.15	.26	.19
11.40	.19	.19	.28
11.50	.13	.20	.55
12.00	.21	.15	.75
12.10	.20	.16	.50
12.20	.22	.19	.56
12.30	.23	.21	1.53
12.40	.14	.18	1.16
12.50	.15	.17	1.18
13.00	.14	.15	.81
13.10	.13	.25	.63
13.20	.19	.14	.38
13.30	.18	.17	.54
13.40	.17	.15	.49
13.50	.22	.20	.48
14.00	.25	.14	.25
14.10	3.92	.17	.30
14.20	1.96	.14	.31
14.30	1.58	.13	.25
14.40	.99	.12	.16
14.50	.89	.15	1.05
15.00	.60	.15	1.12
15.10	.58	.24	.85
15.20	.38	.21	.69
15.30	.33	.18	.63
15.40	.25	.18	.35
15.50	.22	.20	.39
16.00	.24	.17	.42
16.10	.22	.21	.33
16.20	.21	.17	.32
16.30	.14	.18	.34
16.40	.22	.21	.53
16.50	.27	.21	.21
17.00	.17	.21	.34

TABLE 17 (cont'd).

GILT 6	TIME (h)	BLEED A
	09.00	.24
	09.10	.27
	09.20	.23
	09.30	.22
	09.40	.19
	09.50	.20
	10.00	.20
	10.10	.20
	10.20	.26
	10.30	.34
	10.40	.14
	10.50	.19
	11.00	.22
	11.10	.25
	11.20	.32
	11.30	.34
	11.40	.28
	11.50	.29
	12.00	.33
	12.10	.32
	12.20	2.02
	12.30	3.15
	12.40	2.57
	12.50	.76
	13.00	.61
	13.10	.44
	13.20	.23
	13.30	.19
	13.40	.21
	13.50	.21
	14.00	.20
	14.10	.18
	14.20	.13
	14.30	.15
	14.40	.09
	14.50	.14
	15.00	.12
	15.10	.11
	15.20	.09
	15.30	.10
	15.40	.14
	15.50	.10
	16.00	.11
	16.10	.09
	16.20	.14
	16.30	.13
	16.40	.10
	16.50	.09
	17.00	.16

APPENDIX TABLE 18. POST-GnRH SERUM LH LEVELS (ng/ml) OF GILTS IN EXPERIMENT 2.

GILT 1 TIME (h)	BLEED A	B	C
17.10	3.25	2.42	2.31
17.20	5.50	1.80	5.79
17.30	4.84	4.50	4.81
17.40	2.64	5.72	3.47
17.50	1.80	3.65	2.47
18.00	1.95	2.47	2.37
18.10	1.67	2.55	2.39
18.20	.84	2.03	1.98
18.30	1.13	1.98	1.42
18.40	.75	1.85	1.43
18.50	.90	1.31	1.24
19.00	.91	1.35	1.17
19.10	1.55	1.42	.73
19.20	1.41	1.03	.65
19.30	.91	.69	.57
19.40	.76	.87	1.68
19.50	.81	.64	1.31
20.00	.54	.57	.83
20.10	.43	.37	.71
20.20	.18	.41	.63
20.30	.43	.38	.56
20.40	.31	.35	.32
20.50	.30	.27	.33
21.00	.38	.26	.28

TABLE 18 (cont'd).

GILT 2 TIME (h)	BLEED A	B	C
17.10	6.86	8.88	5.02
17.20	5.28	6.64	15.11
17.30	6.65	6.44	12.51
17.40	3.28	5.68	10.88
17.50	2.89	4.95	8.95
18.00	2.71	4.45	6.86
18.10	1.99	3.38	4.91
18.20	1.45	3.08	4.90
18.30	1.10	3.13	3.27
18.40	1.15	2.74	4.00
18.50	.83	2.33	2.98
19.00	.70	2.34	2.13
19.10	2.50	1.63	1.57
19.20	3.33	1.26	1.42
19.30	1.41	1.16	1.34
19.40	1.20	1.26	1.04
19.50	.84	2.52	1.11
20.00	.66	1.53	.80
20.10	.48	1.29	.59
20.20	.49	1.08	.45
20.30	.48	.89	.79
20.40	.44	.88	2.48
20.50	1.74	.75	1.39
21.00	1.36	.62	1.26

TABLE 18 (cont'd).

GILT 3 TIME (h)	BLEED A	B	C
17.10	15.39	3.90	3.83
17.20	11.14	9.41	9.46
17.30	8.72	8.44	8.54
17.40	5.76	7.99	7.33
17.50	5.55	5.89	5.90
18.00	7.08	4.42	5.07
18.10	3.85	3.47	3.96
18.20	2.82	2.96	2.98
18.30	2.05	2.32	2.37
18.40	1.25	1.92	1.99
18.50	.91	1.05	1.58
19.00	1.33	1.38	1.65
19.10	1.08	.90	1.28
19.20	1.02	1.13	1.23
19.30	.70	.74	1.12
19.40	.59	2.34	.79
19.50	3.31	2.70	.85
20.00	2.23	1.50	.57
20.10	1.98	.83	.47
20.20	1.28	1.09	.45
20.30	.83	.97	.58
20.40	.64	.97	.51
20.50	.65	.93	2.25
21.00	.60	.70	2.33

TABLE 18 (cont'd).

GILT 4 TIME (h)	BLEED A	B	C
17.10	9.10	3.19	2.00
17.20	16.84	5.98	5.30
17.30	8.72	5.16	7.45
17.40	9.13	3.77	5.91
17.50	5.39	3.89	5.12
18.00	3.97	3.10	4.21
18.10	3.01	3.08	3.60
18.20	2.41	2.70	2.26
18.30	1.77	1.86	3.53
18.40	1.57	1.72	2.65
18.50	1.34	1.52	2.87
19.00	.94	1.10	2.57
19.10	1.05	.97	1.90
19.20	.81	1.10	1.62
19.30	.63	.74	1.79
19.40	.55	.62	1.53
19.50	.50	.71	1.09
20.00	.42	.54	1.09
20.10	.30	.58	1.03
20.20	.49	.49	1.01
20.30	.42	.46	.90
20.40	.42	.53	.55
20.50	.36	.48	.80
21.00	.43	.49	.60

TABLE 18 (cont'd).

GILT 5 TIME (h)	BLEED A	B	C
17.10	10.75	6.07	2.32
17.20	19.25	6.70	4.93
17.30	9.68	6.53	8.60
17.40	7.56	8.29	3.32
17.50	4.09	8.30	3.60
18.00	3.19	5.46	3.16
18.10	2.74	3.86	2.59
18.20	1.69	3.07	1.78
18.30	1.30	2.64	1.72
18.40	1.41	1.85	1.89
18.50	1.32	1.55	1.50
19.00	1.85	1.16	1.21
19.10	2.24	1.09	1.13
19.20	1.50	.87	1.17
19.30	1.14	1.04	1.05
19.40	.88	.90	1.01
19.50	.77	1.06	.70
20.00	.65	.79	.81
20.10	.52	.64	.61
20.20	.56	.65	.76
20.30	.36	.51	.66
20.40	.41	.52	.79
20.50	.37	.32	.79
21.00	.37	.70	1.32

TABLE 18 (cont'd).

GILT 6

TIME (h)	BLEED A
17.10	7.96
17.20	8.64
17.30	8.35
17.40	6.47
17.50	5.41
18.00	4.83
18.10	4.28
18.20	2.65
18.30	3.80
18.40	3.15
18.50	2.07
19.00	1.94
19.10	1.45
19.20	1.52
19.30	1.46
19.40	.98
19.50	1.16
20.00	1.03
20.10	.90
20.20	.73
20.30	.60
20.40	.64
20.50	.68
21.00	.89

APPENDIX TABLE 19. DATA FOR GILTS IN EXPERIMENT 2.

	GILT					
	1	2	3	4	5	6
AGE(d) BLEED-A	125	102	112	110	109	118
B	155	132	142	140	139	145[u]
C	184	161	171	169	168	
BODY WEIGHT(kg) BLEED-A	47.3	45.5	46.4	47.3	52.7	45.0
B	74.5	68.2	67.3	64.5	73.6	51.8
C	93.6	91.8	89.1	80.9	96.4	
AGE AT SLAUGHTER(d)	205	182	192	199	189	
TOTAL OVARIAN WEIGHT(g)	17.2	7.3	9.6	6.0	6.6	7.7
UTERINE WEIGHT(g)	540	123	461	159	150	149
UTERINE HORN LENGTH(cm)	122	54	85	46	50	58
OVARIAN STATUS[v]						

[u] Died after surgery.

[v] Gilt: 1- 17 CL's
2- prepuberal
3- in proestrus, 16 preovulatory follicles
4- prepuberal
5- prepuberal
6- prepuberal.

APPENDIX TABLE 20. MEAN* SERUM FSH LEVELS (ng/ml) OF GILTS DURING EXPERIMENT 2.

GILT	BLEED		
	A	B	C
1	673	761	753
2	560	645	901
3	831	626	646
4	431	539	488
5	440	396	651
6	500		

* Mean of 4 determinations on each pooled sample.