INFLUENCE OF DIFFERENT LIGHT TYPES ON GROWTH, PHYSIOLOGICAL RESPONSES, PUBERTY ONSET AND EMBRYO SURVIVAL IN GILTS

A Thesis

Submitted to the Faculty

of

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by

Jennifer Hannesson

In Partial Fulfillment of the

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of

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FACULTY OF GRADUATE STUDIES

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ABSTRACT

With the intent of determining the biological effect of lights of different energy efficiencies an experiment was designed to compare four light types - high pressure sodium (HPS), metal halide (MH), a mixture of MH and HPS (MH+HPS), and standard T8 fluorescent (T8). Comparisons included growth, puberty onset, endocrine changes and embryo survival in gilts. The trials were conducted as a completely randomized design with allowance made for gilt age, litter, and weight. Two trials were conducted using a total of 101 Cotswold gilts, randomly assigned to one of four light treatment rooms (n=24 to 26 per room), at an average of 123 ± 1 days. Gilts were maintained under a 10 h photoperiod and 14 h scotoperiod, with light intensities ranging from 256 ± 12 lux (lx) to 311 ± 12 lx, from trial onset to second estrus, or an average of 28.2 ± 0.2 d of gestation. Weights, feed consumption and backfat were measured weekly. Onset and duration of pubertal and second estrus were recorded. Gilts were bred three times by artificial insemination (AI) at 12 h intervals at their second estrus. Reproductive tracts were recovered from gilts at an average of 30.7 ± 1.0 d of gestation and examined for numbers of corpora lutea (CL) and number of viable embryos as measures of ovulation and embryo survival. Serial blood samples were collected from indwelling ear-vein catheters at 20 minute intervals for 24 hours at 148 days of age (148D), day before second estrus (DB42ndE) and day of second estrus (Dof2ndE). Sera, from the blood samples, were analyzed for progesterone (P4), estradiol (E2), cortisol, luteinizing hormone (LH), follicle stimulating hormone (FSH) and melatonin (MEL). All data were analyzed by the Proc GLM and/or Proc MIXED procedures of SAS.

There were no significant differences in total weight gain, average daily gain, backfat (BF), total feed intake, average daily feed intake, feed efficiency, age at 110 kg, or weight at puberty during either the finishing or pubertal periods for any of the four light treatments. Reproductive performance, which examined age at puberty (182 ± 4 d), duration of puberty estrus (28.4 ± 3.3 h), age at second estrus (203 ± 4 d), duration of second estrus (32.6 ± 2.8 h), estrus interval (21.5 ± 0.4 d), ovulation rate (14.6 ± 0.7 CL) and embryo survival rate (80 ± 4 %), showed no differences between the four light treatments. There were minimal trial differences for these growth and reproductive performance data.

With the exception of cortisol, there were no significant endocrine differences at 148D. Cortisol concentration at 148D was significantly greater for MH (21.4 ± 1.4 ng/ml) and MH+HPS (20.9 ± 1.4 ng/ml) compared to HPS (17.0 ± 1.5 ng/ml) and T8 (15.7 ± 1.9 ng/ml) (P=0.0465). At DB42ndE, trends were present for P4, E2 and LH, otherwise there were no treatment differences for this day's endocrine evaluations. For P4, concentration was greater for MH+HPS (0.13 ± 0.02 ng/ml) than T8, or MH (both, 0.07 ± 0.02 ng/ml) (P=0.0839). For E2, concentrations tended to be greater for T8 (10.2 ± 1.1 pg/ml) than MH+HPS (6.1 ± 1.0 pg/ml) (P=0.0546). For average and baseline LH concentrations, these values tended to be greater in HPS and MH than T8 (P=0.0659 and P=0.0793, respectively). There were no treatment differences for Dof2ndE, or end trial GestD. There was a significant treatment difference for P4 at boar-no STH, but this was most likely due to a gilt with a silent estrus. Combined sample days did not show any significance across treatments except for E2 which showed T8 (5.0 ± 0.5 pg/ml) greater in concentration than MH+HPS (3.0 ± 0.4 pg/ml) (P=0.0439). Diurnal profiles were present for MEL at 148D, DB42ndE and

combined sample days (assays were not done at Dof2ndE). There were no significant differences in concentration between these days, or across treatments. Trial differences were minimal for all hormones except for P4.

Subjective evaluation of these four light treatments showed that the colour rendering index of HPS challenges ease of detection of vulva redness due to its spectral absorption of red colour.

Results from this research will permit recommendations for use of light types based on knowledge of their potential biological impact and subjective evaluations.

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To my husband, Mark, I dedicate my Master of Science thesis. Thank you for your love and support, your company during my research and your celebrations as each stage in the process was completed.

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LIST OF ABBREVIATIONS

HPS	high pressure sodium
MH	metal halide
MH+HPS	metal halide mixed with high pressure sodium
T8	T8 fluorescent
n	number
h	hour
d	day
lx	lux
AI	artificial insemination
CL	corpra lutea (plural) / corpus luteum (single)
148D	148 days of age
DB42ndE	day before second estrus
Dof2ndE	day of second estrus
P4	progesterone
E2	estradiol
LH	luteinizing hormone
FSH	follicle stimulating hormone
MEL	melatonin
kg	kilogram
ng	nanogram

ml	millilitre
pg	picogram
STH	standing heat
NPD	non-productive days
mos	months
HPG	hypothalamic-pituitary-gonadal
HPA	hypothalamic-pituitary-adrenal
GnRH	gonadotropin releasing hormone
CRH	corticotropin-releasing hormone
ACTH	adrenocorticotropic hormone
MCR	metabolic clearance rate
CRI	colour rendition index
°C	degrees celsius
IGF-I	insulin-like growth factor-I
mm	millimetre
m	metre
Phason	Phason Environment Control
RT	room temperature
BF	backfat
max	maximum
min	minimum
2ndE	second estrus

PubE	pubertal estrus
CH	corporal hemorrhagica
CA	corpora albicans
ad lib	ad libitum
T1	trial 1
T2	trial 2
RIA	radioimmunoassay
cpm	counts per minute
μl	microlitre
g	gravity
ADG	average daily gain
ADFI	average daily feed intake
FE	feed efficiency
photo	photoperiod
scoto	scotoperiod
GestD	gestation day
WEI	wean to estrus interval
GLM	general linear model
SAS	Statistical Analysis System

CHAPTER 1

INTRODUCTION

Maximization of reproductive performance is a primary goal of management practice in the swine industry. Successful reproductive performance requires few non-productive days (NPD) and an optimum number of piglets produced per sow per year. The introduction of gilts into the breeding herd contributes to NPD until the time of their first conception. Therefore, early management is important for their future performance. Reproductive efficiency of the gilt is an important aspect of herd productivity because they replace the 30 to 40 percent of sows culled annually in a sow herd (Christenson, 1986).

Factors that may influence gilts' age of puberty and/or future reproductive performance have potential economic impact. These influential factors include: genetics, nutrition, socialization, season, and housing environment (Hughes, 1982; Christensen, 1986). As our domesticated swine have descended from the European wild boar, a short day seasonal breeder, it is reasonable to assume that vestiges of this seasonality remain (Mauget, 1982). Detrimental seasonal effects, of which long photoperiod is a main trigger, are manifested in delay of puberty, reduced conception rate and increased embryo mortality (Love et al., 1993). Because one component of light, photoperiod, has an influence on the reproductive axis and its endocrinological mechanisms, it is possible that another component of light, such as spectrum, may also be influential. However, there has been limited investigation into the impact light spectrum has on reproduction.

In Canada, the primary design of swine barns is intensive confinement housing which relies on artificial lighting. The current forms and methods of barn illumination and their cost efficiencies are inconsistent between and within barns. Spectrums vary with type of lamp, such as incandescent, fluorescent, MH, and HPS. Currently, MH and fluorescent, which have a fairly broad light spectrum are commonly used in barn construction even though these are not the most economically efficient. High pressure sodium, which is more cost efficient, is less commonly used because it has a spectrum with low colour rendering. A mix of MH and HPS may provide both the economic efficiency and pleasant working spectrum that are desired by the industry.

The objective of this research was to determine the influence of four light types - MH, HPS, MH+HPS, and T8 fluorescent - upon growth, endocrine activity (E2, P4, cortisol, LH, FSH, and MEL), puberty onset, estrus expression, and embryo survival in gilts maintained under a consistent photoperiod and light intensity from four months of age. It was hypothesized that performance and physiological parameters would not be influenced by light spectrum. This would be demonstrated by performance consistencies across light treatments.

As well, it was anticipated that investigations to clarify the role that light spectrum and quality play in the mechanisms of reproduction and growth performance had the potential to affect economic and production efficiency in both breeding/gestation barns, as well as grower-finisher barns.

CHAPTER 2

REVIEW OF THE LITERATURE

2.1 Introduction

Conventional confinement housing for pigs in Canada requires artificial lighting as the primary light source. The impact that lighting has on the performance of these pigs is not fully understood. Domestic swine have descended from the seasonally breeding European wild boar. Vestiges of this seasonality are routinely observed in domestic production (Claus and Weiler, 1985), and it may be assumed that lighting, a trigger of seasonality, may have an influence on production even in intensively housed animals.

Photoperiod duration appears to be the main environmental factor influencing this seasonality (Delcroix et al., 1990; Love et al., 1993; Peltoniemi et al., 1999). However, in addition to duration, other components of light such as intensity and spectrum may also impact swine performance. Light intensity at 800 lux (lx) have been suggested to entrain gilts to a specific circadian rhythm (Green et al., 1996). Entrainment of a circadian rhythm may also be stronger under natural light than artificial sources (Cook et al., 1998). However, the impact that various artificial light spectrums have on pigs is relatively unknown.

The maintenance of gilt pools as replacements to maintain a consistent breeding herd size can be expensive when considering feed, labor and housing costs over a non-productive period (Christenson, 1986; Dial et al., 2001). Thus, methods of insuring early reproduction and maintaining reproductive efficiency of gilts are economically, as well as biologically

important. This literature review deals with factors influencing growth, onset of puberty and early reproductive performance with emphasis on the impact of light on these factors.

2.2 Seasonality of *Sus scrofa*

2.2.1 European Wild Boar

Domestic swine (*Sus scrofa*) are descended from the European wild boar, which is a short-day, seasonal breeder (Claus and Weiler, 1985). Short-day breeding, occurring during late autumn and early winter, is dependent upon the available feed supply (Delcroix et al., 1990). If nutritional sources are abundant, a second breeding period may occur in April (Love et al., 1993). Over the summer and early autumn sows and gilts remain in an anestrous period, which has been associated with low levels of circulating progesterone (Mauget, 1982).

2.2.2 Seasonal Influence on Reproductive Performance in Domestic Gilts and Sows

Vestiges of the European wild boar's seasonal performance are present in domestic swine. Decreased reproductive performance during, or soon after the summer months are commonly expressed by delayed puberty onset, decreased conception and farrowing rates, increased abortions, reduced litter sizes through increased embryonic and/or fetal mortality, and an increased wean-to-estrus interval (WEI) (Claus and Weiler, 1985; Paterson et al., 1991; Paterson et al., 1992; Peltoneimi et al., 1999). Production and economic returns are directly affected by this reduced performance.

Delay in puberty is often observed during the long days of summer. Puberty onset appears to be similar to that of the onset of seasonal cyclicity. In studies by Paterson et al. (1991) and those reviewed in Christenson (1986), fewer gilts showed normal estrous cycles by an acceptable breeding age (nine months) in the summer months. In a different study, reproductive tracts collected from market weight gilts in the late summer showed less sexual development than tracts collected at other times of the year (as reviewed by Christenson, 1986).

The term "autumn abortion" has resulted from the high incidence of abortions occurring in the period of September through October. These abortions mainly occur in gilts and sows bred between June and September (Claus and Weiler, 1985; Smith et al., 1991). Irregular returns to estrus can result from abortions. Xue et al. (1994) showed that the number of sows with irregular returns to estrus was twice as high as the number occurring in November and December. Another impact on breeding and farrowing performance is litter size. Conceptions from breedings in November, December and January resulted in significantly larger litter sizes than those resulting from July and August breedings (Xue et al., 1994).

Management of conventional pig production units incorporates elements that attempt to overcome reduced seasonal performance. High quality nutrition at adequate feeding levels, environmental controls of temperature, herd health, genetics, and boar exposure are just some of the methods used in herd management. Yet, season is influential in both confinement and non-confinement housed pigs (Christenson, 1986). Different management styles, observed in a study of Finnish sow units, all showed impact of the longer summer

days, such as higher rebreeding rates for the period of August to November (Peltoniemi et al., 1999).

Temperature is considered less of an influence than a long photoperiod (Claus and Weiler, 1985; Delcroix et al., 1990; Love et al., 1993; Peltoniemi et al., 1999). Conception was not improved when cooling methods were employed during hot days (Claus and Weiler, 1985). Consistently, late summer and early autumn showed the poorest reproductive results for gilts and sows.

2.3 Physiological Control of Puberty

Puberty is the process by which animals become both physically and physiologically able to reproduce. There are numerous criteria used to define puberty in the female, such as the first ovulation and/or the first behavioural estrus (Senger, 1997). In gilts, the common age range for puberty onset is 5 to 7 months (mos) (Senger, 1997).

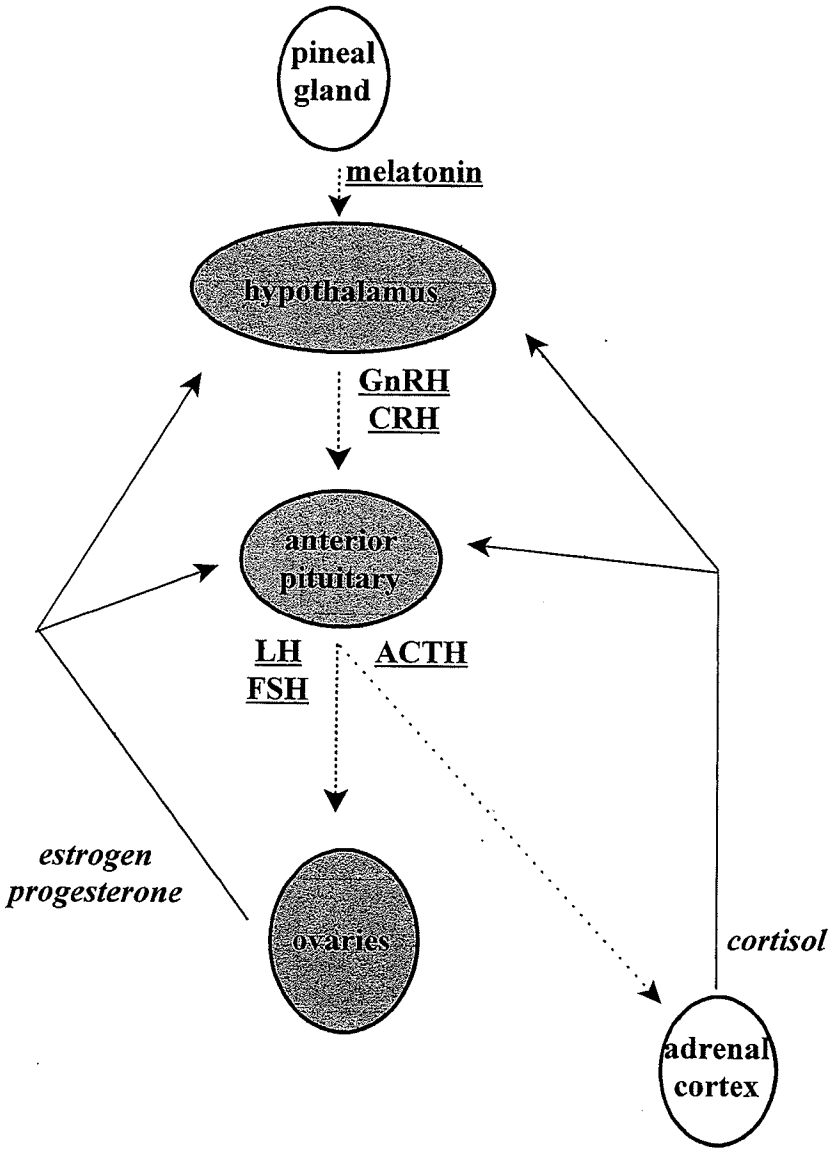
It is the female's ability to successfully conceive and to maintain a full-term pregnancy that are the goals in the management of puberty. These goals are implemented by management of body condition, age at boar exposure and age at first breeding. In order to fully understand the influence of reproductive management, a review of the mechanisms governing puberty is important.

2.3.1 Primary Endocrine Mechanisms

The physiological processes in the body are mediated through the endocrine system. Reproductive processes (puberty and estrous cycles) are regulated by the hypothalamic-

pituitary-ovarian /gonadal (HPG) axis. The HPG axis is in turn influenced by the hypothalamic-pituitary-adrenal (HPA) axis and the pineal gland (Figure 1). Specifically, it is the physiological changes occurring in the HPG axis that result in puberty.

Figure 1. The HPG axis and associated HPA axis and pineal gland (adapted from Binkley, 1995).



The onset of early puberty in the female is not limited by the functions of the anterior pituitary, or the ovaries, but by the production and release of gonadotropin-releasing-hormone (GnRH) from the hypothalamus (Senger, 1997). Stimulation of the anterior pituitary by exogenous GnRH results in secretion of LH and FSH in pre-pubertal females (Senger, 1997). In turn, pre-pubertal ovaries stimulated with LH and FSH produce follicles which produce and release estrogen. It is the maturational change in the sensitivity of the hypothalamus to blood estrogen levels that results in the pre-ovulatory surge of LH culminating in puberty (Hafez, 1993). Prior to this pre-ovulatory surge, the hypothalamus produces low tonic levels of LH and FSH which result in pre-ovulatory follicle development and low levels of E2 in the blood. The hypothalamus is sensitive to these low levels of estrogen, which cause a negative feedback upon the hypothalamus surge center and keeps LH and FSH levels low. Pre-pubertal peripheral P4 remains low (< 0.500 ng/ml) and is often used as an indicator of non-cyclicity (Bollinger et al., 1997; Diekman and Green, 1997).

As the animal matures within its environment, there is a decreased hypothalamic sensitivity to estrogen. As estrogen concentration increases, due to gonadotropin stimulated follicular development, it finally acts as a positive feedback on the hypothalamus resulting in a surge of GnRH and consequent pre-ovulatory surge of LH. Following this first ovulation there is the development of corpora lutea (CL) and the production of progesterone. Serum progesterone levels greater than 3 ng/ml characterize the post-pubertal condition (Diekman and Green, 1997). Post-pubertal cycling still involves negative and positive feedback by estrogen concentrations on the hypothalamus, but the pattern of release of the GnRH and of the gonadotropins provides a regular periodicity for increase in estrogen concentration. This

relationship between estrogen levels and the sensitivity of the hypothalamus constitutes the "gonadostat theory" (as reviewed by Pearce et al.; 1988; Senger, 2003).

An additional factor which may be impacting the onset of puberty is the metabolic clearance rate (MCR) (Christenson et al., 1985). The MCR influences endocrine levels as it is the speed with which an animal metabolizes steroids (i.e. clears them from the body). The MCR for estradiol is greater in pre-pubertal compared to post-pubertal gilts. Perhaps it is the decrease in MCR which initially causes elevated levels of circulating estrogens which contributes to the advancement of sexual development.

2.3.2 Two Secondary Endocrine Mechanisms

In addition to the primary hormones of the HPG axis, there are secondary hormones which have an influence upon metabolic function and/or puberty attainment. Their sites of action lie within the HPG axis, but they are produced outside of this system.

2.3.2.1 Cortisol

Cortisol, a corticosteroid produced and released from the adrenal glands, is a stress related hormone. The normal release of cortisol follows a circadian profile (Evans et al., 1988). In gilts at 21 weeks of age (approximately 148 days) the plasma cortisol profile shows the emergence of morning peaks and an evening trough. By 28 weeks (approximately 196 days) the profile shows one large peak, one small peak and a late evening trough. Prunier et al. (1993) found a diurnal pattern as Evans et al. (1988) did, but these cortisol profiles showed a lowered morning concentration from 4 to 5 days prior to and following

pubertal estrus. However, in a study by Kirkwood et al. (1987) there were no significant differences in cortisol levels across various prepubertal weights and age treatments.

Elevated cortisol secretion occurs when stress increases activity of the HPA axis. Cortisol influences functioning of the HPG axis by immediately affecting the hypothalamus and, with chronic release, other levels of this system (Rivier and Rivest, 1991). The term, "transport stress" is used to explain the positive influence of acute stress (i.e. increased cortisol) upon puberty onset in gilts. In adrenalectomized gilts puberty was delayed, whereas puberty was advanced in those gilts treated with synthetic glucocorticoids (as reviewed by Prunier et al., 1993). As well, Turner et al. (1998) showed that repeated acute stress did not inhibit reproductive performance (e.g. duration of estrus, ovulation rate), but did not see results that found enhancement of this performance. There was no significant difference between the acutely stressed gilts and the non-stressed/control gilts.

The acute stress release of cortisol may play a role in decreasing sensitivity of the hypothalamus and pituitary to estradiol feedback (Pearce et al., 1988). In the absence of exogenous GnRH, cortisol-treated gilts produced more LH than the saline-treated gilts. In a later study, acute levels of cortisol in an estrogen-primed body, were shown to facilitate the release of FSH and LH (Brann and Mahesh, 1991). These results are supported by a decrease in FSH and a disruption in follicular development resulting from adrenalectomy (from Meijs-Roelogs and Kramer, 1977 in Brann and Mahesh, 1991).

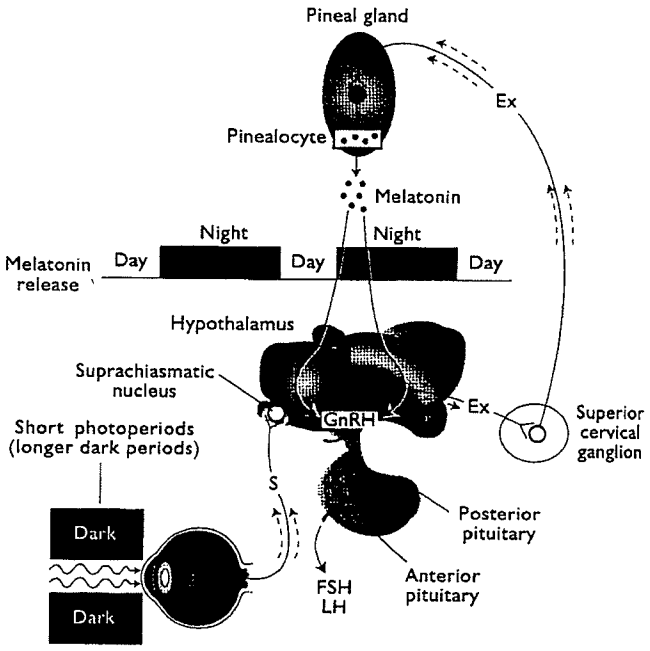
Contrary to acute cortisol release, chronic elevated cortisol levels have a negative impact upon reproductive performance. Changes in the responsiveness of the anterior pituitary gland and ovary result from continuous exposure to cortisol (Rivier and Rivest,

1991). Both GnRH and LH secretion is blocked, thus preventing ovulation (Rivier et al., 1986; Booth, 1990). This inhibition was reversed by the administration of an antagonist of corticotropin-releasing factor (Rivier et al., 1986). These continual elevated cortisol levels have been shown to reduce responsiveness of the pituitary gland to GnRH, as well (Varley, 1994).

2.3.2.2 Melatonin

The regulation of seasonality in the pig and other animals is considered to be mediated through MEL (Paterson et al., 1992a/b; Love et al., 1993). Melatonin, a pineal hormone, is the main transducer of daily and annual photoperiodic information to the HPG (Malpaux et al., 1999). Reproductive performance is influenced by MEL through its effect on the firing rate of hypothalamic GnRH neurons (Reiter, 1991; Malpaux et al., 1999). Figure 2 outlines the model by which short-day breeders, of which pigs are included, may be stimulated by short photoperiods.

Figure 2. Action of short photoperiods on the hypothalamus (as adapted from Senger, 1997).



It is the action of the light upon the retinal cells of the eye that lead to the firing of inhibitory neurons which inhibit release of MEL (Senger, 1997); it is the duration of MEL inhibition that communicates photoperiod. The theory that a short photoperiod (decreased length of MEL inhibition) and long scotoperiod (increased length of MEL release) is influential was supported in results from research by Diekman et al. (1991). With the application of exogenous MEL, gilts reached puberty at an earlier age than the control gilts. These results occurred independent of housing under either a natural increasing, or decreasing day length. Additionally, there were no significant differences in average daily gain (ADG), percentage of carcass muscle, length of estrous cycle, LH, or FSH. Research by Paterson et al. (1992b) showed that overcoming seasonal inhibition of puberty in gilts was more dependent upon the

diurnal rhythm of MEL than its concentration in the blood. MEL implants, controlling overall concentration, had no significant affect on numbers of gilts achieving puberty; whereas, MEL taken orally at afternoon feeding, thereby extending MEL duration, did significantly increase the number of gilts achieving puberty.

The profile of MEL shows elevated concentrations during the scotoperiod and decreased concentrations during the photoperiod. A nocturnal rise in MEL is considered to occur when the mean concentration of the scotophase is greater and significantly different from the photophase mean concentration (Bollinger et al., 1997). In different studies, gilt reproductive status (pre- vs postpubertal; non-pubertal vs pubertal) had no effect on MEL profile. These observations are consistent with those of MEL concentrations throughout the ewe's estrous cycle (Arendt et al., 1981; as reviewed by Diekman and Green, 1997). Change in seasonal photoperiod, as opposed to reproductive stage, produced the change in MEL concentrations in ewes, which are also short-day breeders.

Although MEL profiles are well established for sheep, there are great inconsistencies in the literature regarding MEL secretion in pigs. Studies on the influence of MEL secretory profiles by photoperiod (short, long, equal), light intensity, and light type (artificial, natural) show varied results. For Diekman et al. (1992) no significant changes in MEL serum concentrations were shown between photoperiod treatments. Collections during light, or dark periods, following abrupt light exposure, and within short, or long-days produced similar results. Only with the application of exogenous MEL was a rise detected. In a further study, neither light intensity (50 000 lux vs 700 lux), or type of light (natural vs artificial) affected the secretory pattern of MEL under a long photoperiod (Diekman and

Green, 1997). In a study by Bollinger et al. (1997) ewes were housed with gilts in order to act as controls. The ewes showed significant differences between scoto- and photophase profiles, whereas the gilts did not. Alternatively, research by Paterson et al. (1992a) did observe a significant MEL peak in gilts housed under an equatorial photoperiod, which was then either increased (long-day), or decreased (short-day). Within 1 to 2 hours into the scotoperiod the MEL peak was observed. Further studies have also found significant differences in MEL concentration between photo- and scotophase. Tast et al. (2001) found significant differences between photo- and scotoperiods no matter the photophase light intensity, which ranged from 40 to 10,000 lux within a light-dark cycle of 12 h. As well, Andersson et al. (2000) showed that peripubertal boars expressed a typical circadian MEL profile under natural and artificial lighting and varying photoperiods. In comparisons of two commercial assays, only the Buhlmann Laboratories AG MEL assay was able to differentiate between low and otherwise undetectable levels (Andersson et al., 2000). This suggests that previous contradictions in MEL concentration at scotoperiod, photoperiod and overall profile may have been due to lack of assay sensitivity.

2.4 Management Factors Influencing Puberty

There are numerous factors that can influence the physiological mechanisms of puberty and therefore, future reproductive performance. These factors include: genetics, nutrition, socialization (group size, boar exposure), and environment (lighting, temperature, etc.). Season influences these factors, but to some extent this can be overcome by management.

2.4.1 Genetics

Between purebred and crossbred animals there are significant differences in age of puberty attainment. Crossbred animals tend to attain puberty at younger ages, than do purebred animals due to hybrid vigor (as reviewed by Christenson, 1986; Hughes, 1982).

The influence of sire was believed to be the cause of variation in amplitude of scotoperiod melatonin levels in peri-pubertal, crossbred boars (Andersson et al., 2000).

Knowing when a given gilt genotype is likely to reach puberty assists in making management decisions, such as, age of boar exposure, nutrition, and what age to acquire gilt replacements (Foxcroft and Aherne, 2001).

2.4.2 Nutrition

Industry practice has commonly used ad libitum (ad lib) feeding for replacement gilts up to the time of breeding (VIDO, 1996). Movements to a restricted feeding though, are being considered due to rapid growth performance of genetically lean pigs (Foxcroft and Aherne, 2001).

However, the influence of feeding regime on puberty age is varied. In a study examining feed intake without boar exposure, no significant difference was shown for puberty onset between gilts fed ad lib, or at 50% and 75% of ad lib levels (Newton and Mahan, 1992). However, a trend was observed suggesting that 50% of ad lib intake may reduce ovulation onset. Additionally, it was shown that as feed intake increased, the percentage of gilts showing estrous behaviour was lower and the percentage that ovulated was higher (i.e. silent heat). In comparing puberty onset between gilts fed ad lib and those

fed a moderately restricted diet (60 to 85% ad lib) puberty was delayed, but not prevented (Beltranena et al., 1991a) by the lower intakes. As well, Klindt et al. (1999) found that there were no differences in puberty age, number of CL and pregnancy between gilts fed ad lib, or moderately restricted from 13 to 25 weeks of age. However, the rate of puberty attainment was greatest for gilts fed ad lib. Alternatively, Mauget (1982) showed that European wild boar gilts and sows that were fed liberally showed an earlier onset of sexual activity.

There is some debate as to whether it is body weight, degree of fatness, or age that determines puberty onset in pigs. In one study by Beltranena et al. (1993) no significant difference in LH secretion, follicular development, reproductive tract weight, or E2 concentrations was shown between same age gilts of different body fatness. This study determined that protein deposition, regardless of fat deposition, was most important. As well, innate LH secretion was shown to be the determinant of ovarian function and reproductive tract weight. Accordingly, a study by Kirkwood et al. (1987) showed no difference in basal LH concentration across weight, or age values, although, LH pulse amplitude did show an increase with an increasing weight. As well, some correlation has been found between the weight at boar stimulation and the weight at puberty (Beltranena et al., 1991a). Following realimentation, after feed restriction, Cosgrove et al. (1993) found gilts showed an increase in LH concentration regardless of weight. This suggests that age may be more associated with increased LH secretion. In other studies, gilts reaching puberty and those that did not had no significant difference in weights (Flowers et al., 1989; Paterson et al., 1991). As well, Newton and Mahan (1992) reported that puberty onset was not influenced by a minimum fat content.

Therefore, in evaluating a specific feeding regime, consideration must be made of genetics, age, housing, and many other factors that would enable feeding to either enhance, or delay reproductive performance.

2.4.3 Environment

2.4.3.1 Housing

The recommendation for floor space allowance varies according to pig weight and flooring type, as outlined in the recommended code of practice (Connor, 1993). In a study comparing low (more space than recommended by guidelines) and high (half of low levels) stocking densities, the low density group showed heavier weights for adrenal, pituitary, brain, uterine and ovarian tissues (Rahe et al., 1987). As well, significantly more gilts from the low density group had ovulated at slaughter, which occurred when the average pen weight reached 100 kg.

2.4.3.1.1 Sow/Gilt Socialization

Pigs are highly social and are naturally reared in family social groups (Mauget, 1982). Limiting their intraspecies contact can result in a delay in puberty onset (Hafez, 1993; Sterle and Lamberson, 1996). Gilts that have been individually housed show a higher incidence of silent estrus and irregular estrous cycles compared to those gilts grouped in pens. Group size may also be influential. Christenson (1986) reported on studies demonstrating that the highest percentages of gilts cycling by 9 months of age were found in groups greater than 3

and below 50. Senger (1997) also reported that gilts housed in groups of 2 to 3 will likely show delays in puberty onset, whereas gilts in groups of 10 or more will be more likely to reach puberty at expected ages. These results are similar to those shown by Love et al. (1993) for sows. Seasonal infertility was reduced in groups of 5 to 6 sows, compared to larger groups.

Synchronization of estrus onset is also influenced by gilt/sow groups. In studies by Mauget (1982) and Delcroix et al. (1990) synchronization of reproductive cycling after a summer-autumn anestrus occurred in groups of European wild boar females regardless of the presence of a male in the group. In examination of pubertal attainment in domestic gilts, exposure to an estrual female, or a mature boar decreased age at puberty (Sterle and Lamberson, 1996). Although exposure to an estrual female was effective in inducing puberty, a greater percentage of gilts reached puberty when exposed to a mature boar.

2.4.3.1.2 Boar Exposure

Exposure to a mature boar (12 months of age or older) induces earlier puberty onset in gilts, as well as enhancing synchrony of estrus (Kingsbury et al., 1993; Senger, 1997). These results can occur with continual boar exposure, such as shared wall penning , or with exposure as short as 10 minutes per day (as reviewed by Kingsbury and Rawlings, 1993; Foxcroft and Aherne, 2001). The use of boar exposure may also overcome a seasonal delay in puberty onset. A higher proportion of gilts reached puberty when provided with daily boar exposure (full contact), as compared to isolated gilts (Paterson et al., 1991). In both of these treatment groups, the proportion of gilts reaching puberty was lower when housed under

long-days, as compared to short-days.

Methods of boar exposure may vary between production units. Typically though, there are two main types: fence-line contact, where there is minimal physical contact, but there are visual, olfactory and auditory cues received by the gilt; and physical introduction of gilt and boar, which provides the above cues in addition to physical contact. Whatever method is used for stimulation of estrus, the use of a mature boar is key (Hughes, 1982; Hughes et al., 1990). Between eight and ten months, the submaxillary salivary glands begin to accumulate 3α-androstenol, which is released in the frothy saliva. This pheromone release acts as a primer by altering the endocrine status of the prepubertal gilt.

The physical introduction of a boar seems to be more advantageous. Results from Turner et al. (1998) showed a significant increase in cortisol concentration, indicative of acute stress, 15 minutes after physical boar contact, as compared to fence-line contact and back pressure tests. Within 30 minutes of boar introduction, cortisol levels had returned to normal concentrations. Additional differences between these two groups were a decreased estrous cycle length and an increased ovulation rate for those gilts exposed to physical contact with the boar. There were no differences in duration of estrus, sexual receptivity, or fertility. Results from Pearce and Hughes (1987) also found that full physical contact with the boar (versus fence-line contact) was necessary for cortisol elevation, i.e. the stress response. It is this stress response that is often considered to be instrumental in inducing puberty (as reviewed by Pearce and Hughes, 1987).

Alternatively, results from Kingsbury and Rawlings (1993) suggest that it is not the release of cortisol, per se, that induces the pre-ovulatory pubertal LH surge. Those gilts

showing signs of estrus after exposure to fence-line contact with a mature boar had lower levels of cortisol compared to the gilts with high levels of cortisol, who did not show signs of estrus by 200 days of age.

Boar exposure is not defined through one specific method of exposure, or by one static response in gilts/sows. Its role in stimulation, synchronization and detection of estrus are undeniable, though.

2.4.3.2 Lighting

The role of light involves the entrainment of the body to a specific light dark cycle (Picazo and Lincoln, 1995). It is this circadian profile which determines the impact of different hormones upon the HPG.

The components of light include duration/photoperiod, intensity and spectrum. It appears that the animal's prior exposure to light, what is called the photoperiodic history, also influences the impact of current light's components.

2.4.3.2.1 Photoperiodic History

The mechanism by which photoperiodic history is registered is not completely understood. What has been determined is that an animal may require repeated exposure to a new light regime before the circadian profile reflects it. For example, male Japanese quail previously reared within a specific lighting condition showed a one week delay in expected reproductive performance that should occur under a new specific lighting regime (Meyer and Millam, 1991). It has also been suggested by Green et al. (1996) that the pig requires a long

acclimatization period in order for melatonin rhythms to entrain to a specific (short) photoperiod. Paterson et al. (1991) and Evans et al. (1994) suggest that this period of physiological entrainment to a new photoperiodic environment is one month for the pig.

2.4.3.2.2 Photoperiod

Photoperiod is the duration of light, whether it is circadian (i.e. within a 24 h period), or circannual (i.e. within a year) (Blood and Studdert, 1999). A short photoperiod is considered to be less than 12 h of light, whereas a long photoperiod is considered greater than 12 h of light. It is suggested that it is not the specific duration of light that is critical, but the direction of change in the duration that is important (Reiter, 1991). Specifically, it is whether the photoperiod is interpreted as becoming shorter or longer that influences its impact. If an animal's physiology changes due to changes in either the length, or phase (location within a cycle), of the daily photoperiod, the animal is said to be photoperiodic (Curtis, 1983). The pig is one such animal, as indicated previously.

The influence by photoperiod is through activation of the pineal gland and its release of MEL. This may be how the body entrains to a specific light regime (Paterson et al., 1992).

Ntunde et al. (1979) studied growth, puberty and plasma LH in gilts raised in complete darkness, long-day, or natural short-day. Age of puberty was significantly delayed in gilts housed in complete darkness. Other factors such as ADG, feed efficiency (FE), ovulation rate, LH concentration, and embryo survival showed no differences between groups. In a study by Smith et al. (1991) LH release failed to show any response other than minimal seasonal changes. These seasonal changes were made by exogenous estradiol

though, not photoperiod.

Circadian patterns of LH have been noted in numerous studies. During the scotoperiod, LH pulses are observed as more frequent than during the photoperiod (Paterson et al., 1992a; Cosgrove et al., 1993). Paterson et al. (1992a) suggest that a short photoperiod plays a role in physiological maturation by providing a longer scotoperiod and subsequent increase in LH pulses. In addition, Evans et al. (1994) showed that injection of estradiol during the "subjective night" for pigs (two groups with night and day in reversal of each other) resulted in well-defined LH spikes. Whereas, if exogenous estradiol were given during "subjective day" there were fewer and less defined peaks.

2.4.3.2.3 Intensity

The intensity of light refers to its illumination level (MB Hydro, 1999). Within each species, the intensity of light that is effective for circadian entrainment varies (Reiter, 1991; Griffith and Minton, 1992). For example, animals that are diurnally active, such as the pig, are less sensitive to the inhibition of light on MEL because they have cone-dominated retinas (Reiter, 1991). The recommended light intensity for confinement pig production range from a minimum of 53.8 lx to over 107.6 lx (Connor, 1993; MacDonald, 2003). In a study by Griffith and Minton (1992) the influence of different light intensities upon cortisol, MEL, prolactin, and ACTH was examined in barrows. Cortisol and ACTH concentrations were similar under both control (113 lx) and intense (1783 lx) illuminations of light. However, MEL was not influenced at a light intensity of 113 lx, while prolactin concentration was greater at this intensity. Green et al. (1996) documented entrainment at 800 lx for MEL .

Although, a study by Tast et al. (2001) showed that at 40 lx there was an impact on the MEL pattern.

Alternatively, in male Japanese quail it is not the specific light intensity, but the intensity of accompanying phases that influences MEL levels (Meyer and Millam, 1991). The use of low lx (3 to 7 lx), red lamps in the process of scotoperiod blood collection in swine does not show an influence on MEL (Diekman et al., 1992; Griffith and Minton, 1992). It is unclear at this time whether pigs respond to a specific light intensity, associated phase of intensity, light type, or a combination of all.

2.4.3.2.4 Spectrum

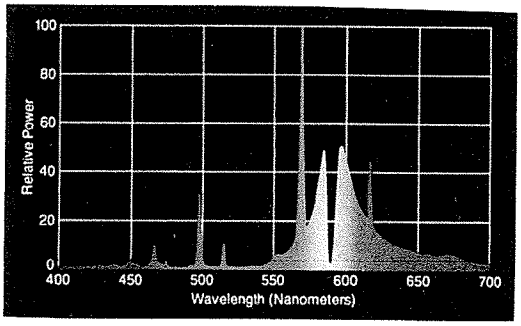
Light spectrum refers to the overall colour produced by various wavelengths. Each lamp, or light source provides a different light spectrum and there is little known about how these different spectra may, or may not, affect the animals. There are numerous lamp types available for use in swine units, all of which may provide similar light intensities, but different light spectra and energy efficiencies (Chastain and Hiatt, 1998; MB Hydro, 1999). Common lamp types used in swine barns are incandescent, metal halide and T8 fluorescent (MB Hydro, 1999).

The spectral property of the lamps produces the visual colour that is perceived by the animals. The measure of this colour, or degree of whiteness, of a light source is defined by the colour rendition index (CRI) (Chastain and Hiatt, 1998; Economopoulos and Chan, 1991). A range of 0 to 100 is used for the CRI, with 100 being the greatest degree of whiteness. MH lamps have a CRI of 60 to 80, producing a visually white light (Figure 3 (a))

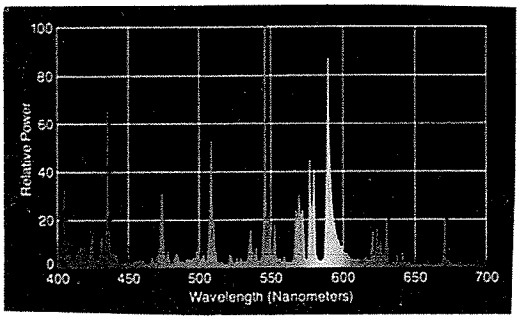
(Chastain and Hiatt, 1998; Economopoulos and Chan, 1991). T8 lamps are within the range of 80 to 85 for CRI and also produce a visual white light (Figure 3 (c) (Chastain and Hiatt, 1998; Economopoulos and Chan, 1991). High pressure sodium (HPS) lamps, not commonly used in barns, have a CRI range of 40 to 60 and produce a yellow-orange coloured light (Figure 3 (b)) (Chastain and Hiatt, 1998; Economopoulos and Chan, 1991). In comparison, sunlight provides a very bright, white light (Figure 3 (d)) (Economopoulos and Chan, 1991).

Figure 3. Light spectrum for (a) MH, (b) HPS, (c) fluorescent and (d) natural light.

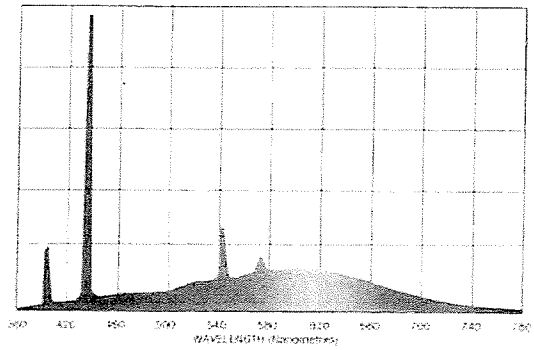
(a)



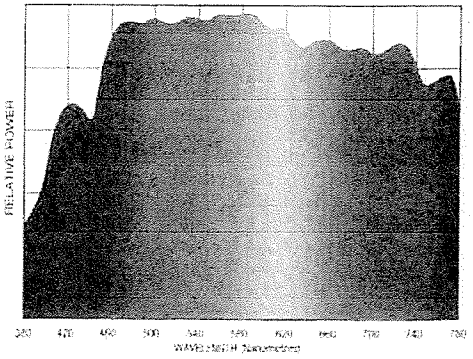
(b)



(c)



(d)



In a study by Cook et al. (1998) natural and fluorescent lighting were compared for their effects on cortisol levels and meat quality. In both treatments, handling did cause an elevation in cortisol, with pigs showing no significant difference at pre-wean handling. However, at post-wean handling, cortisol levels were significantly higher for weanlings housed under fluorescent lighting. It was suggested that under natural light pigs are better entrained to the circadian rhythm of cortisol and therefore, better able to recover, or not react as intensely, as those under artificial lighting. As well, meat quality of male pigs was affected, showing a significantly greater fat depth with artificial lighting than with natural light.

There is some debate about whether, or not the visible wavelengths (i.e. colours) that make up a lamp's spectrum may influence the subjects sensitivity to light (Brainard et al., 1984). As pigs do possess colour vision (Jacobs, 1981), they may be more sensitive to different light spectra.

2.4.3.3 Temperature

The role of temperature in the seasonal performance of reproduction is not clear. Because ambient temperature can be a highly variable factor, it seems unlikely as a primary factor influencing seasonal variation of puberty attainment (Paterson et al., 1991). In European wild boar sows cessation of ovarian activity begins when ambient temperatures are still low (approximately 10 °C), but within the thermal neutral zone for sows (Connor, 1993; Mauget, 1982).

However, temperature can affect reproductive performance. Gilts under chronic heat

stress (33.3°C) reached puberty at a later age and in a lower proportion (4 versus 18 out of 20) than gilts under control conditions (15.6°C) (Flowers et al., 1989). As well, the influence of temperature on sow (and litter) performance was stronger than photoperiod in results from Prunier et al. (1994). More sows showed a weaning to estrus interval within 10 days under short photoperiod. However, under the same photoperiod, a higher percentage of sows showed this interval in cooler (January: 18 to 25°C) temperatures than in warmer (July temperatures: 25 to 38°C) temperatures. These results suggest that temperature may have a role to play in the seasonal impact on reproduction.

2.5 Embryo Survival

Litter size is largely determined by conception rate and embryo survival. Less than perfect conception, or a loss of embryos after fertilization will influence actual embryo numbers that result in viable fetuses. Implantation of the pig embryo begins 10 to 13 days post-breeding and is completed around days 24 to 30 of gestation (Hafez, 1993). The majority of embryonic mortality (30%) occurs 7 to 30 days post-breeding (Pope and First, 1985).

2.5.1 Estrus Detection

Estrus detection is vital for the efficient management and operation of the breeding herd. It is a process of detecting puberty onset, estimating future estrous activity and estimating the timing of breeding procedures (Christenson, 1986). With gilts demonstrating a shorter estrus than sows (by approximately 8 h) (Steverink et al., 1999), good estrus

detection for correct insemination time is especially important.

In selecting replacement gilts, their future estrus performance may be determined by their pubertal estrus (Sterning et al., 1998). Gilts with early puberty onset (about 186 d) demonstrated a significantly greater chance of manifesting a WEI within 10 days, compared to gilts expressing puberty at a later age (approximately 226 d). Additionally, signs of estrus onset at puberty, such as reddening and swelling of the vulva and STH response were positively correlated with strong estrus signs occurring within 10 d of WEI.

2.5.2 Timing of Insemination

Ovulation occurs from 10 h to 85 h after the onset of estrus, or approximately 70 per cent of the way through estrus (Soede and Kemp, 1997). The timing of insemination is typically based on the onset and expected duration of STH, both of which can be influenced by many factors. Generally, breeding between 0 and 24 h before ovulation gives good fertilization results (Kemp and Soede, 1997). Breeding too early, or too late results in decreased litter size and farrowing rate. Double inseminations, 12 to 24 h apart, helps ensure higher farrowing rates and litter sizes (Steverink et al., 1999).

2.5.3 Insemination at Pubertal vs Post-Pubertal Estrus

By waiting to breed until the third estrus there is an increase of non-productive days (NPD) with the concomitant costs associated with feeding and housing a 'non-productive' animal. However, it is important to consider that physiological development is not necessarily complete at puberty. In the study by Young and King (1981) breeding at the third

estrus resulted in 44 additional NPD days, but improved conception rate and resulted in 1.4 additional pigs per sow over 3 parities. In addition, three week weights of piglets were heavier for gilts bred on third estrus. This was likely a function of the gilts' greater size and body stores by the time of breeding and gestation. Additionally, a greater increase in ovulation rate was seen in gilts reaching puberty at 5 to 7 months and bred at their third estrus than in gilts reaching puberty at 8 to 10 months and subsequently bred (as reviewed by Christenson, 1986).

However, some gilts may be physiologically ready for breeding and pregnancy maintenance at puberty estrus (Young and King, 1981). The extent to which chronological age influences physiological maturity is not exactly known. In a study by Young and King (1981) conception rate was lower in gilts bred at their first estrus as compared to third observed estrus. In contrast Grigoriadis et al. (2001) reported that gilts bred at 215 days on either their second, or third estrus had similar conception rate and litter sizes. As well, no significant differences were found for ovulation rate and embryo survival between three different weight groups bred at either their first or third estrus (low weight, or intermediate and bred at third estrus; finish weight and bred at first estrus) (Knott et al., 1984). There are likely genotype and environmental impacts which also confound differences between these various studies.

2.5.4 Nutrition

Nutrition can impact ovulation rate and therefore, available ova for fertilization. Ad lib feeding, till the time of mating, is believed to maximize ovulation rate (as reviewed by

Christenson, 1986). In restricted fed animals, nutritional flushing 10 to 14 days before expected breeding can have the same result (Christenson, 1986), especially if gilts were previously restricted fed. However, this may not translate into improved litter sizes. In a later study, Beltranena et al. (1991) found that the ovulation rate of nutritionally flushed gilts that were previously restricted fed was at the same level as gilts fed ad lib. Feeding level showed no impact on concentrations of E2, P4, LH, or FSH.

Restricted feeding post-mating, specifically during the first 30 days, may affect embryo survival especially during the long days of summer and early autumn. In some cases it may be causing a return to estrus 25 to 30 days post-breeding (Peltoniemi et al., 2000). Lower levels of LH during this long-day photoperiod may be further suppressed with restricted feeding. The reduced levels of LH, though not severe enough to completely regress CL development, did influence P4 production and its effect on development of the endometrial environment. The compromised endometrial environment may be retarding embryo quality and therefore, embryo production of E2 which is a necessary signal for pregnancy maintenance. Research by Dyck et al. (1980) found that LH concentration was highest for gilts fed 2.25 kg daily and lower for gilts fed 1.50 kg, or 3.00 kg daily. While P4 was highest at 1.50 kg feed intake daily and lowest at 3.00 kg fed daily. However, research by Klindt et al. (1999, 2001) found no significant impact on gilts' reproductive performance through first pregnancy, or piglet production when fed a moderately restricted diet (74% of ad lib intake) from 13 to 25 weeks of age.

2.5.5 Genetics

While the European wild boar averages 5 piglets per sow, compared to an average of 11 piglets per sow in domestic breeds, intrauterine mortality is significantly lower than in the domestic pig (Mauget, 1982). In the domestic pig, hybrid vigor results in a significant increase in ovulation rate as compared to selection for ovulation rate in purebred lines (as reviewed by Christenson, 1986). Additionally, a study by Wettemann et al. (1980) showed crossbred gilts had significantly more CL and embryos than did purebred gilts. Management, specifically feeding, of certain genetic lines may also influence litter size and weight (Stalder et al., 2000). In comparing purebred Meishan gilts with purebred Large White gilts, embryo survival is significantly greater in the Meishan breed. Part of this may be due to the levels of insulin-like growth factor-I (IGF-I) in the uterine fluids, which have been strongly correlated with embryo survival (Yu et al., 1999).

2.5.6 Endocrine Mechanisms

2.5.6.1 Progesterone

Progesterone secreted by CL, is the primary hormone associated with pregnancy. Characteristically, P4 levels peak by day 12 of gestation and then gradually decrease throughout pregnancy (Hafez, 1993). Research has been conducted to determine the correlation between P4 concentration and embryo survival. In a study by Wettemann et al. (1980), P4 concentrations were not confident predictors for the number of CL and embryos at 30 days of pregnancy. As well, Yu et al. (1999) found that exogenous P4 treatment for 12

to 14 days, either two, or four days post breeding, neither increased embryo survival rate, or affected IGF-I mRNA in endometrial tissues. Additionally, irrespective of feed intake levels, embryo survival differed between high (91%), intermediate (65%) and low (69%) blood P4 levels (Jindal et al., 1996). However, a study by Archibong et al. (1987) suggests that for gilts bred at first or third estrus, the difference in embryonic mortality, but not in P4 concentration, may be due to the lack of progesterone priming at the pubertal estrus.

Prior to pregnancy, systemic and follicular levels of P4 were not significantly different between gilts at their first, or third estrus (Smith et al., 1992). This may also suggest that the important role for P4 is its priming of the uterus.

2.5.6.2 Estradiol

Estrogen is another important hormone associated with pregnancy in the gilt/sow. The embryo, by day 12 of gestation produces E2 which provides the signal for maternal recognition of pregnancy (Hafez, 1993). Results from Archibong et al. (1987) showed no significant differences in plasma E2 levels at day 3 of gestation between gilts bred at their first, or third estrus. However, at day 30 significant differences were observed, showing a greater E2 concentration for gilts bred at their third estrus. Correlation between lower embryonic mortality and greater E2 levels was not determined.

Prior to pregnancy, follicular and systemic levels of E2 were greater at gilts' third estrus, compared to first estrus (Smith et al., 1992). This may be correlated with the average size of follicles present at first estrus (4 to 10 mm) and third estrus (10 to 12 mm).

2.6 Conclusion

This review has focused on some of the important factors involved in the growth and reproductive development of the replacement gilt. Particular emphasis was placed on the seasonality of the domestic pig and factors that communicate these annual periods. Specifically, the potential impact of light on growth and reproductive performance in the pig was examined. Although there is some research on the impact by photoperiod, other components of light, such as intensity and spectrum/source have been less well investigated in pigs. It is important to understand the biological impacts before discarding the role that lighting might play.

Management can make up for many of the environmental challenges that face the replacement gilt, but not always completely. By understanding how these challenges impact the gilt, management can work with these effects and thus, enhance their efforts. Whether this effort improves animal performance, or at least benefits barn economics, it is worthy of consideration.

CHAPTER 3

MATERIALS AND METHODS

3.1 Animal Management

A total of 101 Cotswold gilts (Cotswold Canada Ltd., Winnipeg, MB, Canada) were used in 2 trials: Trial 1(May to September 1999; n=51 gilts) and Trial 2 (January to May 2000; n=50 gilts). At an average age of 123 ± 0.6 days gilts were transported approximately 2 hours from the source barn to the Animal Science Research Unit (ASRU) at the University of Manitoba. Gilts were balanced by age, litter and weight then randomly assigned to 1 of 4 light treatment rooms (n=24 to 26 per room) where they were housed in individual pens (1.125 metre (m) x 1.450 m) on plastic-coated metal mesh flooring. Room temperatures (RT) were set at 16 degrees Celsius (°C) using the Phason Proportional Environment Control (Phason) (Phason, Steinbach, MB, Canada). RT were recorded twice daily at approximately 0800 h and 1600 h from a maximum/minimum thermometer and Phason control panel in each treatment room. When RT exceeded 27°C on the Phason control panel gilts were misted with cold water.

Gilt body weights were recorded weekly. Beginning at 80 kilogram (kg) body weight, weekly P_2 BF measurements were taken using a Renco Lean-Meat ultrasound (Renco Corporation, Minneapolis, MN, USA). Protocols of animal care adhered to the Recommended Code of Practice for the Care and Handling of Farm Animals: Pigs (Connor, 1993) and the Guidelines of the Canadian Council on Animal Care (1993).

3.2 Light Program

Gilts were maintained under a 10 h photoperiod (0700 h to 1700 h) and 14 h scotoperiod throughout the trials by use of timers (Paragon Electric Company, Inc., Two Rivers, WI, USA). Extraneous light was eliminated by window covers and door curtains. Each of the rooms had one of the four light treatments: HPS; MH; MH+HPS; and T8 as control. Figures 4, 5, 6 and 7 show the light fixture layout in each of the treatment rooms. Light fixtures were situated with the intent to provide equal intensity distribution to all pens. Intensities were judged to be equal upon initiation of trials. Measurements of light intensity were recorded at approximately 0.6 m above the pen floor with an Illuminance Meter IM1 (Control Company, Friendswood, TX, USA). Light intensities were measured at the beginning, mid and end of each trial.

During the scotoperiod, any work carried out in the treatment rooms was facilitated by red light from a head lamp or treble light (approximately 3.44 lx).

3.3 Feeding Schedule

Gilts were fed commercial barley-based diets on an ad lib basis with feed added and amounts recorded throughout each trial. The ration, as outlined in Table 1, was changed to a finisher ration at an average gilt weight of 90 kg and then to a dry sow ration at the second estrus (2ndE) if the gilt was bred.

Figure 4. HPS light treatment room.

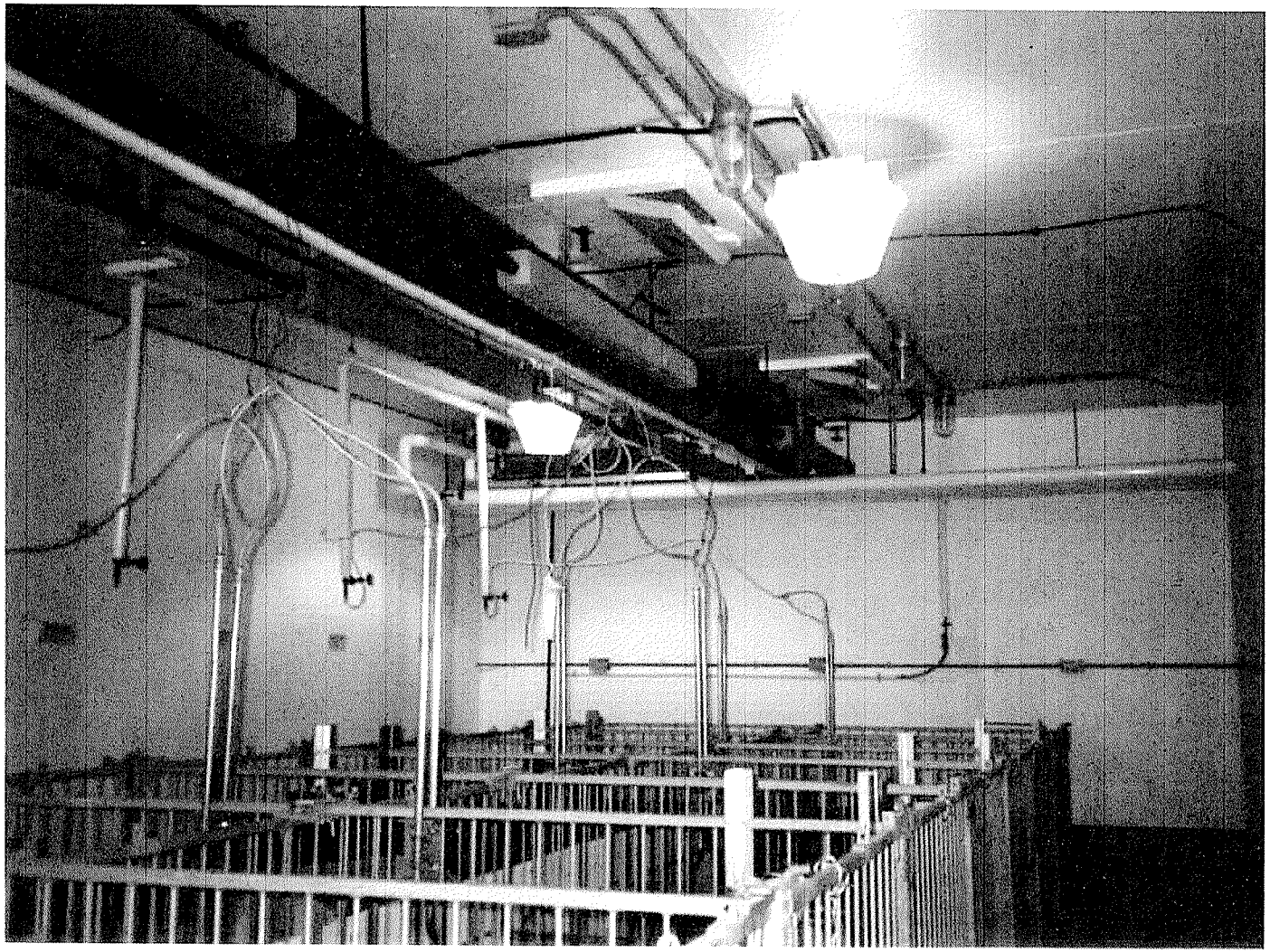


Figure 5. MH light treatment room.

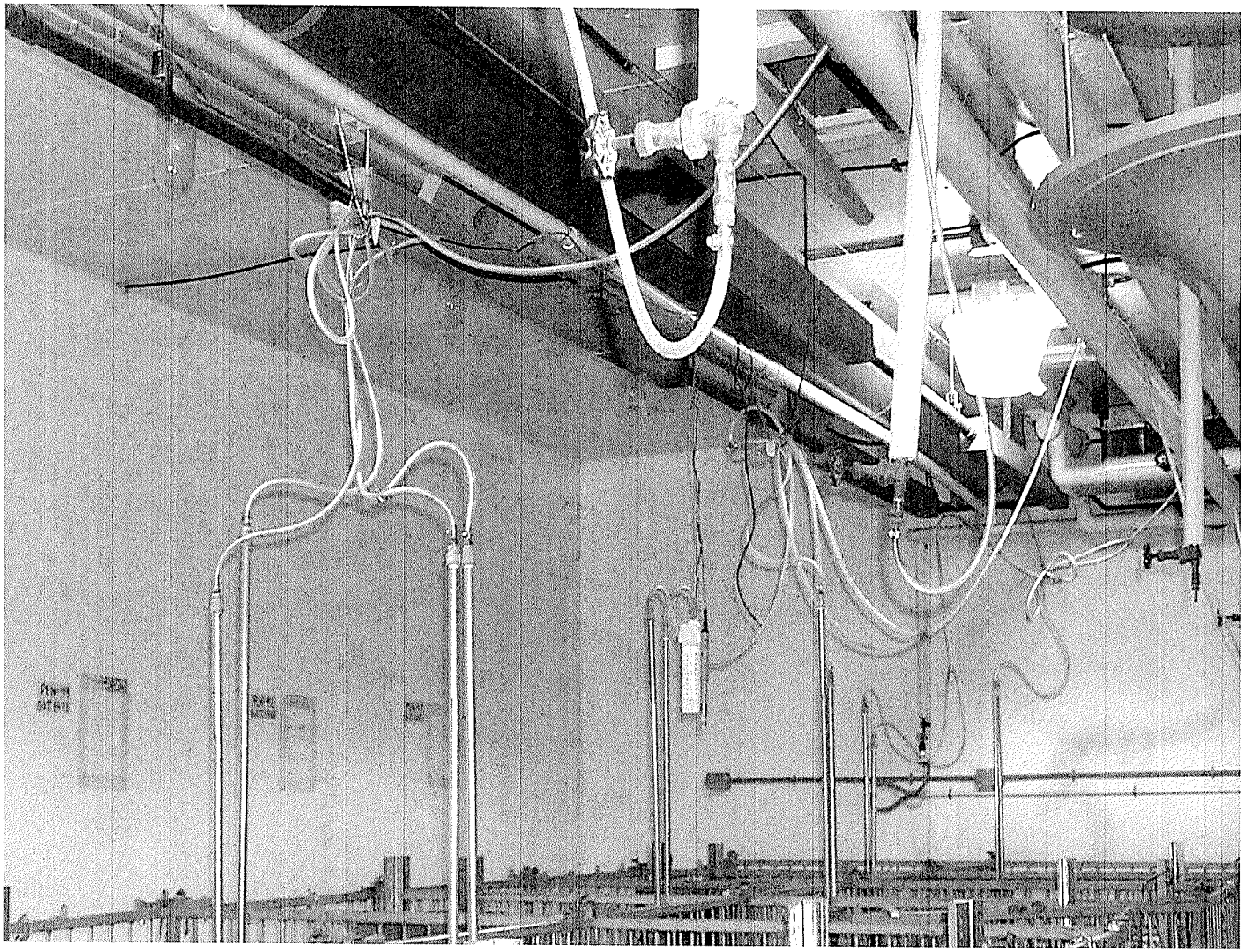


Figure 6. MH+HPS light treatment room.

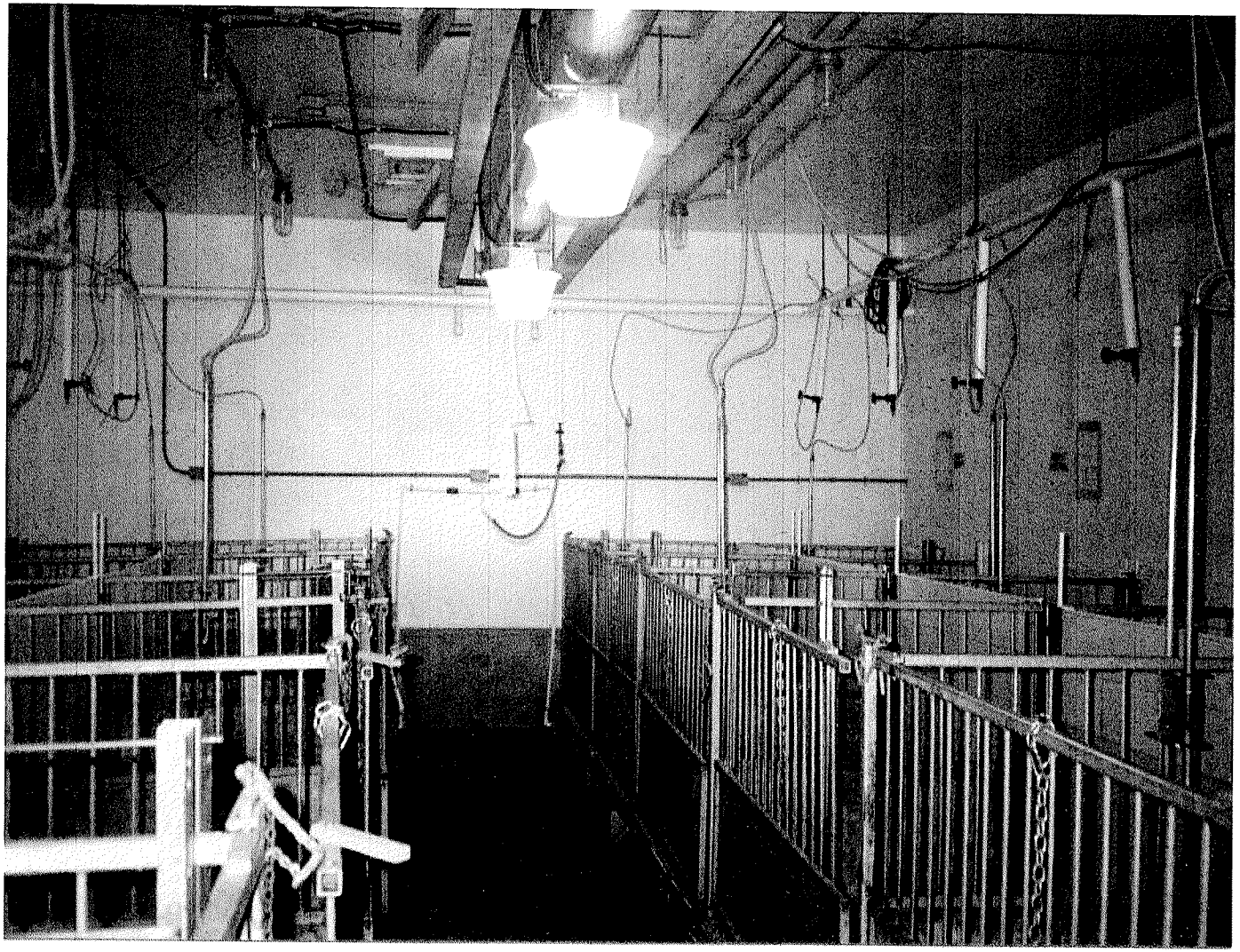


Figure 7. T8 light treatment room.

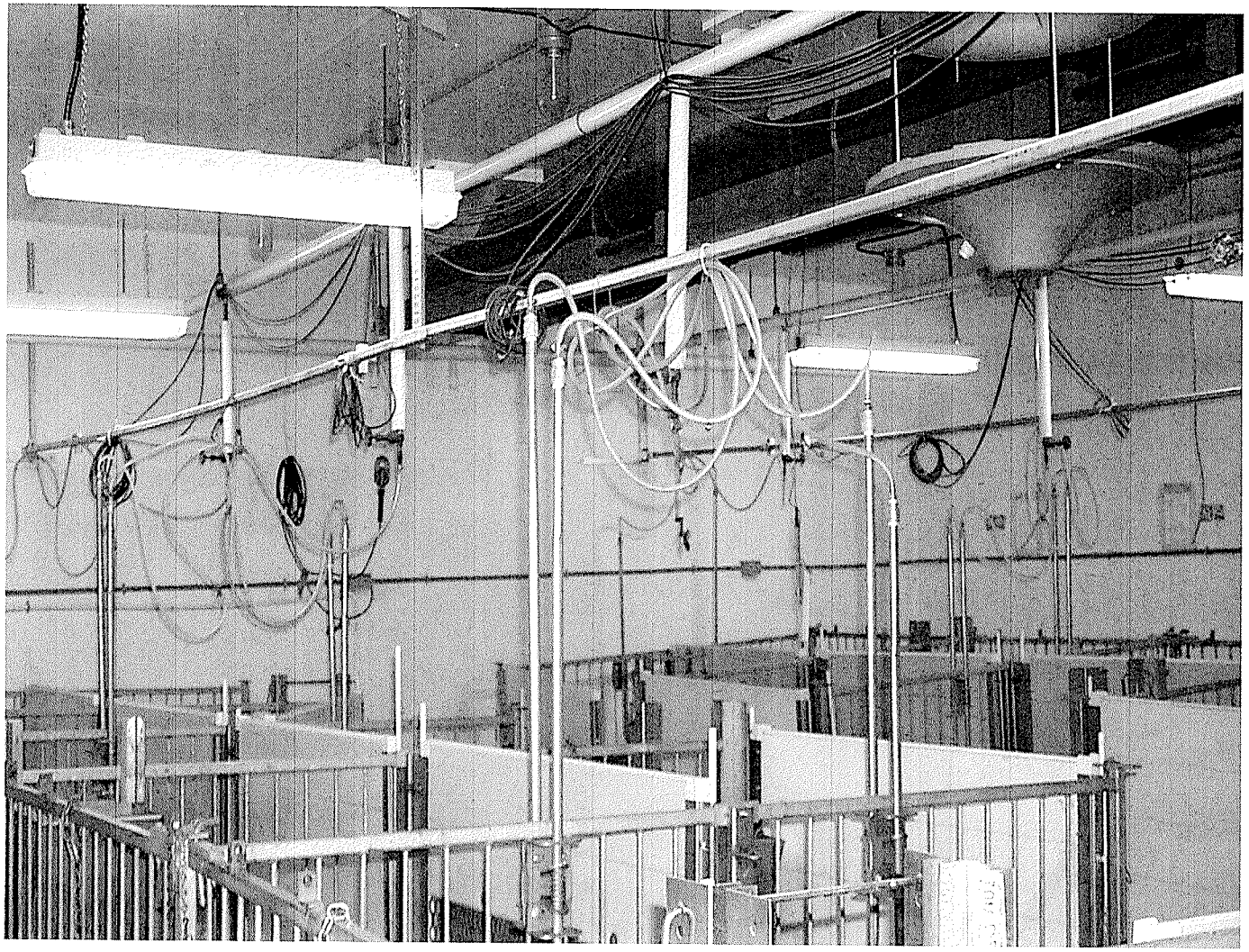


Table 1. Fed rations for the trials.

	Trial Onset	Average of 90 kg	2ndE Breeding
Commercial Diet	Gilt Grower	Gilt Finisher	Dry Sow
Crude Protein (%)	17.0	16.5	14.0

Rations met or exceeded the National Research Council (NRC) (1998) recommendations. Gilts had free access to water at all times through a nipple waterer in their pen.

3.4 Estrus Detection and Breeding

3.4.1 Boar Exposure & Estrus Stimulation/Detection

Beginning at an average age per room of 152 d, gilts were allowed fence-line contact with 1 of 2 mature boars (> 15 months of age), which were used in rotation. Boar exposure was initially used for estrus stimulation in the gilt rooms and occurred daily for approximately 10 minutes per room once a day for 3 weeks. Estrus detection and further stimulation was carried out with, or without boar exposure and/or Sex Odour Aerosol (SOA) (Intervet Canada Ltd., Whitby, ON, Canada). The total numbers of day that each treatment received boar exposure is outline in Table 2.

Table 2. Total boar exposure days per treatment and trial.

	HPS	MH	MH+HPS	T8
T1	51	53	50	52
T2	56	59	58	61

Signs of estrus and standing heat (STH) in the gilts were monitored twice daily at approximately 0900 h and 1500 h. To reduce variability, estrus checks were conducted consistently by the same trained individuals. Physical indicators assessed included: redness and swelling of the vulva with (or without) a sticky, mucous vaginal discharge and willingness to stand to back pressure. Behavioural signs observed included; increased nervousness/restlessness and characteristic agitated grunting. Start of estrus onset was defined by the first observed STH. End of estrus was recorded as the last observed STH. Onset and duration of the pubertal estrus (PubE) and 2ndE were recorded.

3.4.2 Estrous Cycles

Gilts not displaying a PubE by 216 to 222 d of age were shipped to the abattoir and their reproductive tracts recovered. This cut-off age range was used to allow for observation of puberty onset beyond the average age of 180 days, while ensuring a 2ndE would be observable within the trial period.

Gilts not displaying a 2ndE by an average of 32 d (range of 26 to 37 d) after the first STH of PubE were shipped to the abattoir and their reproductive tracts recovered. Gilts not bred at 2ndE were observed for onset and duration of the 2ndE and then shipped to the abattoir and their reproductive tracts recovered.

3.4.3 Breeding & Gestation

At the 2ndE gilts were bred by AI. All semen used was provided by Cotswold Canada Ltd. (Winnipeg, Manitoba, Canada); stored at 18 °C and used within the

recommended 4 d (42 h if not in a cool room). To reduce variability, AI was conducted during the trial by 1 of 4 specifically trained people. Gilts were inseminated at first observed STH then twice more at 12 h intervals.

Gilts were checked for pregnancy status by Renco Preg-Tone ultrasound (Renco Corporation, Minneapolis, MN, USA) between 26 to 32 d after breeding and were shipped to the abattoir where they were killed the following day. The complete reproductive tract (vulva, vagina, cervix, uterus, ovaries) was recovered and returned to the laboratory for examination.

3.5 Ovulation Rate and Embryo Survival

Reproductive tracts were kept at 4 °C until examined within 72 h of slaughter. Ovaries were inspected for numbers of corpora hemorrhagica (CH), corpora lutea (CL), corpora albicans (CA), cysts (follicular or luteal) and overall appearance. To ensure that all CL were counted, random slices were made into the ovary. Ovulation rate was defined as the number of CL.

Each uterine horn was examined for external indications of placentation sites. Incisions began at the oviduct and continued to the cervix, cutting through the myometrium and endometrium to reach the embryos in the lumen. Embryos were removed from the uterus and counted. The allantoic membrane was removed and each embryo was measured for crown-rump length. Gross evaluation of each embryo was made in order for classification as normal, or abnormal. Abnormal embryos were those with blood in their amniotic fluid, were obviously degenerating, or were less than half the size of the other

uterine embryos. Using the assumption that fertilization was 100 per cent, embryo survival for each gilt was calculated as the number of normal embryos divided by the ovulation rate.

3.6 Ear Vein Catheterization for Frequent Blood Sampling

Randomly selected gilts in each treatment underwent ear vein cannulation at 24 to 72 h prior to 148D and/or 19 d after first puberty STH, which was presumed proestrus. Each procedure was completed within 30 minutes and followed aseptic conditions. At the time of cannulation an intramuscular injection of Pen G Procaine (Langford, ON, Canada) was given at a weight appropriate dose to further reduce risk of infection.

For cannulation, gilts were restrained in their pens by a nose snare. The selected ear, crown of head and dorsal region of neck and shoulders were shaved. The ear was washed with a Hibitane (Wyeth-Ayerst Canada, Inc., Montreal, PQ, Canada) and water mixture, dried, swabbed with Betadine (Purdue Frederick Inc., Pickering, ON, Canada), dried and an elastic band tourniquet was placed around the base of the ear. The ear was then wiped with 70% isopropyl alcohol. A 3.8 centimetres (cm) 16 gauge needle (Becton Dickinson and Company, Franklin Lakes, NJ, USA) was inserted into a prominent ear vein through which a guide wire (A Cook Group Company, Bloomington, IN, USA, O.D. 0.032 in) was inserted. The guide wire was fed to the base of the ear, the tourniquet removed and the wire was continued through the ear-head junction. Once visual recognition identified that the guide wire had cleared the ear-head junction the needle was removed and a 1.005 m piece of catheter tubing(Dural Plastics and Engineering, Auburn, NSW, 2128, Australia, O.D. 1.5 mm, I.D. 1.00 mm) was threaded over the wire with beveled end leading. When

approximately 0.45 m of the catheter tubing was inserted, the guide wire was slowly drawn out. The catheter was fitted with a 19 gauge blunt tipped needle (Becton Dickinson and Company, Franklin Lakes, NJ, USA) and immediately tested for patency by injecting 3 millilitres (ml) of sterile 0.9% normal saline and withdrawing blood. Upon withdrawal of full blood, 2.5 ml of 100 (USP) heparanized saline was injected and the catheter plugged by a PRN adapter (Becton Dickinson, Sandy, UT, USA). Catheter patency was maintained with 1.5 ml of 10 USP heparanized saline injected into the cannula between samples.

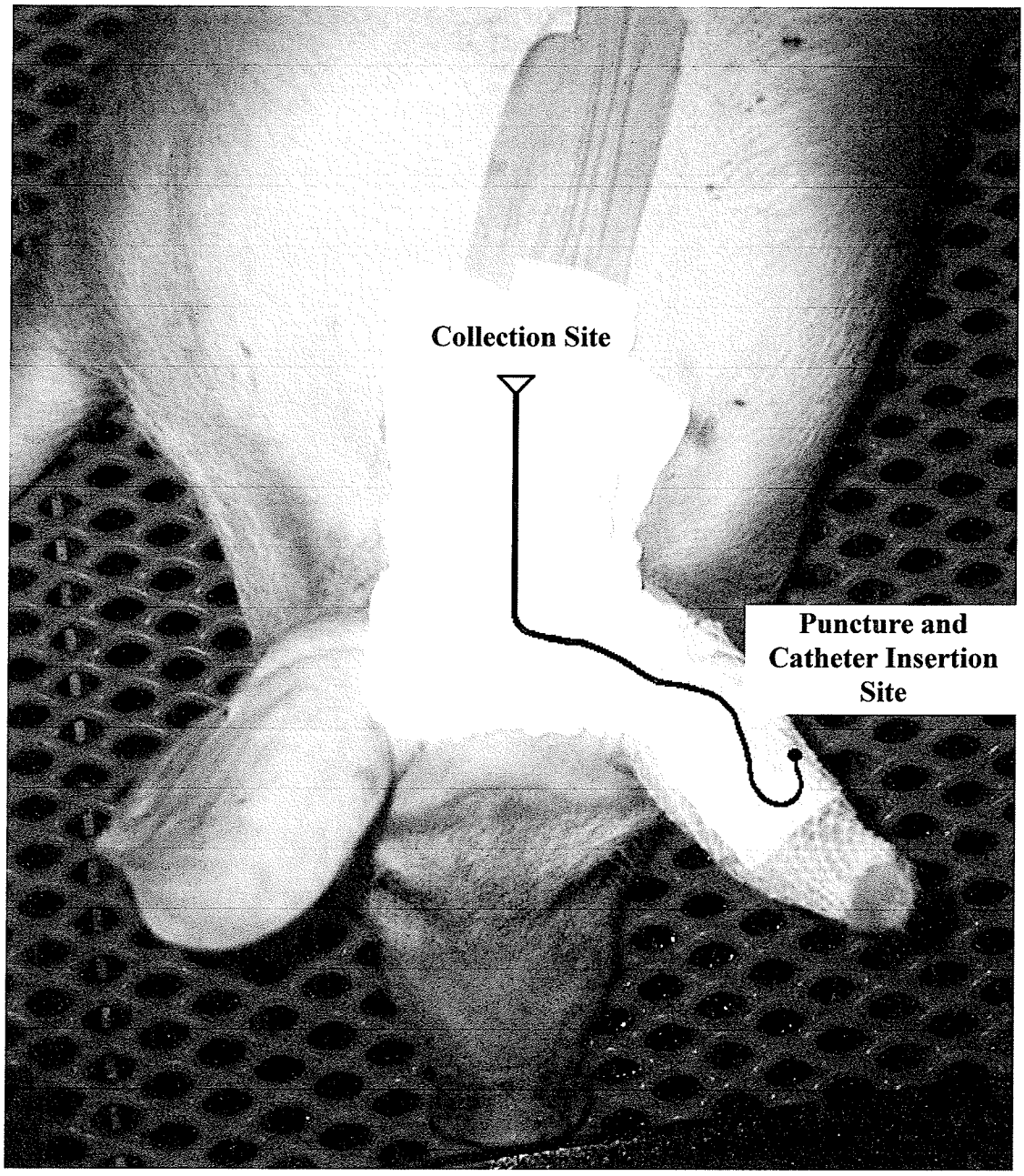
The 10 000 USP stock solution of heparin was prepared with 1.330 g of Sigma Heparin Sodium Salts from porcine intestinal mucosa (188 USP/mg) (Sigma Chemical Co., St. Louis, MO, USA, H-9399, Lot # 117H0805) in 25 ml sterile normal 0.9% saline. Saline and heparinized saline were kept in a 4 °C refrigerator and warmed to room temperature prior to use.

Figure 8 illustrates the puncture and collection sites and how the catheter was attached to the gilt. The catheter insertion site was sealed by a few drops of Vetbond (3M Animal Care Products, St. Paul, MN, USA) to help close the wound and secure the catheter. A small strip of Kendall Curity porous fabric tape (The Kendall Company, Mansfield, MA, USA) was used to create a butterfly bandage that was adhered to the ear by Vetbond to further anchor the catheter. Pieces of 7.6 cm wide Elastoplast tape (Smith and Nephew Inc., Lachine, PQ, Canada) were glued to the shaved regions on the back of the ear, head, neck and shoulders by Ag-Tek cement (Kane Enterprises, Sioux Falls, SD, USA). The remaining catheter was laid out over the Elastoplast and secured by 2.5 and 5.0 cm wide pieces of Kendall Curity tape until it was collected into a 57 gram sample bag (Fisher Scientific,

Nepean, ON, Canada) secured on the dorsal region of the shoulders. From this site, the catheter was readily manipulated for sampling. To further protect the taping of the catheter insertion site on the ear, a piece of Surgilast surgical netting (Glenwood Laboratories, Oakville, ON, Canada), cut to the same length of the ear, was placed over the ear and glued with Ag-Tek cement at ear base and tip.

The catheter was removed between 24 and 72 hours after the last serial blood sample.

Figure 8. Dorsal view of site of puncture and view of tape and catheter attachment.



3.7 Blood Collection

3.7.1 Serial Blood Samples

A total of 108 gilts were serially blood sampled at 148D and 2ndE. The specific number of gilts collected from on each sample day, trial and treatment are outlined in Table 3.

Table 3. Number of gilts used for serial blood collection by day, trial and treatment.

	Sample Day	HPS	MH	MH+HPS	T8
	148D	7	6	6	6
T1	DB42ndE	4	3	3	2
	Dof2ndE	4	2	4	2
	148D	4	5	5	2
T2	DB42ndE	5	7	5	5
	Dof2ndE	6	6	5	6
	148D	11	11	11	8
Total	DB42ndE	9	10	8	7
	Dof2ndE	10	8	9	8

At 148D gilts were serially blood sampled for 24 h to provide samples of pre-pubertal endocrine levels. Collection for 2ndE began at 20 days after beginning of PubE STH and ended after Dof2ndE collection in order to provide post-pubertal samples at the day before and day of estrus . Four gilts were sampled beginning at 19 d post puberty STH at 1600 h

(approximately 4 to 5 h after catheter insertion) because of indications of imminent estrus. At 2ndE blood collection the longest duration of sampling was 5 days. Serial blood collection for each sample day occurred over a 24 h period. Blood samples (5 ml) were collected every 20 minutes during 0800 h to 1600 h and 2000 h to 0400 h and every hour (10 ml) during 1600 h to 2000 h and 0400 h to 0800 h.

Serial blood samples were collected from catheters in 3 steps: 1. Heparinized saline present in the catheter was drawn up with full blood (approximately 2 ml). 2. A new, sterile syringe (Becton Dickinson and Company, Franklin Lakes, NJ, USA) was then used to collect the appropriate blood sample volume. 3. The catheter was flushed with 1.5 ml of 10 USP heparanized saline. Collected blood samples were stored in 16 x 100 mm borosilicate glass tubes (Fisher Scientific, Pittsburgh, PA, USA), covered with tin foil and set in a cool water bath. After approximately 8 hours, the samples were placed in a 4 °C cold room to await processing.

3.7.2 Single Blood Samples

Single blood samples were collected from all gilts not catheterized at 148D (except two gilts that were single sampled at 149 days of age) to provide confirmation of pre-pubertal status (<0.500 ng/ml of P4). Single blood samples were also taken from gilts not displaying PubE or 2ndE by specific trial dates. Criteria for these gilts were: no display of pubertal STH after a set number of days of boar exposure (T1: 28 days, T2: 47 days), no display of PubE by 216 to 222 days of age, no display of 2ndE by 32 days post PubE STH. These samples provided hormone profiles for detection of silent heat. Single blood samples were also taken

at day 27 to 32 of gestation to provide hormone profiles that could relate to embryo survival.

Single blood samples were collected by jugular venipuncture using a 10 ml Vacutainer tube with no additives (Becton Dickinson and Company, Franklin Lakes, NJ, USA), a 1.5 inch 20 gauge Vacutainer needle (Becton Dickinson and Company, Franklin Lakes, NJ, USA), and a Vacutainer needle holder (Becton Dickinson and Company, Franklin Lakes, NJ, USA). Gilts were restrained by a nose snare during the collection process.

3.7.3 Blood Processing

Both serial and single blood samples were stored in a 4 °C cold room for up to 30 hours after which they were centrifuged for 20 minutes at 1400 x g. Serum was removed and placed in labeled scintillation vials and stored at -20 °C until analysis.

3.8 Hormone Analyses

3.8.1 Progesterone

Serum P4 was analyzed in 6 assays by means of a commercial solid-phase radioimmunoassay (RIA) in a commercial kit (Coat-A-Count, Diagnostic Products Corporation, Los Angeles, CA, USA). All assay components were at room temberature for standard and sample serum, 100 microlitres (μl) aliquots were micropipetted into polypropylene tubes coated with P4 antibodies. One ml of ^{125}I labeled P4 tracer was added within a 10 minute period. This mix was incubated for 3 hours at room temperature after which the supernatant was decanted, thus isolating the precipitated antibody-bound fraction.

Radioactivity was counted for 1 minute in a gamma counter (LKB Wallac 1282 Compu Gamma Universal Gamma Counter). The working range of the standard curve was 0.1 to 10 ng/ml of P4 standard with a sensitivity of 0.058 ng/ml. The total counts were 57 393 counts per minute (cpm) with a maximum binding (MB) of 50.87% and non-specific binding (NSB) of 1.48%. The intra- and inter-assay coefficients of variation were 12.48% and 27.76%, respectively.

3.8.2 Estradiol

Estradiol was analyzed by a double-antibody RIA commercial kit (Ultra-Sensitive Estradiol RIA, Diagnostic Systems Laboratories, Inc., Webster, TX, USA) in 8 assays. Two hundred μl of room temperature serum from each of standards, controls and samples were pipetted into polypropylene tubes (VWR Scientific Products, West Chester, PA, USA). After 100 μl of rabbit anti-E2 serum was pipetted into the tubes (excluding the NSB and total count (T) tubes), they were vortexed and incubated for 1 hour at room temberature. A volume of 100 μl of ^{125}I-labelled E2, was pipetted into all the tubes, which were then vortexed, and incubated for 2 hours at room temperature. One ml of precipitating reagent (2nd antibody) was added to the tubes, which were then vortexed and left to stand for 15 minutes. The tubes were then centrifuged at 1500 x g for 20 minutes. The supernatant was decanted and tubes were read for radioactivity in a gamma counter for 1 minute each. The working range of the standard curve was 2.5 to 250 pg/ml of E2 standard with a test sensitivity of 1.34 pg/ml. The total count was 55 932 cpm with a MB of 28.09% and a NSB of 3.91%. The intra- and inter-assay coefficients of variation were 16.28% and 23.87%,

respectively.

3.8.3 Cortisol

Serum cortisol was analyzed in 8 assays by a commercial solid-phase RIA (Coat-A-Count, Diagnostics Products Corporation, Los Angeles, CA, USA). For all room temperature standards, controls and samples, 25 μl volumes were pipetted into polypropylene tubes coated with cortisol antibodies. To these tubes 1.0 ml of ^{125}I-labelled cortisol tracer was added. Tubes were incubated for 45 minutes in a 37 °C waterbath. The supernatant was decanted and radioactivity within the tube was read for 1 minute in a gamma counter. The working range of the standard curve was 5 to 200 ng/ml of cortisol standard with a sensitivity of 2.7 ng/ml. The total counts were 64 589 cpm with a MB of 59.56% and NSB of 1.50%. The intra- and inter-assay coefficients of variation were 7.41% and 11.35%, respectively.

3.8.4 Luteinizing Hormone

Luteinizing hormone was analyzed by Prairie Diagnostic Services in the Department of Veterinary Physiological Sciences in the Western College of Veterinary Medicine at the University of Saskatchewan. As described by Kingsbury and Rawlings (1993), the method of analysis was a heterologous double antibody RIA using lyophilized porcine LH standard (USDA-pLH-B:I). The primary antibody was raised in rabbits against bovine LH. The secondary antibody was raised in sheep against rabbit gamma-globulins. The working range of the standards was 0.0625 to 8.0000 ng/ml. The tracer, ^{125}I-labelled bovine LH provided 13 000 to 18 000 cpm in 200 μl.

All pools and samples were analyzed in 28 individual assays. Individual assay completion required 4 days:

Day 1: Two hundred μl aliquots of standard, sample and primary antibody were added to all tubes except the total and NSB tubes. The NSB tube received 200 μl of 0.2% NRS. All tubes were vortexed and incubated over night.

Day 2: All tubes received 200 μl of tracer, were then vortexed and left to incubate in a cold room overnight.

Day 3: All tubes, except the total counts tube receive 500 μl aliquots of secondary antibody and 5% polyethylene glycol. These were then vortexed and incubated in a cold room overnight.

Day 4: All tubes, except total count, were centrifuged for 15 minutes and the supernatant decanted. The pellets were measured for radioactivity in a gamma counter where each tube was read for 1 minute.

The intra- and inter- assay coefficients of variation were 8.28% and 25.41%, respectively. The NSB was < 5% and the average sensitivity of the assays, defined as the lowest standard concentration different than zero, was 0.01 ng/ml.

3.8.5 Follicle Stimulating Hormone

Follicle stimulating hormone was analyzed by Prairie Diagnostic Services in the Department of Veterinary Physiological Sciences in the Western College of Veterinary Medicine at the University of Saskatchewan. The method of analysis was a homologous double antibody RIA as described by Kingsbury and Rawlings (1993) using lyophilized

porcine FSH standard (USDA-pFSH-1-2). The primary antibody for all of Trial 1 analysis and 148D period of Trial 2 had an initial working dilution of 1:10 000 (USDA-398-04P); for the remaining sample periods of Trial 2 the initial working dilution was 1:50 000 (AFP2062096). The primary antibody was prepared in 0.2% NRS. The secondary antibody was raised in sheep against rabbit gamma-globulin in PBS with gel at a dilution of 1:130. The working range of the standards was 0.125 to 16.000 ng/ml. The tracer, I^{125} porcine FSH (USDA-pFSH-1-2) provided 12 000 to 15 000 cpm in 100 μL.

The assay procedure for FSH was similar for that of LH. The pools and samples were analyzed in 19 individual assays. The intra- and inter- assay coefficients of variation were 7.99% and 22.87%, respectively. NSB for the assay was less than 5% and the sensitivity of the assay, defined as the lowest standard concentration not equal to zero, was 0.01 ng/ml.

3.8.6 Melatonin Analysis

Melatonin was analyzed in 4 assays by means of a commercial double-antibody RIA (BÜHLMANN, American Laboratory Products Co., Windham, NH, USA). Four hundred microlitres (μl) of incubation buffer, standards, controls or samples were pipetted into labelled polystyrene tubes (Sarstedt, PQ, Canada) to which 100 μl of MEL antibody was added, followed by 100 μl of ^{125}I-labelled MEL tracer. The tubes were incubated for 20 hours in a 4 °C cold room. The second antibody was added in 100 μl volumes to all tubes and tubes were then incubated for 15 minutes in a 4 °C cold room. Cold, nanopure water (1 ml) was then added to the tubes and these centrifuged for 2 minutes at 2000 x g. The supernatant was decanted and the precipitate counted for 2 minutes in a gamma counter. The

standard curve ranged from 0.5 to 50 pg/ml of MEL standard with a sensitivity of 0.44 pg/ml. The total counts were 10 952 cpm with a MB of 36.41% and NSB of 3.48%. The intra- and inter- assay coefficients of variation were 8.78% and 10.22%, respectively.

Prior to analysis, MEL was extracted from serum by means of C18 reversed phase extraction columns (BÜHLMANN, American Laboratory Products Co., Windham, NH, USA). Columns were prepared for extraction by flushing of the sorbent material as outlined in the instructions. Immediately following column preparation, 0.8 ml of sample was pipetted into the column, which was centrifuged for 1 minute at 200 x g. Extraneous serum components were washed twice out of the column with the addition of 1 ml of 10% methanol in water and centrifuged at 500 x g for 1 minute, followed by 1 ml of HPLC grade hexane (Fisher Scientific, Fair Lawn, NJ, USA) and centrifuged at 500 x g for 1 minute. Elution of MEL from the column was achieved by washing it with 1 ml of methanol into a clean 12 x 75 mm borosilicate glass tube (Fisher Scientific, Pittsburgh, PA, USA). The methanol was evaporated to dryness using a 37 °C heating block and a stream of particle free nitrogen (Welders Supply, Winnipeg, MB, Canada). The sample was reconstituted for analysis with 0.8 ml of incubation buffer.

3.9 Statistical Analysis

Level of significance was defined as $P<0.0500$ and a trend was defined as $0.5000<P<0.1000$.

3.9.1 Treatment Rooms

Light intensity was analyzed as a complete randomized design using the General Linear Model (GLM) procedure of the Statistical Analysis System 8 (SAS). Trial, treatment and trial by treatment were tested with an error term of pen within trial by treatment.

Room temperature was analyzed using the GLM procedure of the SAS. The analysis was a split plot design testing trial and treatment using an error term of trial by treatment. The sub plot tested time of day alone and against trial and treatment with an error term of time by trial by treatment.

3.9.2 Growth Performance and Feed Intake

Weight, feed intake and BF were arranged as a split plot design. These results were corrected using a model 3 quadratic polynomial to provide a value for age at puberty and an age/value at 110 kg. These results were used to calculate average daily gain (ADG), average daily feed intake (ADFI) and feed efficiency (FE).

All results were of a completely randomized design and analyzed by the GLM procedure of SAS. Analysis was of trial, treatment and trial by treatment with a default error term.

3.9.3 Reproductive Performance

The specific numbers of gilts per treatment that did not achieve pubertal or post-pubertal estrus were analyzed by chi square.

Age and duration of the pubertal and postpubertal estrus, gestation length, ovulation

rate, embryo survival and estrus interval were analyzed by the GLM procedure of SAS as a completely randomized design. Trial, treatment and trial by treatment were analyzed with error as a default.

The influence of frequent blood sampling on pubertal and post-pubertal duration, ovulation rate and embryo survival was analyzed by the GLM procedure. Sample status and treatment were analyzed with error as a default.

3.9.4 Endocrine Values

For all hormones, single blood samples and overall means from frequent blood sampling periods were analyzed as a complete randomized design by the GLM procedure, except for LH and FSH which were analyzed by the MIXED procedure. Analysis of trial, treatment and trial by treatment was conducted with error as a default. Cortisol showed a significant difference by treatment, therefore the least square difference (LSD) multiple comparison test was used. LSD was used because other multiple comparison tests did not provide results at an alpha equal to 0.05. As well, contrasts were conducted between treatments for cortisol.

Endocrine results from frequent collection over individual sample periods were analyzed as a split plot by the GLM procedure, except for LH and FSH which were analyzed as a split plot by the MIXED procedure. Analysis was of trial, treatment and trial by treatment with an error term of gilt within trial by treatment. For cortisol there was further analysis by the sub-plot of time, time by trial and time by treatment. For melatonin there was further analysis by period, period by trial and period by treatment. As well, contrasts for

melatonin at the DB4E sample period were conducted between treatments and trials.

For examination of endocrine results between all three sample periods a further effect of day, day by trial and day by treatment was analyzed with error as default. At this point in analysis, another error term was used for MEL which was gilt within period by trial by treatment.

Analysis of LH and FSH for number and amplitude of peaks followed the method of Evans et al (1994). Hormone peaks within a sample period profile were defined as 3 standard deviations above the mean for the specific animal. Baseline concentration was determined from the resulting mean after the removal of the peaks. Baseline concentration and mean peak amplitude was analyzed by the MIXED procedure. Number of pulses per treatment were analyzed by chi-square.

CHAPTER 4

RESULTS

4.1 Treatment Rooms

4.1.1 Light Intensity

Based on manufacturer specifications, lights were initially installed to obtain similar light intensities. However, analysis revealed that overall light intensity was significantly different (P=0.0165) between HPS and MH, as were Start (P=0.0393) and Mid (P=0.0056) light intensities. The end of trial light intensity measurement did not show significant treatment differences (P=0.1013). Light intensity treatment results are shown in Table 4.

Table 4. Light intensity across treatments.

Light Intensity (lx)	HPS	MH	MH+HPS	T8	P-Value
Overall	256 ± 12^{a}	311 ± 11^{b}	$287 \pm 12^{a,b}$	$280 \pm 12^{a,b}$	0.0165
Start	266 ± 13^{a}	322 ± 13^{b}	$304 \pm 14^{a,b}$	$290 \pm 14^{a,b}$	0.0393
Mid	252 ± 13^{a}	319 ± 12^{b}	$278 \pm 13^{a,b}$	$280 \pm 13^{a,b}$	0.0056
End	249 ± 12	293 ± 12	280 ± 13	274 ± 12	0.1013

values are LS mean ± SEM
$^{a, b}$ significant difference in a row at P<0.0500

Light intensities were similar between trials except for the mid trial measurement in

which T1 light intensity was greater than T2 (P=0.0110) (Table A1.1, Appendix 1). There were no significant differences in trial by treatment interactions.

4.1.2 Temperature

The daily average maximum(max) temperature reading showed a tendency towards treatment differences (P=0.0713). Multiple comparison tests (Bonferroni and Contrasts, at alpha = 0.05 to 0.10) were unable to show the specific treatment difference. For the daily average minimum (min) temperature reading MH was significantly warmer than T8 (P=0.0269) but, was still within the gilts thermocomfort zone (Connor, 1993). Table 5 shows the daily average max and min temperature results across all treatments.

Table 5. Daily average maximum and minimum temperature results.

Temperature Readings (°C)	HPS	MH	MH+HPS	T8	P-Value
Max	21.9 ± 0.2	22.4 ± 0.2	22.2 ± 0.2	21.3 ± 0.2	0.0713^
Min	18.5 ± 0.1^{a,b}	19.1 ± 0.1^{a}	18.6 ± 0.1^{a,b}	17.9 ± 0.1^{b}	0.0269

values are LS mean ± SEM
^{a,b} significant difference at P<0.0500
^trend not indicated by Bonferroni set between 0.0500 and 0.1000

The daily AM and PM temperature readings for max and min were significantly different (P=0.0002 and P=0.0027, respectively. Figures A1.1 to A1.8 in Appendix 1 illustrate the AM and PM temperature readings of each treatment room within each trial.

Between trials, daily temperature readings were significantly higher and lower in T1

(max: 24.0 ± 0.1; min: 18.3 ± 0.1), conducted May to September, than T2 (max: 20.0 ± 0.1; min: 18.8 ± 0.1), conducted January to May (max: P=0.0002; min: P=0.0334).

As well, there were significant interactions in treatment by time for daily max temperature (P=0.0002) and trial by time for both max (P=0.0006) and min (P=0.0046) temperatures (Tables A1.2 and A1.3, Appendix 1).

Daily Phason temperatures readings were within the ranges for AM and PM maximum/minimum thermometer readings (Tables A1.4 to A1.8, Appendix 1).

4.2 Growth Performance & Feed Intake

Gilts were of similar age and weight at the start of both trials and showed no differences in these measurements by the end of the trials (Table A2.1, Appendix 2).

4.2.1 Finishing Period

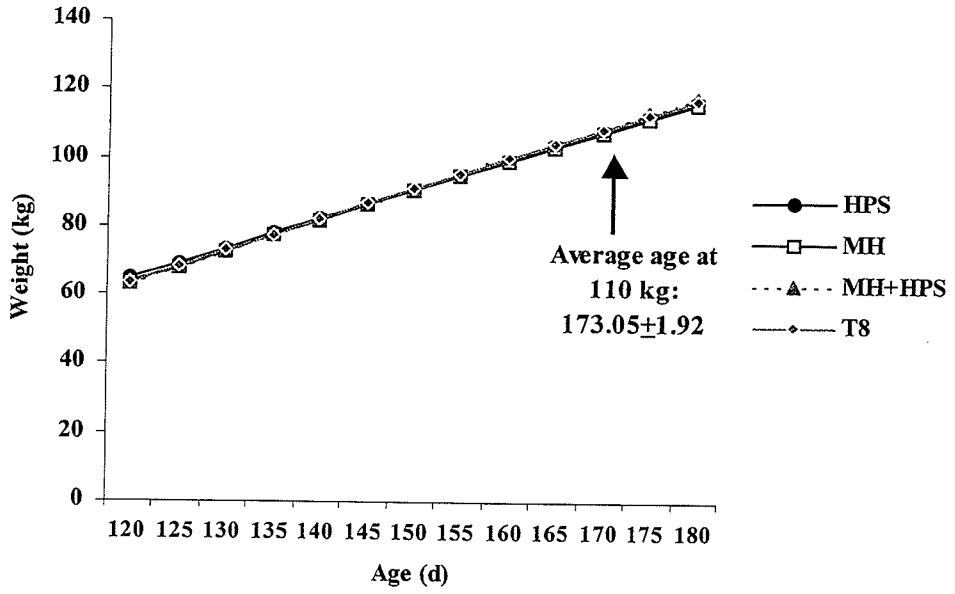
The finishing period is defined as that time period from the start of the trial to a finisher weight of 110 kg, which approximates market weight. There were no significant treatment differences in age at 110 kg, total gain, average daily gain (ADG), and BF (Table A2.2, Appendix 2). Figure 9 illustrates the pattern of weight gain and BF depth throughout the finishing period of combined trials.

Average growth performance across trials for the finishing period is shown in Table A2.3 of Appendix 2. Although gilts in T1 were approximately 2 d older at the start of the trial than those in T2 (P=0.0004), T2 gilts took an average of 6.5 more days to reach 110 kg (P=0.0001). Gilts in T1 had a starting weight approximately 5.5 kg heavier than T2

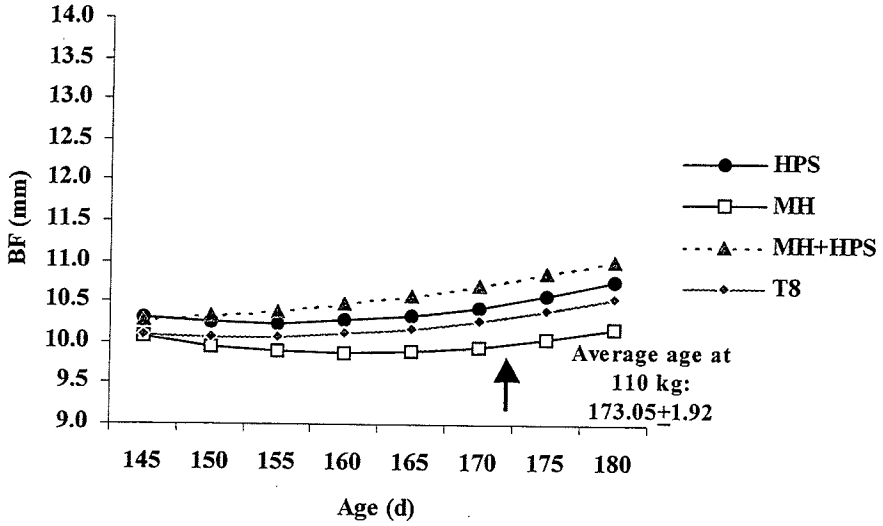
(P=0.0001), with ADG similar between trials. Final BF was about 1.1 mm less for gilts in T1 (P=0.0001). There were no significant differences in trial by treatment interactions for growth measurements in the finishing period.

Figure 9. (a) Growth curve and (b) BF measurements throughout the finishing period. Back fat measures began at 80 kg weight.

(a)

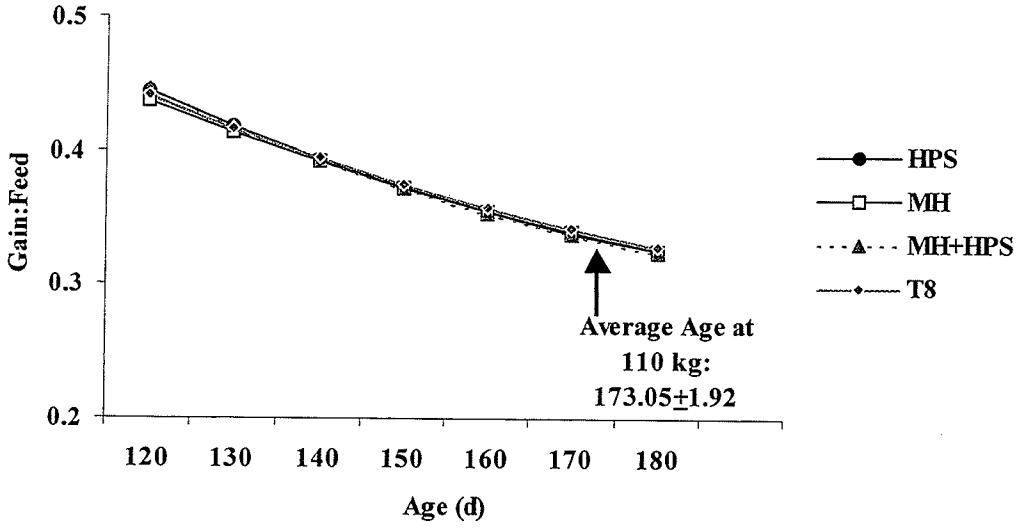


(b)



Although total feed intake was greater for T2 gilts (P=0.0001), there were no significant differences in average daily feed intake (ADFI) and gain:feed between treatments or trials (Tables A2.4 and A2.5, Appendix 2). Figure 10 illustrates Gain:feed throughout the finishing period. There were no significant differences in trial by treatment interactions for feed intake in the finishing period.

Figure 10. Gain:feed throughout the finishing period.



4.2.2 Pubertal Period

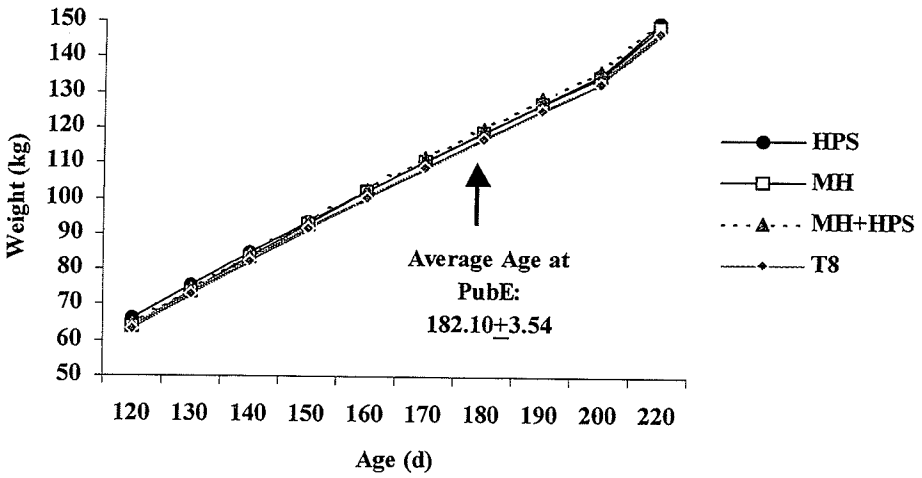
The pubertal period is defined as that time period from the start of the trial to the first estrus.

There were no significant treatment differences in weight at puberty, BF, total gain, and ADG (Table A2.6, Appendix 2). Figure 11 illustrates the patterns of weight gain and BF deposition throughout the pubertal period.

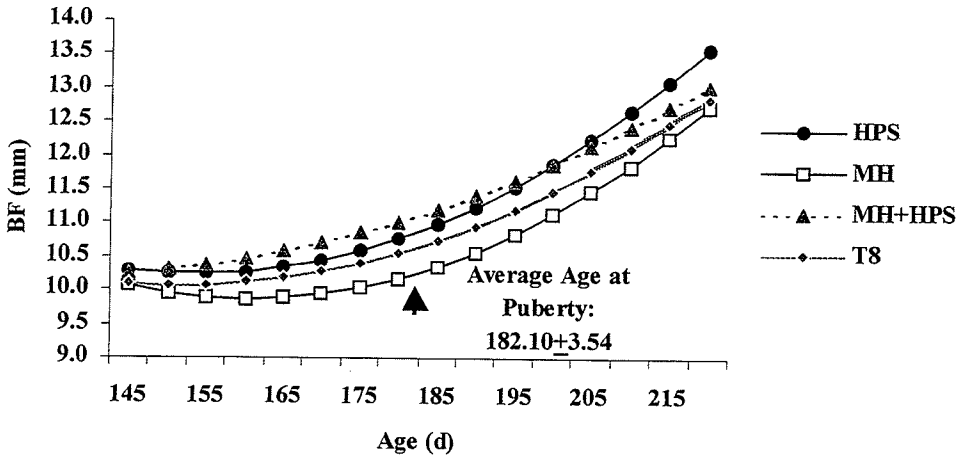
Across trials, there were no significant differences for weight at puberty, total gain, or ADG, although T1 BF was significantly lower than T2 (P=0.0002). Table A2.7 in Appendix 2 shows the trial differences for the pubertal period. There were no significant differences for trial by treatment interactions in growth during the pubertal period.

Figure 11. (a) Weight and (b) BF measurements for the pubertal period. Back fat was measured beginning at approximately 80 kg.

(a)

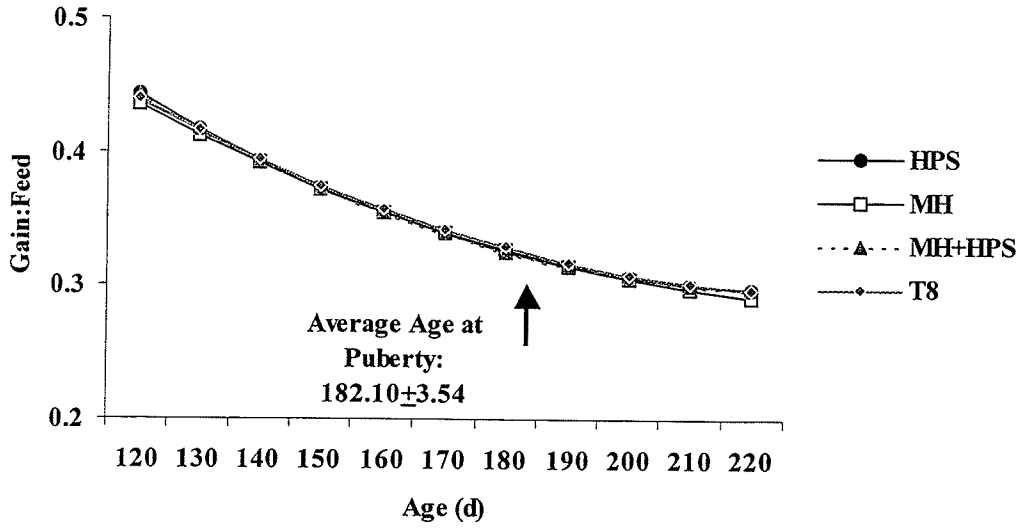


(b)



There were no significant treatment differences in total intake, ADI, and Gain:feed during the pubertal period (TableA2.8, Appendix 2). Figure 12 illustrates the pattern for Gain:feed throughout the pubertal period. There were no significant trial differences nor trial by treatment interactions in feed intake during the pubertal period (Table A2.9, Appendix 2).

Figure 12. Gain:feed throughout pubertal period.



4.3 Reproductive Performance

Age at PubE, duration of PubE, age at 2ndE, duration of 2ndE, estrus interval, end trial GestD, tract exam GestD, ovulation rate, and embryo survival rate were not different between treatments (Table 6). Note, that GestD is divided into end trial and tract exam due to the one day difference in end of trial shipping and slaughter. Figures 13 to 15 show

examples of gravid and prepubertal uteri, cycling and prepubertal ovaries, and an embryo around 30.7 ± 1.0 d. As well, numbers of gilts not attaining PubE, or 2ndE (i.e. behavioural signs of estrus) were not significant across treatments (Table 7).

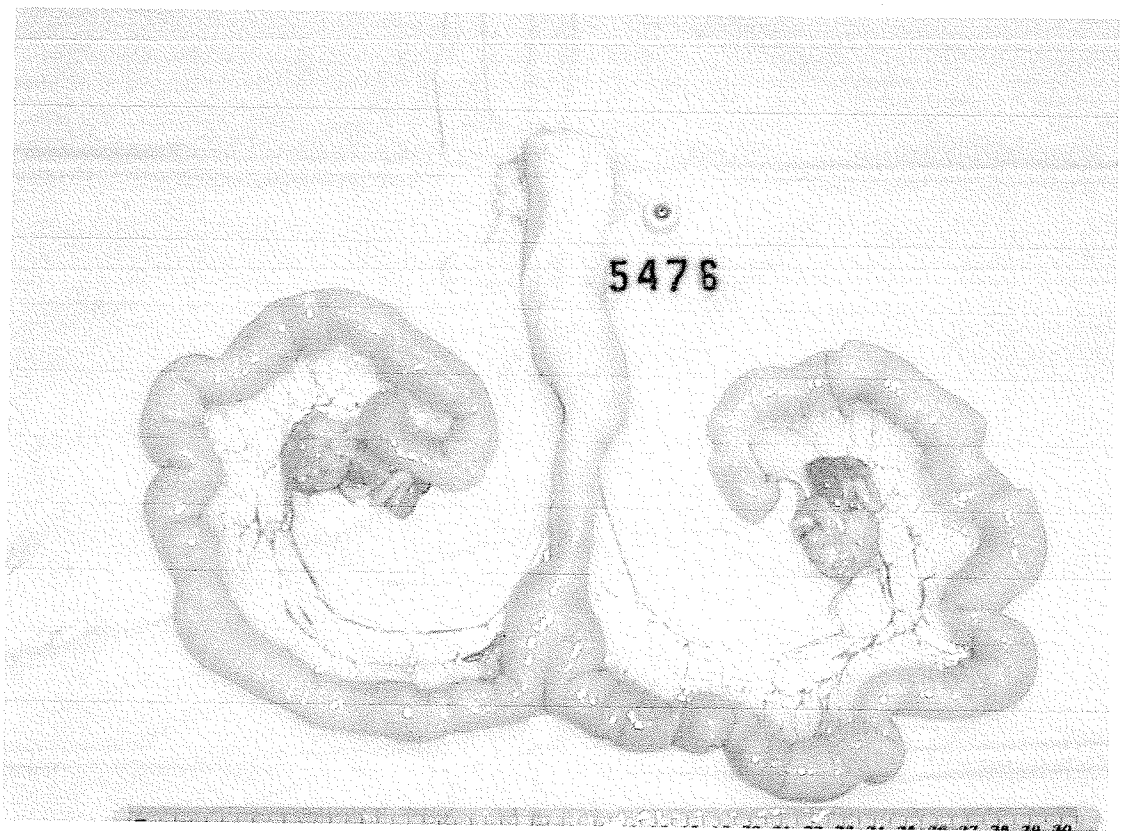
Table 6. Reproductive performance across treatments.

T1 & T2	HPS	MH	MH+HPS	T8	P-Value
Age at PubE (d)	182 ± 3	182 ± 4	178 ± 4	186 ± 4	0.4970
Duration of PubE (h)	33.8 ± 3.1	27.0 ± 3.4	27.3 ± 3.2	25.5 ± 3.4	0.2696
Age at 2ndE (d)	201 ± 4	204 ± 4	201 ± 4	207 ± 4	0.6245
Duration of 2ndE (h)	34.4 ± 2.7	30.7 ± 2.6	30.4 ± 3.0	35.0 ± 3.1	0.5464
Estrus Interval (d)	21.5 ± 0.4	21.7 ± 0.4	21.3 ± 0.4	21.5 ± 0.4	0.8834
End Trial GestD (d)	28.4 ± 0.3	28.0 ± 0.3	28.2 ± 0.3	28.1 ± 0.4	0.8174
Tract Exam GestD (d)	30.4 ± 1.0	29.3 ± 1.0	31.5 ± 1.0	31.5 ± 1.0	0.3420
Ovulation Rate	14.3 ± 0.7	13.6 ± 0.7	15.5 ± 0.7	15.1 ± 0.7	0.2449
Embryo Survival Rate (%)	86 ± 4	78 ± 4	80 ± 4	80 ± 4	0.2190

values are LS mean ± SEM

Figure 13. Photograph of prepubertal reproductive tract: (a) uterus and ovaries, and (b) ovary.

(a)



(b)

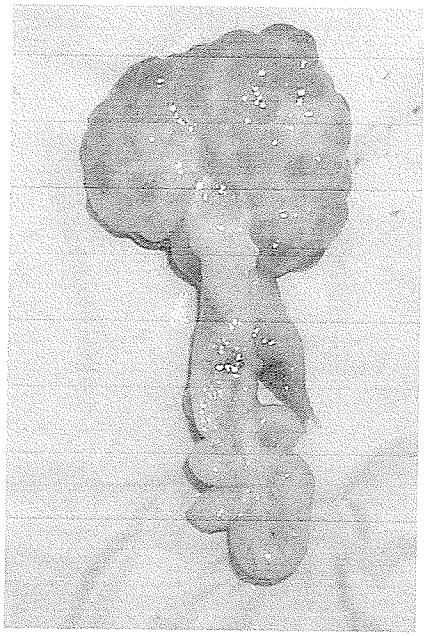
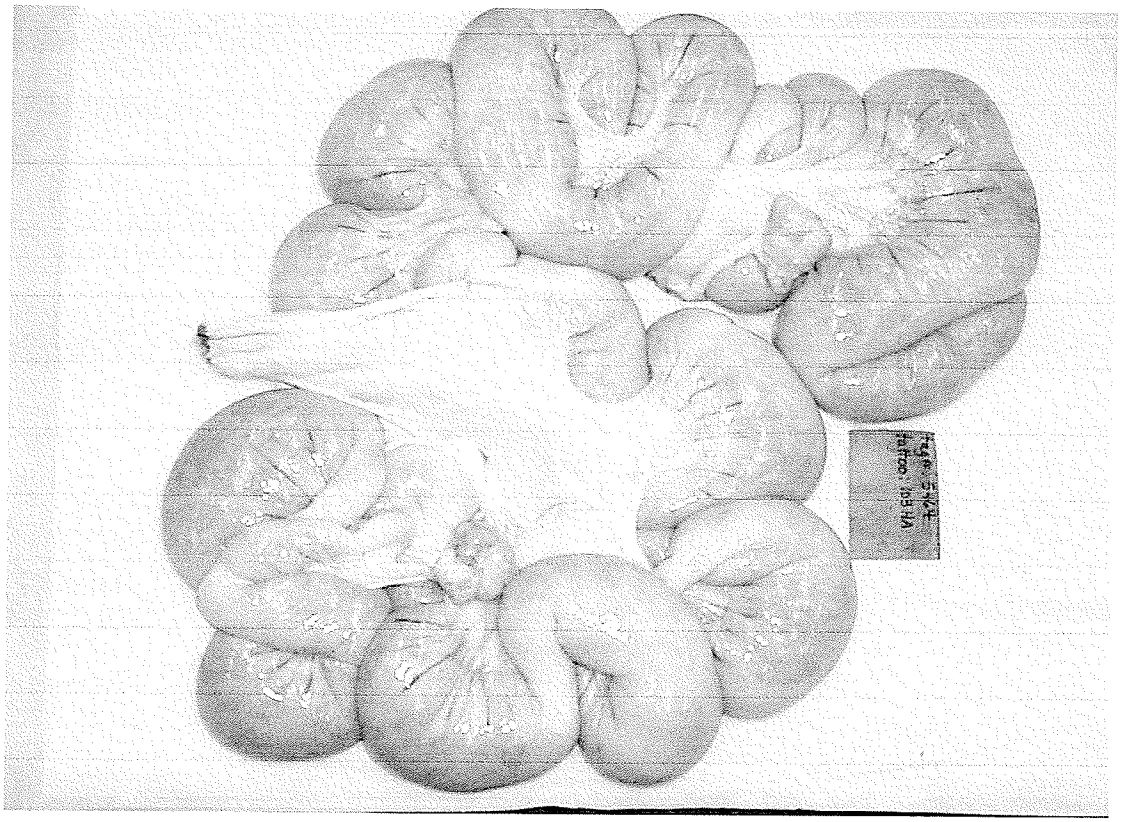


Figure 14. Photograph of gravid reproductive tract at an average gestation of 30.7 ± 1.0 d: (a) uterus and ovaries, and (b) ovary.

(a)



(b)

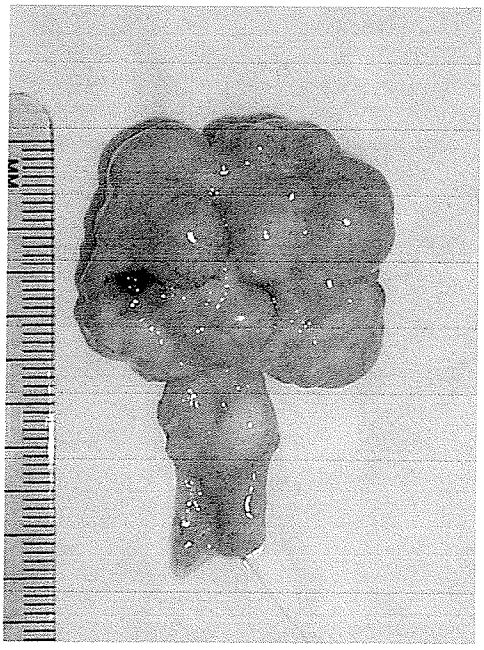


Figure 15. Photograph of embryo examined at an average of 30.7 ± 1.0 d.

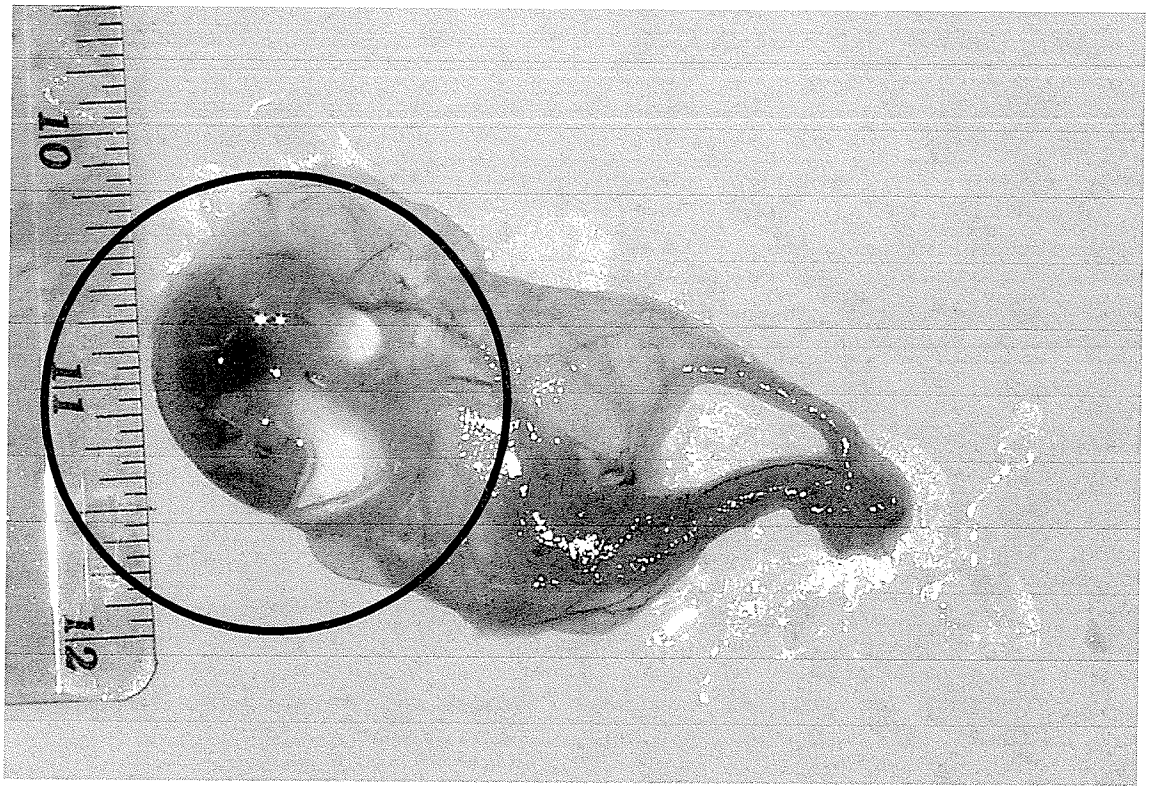


Table 7. Proportion of gilts not showing PubE and those not showing 2ndE.

	HPS	MH	MH+HPS	T8	Significance*
N per Treatment	26	26	24	25	No
Showing PubE	25	23	23	21	No
Showing 2ndE	22	23	21	19	No

*significance at chi-square value of 7.815

Reproductive performance by trial (Table A2.10, Appendix 2) showed no significant differences for age at PubE, age at 2ndE, ovulation rate, end trial GestD, or embryo survival rate. However, T2 gilts tended to have a longer PubE (P=0.0852), inter-estrus interval (P=0.0632), and did show a significantly longer 2ndE (P=0.0010). Because of a late slaughter date for 5 gilts in T1 (HPS: 5481; MH+HPS: 5468, 5450; T8: 5474, 5453) due to a holiday break in the slaughter plant, tract exam GestD was significantly greater than T2(P=0.0048); although over treatments there was no difference in this day. There were no trial by treatment interactions.

Comparisons of reproductive data from gilts serially blood sampled at 148D, DB42ndE and/or Dof2ndE and those not serially blood sampled demonstrated no significant differences for duration of 2ndE, ovulation rate, or embryo survival rate. Only duration of PubE was significantly different between gilts serially blood sampled at 148D (24.2 ± 2.4 hr) and those not serially blood sampled at 148D (31.7 ± 2.1 hr) (P=0.0228).

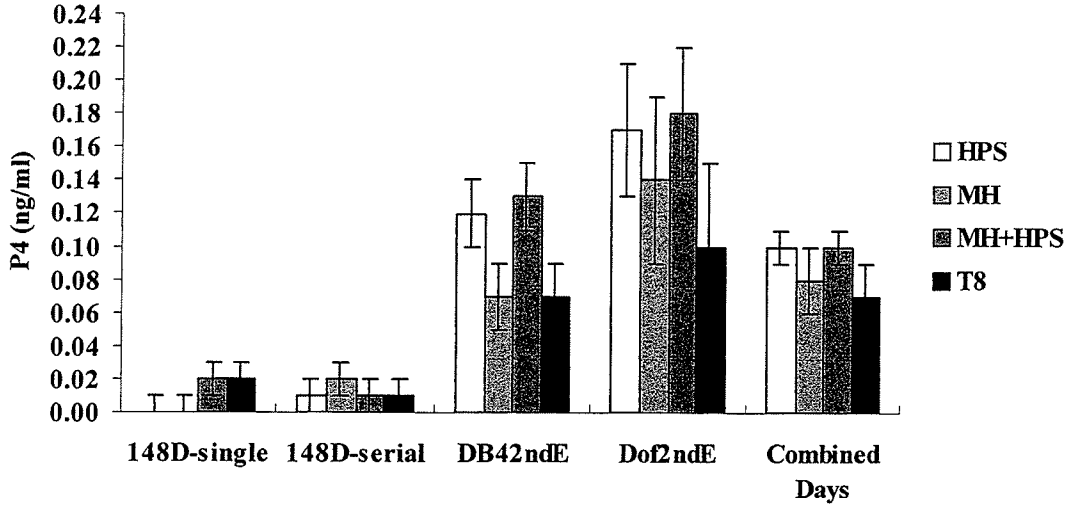
4.4 Endocrine Profiles

4.4.1 Progesterone

Progesterone concentrations were not affected by light treatments (Figure 16; Table A2.11, Appendix 2). However, gilts in MH+HPS tended to have higher P4 than gilts in MH, or T8 at DB42ndE (P=0.0839). There was no significant difference between P4 in single blood samples collected at 148D (P=0.4237), nor between these P4 concentrations (0.01 ± 0.01 ng/ml) and serial blood samples (0.02 ± 0.01 ng/ml) (P=0.7059). The average P4 concentration from combined sample days was not different across treatments (P=0.3623). Compared to 148D P4 (0.02 ± 0.02 ng/ml), P4 was significantly higher on DB42ndE (0.09 ± 0.02), and Dof2ndE (0.15 ± 0.02 ng/ml) (P=0.0011). The significant treatment difference of P4 in boar-no STH was, more than likely, due to gilt 3969 (HPS in T2) whose elevated P4 concentration and CL present at slaughter suggest that she had a silent ovulation. There was no significant treatment difference for P4 concentration in pregnant gilts at the end of the trial (P=0.1272).

With the exception of 148D-single, significant trial differences were present in all P4 measures, although all results were within an expected range and below 0.5 ng/ml (Table A2.12, Appendix 2). Pregnant gilts at end trial GestD, showed a trend (P=0.0974) and gilts for boar-no STH showed a significant (P=0.0020) interaction in trial by treatment, where as all other measures did not. As well, differences in P4 concentration between 148D, DB42ndE, and Dof2ndE P4 concentrations were greater in T1 trial by day analysis (Table A2.13, Appendix 2).

Figure 16. P4 concentration±SEM for 148D, DB42ndE, Dof2ndE and combined sample days across treatments.



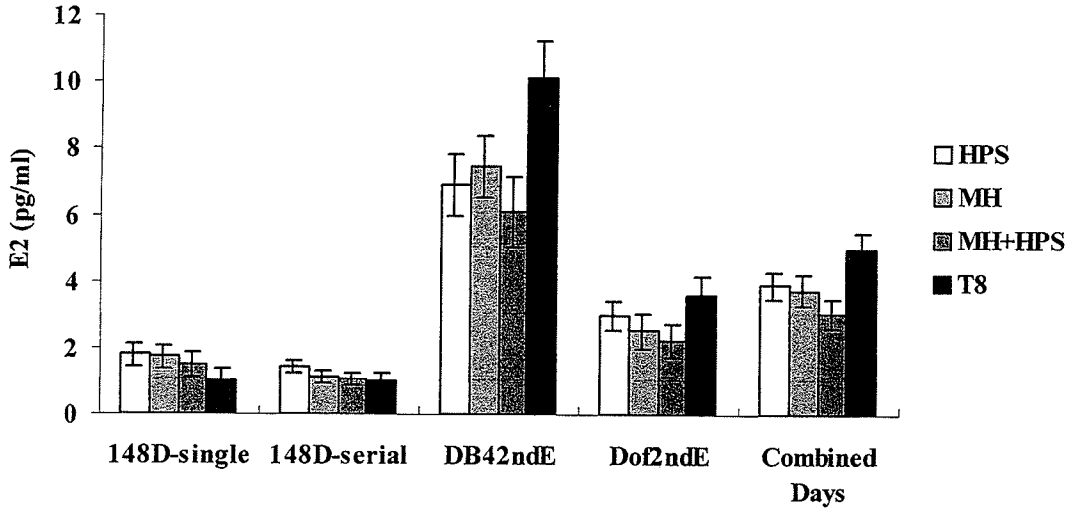
4.4.2 Estradiol

The majority of E2 concentrations were not affected by the light treatments (Figure 17; Table A2.14, Appendix 2). However, T8 E2 concentration tended to be greater than MH+HPS at DB42ndE (P=0.0546) and significantly greater for combined sample days (P=0.0439). In treatment by day interactions, the difference in E2 concentrations between days tended to be greater in T8 as compared to HPS and MH+HPS (P=0.0574) (Table A2.15, Appendix 2). As would be expected with puberty onset E2 was significantly higher in DB42ndE (7.6 ± 0.3 pg/ml) and Dof2ndE (3.1 ± 0.3 pg/ml) as compared to 148D (1.1 ± 0.3 pg/ml) (P=0.0001). Although, single blood sampled gilts (2.0 ± 0.2 pg/ml) showed a significantly higher E2 concentration than serial blood sampled gilts (1.0 ± 0.3 pg/ml) on 148D (P=0.0192), the averages were still under the 2 pg/ml associated with the pre-pubertal

state.

With the exceptions of single blood sampled 148D (P=0.0037), boar-no STH (P=0.0159), and end trial GestD (P=0.0094), there were no significant trial differences (Table A2.16, Appendix 2).

Figure 17. E2 concentration ± SEM for 148D, DB42ndE, Dof2ndE, and combined sample days across treatments. There were no differences between treatments within sample day.



4.4.3 Cortisol

Cortisol concentration was greater in MH (21.4 ± 1.4 ng/ml) and MH+HPS (20.9 ± 1.4 ng/ml) than in HPS (17.0 ± 1.5 ng/ml) and T8 (15.7 ± 1.9 ng/ml) (P=0.0465). There were no significant differences in trial, or trial by treatment interactions.

Figure 18 illustrates a diurnal pattern for cortisol concentrations over the 24 h period of 148D. Time was significantly different (P=<0.0001) and contrasts were used to outline differences between specific periods (Table 8).

Figure 18. Diurnal pattern of cortisol concentration at 148D. Boxed area indicates scotoperiod; open area indicates photoperiod.

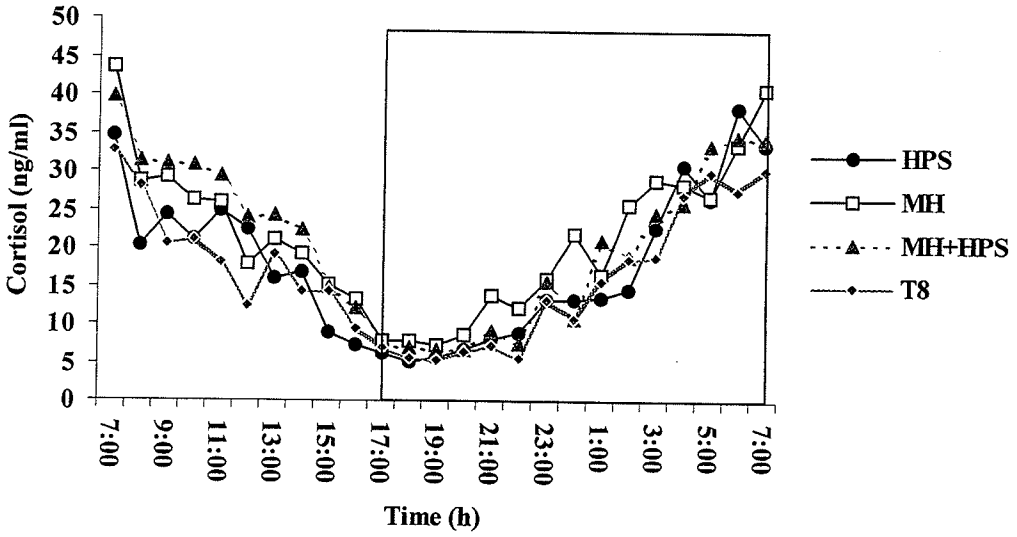


Table 8. Contrasts between cortisol concentrations within specific time periods at 148D.

Contrast	P-Value
0500 to 0700 vs 0800 to 1100	<0.0001
0500 to 0700 vs 1700 to 2000	<0.0001
1300 to 1600 vs 1700 to 2000	<0.0001
0700 to 1600 vs 1700 to 0700	<0.0001

4.4.4 Luteinizing Hormone

The mean concentration of LH throughout the 24 h periods of 148D, DB42ndE, and Dof2ndE are illustrated in Figure 19. Average and baseline concentrations, as well as peak amplitude at 148D, were not influenced by treatment on 148D, Dof2ndE, and combined sample days (Table 9). However, for DB42ndE, average and baseline LH concentration in

T8 tended to be lower than HPS (P=0.0659 and P=0.0793, respectively). The number of pulses across treatments were similar on 148D, but were significantly different on DB42ndE and Dof2ndE (Table 10). Treatment by day interactions were significant for average and baseline LH concentrations (P<0.0001 and P=0.0178, respectively) (Table A2.17, Appendix 2). Figure A2.1 in Appendix 2 illustrate LH profiles for 148D, DB42ndE and Dof2ndE. Compared to 148D (0.5 ± 0.1 ng/ml), average LH was significantly higher on DB42ndE (2.4 ± 0.1 ng/ml) and Dof2ndE (2.3 ± 0.1 ng/ml) (P<0.0001). As well, for baseline LH, 148D (0.3 ± 0.2 ng/ml) was significantly lower than DB42ndE (2.4 ± 0.2 ng/ml) and Dof2ndE (2.3 ± 0.2 ng/ml) (P<0.0001), again supporting their pre-pubertal status.

Figure 19. Average LH ± SEM concentration across sample days.

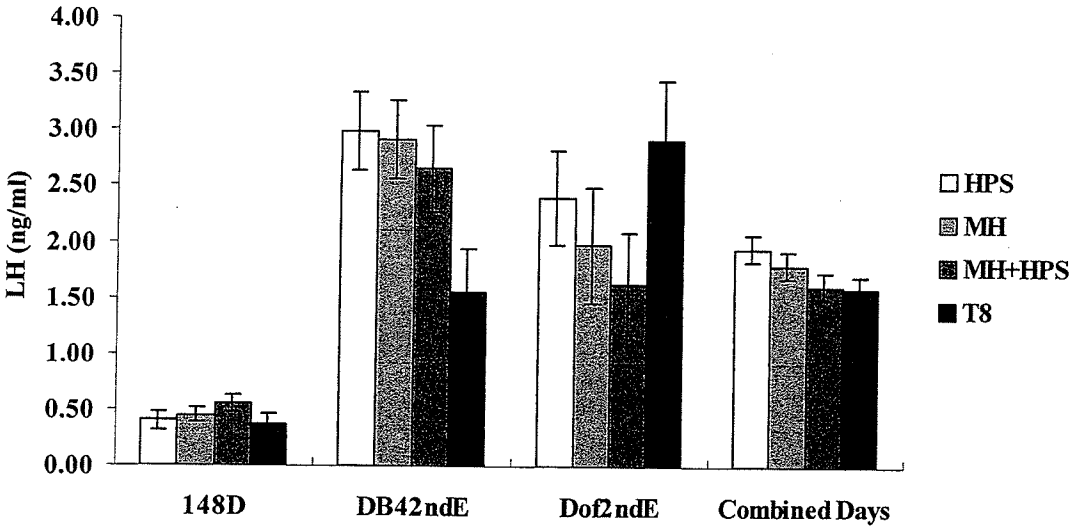


Table 9. Treatment values for LH concentration (ng/ml) across sample days.

Sample Day	LH (ng/ml)	HPS	MH	MH+HPS	T8	P-Value
148D	Average	0.4 ± 0.1	0.4 ± 0.1	0.6 ± 0.1	0.4 ± 0.1	0.4054
	Baseline	0.3 ± 0.1	0.4 ± 0.1	0.4 ± 0.1	0.3 ± 0.1	0.5949
	Amplitude	1.6 ± 0.3	1.7 ± 0.3	2.1 ± 0.3	1.8 ± 0.4	0.6914
DB42ndE	Average	3.0 ± 0.4^{y}	2.9 ± 0.4^{y}	2.6 ± 0.4^{yz}	1.5 ± 0.4^{z}	0.0659
	Baseline	2.9 ± 0.4^{y}	2.9 ± 0.4^{y}	2.6 ± 0.4^{yz}	1.5 ± 0.5^{z}	0.0793
	Amplitude	Non-est	Non-est	Non-est	Non-est	
Dof2ndE	Average	2.4 ± 0.4	2.0 ± 0.5	1.6 ± 0.4	2.9 ± 0.5	0.2890
	Baseline	2.4 ± 0.4	2.0 ± 0.5	1.6 ± 0.4	2.9 ± 0.5	0.2998
	Amplitude	Non-est	Non-est	Non-est	Non-est	
Combined Sample Days	Average	1.9 ± 0.1	1.8 ± 0.1	1.6 ± 0.1	1.6 ± 0.1	0.1371
	Baseline	1.9 ± 0.2	1.7 ± 0.2	1.6 ± 0.2	1.5 ± 0.2	0.6290
	Amplitude	Non-est	Non-est	Non-est	Non-est	

values are LS mean ± SEM
$^{y, z}$ trend at 0.0500<P<0.1000

Table 10. Number of LH pulses across treatments for 148D, DB42ndE and Dof2ndE.

Pulses	Total	HPS	MH	MH+HPS	T8	Significance*
148D	125	30	33	40	22	No
DB42ndE	16	4	2	1	9	Yes
Dof2ndE	8	0	0	5	3	Yes

*significance at chi-square value of 7.815

Across trials, gilts in T1 at Dof2ndE had a significantly lower average (P=0.0407) and baseline (P=0.0433) LH concentration. However, there were no significant trial differences at 148D, DB42ndE, or combined sample days (Table A2.18, Appendix 2). The number of pulses were significant across trials for DB42ndE, but not for 148D, or Dof2ndE

(Table A2.19, Appendix 2). Trial by day interactions were significant for both average (P=<0.0001) and baseline (P=0.0265), with T1 greater than T2 (Table A2.20, Appendix 2). There were no significant differences in trial by treatment interactions for average and baseline LH concentrations except at DB42ndE (P=0.0162 and P=0.0173, respectively) (Table A2.21 (a) & (b), Appendix 2).

4.4.5 Follicle Stimulating Hormone

Figure 20 illustrates the mean FSH concentrations across treatments for each sample day. There were no significant treatment differences for average, or baseline FSH concentrations in 148D, DB42ndE, Dof2ndE, or combined sample days (Table 11). FSH pulses were infrequent over the sampling days therefore peak amplitude analysis could not be conducted through the GLM, or Mixed procedures of SAS. The number of pulses in each sample day, as outlined in Table 12, were not significantly different between treatments. Treatment by day interactions were significant for average FSH concentration (P<0.0001), but not baseline (Table A2.22, Appendix 2). Figure A2.2 in Appendix 2 illustrate FSH profiles for 148D, DB42ndE and Dof2ndE. Average FSH concentration at 148D (0.9 ± 0.1 ng/ml) was significantly greater than at DB42ndE (0.8 ± 0.1 ng/ml), but significantly lower than Dof2ndE (1.3 ± 0.1 ng/ml) (P<0.0001). Baseline FSH concentration at 148D (1.9 ± 0.1 ng/ml) and DB42ndE (0.9 ± 0.1 ng/ml) was significantly lower than Dof2ndE (1.4 ± 0.1 ng/ml) (P=0.0005)

Figure 20. Average FSH concentration ± SEM at 148D, DB42ndE, Dof2ndE and combined sample days. No significant differences were noted for treatments within days.

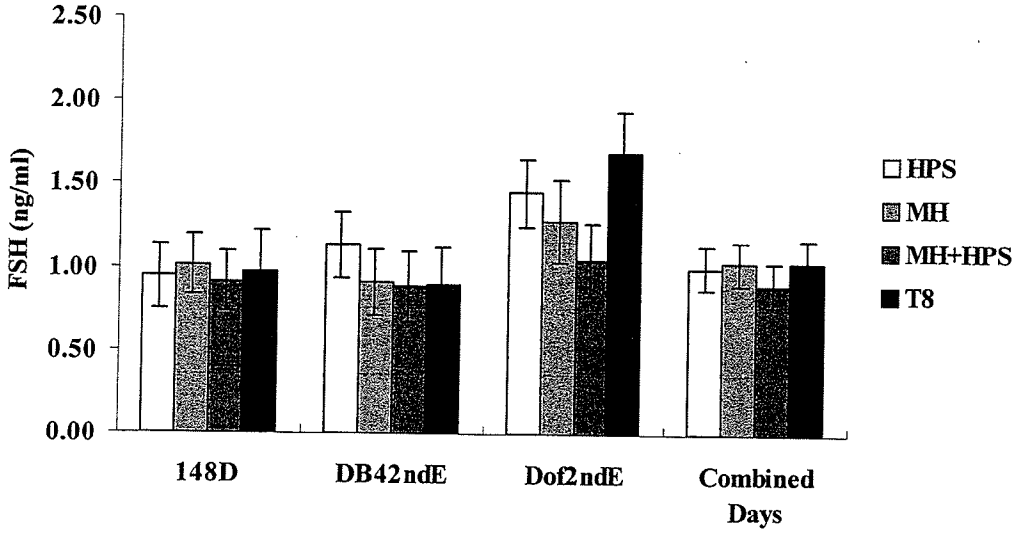


Table 11. Average and baseline FSH concentrations at each sample day across treatments.

Sample Day	FSH (ng/ml)	HPS	MH	MH+HPS	T8	P-Value
148D	Average	0.9 ± 0.2	1.0 ± 0.2	0.9 ± 0.2	1.0 ± 0.2	0.9814
	Baseline	0.9 ± 0.2	1.0 ± 0.2	0.9 ± 0.2	1.0 ± 0.2	0.9747
DB42ndE	Average	1.1 ± 0.2	0.9 ± 0.2	0.9 ± 0.2	0.9 ± 0.3	0.8088
	Baseline	1.1 ± 0.2	0.9 ± 0.2	0.9 ± 0.2	0.9 ± 0.2	0.8048
Dof2ndE	Average	1.4 ± 0.2	1.3 ± 0.3	1.0 ± 0.2	1.7 ± 0.3	0.3100
	Baseline	1.4 ± 0.2	1.3 ± 0.3	1.0 ± 0.2	1.7 ± 0.3	0.3052
Combined Sample Days	Average	1.0 ± 0.1	1.0 ± 0.1	0.9 ± 0.1	1.0 ± 0.1	0.8851
	Baseline	1.1 ± 0.1	1.1 ± 0.1	0.9 ± 0.1	1.1± 0.2	0.7593

values are LS mean ± SEM

Table 12. Pulse numbers for FSH across treatments for 148D, DB42ndE, and Dof2ndE.

Pulses	Total	HPS	MH	MH+HPS	T8	Significance*
148D	2	0	0	2	0	No
DB42ndE	2	0	1	1	0	No
Dof2ndE	3	1	0	2	0	No

*significance at chi-square value of 7.815

With the exception of 148D, analysis of DB42ndE, Dof2ndE, and combined sample days showed no significant trial differences for average and baseline FSH concentration, or pulse numbers (Tables A2.23 & A2.24, Appendix 2). At 148D, average and baseline FSH concentrations at T1 tended to be greater than T2 (P=0.0656 and P=0.0704, respectively). Also, trial by day interactions for both average (P<0.0001) and baseline (P=0.0247) were greater at T1 than T2 (Table A2.25, Appendix 2). There were no significant differences for trial by treatment interactions for any of these days.

4.4.6 Melatonin

MEL concentrations and diurnal profiles were not affected by the light treatments (Table 13; Figure 21). Photoperiod melatonin levels remained below 2.4 pg/ml, while the scotoperiod was characterized by significantly elevated MEL concentrations above 3.7 pg/ml ($P \leq 0.0001$).

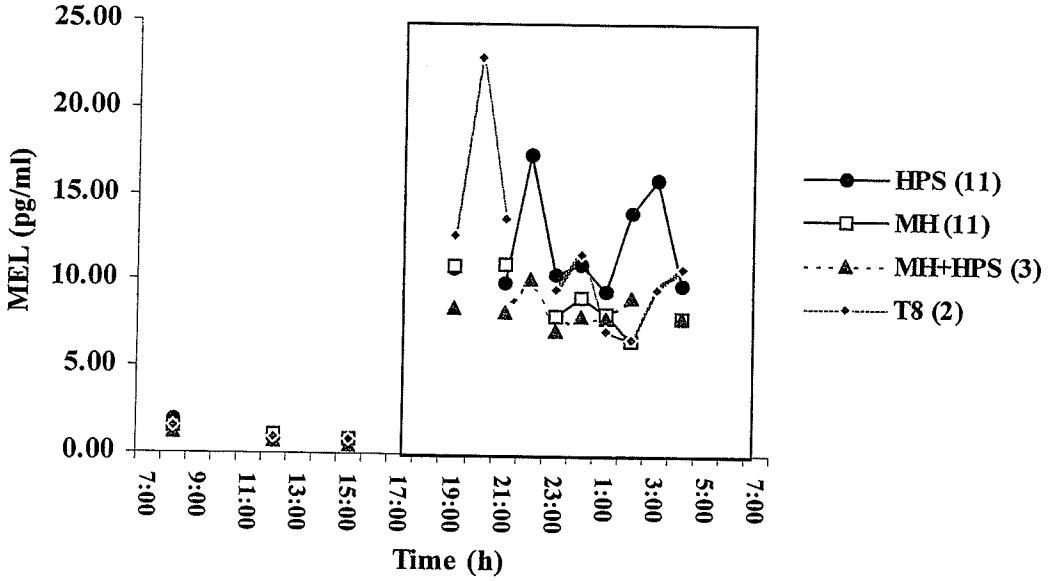
Table 13. MEL concentrations across treatments for 148D, DB42ndE and combined sample days.

MEL (pg/ml)	Period	HPS	MH	MH+HPS	T8	P-Value
148D	Photo	1.2 ± 0.2	1.2 ± 0.2	0.8 ± 0.3	1.1 ± 0.4	0.7436
	Scoto	11.0 ± 1.6	8.9 ± 1.6	8.8 ± 3.2	11.4 ± 3.7	0.7663
	24h	6.1 ± 1.3	5.0 ± 1.2	4.9 ± 2.4	6.2 ± 2.8	0.9127
DB42ndE	Photo	1.4 ± 0.3	1.2 ± 0.3	1.7 ± 0.5	1.8 ± 0.5	0.6884
	Scoto	11.0 ± 2.0	8.2 ± 2.2	7.7 ± 4.0	14.4 ± 4.0	0.5121
	24h	5.7 ± 1.7	4.6 ± 1.9	4.7 ± 3.2	8.1 ± 3.2	0.8200
Combined Sample Days	Photo	1.2 ± 1.2	1.5 ± 1.2	1.1 ± 2.1	1.5 ± 2.2	0.6706
	Scoto	9.7 ± 0.7	8.6 ± 0.7	8.4 ± 1.4	12.9 ± 1.5	0.6706
	24h	5.4 ± 0.8	5.0 ± 0.8	4.8 ± 1.5	7.2 ± 1.6	0.6913

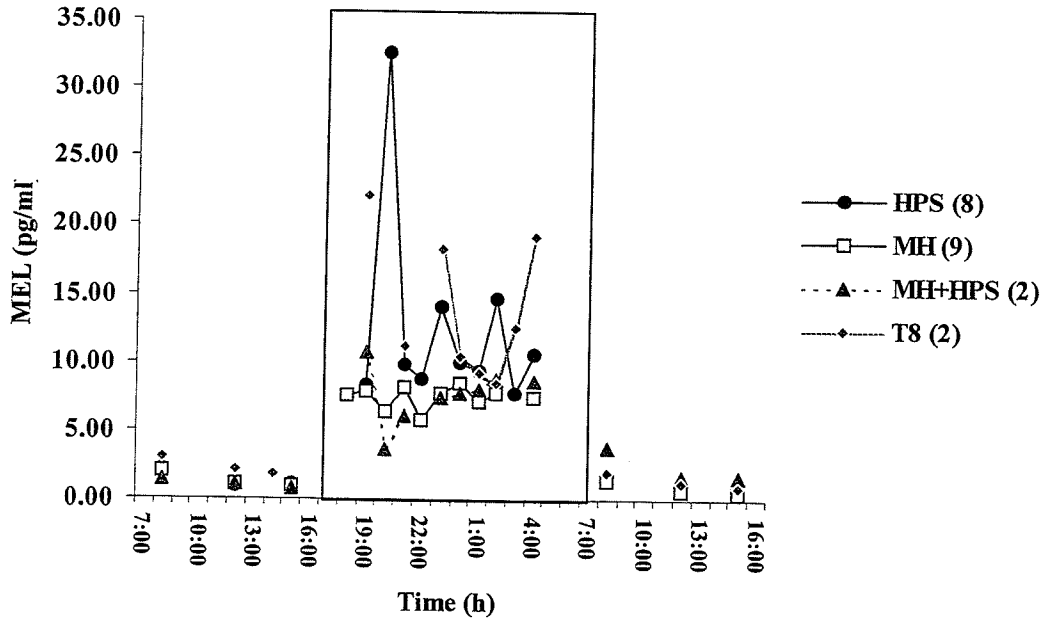
values are LS mean ± SEM

Figure 21. Diurnal pattern of MEL for (a) 148D (N=27) and (b) DB42ndE (N=21).

(a)

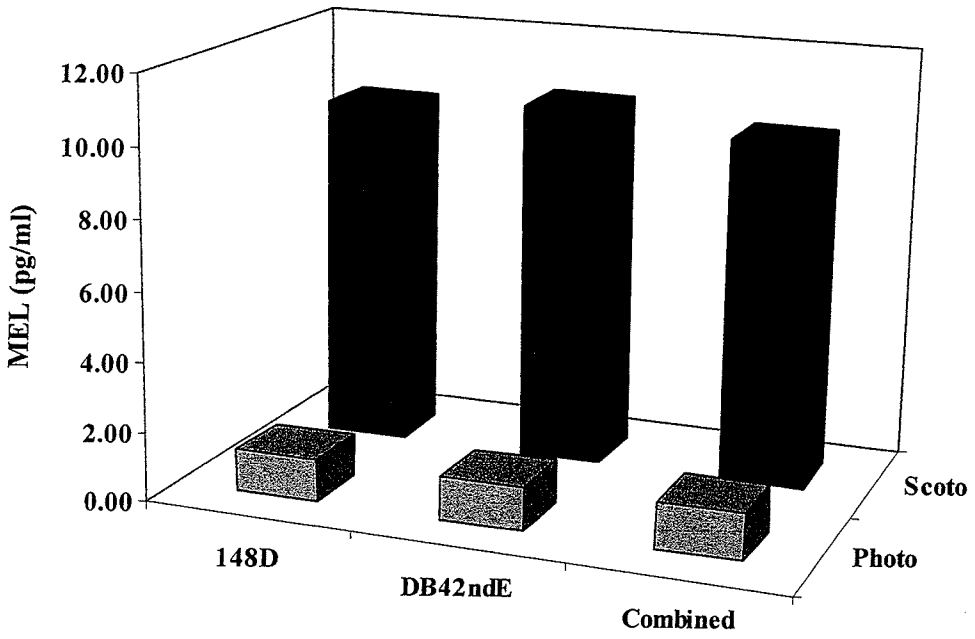


(b)



For 148D, significant differences between photo- and scotoperiod were present in 26 out of 27 gilts sampled across treatments. For DB42ndE, 19 out of 19 gilts showed significant photo- and scotoperiod differences across treatments (2 out of 21 gilts had scotoperiod values, only) (Figure 22). Figures A3.1 to A3.4 of Appendix 3 illustrate the photo- and scotoperiod differences for individual gilts within each treatment. (P=0.0129).

Figure 22. MEL concentration in photo- and scotoperiods during 148D, DB42ndE, and combined sample days.



Treatment by period interactions were significant for DB42ndE (P=0.0129) (Table 14). Light treatments containing MH (i.e. MH and MH+HPS) had significantly smaller differences between photo- and scotoperiod, as compared to HPS and T8 (Table A3.1, Appendix 3).

Table 14. Treatment by period interactions for 148D, DB42ndE, and combined sample days

MEL (pg/ml)	Period	HPS	MH	MH+HPS	T8	P-Value
148D	Photo	1.3 ± 0.6	1.2 ± 0.6	1.2 ± 1.2	1.1 ± 1.5	0.1781
	Scoto	11.0 ± 0.4	8.9 ± 0.4	8.7 ± 0.8	11.4 ± 1.0	
DB42ndE	Photo	0.4 ± 0.9[a]	0.9 ± 0.9[a]	1.7 ± 1.6[a]	1.8 ± 1.6[a]	0.0129
	Scoto	11.0 ± 0.5[b]	8.3 ± 0.6[a]	7.7 ± 1.0[a]	14.4 ± 1.0[c]	
Combined Sample Days	Photo	1.2 ± 1.2	1.5 ± 1.2	1.1 ± 2.1	1.5 ± 2.2	0.6706
	Scoto	9.7 ± 0.7	8.6 ± 0.7	8.4 ± 1.4	12.9 ± 1.5	

values are LS mean ± SEM

[a, b] significant difference across rows at P<0.0500

For 148D and DB42ndE there were no significant differences in trial, or treatment by trial interactions for photoperiod, scotoperiod, or 24h values. However, there was a significant trial by period interaction for DB42ndE, which showed the difference between photo- and scotoperiods was greater in T1 (P=0.0011) (Table A3.3, Appendix 3). Significance by day, or treatment by day was not present, but trial by day interaction showed that T1 had a greater difference between the sample days (about 1.0 pg/ml) than in T2 (about 0.9 pg/ml) (P=0.0217).

4.5 Correlations

4.5.1 Correlations of Growth Performance

A significant correlation was present between feed intake, growth and BF depth overall (P<0.0001) and for treatment (P<0.0001) correlation analysis.

4.5.2 Age at Estrus Correlations

Correlation between BF and puberty age was not present overall (P=0.3628), but for treatments a positive correlation was present for MH (P=0.0132).

With the exception of MH showing a tendency for correlation (P=0.0993), puberty age and the mean cortisol level at 148D were not significantly correlated.

Overall, the influence of temperature on puberty age and age at second estrus was not significantly correlated. As would be expected, significant correlation was present between puberty age and age at second estrus (P<0.0001).

4.5.3 Endocrine Correlations

A significant correlation was present overall between the 148D mean concentrations of MEL and cortisol (P=0.0033). However, in further analysis by treatment significant correlations were present for MH (P=0.0385) only.

Significant correlations for LH, FSH, E2 and P4 for the overall group are shown in Table 14. Significant correlations from further analysis by treatment are found in Tables A2.26 to A2.29 in Appendix 2.

Table 15. Significant endocrine correlations across treatments.

Correlation	r	P-Value
148D LH and 148D FSH	0.33084	<0.0001
148D LH and DB42ndE E2	-0.25369	0.0260
148D LH and Dof2ndE P4	0.31286	0.0047
DB42ndE E2 and Dof2ndE E2	0.34101	0.0003
DB42ndE E2 and Dof2ndE P4	-0.48363	<0.0001
DB42ndE P4 and Dof2ndE P4	0.21693	0.0248

*significant correlation at P<0.0500

4.5.4 Correlations for Embryo Survival

Overall, between CL number and embryo survival there was a significant correlation (P=0.0006). In further correlation analysis, HPS and T8 treatments supported a significant correlation, while MH showed a trend towards correlation (P=0.0516) and MH+HPS showed no correlation (P=0.6483).

4.5.4.1 Progesterone

Correlations between P4 levels, embryo survival and/or CL number were carried out at DB42ndE, Dof2ndE and end trial GestD. No significant correlations were present between DB42ndE P4 and embryo survival (P=0.1388-0.8222), Dof2ndE P4 and embryo survival (P=0.1816-0.9588), and end trial GestD P4 and embryo survival (P=0.1539-0.9556). Overall, there was a significant (negative) correlation between Dof2ndE P4 and CL number (P=0.0146).

4.5.4.2 Estradiol

Correlations between E2 levels, embryo survival and/or CL number were carried out at DB42ndE, Dof2ndE and end trial GestD. At DB42ndE, E2 was not significantly correlated with embryo survival (P=0.1099-0.8508). Trends in correlation between E2 levels and embryo survival were present at end trial GestD E2 for the overall group (P=0.0533). However, a significant correlation was present for embryo survival at end trial GestD E2 for MH (P=0.0280). Overall, there was a significant correlation between DB42ndE E2 and CL number (P=0.0129) and Dof2ndE E2 and CL number (P=0.0194). Additionally, Dof2ndE E2 tended to be correlated with CL number for MH+HPS (P=0.0860).

4.5.4.3 LH and FSH

LH and FSH concentration values at DB42ndE were analyzed for correlation between embryo survival and/or CL number. DB42ndE LH showed no significant correlation with embryo survival (P=0.2074-0.8062), where as DB42ndE FSH showed a trend towards

correlation with embryo survival for MH+HPS (P=0.0778). With the exception of MH (P=0.0416), DB42ndE LH was not significantly correlated with CL number. Correlations between DB42ndE FSH and CL number showed a trend for MH+HPS (P=0.0926).

CHAPTER 5

DISCUSSION

Reproductive and growth performance can be affected by a variety of different environmental factors, one of which is light. While effects of light duration (photoperiod) and intensity have been documented, to some extent, for the domestic pig, the influence of light type on growth, reproduction and physiological response remains unclear. In this study, groups of gilts were housed under one of four light types to investigate and define the role that light type has on endocrine responses, growth and early reproductive performance. The light types used as treatments in this study were ones commercially available, but differing in colour spectra, colour rendering index, and energy efficiencies. They were HPS, MH, MH+HPS and T8 fluorescent, all of which varied in their colour rendering index and their spectrum.

Growth performance and feed intake within both the finishing and pubertal periods were not influenced by light type. Availability of good quality food throughout the year can overcome some of the seasonal light influences that affect the European wild boar (Love et al., 1993). Gilts in this study were provided a balanced commercial ration ad lib throughout each trial, which eliminated any influence of restricted feed intake. As well, the consistency of performance shows that temperature fluctuations, believed to be part of the seasonal influence (Prunier et al., 1994), had no significant affect on performance parameters.

The ADG of 0.9 kg/d and age of 173.0 ± 2 d at 110 kg were within industry averages

for market hogs (PigChamp, 1999). Similarly, gain:feed at 0.3 ± 0.0 and ADFI at 2.6 ± 0.0 kg/d for both periods were well within industry standards. The average weight at puberty was 118.2 ± 3.2 kg and BF of 11.2 ± 0.5 mm. At their rate of gain gilts were within the range of 125 kg and 15 mm backfat, as recommended by Foxcroft and Aherne (2001), in order to ensure good lifetime performance.

Overall, reproductive performance through puberty to early gestation was unaffected by light type, either positively or negatively. Gilts were maintained under a short day photoperiod (10 h photoperiod and 14 h scotoperiod) throughout the trials, which was similar to their photoperiodic history. Any influence of season, outside of photoperiod, was not obvious between the two trials (T1, May to September; T2, January to May). As well, there were no significant correlations between temperature and age at puberty, or age at second estrus. Although the tendency for estrus periods to be of longer duration in gilts reaching puberty in spring (T2) is consistent with Paterson et al. (1991), this cannot be confidently attributed to season.

Reproductive performance was well within acceptable ranges. The average age at puberty in the gilt is 6 to 8 months (Hafez, 1993). Gilts in this study reached puberty at an average age of 182.10 ± 3 .54 d (i.e. a little over 6 months). Ovulation rate (14.61 ± 0.71) and embryo survival rate (80 ± 4 %) were greater than values reported by Ntunde et al. (1979) and Turner et al. (1998), but are consistent with this modern genotype seen commercially.

Puberty in the gilt is often considered to be a function of age rather than body weight, or backfat (Beltranena et al., 1991 & 1993; Newton and Mahan, 1992). Results of this study

are in agreement. There was no correlation between age of puberty and backfat, nor with weight. This supports results of Newton and Mahan (1992) in which the effect of different levels of feed intake had on puberty onset and subsequent body composition.

In terms of routine estrus checks we did note an impact of light type. Light type did affect the subjective evaluation of estrus. Detection of vulva redness was initially more difficult under the HPS spectrum. The orange-red spectral colour of HPS absorbed red colour present in the room. The addition of MH (in MH+HPS) produced a whiter spectrum which eliminated this visual problem. The CRI, which ranges from 0 to 100, where 100 is the greatest degree of whiteness, rates HPS within the range of 40 to 60, and MH in the range of 60 to 80. Initially, when working between the four light types, HPS was obviously more orange and appeared duller, but with time the difference was no longer consciously noticeable.

Ad lib feeding was continued throughout gestation due to the availability of labour, and in order not to confound results by the potential influence of having some gilts in the same room restricted fed after estrus while others at different reproductive stages were fed ad lib. Gilts/sows are typically restricted fed at the time of breeding and throughout gestation. This practice is followed to ensure good feeding levels during lactation and to maintain a good body composition and future reproductive performance of the sow (VIDO, 1996). Restricted feeding may also be influence P4 levels by preventing increased metabolic clearance rate, (i.e. low levels of this hormone) and any consequent impact on embryo survival (Symonds and Prime, 1988). In this study though, embryo survival was comparable to gilts/sows that are typically restricted fed at breeding (Beltranena et al., 1991).

Venous catheterization and frequent blood sampling, which could be considered an acute stress, had no apparent impact on duration of estrus at either puberty, or second estrus. No analysis was completed for these sources of acute stress on ovulation rate and embryo survival. However, Turner et al. (1988) reported a study in which acute stress created by the handler did not cause any significant difference in embryo survival and ovulation rate.

As with growth and reproductive performance, endocrine profiles and concentrations were not dramatically affected by light type. However, there were some exceptions for cortisol, E2, P4, LH and MEL at different points in the study.

The role of cortisol in the onset of reproduction is associated with acute and chronic levels (Hafez, 1993). The general theory is that acute levels of cortisol, such as may occur through transport, advance the age at puberty onset by influencing hypothalamic sensitivity to steroid negative feedback (as reviewed by Prunier et al., 1993; Brann and Mahesh, 1991; Pearce et al., 1988). Though significant differences were observed in cortisol from gilts in MH and MH+HPS over HPS and T8, it was not reflected in differences in age at puberty onset, nor other parameters measured. Cook et al. (1998) suggested that entrainment to a circadian cortisol profile may be due to light intensity and photoperiod. In this study, the difference could have been associated with light intensity, or light spectra. One theory proposed by Lehrer (as reviewed by Evan et al, 1988) is that the hormone release from the hypothalamus is dependent on both a circadian rhythm and an endogenous age-dependent rhythm. Evans et al. (1988) observed an age-dependent rhythm for cortisol that developed into the accepted circadian rhythm. A circadian rhythm was already established in this study's prepubertal gilts at 148 d of age. Therefore, although cortisol may have been

influenced by light spectrum, it was not in such a way as to obviously affect the circadian profile or biological performance.

Other hormone concentrations and profiles showed no significant treatment differences at 148D. Progesterone levels below 0.500 pg/ ml, and E2 below 2 pg/ml indicated that gilts were prepubertal at this time (Bollinger et al., 1997; Diekman and Green, 1997). Low levels of LH were released in characteristic high frequency pulses (Hafez, 1993).

At boar exposure but no standing heat, a treatment difference was noted for P4. However, this difference was likely the result of one gilt, which underwent a silent ovulation. This was supported not only by her P4 concentration at this time, but by the presence of CL on her ovaries. She did show signs of estrus onset, but never came into standing heat, the indicator used to define estrus onset, before losing redness and vulval swelling. Estradiol, was not significantly different between treatments for boar-no STH.

Just before second estrus (DB42ndE), treatment differences were present for P4, E2 and LH. Progesterone concentration tended to be greater for MH+HPS when compared to T8, or MH. Where as for E2, concentrations in T8 tended to be greater than in MH+HPS. The concentrations of P4 and E2 in T8 at DB42ndE behaved as would be expected in the follicular phase when P4 concentration is low and E2 concentration is high (Senger, 1997; Hafez, 1993). As well, for LH, average and baseline concentrations at DB42ndE were different between treatments. Concentrations in T8 tended to be lower than those in HPS and MH, which would be consistent with the increasing E2 (Hafez, 1993; Senger, 1997).

The high pre-ovulatory surge peak of LH was not readily captured by our sampling schedule. This may have been due to the onset of blood sampling, which was based on

expected estrus onset and heat checks, which only occurred twice daily. The number of LH pulses at DB42ndE were significantly different between treatments and between trials. Whether these differences were due to collection onset, or some other variable could not be ascertained. Hormone concentrations between treatments were not different on the day of second estrus. This further supports no significant effect of light type.

Analysis of combined sample days showed no significant difference, except for E2, which was greater with fluorescent light (T8) than in MH+HPS at DB42ndE. If these results reflect an influence of light type it is not clear. The fact that other hormones, as well as reproductive performance were unaffected does not lend support for any influence of light type. However, that there could be more subtle affects on follicular growth and steriodogenesis cannot be dismissed.

The lack of any significant treatment, or trial differences for FSH also disputes light type effects on this hormone.

Trial differences present for all but one of the P4 sample times could be interpreted as a seasonal influence. However, since other hormones measured and animal performance were not similarly affected, seasonality is unlikely to be the cause.

Melatonin is considered to be the transducer of daily and annual photoperiodic information to the HPG in many species (Malpaux et al., 1999). Gilts, considered to be short-day seasonal breeders (Claus and Weiler, 1985; Love et al., 1993), reached puberty at an earlier age when administered exogenous MEL (Diekman et al., 1991). Until fairly recently, controversy existed as to whether pigs showed a clear diurnal MEL profile (Diekman et al., 1992). Results of the current study demonstrate a clear diurnal variation in

MEL release in gilts, which supports observations of Paterson et al. (1992a) and Tast et al. (2001). We have extended these observations to include different light types. Previous controversy about the presence of a diurnal pattern for MEL in swine seems to have been influenced by assay techniques (Andersson et al., 2000).

The significant differences present for light intensity between HPS and MH are a potential factor of influence in this study. All readings, except the end, were significantly greater for MH, compared to HPS, by approximately 60 lx . The fact that differences were not present in performance and were minimal in the endocrine measures suggest that this difference in intensity may be of negligible impact. However, differences noted in cortisol between those treatments with MH (MH and MH+HPS) versus those without (HPS and T8) may be a function of the difference in light intensity between MH and HPS.

Research on the influence of light type on pig performance is limited. Some investigations have been made comparing artificial versus natural light, but variables such as photoperiod exist. Considering that conventional housing uses artificial light types, a comparison between the various types available is critical. Not only is it important for pig performance, but also for the working environment it creates for individuals working in it. For example, the use of HPS in a breeding barn creates a challenge for heat detection, but it would be satisfactory in a finisher barn. There is room for further study into the influences of light and its various components. Photoperiod is relatively well researched, but intensity and type have been less well examined. By examining these components together, results would help provide an understanding of what specific aspect of light has a stronger influence upon pig performance.

From a practical standpoint, results from this study show that of the light types tested, selection can be based on economics and personal preference because impact on performance and endocrine changes were negligible.

CHAPTER 6

CONCLUSIONS

It is important to consider how the managed environment may impact animal performance, both current and future. The fact that pigs retain vestiges of seasonality suggest that light, either duration, intensity, or type, may be one such source of environmental impact. In this study, a short-day photoperiod was constant across treatments and trials. Light intensity was intended to be constant, however there was a significant difference between HPS and MH. The majority of results show that the minimal difference between these two intensities was not significant.

In this study, no significant differences were observed in growth, gain:feed or reproductive performance in gilts housed within HPS, MH, MH+HPS, or T8 light types. Trial differences in growth were due to animals' weight and age at trial start. Whereas, the trial difference in duration of 2ndE may have been caused by seasonal influence, but the constancy of photoperiod does not lend support to this influence.

With the exception of cortisol, the remaining hormones did not show consistent treatment differences. The greater concentration of cortisol for MH and MH+HPS may indicate an influence by MH light type, but this was not reflected in performance. Trial differences were present for P4 and E2, but these cannot be confidently attributed to seasonal differences because there is a lack of support in results from performance and other hormones.

Subjective observations of these light types showed that HPS may be a poor light to use in a breeding, or farrowing barn. This is due to the absorption of red colour by this light's spectrum; a colour essential for detecting vulva redness.

Further investigation is recommended for examination of these hormonal actions at puberty onset. The use of ultrasound techniques would benefit in the determination of puberty onset and allow timely collection to provide analysis of these hormones.

As well, research examining light type with either varying photoperiods, or different intensities would provide additional understanding as to the influence light type has upon performance.

Knowledge regarding the influence of each of light's components has the potential to cut industry costs. By enhancing managements ability to use the environment in such a way as to benefit, or at least remove negative influence upon animal performance will save on unnecessary costs.

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APPENDIX 1

TREATMENT ROOMS

Table A1.1. Trial differences in light intensity measures.

Light Intensity (lx)	Trial 1	Trial 2	P-Value
Overall	291 ± 9	277 ± 9	0.2724
Start	298 ± 10	295 ± 10	0.8374
Mid	300 ± 9[a]	265 ± 9[b]	0.0110
End	276 ± 9	273 ± 9	0.7869

values are LS mean ± SEM
[a, b] significant differences at P<0.0500

Table A1.2. Temperature results for treatment by time interaction.

Temperature (°C)	Reading Time	HPS	MH	MH+HPS	T8	P-Value
Max	AM	21.7 ± 0.0	22.3 ± 0.0	22.1 ± 0.0	21.1 ± 0.0	0.0002^
	PM	22.1 ± 0.0	22.6 ± 0.0	22.4 ± 0.0	21.6 ± 0.0	
Min	AM	18.4 ± 0.0	19.0 ± 0.0	18.4 ± 0.0	17.7 ± 0.0	0.3243
	PM	18.7 ± 0.0	19.2 ± 0.0	18.8 ± 0.0	18.1 ± 0.0	

values are LS mean ± SEM
^significance not indicated by Bonferroni set between 0.0500 and 0.1000

Table A1.3. Max and min temperature results for trial by time interactions.

Temperature (°C)	Reading Time	T1	T2	P-Value
Max	AM	23.7 ± 0.0[a]	19.9 ± 0.0[b]	0.0006
	PM	24.3 ± 0.0	20.0 ± 0.0	
Min	AM	18.0 ± 0.0[a]	18.7 ± 0.0[b]	0.0046
	PM	18.6 ± 0.0	18.8 ± 0.0	

values are LS mean ± SEM
[a, b] significant difference at P<0.0500

Figure A1.1. Daily AM and PM temperature readings for HPS in Trial 1 (● High, ○Low).

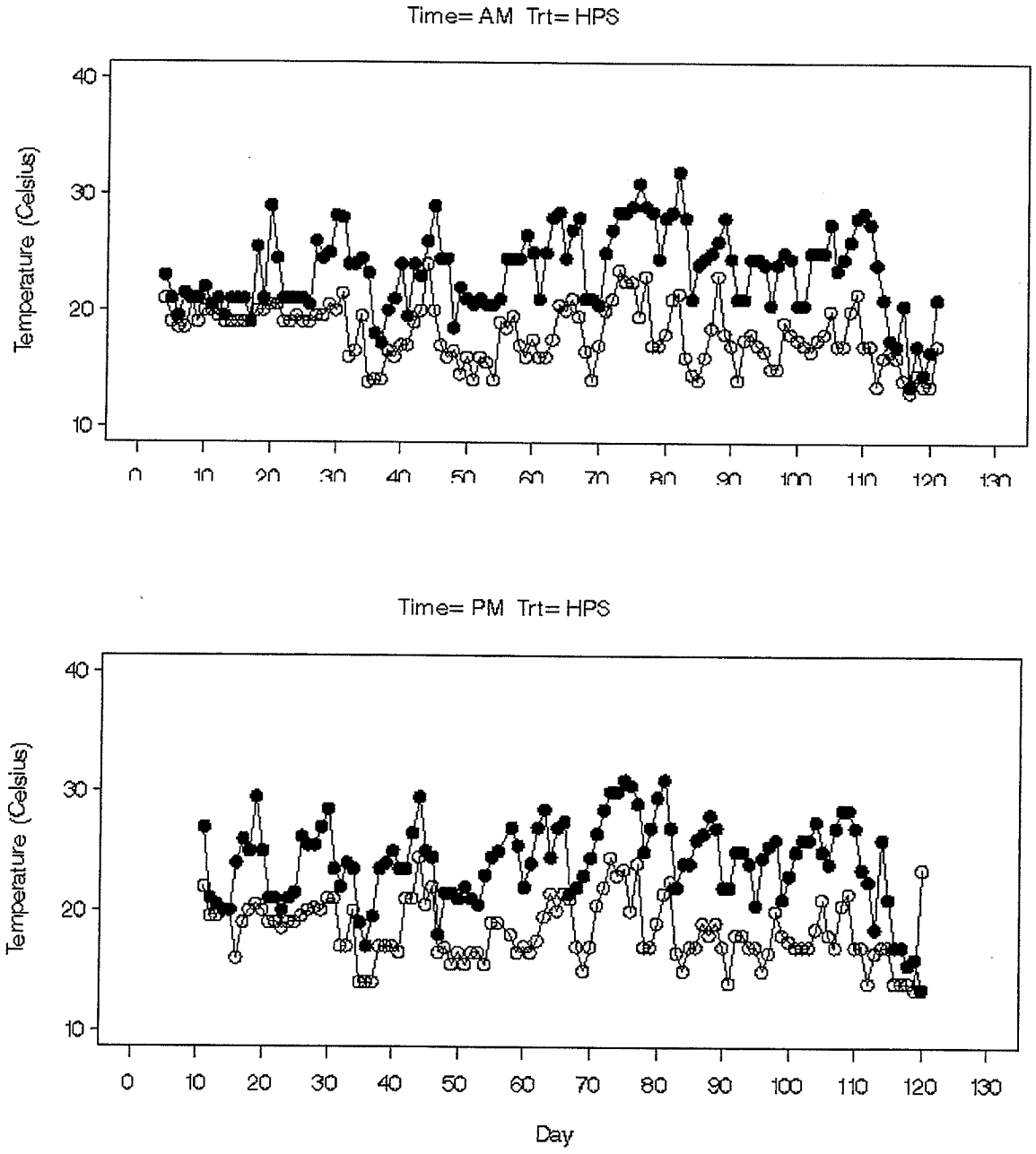
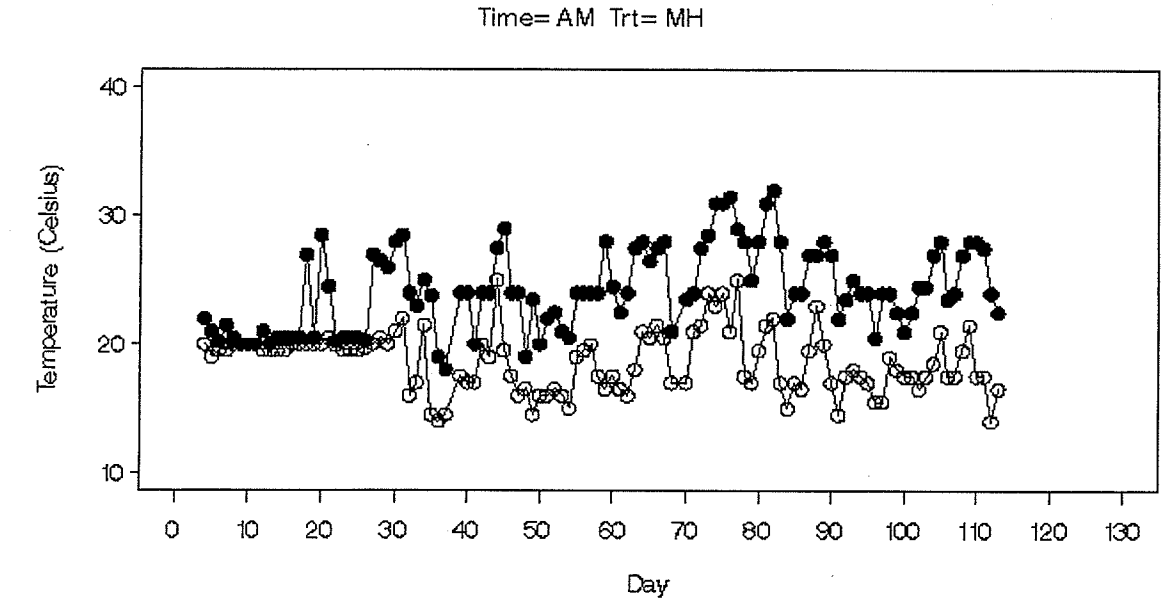


Figure A1.2. Daily AM and PM temperature readings for MH in Trial 1 (● High, ○ Low).



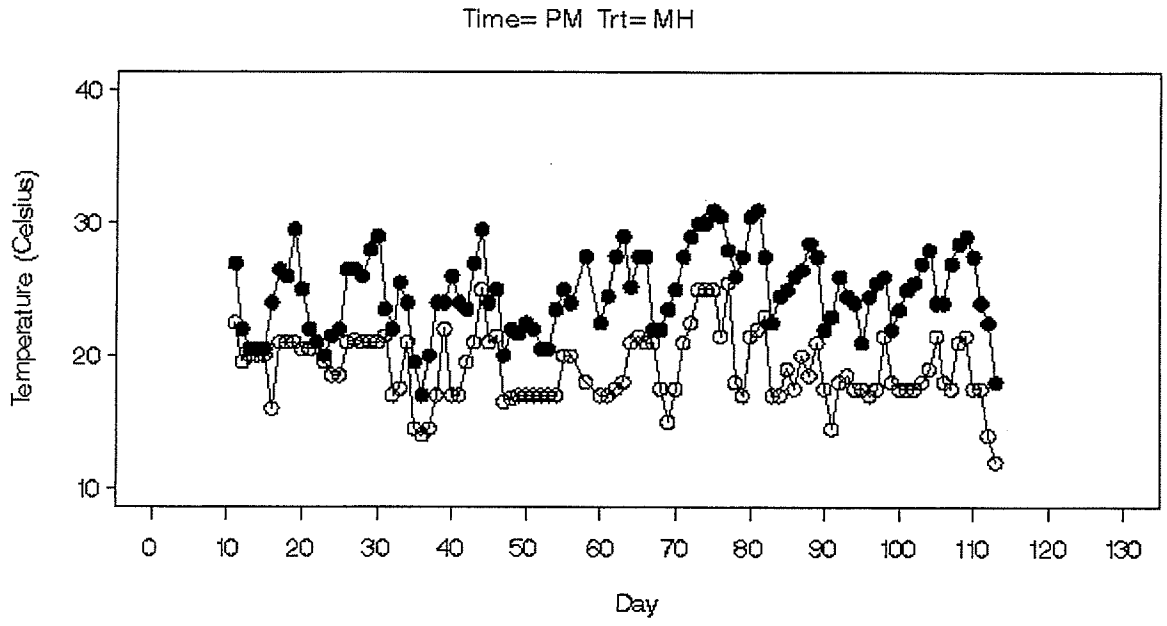
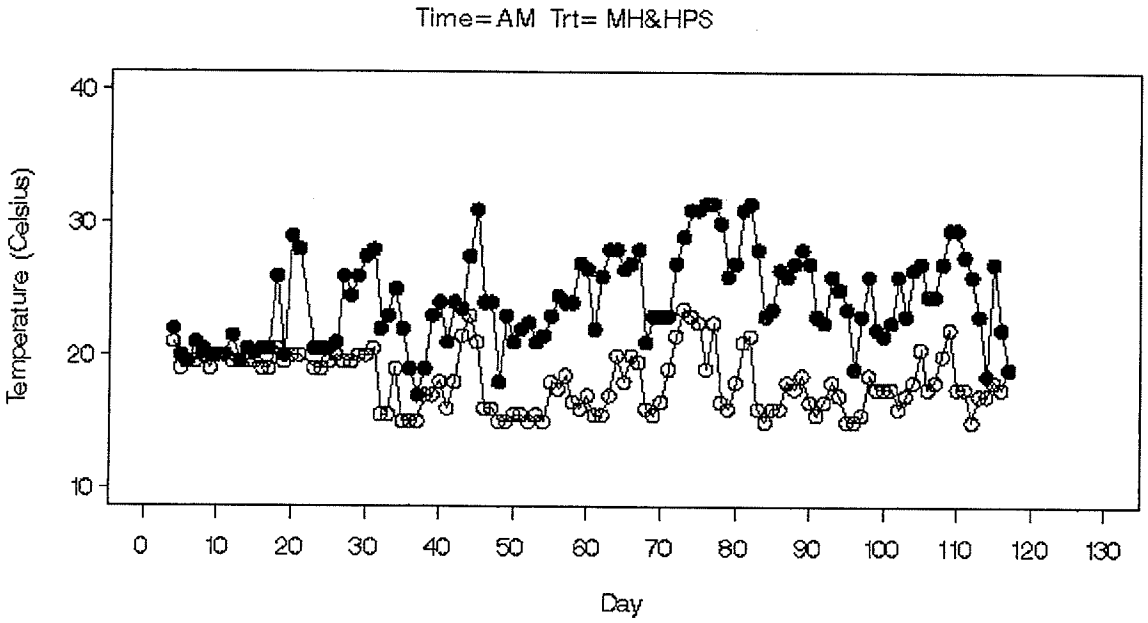


Figure A1.3. Daily AM and PM temperature reading for MH+HPS in Trial 1 (● High, ○ Low).



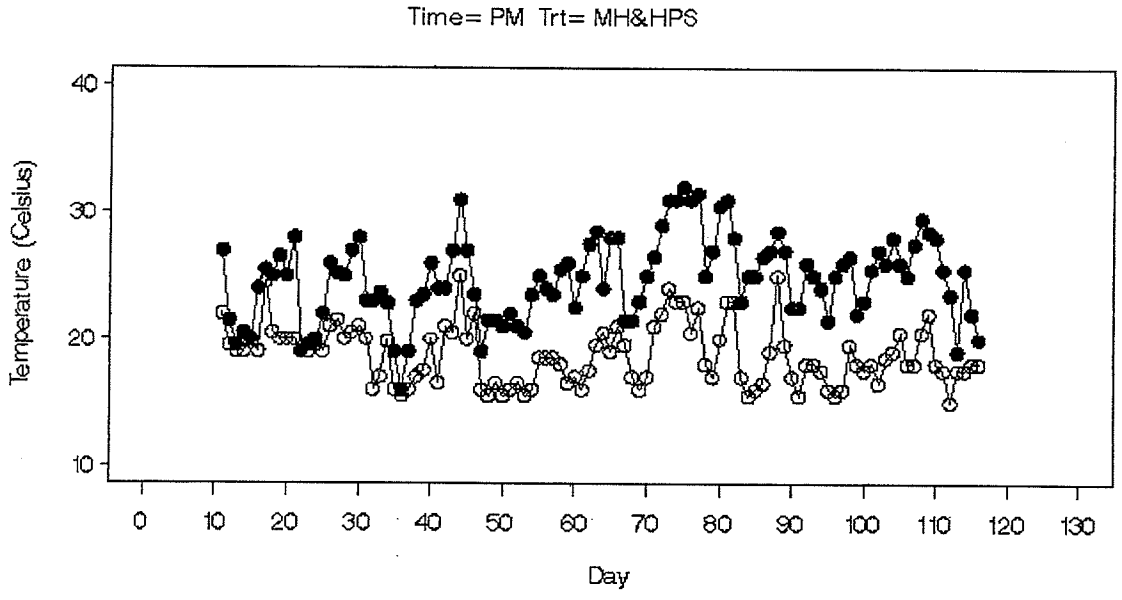
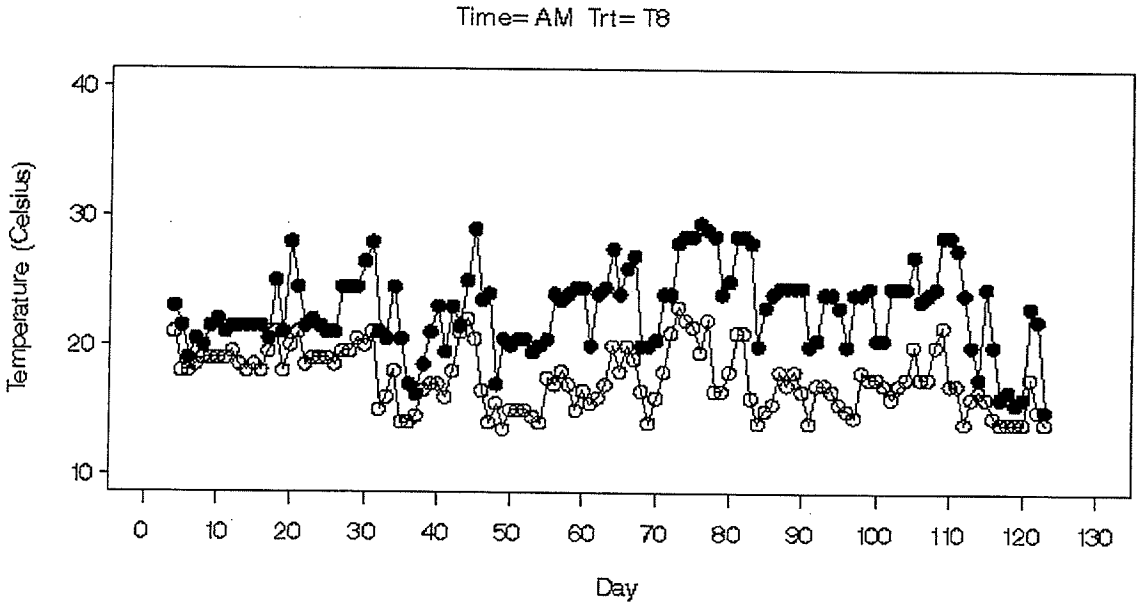


Figure A1.4. Daily AM and PM temperature readings for T8 in Trial 1 (● High, ○ Low).



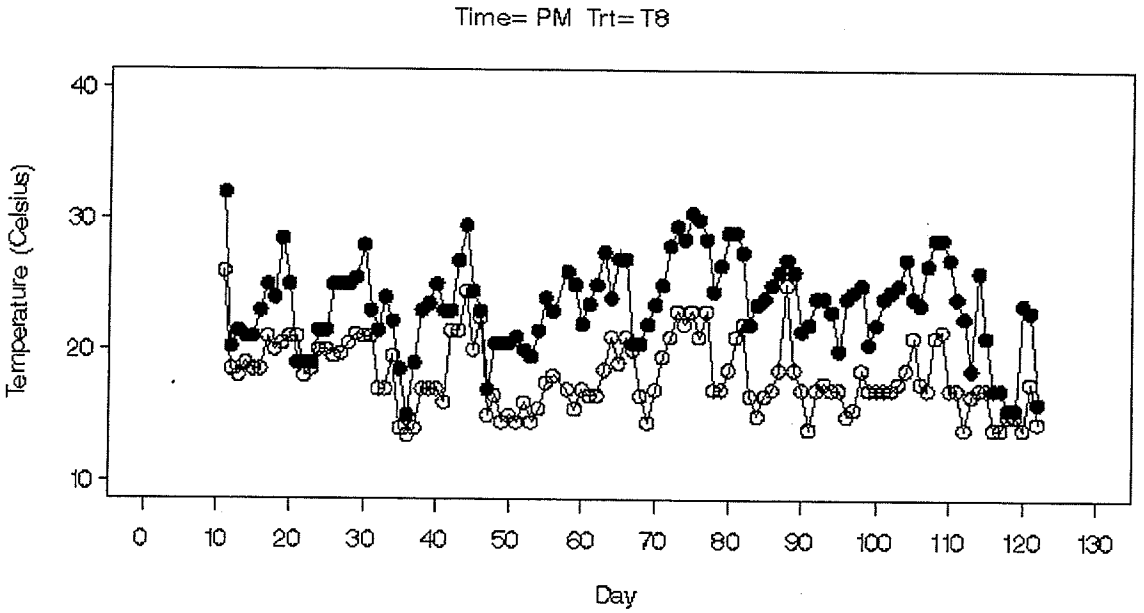
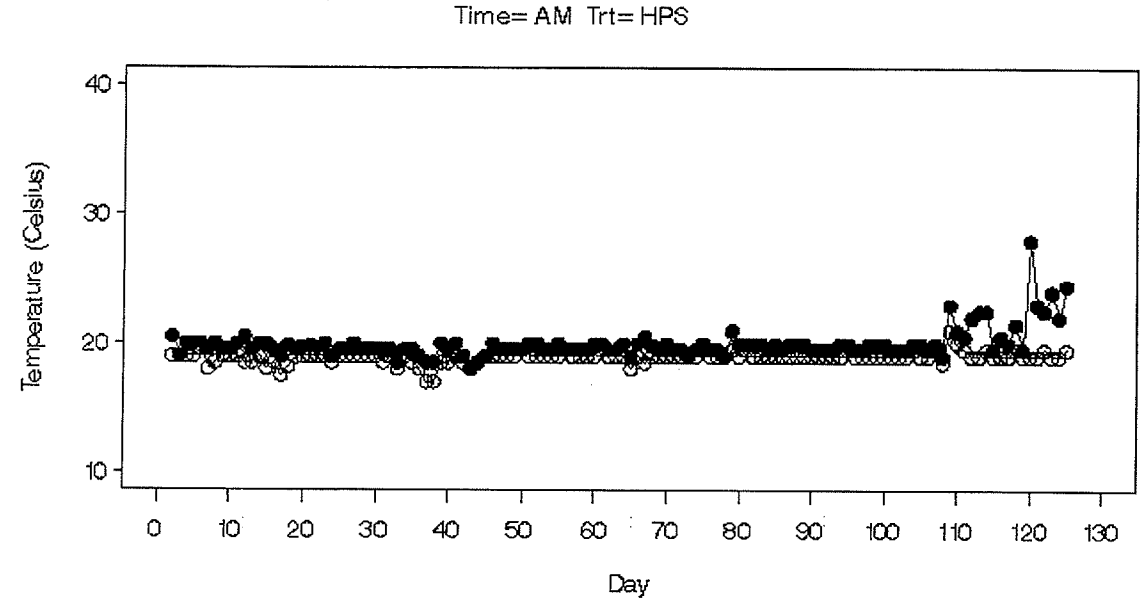


Figure A1.5. Daily AM and PM temperature readings for HPS in Trial 2 (● High, ○ Low).



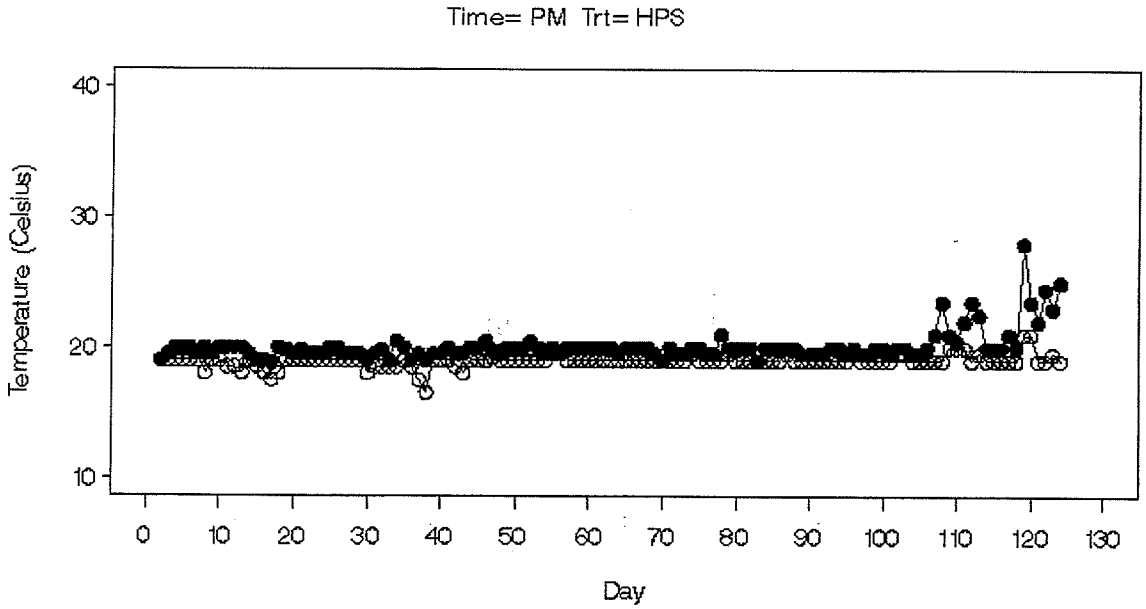
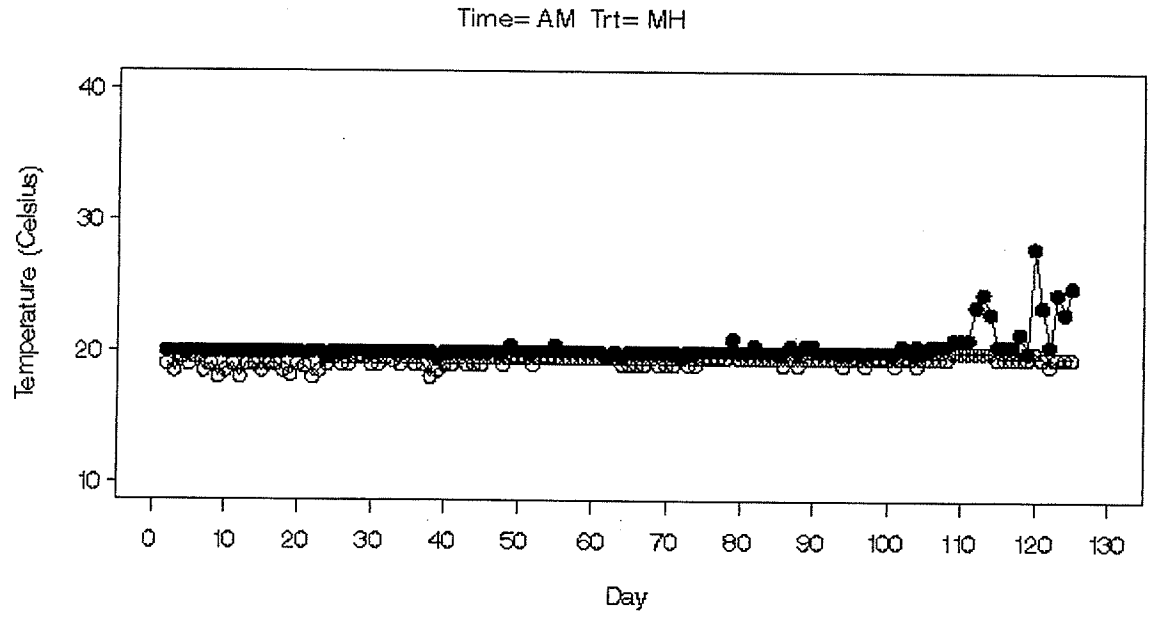


Figure A1.6. Daily AM and PM temperature readings for MH in Trial 2 (● High, ○ Low).



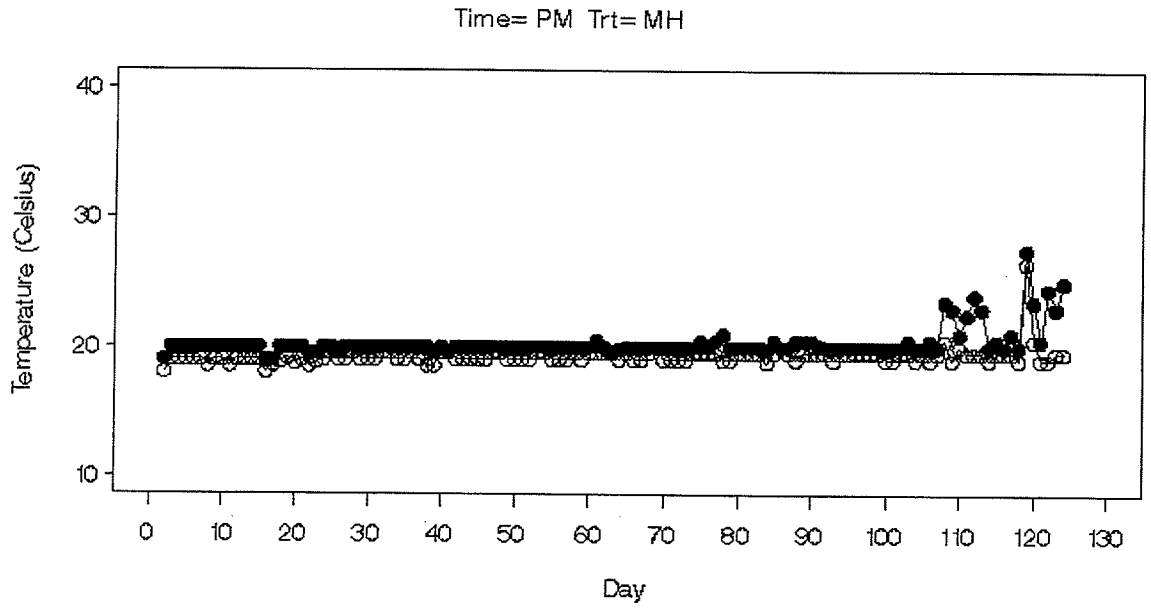


Figure A1.7. Daily AM and PM temperature readings for MH+HPS in Trial 2 (● High, ○ Low).

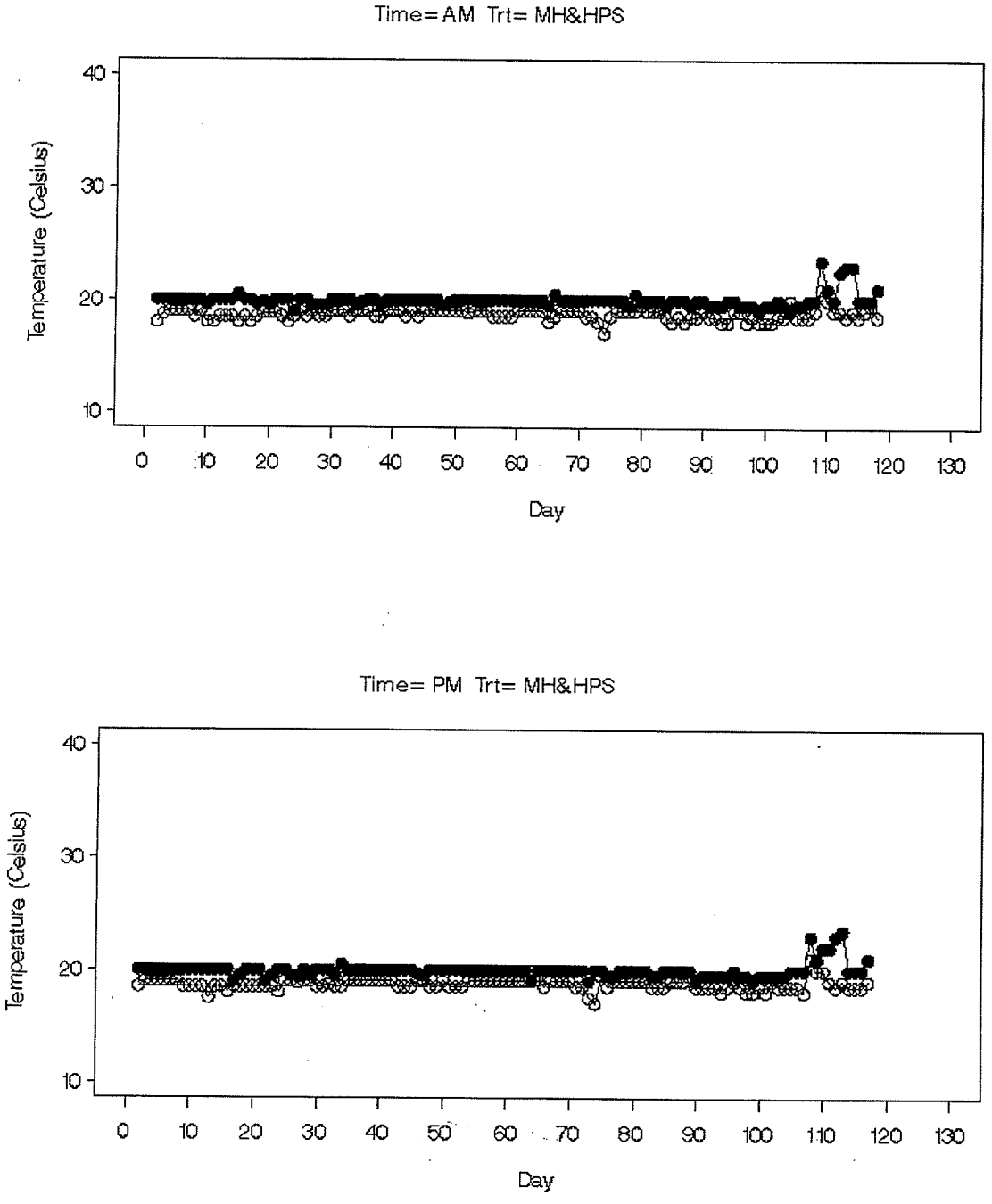
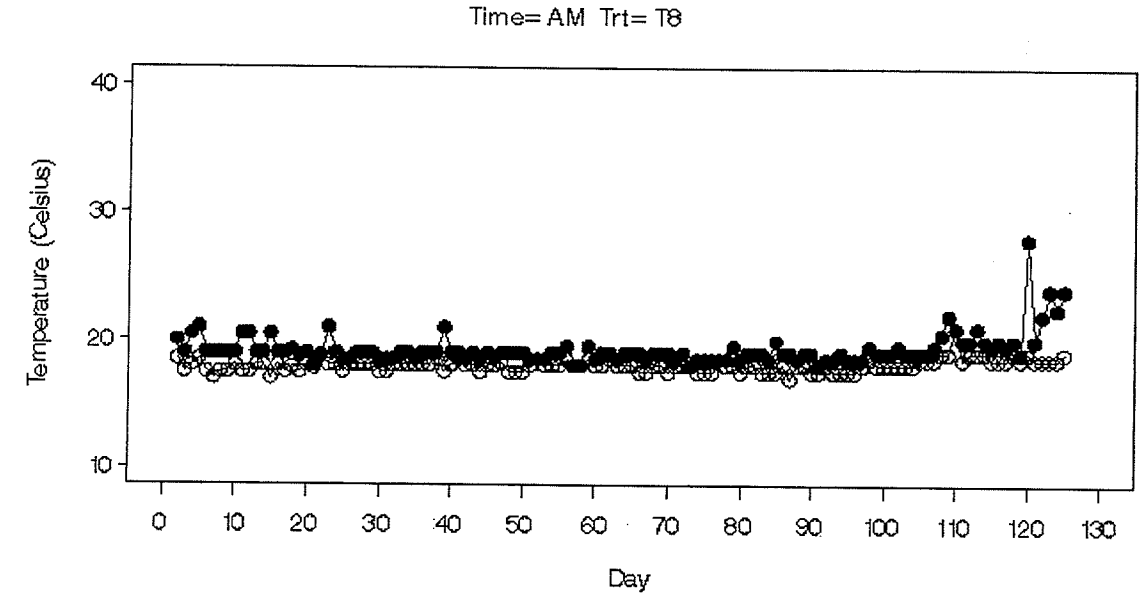


Figure A1.8. Daily AM and PM temperature readings for T8 in Trial 2 (● High, ○ Low).



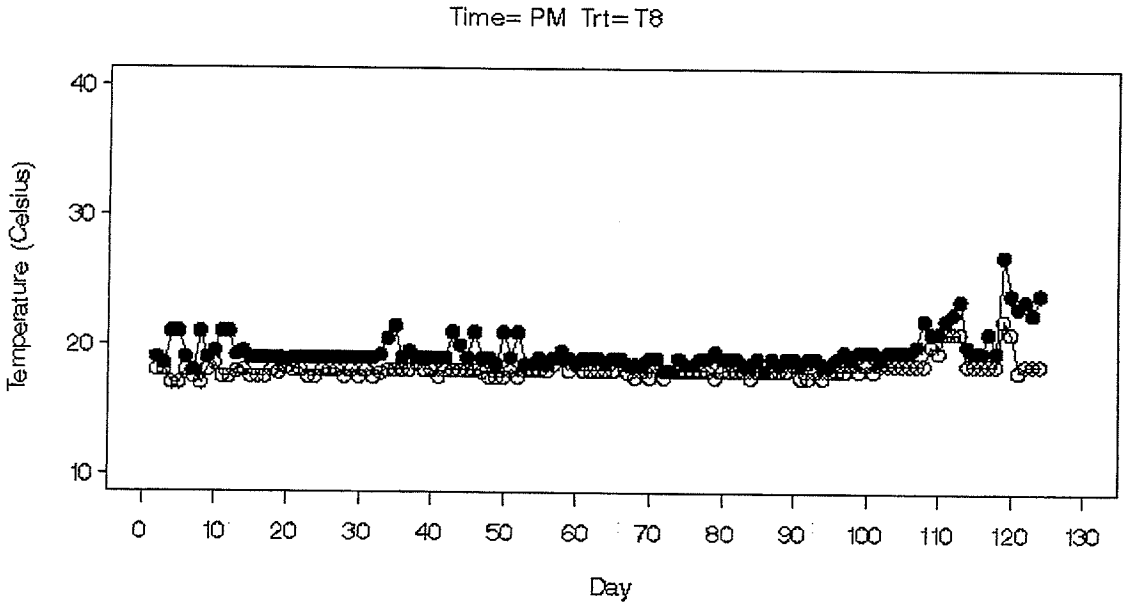


Table A1.4. Phason temperature across treatments.

Temperature (°C)	HPS	MH	MH+HPS	T8	P-Value
Phason	20.9 ± 0.1	21.1 ± 0.2	20.6 ± 0.2	20.8 ± 0.1	0.3166

values are LS mean ± SEM

Table A1.5. Phason temperature between trials.

Temperature (°C)	T1	T2	P-Value
Phason	21.8 ± 0.1[a]	19.9 ± 0.1[b]	0.0010

values are LS mean ± SEM
[a, b] significant difference at P<0.0500

Table A1.6. Phason temperature between AM and PM readings.

Temperature (°C)	AM	PM	P-Value
Phason	19.3 ± 0.1[a]	22.4 ± 0.1[b]	<0.0001

values are LS mean ± SEM
[a, b] significant difference at P<0.0500

Table A1.7. Phason temperature results for treatment by time interaction.

Temperature (°C)	Time	HPS	MH	MH+HPS	T8	P-Value
Phason	AM	19.4 ± 0.2	19.6 ± 0.2	18.9 ± 0.2	19.3 ± 0.2	0.6889
	PM	22.4 ±0.2	22.7 ± 0.2	22.3 ± 0.2	22.4 ± 0.2	

values are LS mean ± SEM

Table A1.8. Phason temperature results for trial by time interactions.

Temperature (°C)	Reading Time	Trial 1	Trial 2	P-Value
Phason	AM	18.9 ± 0.1[a]	19.7 ± 0.1[b]	0.0002
	PM	24.7 ± 0.1	20.1 ± 0.1	

values are LS mean ± SEM

[a, b] significant difference at P<0.0500

APPENDIX 2

GROWTH & REPRODUCTIVE PERFORMANCE

Table A2.1. Age and weight across treatments at start and end of trials.

T1 & T2	HPS	MH	MH+HPS	T8	P-Value
Start Age (d)	123 ± 1	123 ± 1	123 ± 1	123 ± 1	0.9595
Start Weight (kg)	67.6 ± 1.0	67.4 ± 1.0	67.1 ± 1.0	67.4 ± 1.0	0.9887
End Age (d)	227 ± 2	226 ± 2	222 ± 3	228 ± 3	0.3821
End Weight (kg)	157.3 ± 2.8	150.4 ± 2.8	152.2 ± 2.9	155.3 ± 2.8	0.3029

values are LS mean ± SEM

Table A2.2. Growth performance across treatments in finishing period.

110 kg	HPS	MH	MH+HPS	T8	P-Value
Age (d)	173 ± 2	174 ± 2	172 ± 2	173 ± 2	0.9501
Total Gain (kg)	42.4 ± 1.0	42.6 ± 1.0	42.9 ± 1.0	42.6 ± 1.0	0.9887
ADG (kg/d)	0.9 ± 0.0	0.9 ± 0.0	0.9 ± 0.0	0.9 ± 0.0	0.6406
BF (mm)	10.4 ± 0.2	10.1 ± 0.2	10.8 ± 0.3	10.4 ± 0.2	0.2819

values are LS mean ± SEM

Table A2.3. Average growth performance across trials for finishing period.

	T1	T2	P-Value
Trial Start Age (d)	124 ± 0^{a}	122 ± 0^{b}	0.0004
Age at 110 kg (d)	170 ± 1^{a}	176 ± 1^{b}	0.0010
Trial Start Weight (kg)	70.1 ± 0.7^{a}	64.6 ± 0.7^{b}	0.0001
Total Gain (kg)	39.9 ± 0.7^{a}	45.4 ± 0.7^{b}	0.0001
ADG (kg/d)	0.9 ± 0.0^{y}	0.8 ± 0.0^{z}	0.0824
BF (mm)	9.9 ± 0.2^{a}	11.0 ± 0.2^{b}	0.0001

values are LS mean ± SEM

^{y,z} trend at $0.0500<P<0.1000$

^{a,b} significant difference at $P<0.0500$

Table A2.4. Feed intake across treatments in finishing period.

110 kg	HPS	MH	MH+HPS	T8	P-Value
Total Intake (kg)	127.4 ± 4.1	128.1 ± 4.1	128.7 ± 4.2	126.0 ± 4.2	0.9732
ADFI (kg/d)	2.6 ± 0.0	2.6 ± 0.0	2.6 ± 0.0	2.6 ± 0.0	0.4854
FE	0.3 ± 0.0	0.3 ± 0.0	0.3 ± 0.0	0.3 ± 0.0	0.8323

values are LS mean ± SEM

Table A2.5. Feed intake across trials in finishing period.

110 kg	T1	T2	P-Value
Total Intake (kg)	117.0 ± 2.9[a]	138.1 ± 2.9[b]	0.0001
ADFI (kg/d)	2.1 ± 0.0	2.6 ± 0.0	0.4474
FE	0.3 ± 0.0[y]	0.3 ± 0.0[z]	0.0724

values are LS mean ± SEM
[y, z] trend at 0.0500<P<0.1000
[a, b] significant difference at P<0.0500

Table A2.6. Growth performance across treatments in the pubertal period.

Puberty	HPS	MH	MH+HPS	T8	P-Value
Age (d)	182 ± 3	182 ± 4	178 ± 4	186 ± 4	0.4970
Weight (kg)	118.4 ± 3.1	117.4 ± 3.2	115.0 ± 3.2	121.9 ± 3.4	0.5215
Total Gain (kg)	50.6 ± 2.9	50.0 ± 3.0	48.2 ± 3.0	54.2 ± 3.2	0.5847
ADG (kg/d)	0.9 ± 0.0	0.8 ± 0.0	0.9 ± 0.0	0.9 ± 0.0	0.6867
BF (mm)	10.9 ± 0.5	10.6 ± 0.5	11.9 ± 0.5	11.5 ± 0.5	0.2849

values are LS mean ± SEM

Table A2.7. Growth performance across trials in the pubertal period.

Puberty	T1	T2	P-Value
Age (d)	181 ± 2	183 ± 3	0.6484
Weight (kg)	120.0 ± 2.2	116.3 ± 2.3	0.2479
Total Gain (kg)	50.0 ± 2.1	51.4 ± 2.2	0.6338
ADG (kg/d)	0.9 ± 0.0	0.8 ± 0.0	0.1167
BF (mm)	10.2 ± 0.3[a]	12.2 ± 0.4[b]	0.0002

values are LS mean ± SEM

[a, b] significant difference at P<0.0500

Table A2.8. Feed intake across treatments in the pubertal period.

Puberty	HPS	MH	MH+HPS	T8	P-Value
Total Intake (kg)	155.5 ± 10.0	155.3 ± 10.5	148.3 ± 10.5	166.7 ± 11.0	0.6853
ADFI (kg/d)	2.6 ± 0.0	2.6 ± 0.1	2.7 ± 0.1	2.7 ± 0.1	0.7111
FE	0.3 ± 0.0	0.3 ± 0.0	0.3 ± 0.0	0.3 ± 0.0	0.9713

values are LS mean ± SEM

Table A2.9. Feed intake across trials in pubertal period.

Puberty	T1	T2	P-Value
Total Intake (kg)	152.7 ± 7.2	160.1 ± 7.6	0.4833
ADFI (kg/d)	2.7 ± 0.0	2.6 ± 0.0	0.3588
FE	0.3 ± 0.0	0.3 ± 0.0	0.1742

values are LS mean ± SEM

Table A2.10. Reproductive performance across trials.

	T1	T2	P-Value
Age at PubE (d)	181 ± 2	183 ± 3	0.6484
Duration of PubE (hr)	25.5 ± 2.3[y]	31.2 ± 2.3[z]	0.0852
Age at 2ndE (d)	202 ± 3	204 ± 3	0.5724
Duration of 2ndE (hr)	27.7 ± 2.2[a]	37.5 ± 1.8[b]	0.0010
Estrus Interval (d)	21.2 ± 0.3[y]	21.9 ± 0.3[z]	0.0632
End Trial GestD (d)	28.1 ± 0.2	28.2 ± 0.2	0.6034
Tract Exam GestD (d)	32.1 ± 0.7[a]	29.2 ± 0.7[b]	0.0048
Ovulation Rate	14.3 ± 0.5	14.9 ± 0.5	0.3938
Embryo Survival Rate (%)	80 ± 3	79 ± 3	0.7549

values are LS mean ± SEM
[y, z] trend at $0.0500<P<0.1000$
[a, b] significant difference at $P<0.0500$

Table A2.11. P4 concentration across treatments.

P4 (ng/ml)	HPS	MH	MH+HPS	T8	P-Value
148D - single	0.00±0.01	0.00±0.01	0.02±0.01	0.02±0.01	0.4237
148D - serial	0.01±0.01	0.02±0.01	0.01±0.01	0.01±0.01	0.4341
DB42ndE	0.1 ± 0.0^{yz}	0.1 ± 0.0^{z}	0.1 ± 0.0^{y}	0.1 ± 0.0^{z}	0.0839
Dof2ndE	0.2 ± 0.0	0.1 ± 0.0	0.2 ± 0.0	0.1 ± 0.0	0.6922
Combined Sample Days	0.1 ± 0.0	0.1 ± 0.0	0.1 ± 0.0	0.1 ± 0.0	0.3623
Boar-No STH	9.2 ± 1.9^{a}	0.0 ± 1.6^{b}	0.0 ± 1.7^{b}	0.0 ± 1.5^{b}	0.0019
End Trial GestD	21.3 ± 1.1	23.6 ± 1.2	22.6 ± 1.0	19.9 ± 1.2	0.1272

values are LS mean ± SEM
$^{y, z}$ trend at 0.0500<P<0.1000
$^{a, b}$ significant difference at P<0.0500

Table A2.12. P4 concentration across trials.

P4 (ng/ml)	T1	T2	P-Value
148D - single	0.02±0.01	0.01±0.01	0.2000
148D - serial	0.02 ± 0.00^{a}	0.00 ± 0.01^{b}	0.0052
DB42ndE	0.1 ± 0.0^{a}	0.1 ± 0.0^{b}	0.0007
Dof2ndE	0.2 ± 0.0^{a}	0.1 ± 0.0^{b}	0.0004
Combined Sample Days	0.1 ± 0.0^{a}	0.0 ± 0.0^{b}	0.0001
Boar-No STH	0.0 ± 0.9^{a}	4.6 ± 1.4^{b}	0.0114
End Trial GestD	25.0 ± 0.8^{a}	18.7 ± 0.7^{b}	<0.0001

values are LS mean ± SEM
$^{a, b}$ significant difference at P<0.0500

Table A2.13. P4 concentration results for trial by day interactions.

P4 (ng/ml)	Sample Day	Trial 1	Trial 2	P-Value
Combined	148D	0.02±0.02[a]	0.02±0.03[b]	
Sample	DB42ndE	0.1 ± 0.0	0.0 ± 0.0	0.0011
Days	Dof2ndE	0.2 ± 0.0	0.1 ± 0.0	

values are LS mean ± SEM

[a, b] significant difference at P<0.0500

Table A2.14. E2 concentration across treatments.

E2 (pg/ml)	HPS	MH	MH+HPS	T8	P-Value
148D - single	1.8 ± 0.4	1.7 ± 0.4	1.5 ± 0.4	1.0 ± 0.4	0.4480
148D - serial	1.4 ± 0.2	1.1 ± 0.2	1.0 ± 0.2	1.0 ± 0.2	0.4492
DB42ndE	6.9 ± 0.9^{yz}	7.4 ± 0.9^{yz}	6.1 ± 1.0^{y}	10.2 ± 1.1^{z}	0.0546
Dof2ndE	3.0 ± 0.4	2.5 ± 0.5	2.2 ± 0.5	3.6 ± 0.5	0.2196
Boar-No STH	2.4 ± 0.7	3.4 ± 0.6	2.3 ± 0.6	2.3 ± 0.6	0.5327
End Trial GestD	22.5 ± 2.1	22.6 ± 2.3	24.0 ± 2.0	20.0 ± 2.2	0.6307
Combined Sample Days	3.9 ± 0.4^{ab}	3.8 ± 0.4^{ab}	3.0 ± 0.4^{a}	5.0 ± 0.5^{b}	0.0439

values are LS mean ± SEM
^{y,z} trend at 0.0500<P<0.1000
^{a,b} significant difference at P<0.0500

Table A2.15. E2 concentration results for treatment by day interaction.

E2 (pg/ml)	Day	HPS	MH	MH+HPS	T8	P-Value
Combined	148D	1.6 ± 0.5^{y}	$0.7 \pm 0.5^{y,z}$	1.1 ± 0.5^{y}	0.8 ± 0.6^{z}	
Sample	DB42ndE	7.0 ± 0.5	7.5 ± 0.6	5.9 ± 0.6	10.0 ± 0.7	0.0574
Days	Dof2ndE	3.0 ± 0.5	3.0 ± 0.6	2.2 ± 0.6	4.1 ± 0.7	

values are LS mean ± SEM
^{y,z} trend at 0.0500<P<0.1000

Table A2.16. Trial differences in E2 concentration.

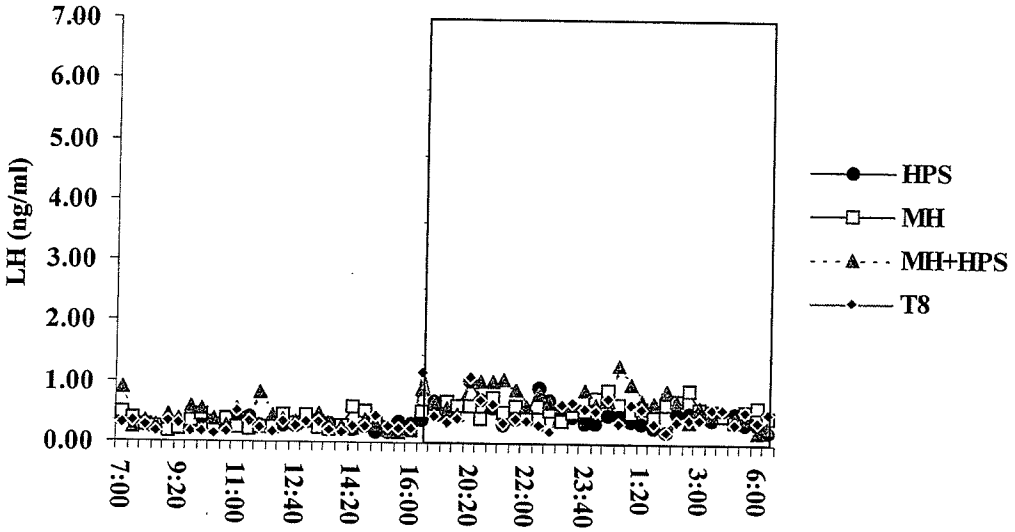
E2 (pg/ml)	Trial 1	Trial 2	P-Value
148D - single	2.0 ± 0.3^{a}	0.9 ± 0.3^{b}	0.0037
148D - serial	1.3 ± 0.1	1.0 ± 0.2	0.2883
DB42ndE	7.2 ± 0.8	8.1 ± 0.6	0.4062
Dof2ndE	2.7 ± 0.4	3.0 ± 0.3	0.4879
Combined Sample Days	3.8 ± 0.4	4.1 ± 0.3	0.4774
Boar-No STH	3.4 ± 0.3^{a}	1.8 ± 0.5^{b}	0.0159
End Trial GestD	25.2 ± 1.6^{a}	19.4 ± 1.4^{b}	0.0094

values are LS mean ± SEM

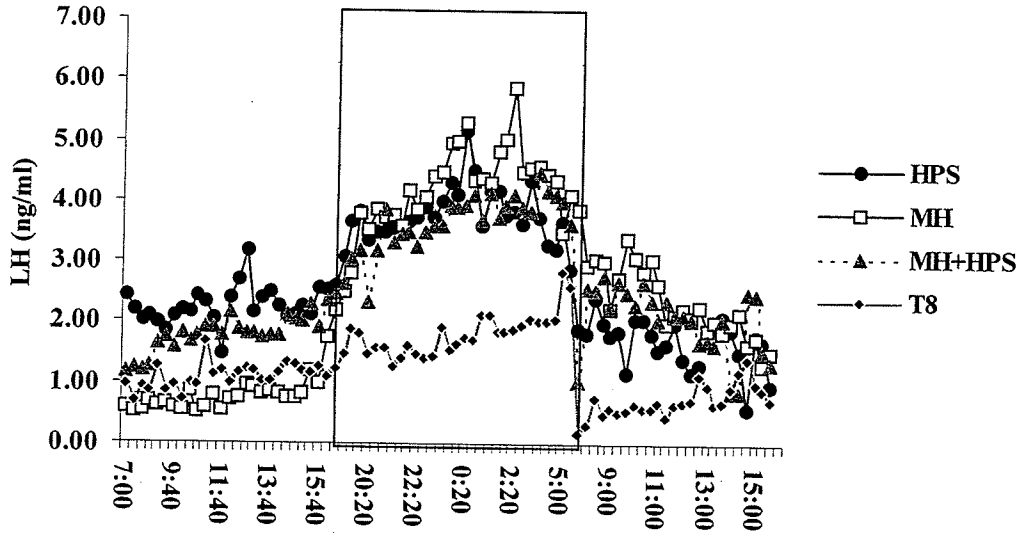
[a, b] significant difference at $P<0.0500$

Figure A2.1. Average diurnal LH concentrations for HPS, MH, MH+HPS and T8 at (a) 148D, (b) DB42ndE and (c) Dof2ndE. Boxed area indicates scotoperiod; open area indicates photoperiod.

(a)



(b)



(c)

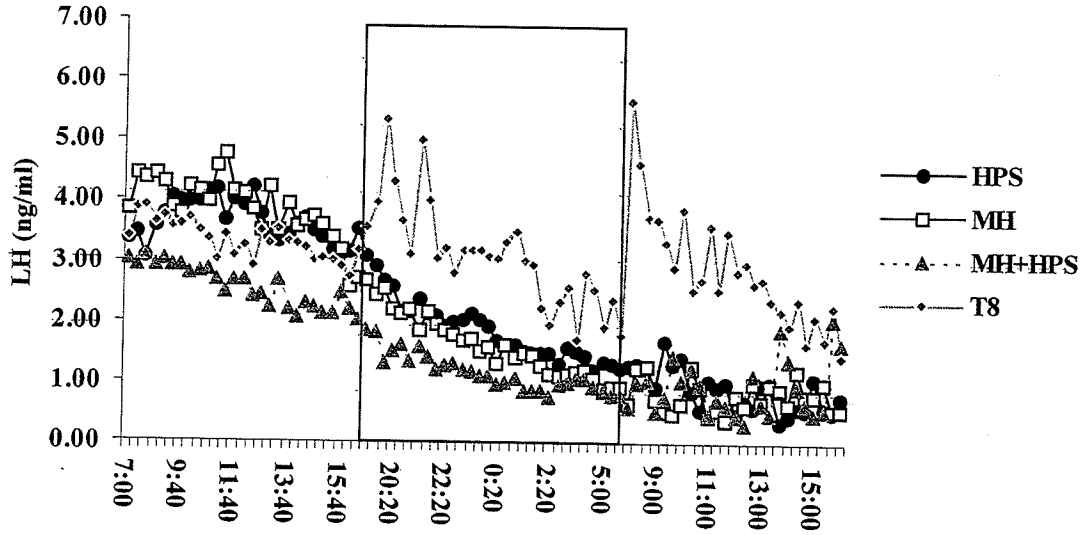

7.00
6.00
5.00
4.00
3.00
2.00
1.00
0.00
LH (ng/ml)
7:00
9:40
11:40
13:40
15:40
20:20
22:20
0:20
2:20
5:00
9:00
11:00
13:00
15:00
HPS
MH
MH+HPS
T8

Table A2.17. LH concentration±SEM for treatment by day interaction.

	LH (ng/ml)	Day	HPS	MH	MH+HPS	T8	P-Value
Combined Sample Days	Average	148D	0.5 ± 0.1^{a}	0.5 ± 0.1^{a}	0.6 ± 0.1^{a}	0.4 ± 0.1^{a}	<0.0001
		DB42ndE	3.0 ± 0.1^{a}	2.7 ± 0.1^{a}	2.7 ± 0.1^{a}	1.4 ± 0.2^{b}	
		Dof2ndE	2.3 ± 0.1^{b}	2.2 ± 0.1^{b}	1.6 ± 0.1^{c}	3.0 ± 0.1^{a}	
	Baseline	148D	0.3 ± 0.3^{a}	0.3 ± 0.3^{a}	0.5 ± 0.3^{a}	0.3 ± 0.4^{a}	0.0178
		DB42ndE	2.9 ± 0.3^{a}	2.6 ± 0.3^{a}	2.8 ± 0.4^{a}	1.4 ± 0.4^{b}	
		Dof2ndE	$2.4 \pm 0.3^{a,b}$	$2.2 \pm 0.4^{a,b}$	1.6 ± 0.3^{b}	2.9 ± 0.4^{a}	

values are LS mean ± SEM

[a, b] significant difference across rows at P<0.0500

Table A2.18. Trial differences in LH concentration at 148D, DB42ndE, Dof2ndE and combined sample days.

Sample Day	LH (ng/ml)	Trial 1	Trial 2	P-Value
148D	Average	0.5 ± 0.0	0.4 ± 0.1	0.2481
	Baseline	0.4 ± 0.0	0.3 ± 0.1	0.1106
	Amplitude	1.9 ± 0.2	1.6 ± 0.3	0.4726
DB42ndE	Average	2.8 ± 0.3	2.3 ± 0.2	0.2401
	Baseline	2.7 ± 0.3	2.2 ± 0.2	0.2884
	Amplitude	Non-est	Non-est	
Dof2ndE	Average	1.7 ± 0.4[a]	2.8 ± 0.3[b]	0.0407
	Baseline	1.7 ± 0.4[a]	2.7 ± 0.3[b]	0.0433
	Amplitude	Non-est	Non-est	
Combined Sample Days	Average	1.6 ± 0.1	1.8 ± 0.1	0.1332
	Baseline	1.6 ± 0.2	1.7 ± 0.1	0.6196
	Amplitude	Non-est	Non-est	

values are LS mean ± SEM
[a, b] significant difference at P<0.0500

Table A2.19. Trial differences in LH pulse numbers.

Pulses	Trial 1	Trial 2	Significance*
148D	63	62	No
DB42ndE	3	13	Yes
Dof2ndE	4	4	No

*significance at chi-square value of 3.841

Table A2.20. LH concentration ± SEM for trial by day interactions.

	LH (ng/ml)	Day	Trial 1	Trial 2	P-Value
		148D	0.5 ± 0.1[a]	0.5 ± 0.1[b]	
	Average	DB42ndE	2.6 ± 0.1	2.2 ± 0.1	<0.0001
Combined Sample Days		Dof2ndE	1.8 ± 0.1	2.7 ± 0.1	
		148D	0.4 ± 0.2[a]	0.3 ± 0.3[b]	
	Baseline	DB42ndE	2.7 ± 0.3	2.2 ± 0.2	0.0265
		Dof2ndE	1.8 ± 0.3	2.7 ± 0.2	

values are LS mean ± SEM

[a, b] significant difference at P<0.0500

Table A2.21(a). Trial by treatment interaction for LH.

Sample Day	LH (ng/ml)	Trial	HPS	MH	MH+HPS	T8	P-Value
	Average	T1	0.4 ± 0.1	0.5 ± 0.1	0.7 ± 0.1	0.4 ± 0.1	0.5212
		T2	0.4 ± 0.1	0.4 ± 0.1	0.4 ± 0.1	0.3 ± 0.2	
148D	Baseline	T1	0.3 ± 0.1	0.4 ± 0.1	0.6 ± 0.1	0.3 ± 0.1	0.3219
		T2	0.4 ± 0.1	0.3 ± 0.1	0.3 ± 0.1	0.2 ± 0.2	
	Amplitude	T1	1.5 ± 0.4	1.8 ± 0.5	2.4 ± 0.5	2.0 ± 0.4	0.9044
		T2	1.6 ± 0.5	1.6 ± 0.5	1.8 ± 0.5	1.6 ± 0.7	
	Average	T1	3.2 ± 0.5^{ab}	4.0 ± 0.6^{a}	1.9 ± 0.6^{ab}	1.9 ± 0.7^{ab}	0.0162
		T2	2.8 ± 0.5^{ab}	1.8 ± 0.4^{ab}	3.4 ± 0.5^{a}	1.2 ± 0.5^{b}	
DB42ndE	Baseline	T1	3.1 ± 0.5^{ab}	4.0 ± 0.6^{a}	1.8 ± 0.6^{ab}	1.8 ± 0.8^{ab}	0.0173
		T2	2.7 ± 0.5^{ab}	1.8 ± 0.4^{ab}	3.4 ±0.5^{a}	1.1 ± 0.5^{b}	
	Amplitude	T1	Non-est	Non-est	Non-est	Non-est	
		T2	Non-est	Non-est	Non-est	Non-est	

values are LS mean ± SEM

[a, b] significant difference at P<0.0500

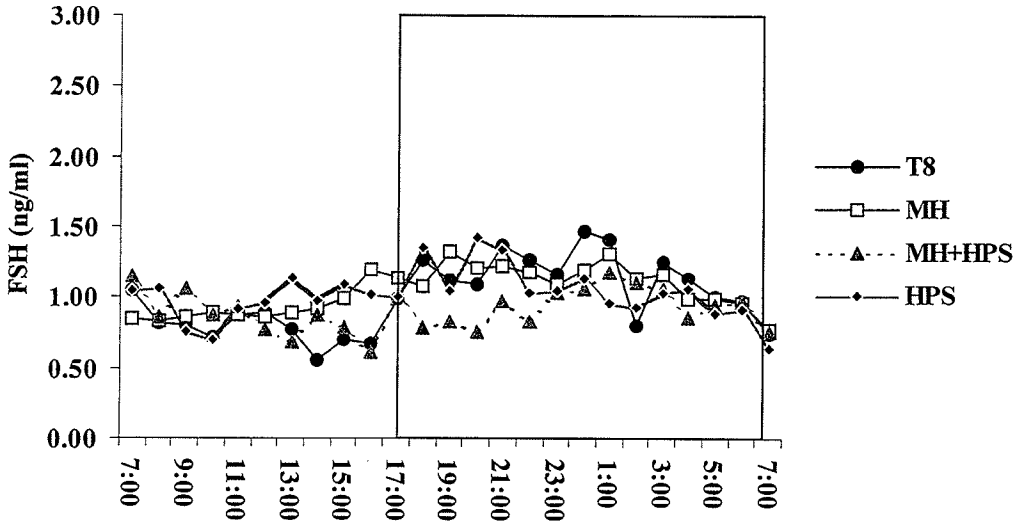
Table A2.21(b). Trial by treatment interaction for LH.

Sample Day	LH (ng/ml)	Trial	HPS	MH	MH+HPS	T8	P-Value
	Average	T1 T2	2.1 ± 0.6 2.7± 0.5	1.2 ± 0.9 2.7 ± 0.5	1.2 ± 0.6 2.1 ± 0.6	2.3 ± 0.9 3.5 ± 0.5	0.9012
Dof2ndE	Baseline	T1 T2	2.1 ± 0.6 2.7 ± 0.5	1.2 ± 0.9 2.7 ± 0.5	1.1 ± 0.6 2.1 ± 0.6	2.3 ± 0.9 3.4 ± 0.5	0.9070
	Amplitude	T1 T2	Non-est Non-est	Non-est Non-est	Non-est Non-est	Non-est Non-est	
	Average	T1 T2	1.8 ± 0.2 2.1± 0.2	1.9 ± 0.2 1.7 ± 0.1	1.4 ± 0.2 1.9 ± 0.2	1.6 ± 0.2 1.6 ± 0.2	0.2504
Combined Sample Days	Baseline	T1 T2	1.8 ± 0.3 1.9 ± 0.3	1.9 ± 0.3 1.6 ± 0.2	1.3 ± 0.3 1.9 ± 0.2	1.5 ± 0.3 1.6 ± 0.3	0.3895
	Amplitude	T1 T2	Non-est Non-est	Non-est Non-est	Non-est Non-est	Non-est Non-est	

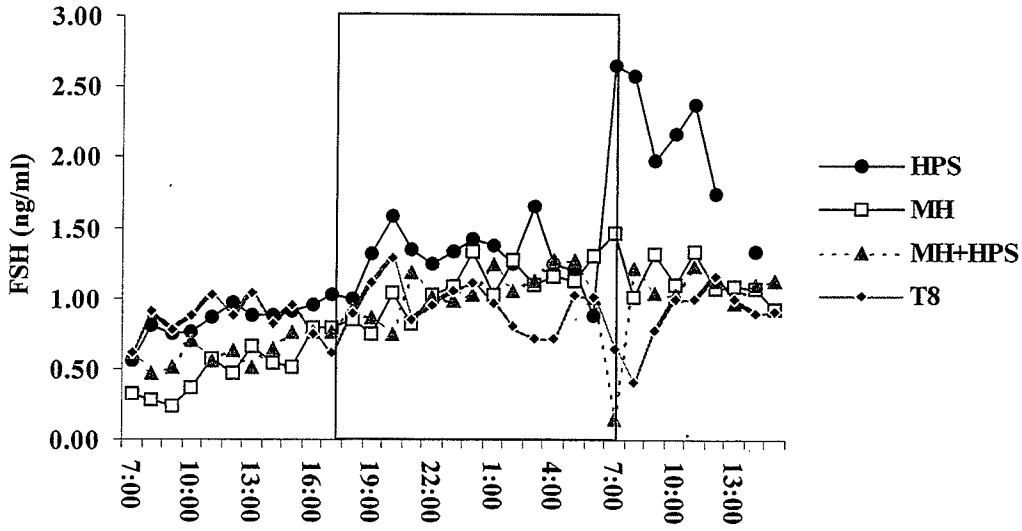
values are LS mean ± SEM

Figure A2.2. Average diurnal FSH concentrations for HPS, MH, MH+HPS and T8 at (a) 148D, (b) DB42ndE and (c) Dof2ndE. Boxed area indicates scotoperiod; open area indicates photoperiod.

(a)



(b)



(c)

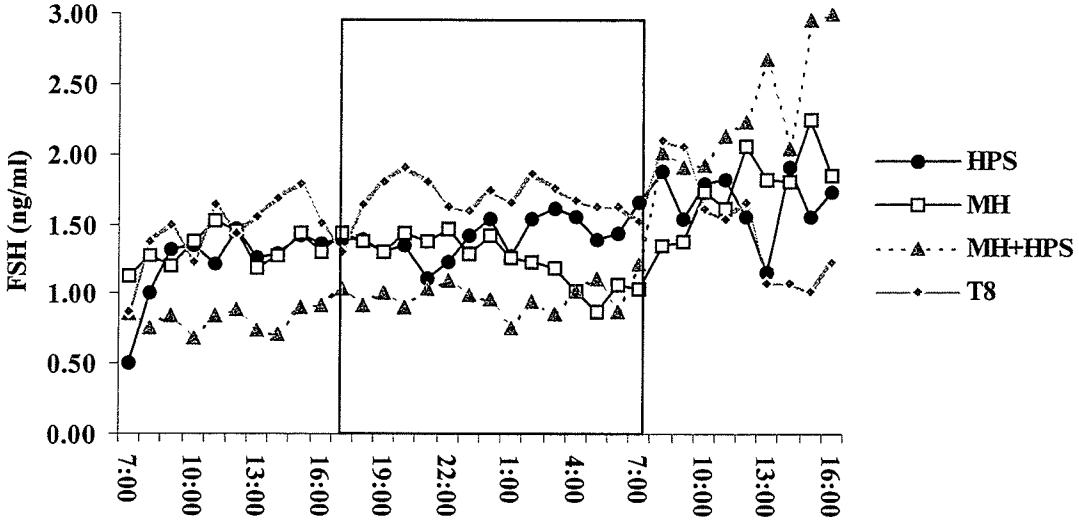

3.00
2.50
2.00
1.50
1.00
0.50
0.00
FSH (ng/ml)
7:00
10:00
13:00
16:00
19:00
22:00
1:00
4:00
7:00
10:00
13:00
16:00
HPS
MH
MH+HPS
T8

Table A2.22. FSH concentration±SEM for treatment by day interaction.

	FSH (ng/ml)	Day	HPS	MH	MH+HPS	T8	P-Value
Combined Sample Days	Average	148D	0.8 ± 0.1^{a}	1.0 ± 0.1^{a}	0.9 ± 0.1^{a}	0.8 ± 0.1^{a}	<0.0001
		DB42ndE	0.9 ± 0.1^{a}	0.8 ± 0.1^{a}	0.7 ± 0.1^{a}	0.8 ± 0.2^{a}	
		Dof2ndE	$1.3 \pm 0.1^{a,b}$	$1.3 \pm 0.1^{a,b}$	1.1 ± 0.1^{b}	1.5 ± 0.2^{a}	
	Baseline	148D	0.9 ± 0.2	1.0 ± 0.2	0.9 ± 0.2	0.9 ± 0.2	0.6859
		DB42ndE	1.0 ± 0.2	0.8 ± 0.2	0.8 ± 0.2	0.8 ± 0.2	
		Dof2ndE	1.4 ± 0.2	1.3 ± 0.2	1.1 ± 0.2	1.6 ± 0.2	

values are LS mean ± SEM

[a, b] significant difference across rows at P<0.0500

Table A2.23. Trial differences in FSH concentration across 148D, DB42ndE, Dof2ndE, and combined sample days.

Sample Days	FSH (ng/ml)	Trial 1	Trial 2	P-Value
148D	Average	1.2 ± 0.1^{y}	0.8 ± 0.2^{z}	0.0656
	Baseline	1.1 ± 0.1^{y}	0.8 ± 0.2^{z}	0.0704
DB42ndE	Average	0.9 ± 0.2	1.0 ± 0.1	0.5584
	Baseline	0.9 ± 0.2	1.0 ± 0.1	0.5849
Dof2ndE	Average	1.4 ± 0.2	1.4 ± 0.1	0.9838
	Baseline	1.4 ± 0.2	1.4 ± 0.1	0.9972
Combined Sample Days	Average	1.0 ± 0.1	1.0 ± 0.1	0.9392
	Baseline	1.1 ± 0.1	1.0 ± 0.1	0.6619

values are LS mean ± SEM
$^{y, z}$ trend at $0.0500<P<0.1000$

Table A2.24. Pulse numbers across trials for FSH at 148D, DB42ndE, and Dof2ndE.

Pulses	Trial 1	Trial 2	Significance*
148D	2	0	No
DB42ndE	0	2	No
Dof2ndE	2	1	No

*significance at chi-square value of 3.841

Table A2.25. FSH concentration ± SEM for trial by day interactions.

	FSH (ng/ml)	Day	Trial 1	Trial 2	P-Value
Combined Sample Days	Average	148D	1.2 ± 0.1[a]	0.6 ± 0.1[b]	<0.0001
		DB42ndE	0.6 ± 0.1	0.9 ± 0.1	
		Dof2ndE	1.2 ± 0.1	1.4 ± 0.1	
	Baseline	148D	1.2 ± 0.1[a]	0.7 ± 0.2[b]	0.0247
		DB42ndE	0.8 ± 0.2	1.0 ± 0.1	
		Dof2ndE	1.3 ± 0.2	1.4 ± 0.1	

values are LS mean ± SEM

[a, b] significant difference at P<0.0500

Table A2.26. Significant correlations and trends for HPS.

Trial	Correlation	r	P-Value*
T1 & T2	148D LH and 148D FSH	0.15594	0.0104
T1 & T2	DB42ndE E2 and Dof2ndE P4	-0.45305	0.0201
T1 & T2	DB42ndE E2 and Dof2ndE E2	0.35674	0.0736

*significant correlation at P<0.0500
*trend at 0.0500<P<0.1000

Table A2.27. Significant correlations and trends for MH.

Trial	Correlation	r	P-Value*
T1 & T2	148D LH and 148D FSH	0.50237	<0.0001
T1 & T2	DB42ndE E2 and Dof2ndE P4	-0.45881	0.0108
T1 & T2	148D and DB42ndE P4	0.53713	0.0120

*significant correlation at P<0.0500
*trend at 0.0500<P<0.1000

Table A2.28. Significant correlations and trends for MH+HPS.

Trial	Correlation	r	P-Value*
T1 & T2	148D LH and 148D FSH	0.30680	<0.0001
T1 & T2	148D LH and Dof2ndE P4	0.41593	0.0484
T1 & T2	DB42ndE E2 and Dof2ndE E2	0.56903	0.0037
T1 & T2	DB42ndE E2 and Dof2ndE P4	-0.61380	0.0011
T1 & T2	DB42ndE P4 and Dof2ndE P4	0.56798	0.0031

*significant correlation at P<0.0500
*trend at 0.0500<P<0.1000

Table A2.29. Significant correlations and trends for T8.

Trial	Correlation	r	P-Value*
T1 & T2	148D LH and 148D FSH	0.44488	<0.0001
T1 & T2	148D LH and Dof2ndE E2	0.59713	0.0404
T1 & T2	Dof2ndE E2 and Dof2ndE P4	0.46316	0.0076

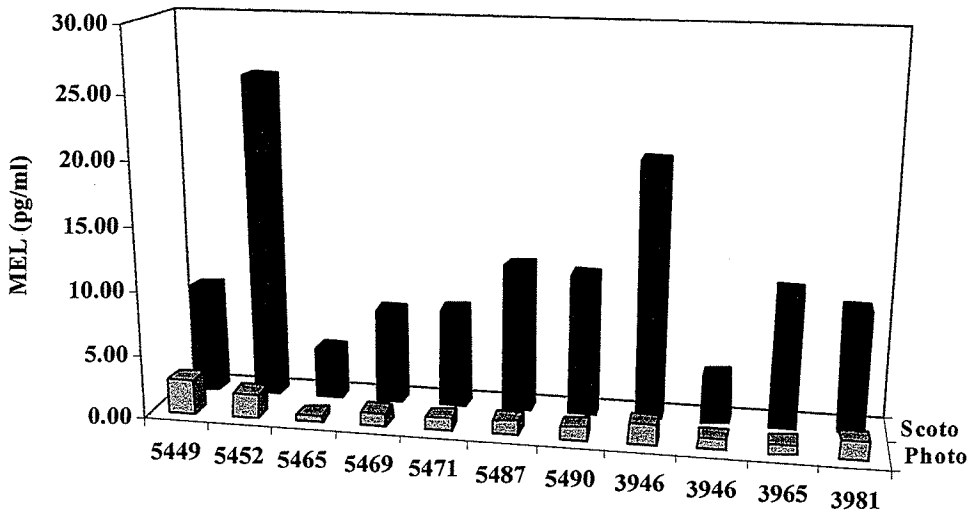
*significant correlation at P<0.0500
*trend at 0.0500<P<0.1000

APPENDIX 3

MELATONIN

Figure A3.1. HPS MEL concentration for photoperiod and scotoperiod during (a) 148D and (b) DB42ndE.

(a)



(b)

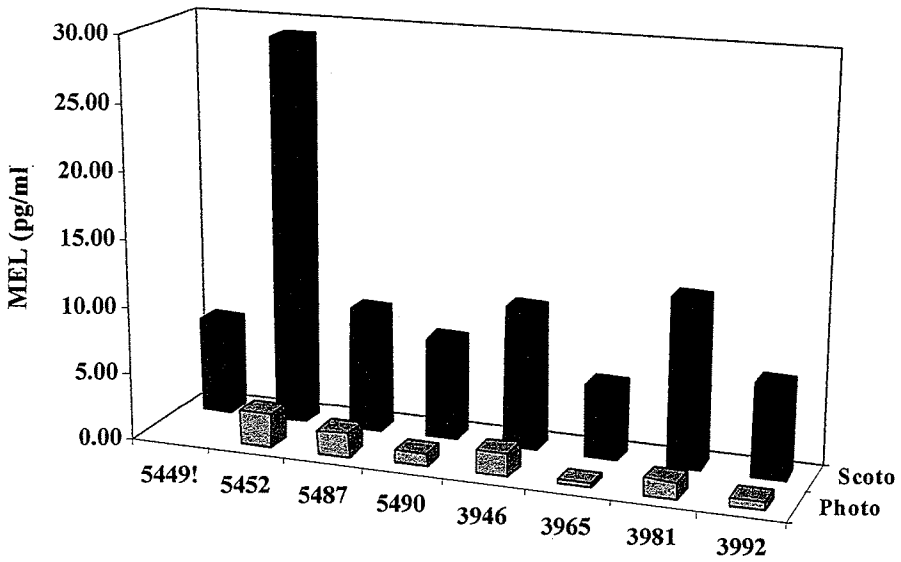
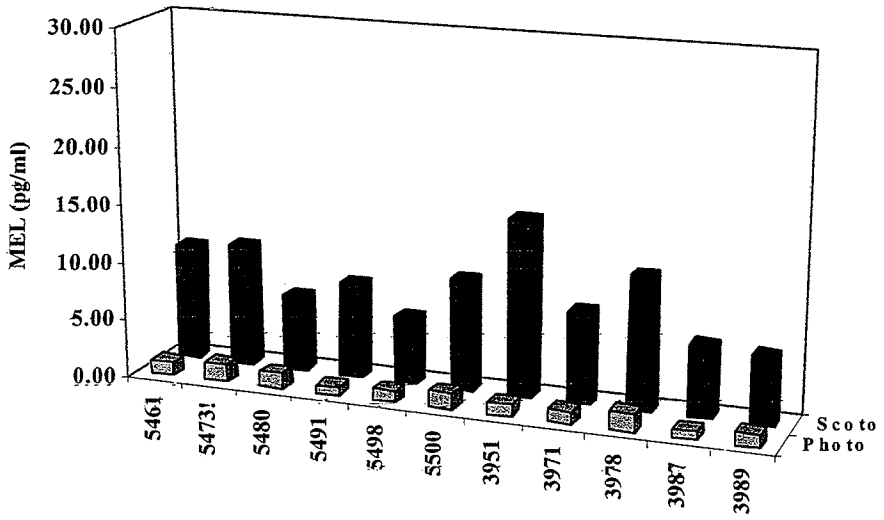


Figure A3.2. MH MEL concentration for photoperiod and scotoperiod during (a) 148D and (b) DB42ndE.

(a)



(b)

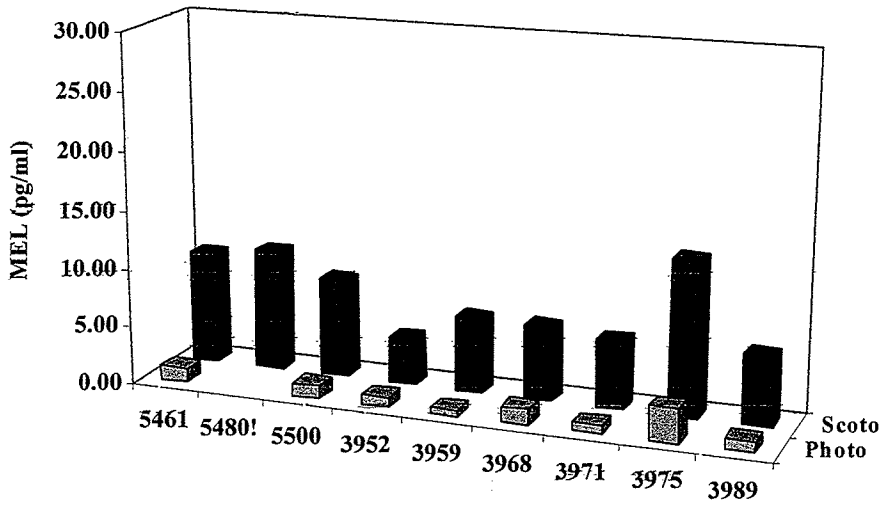
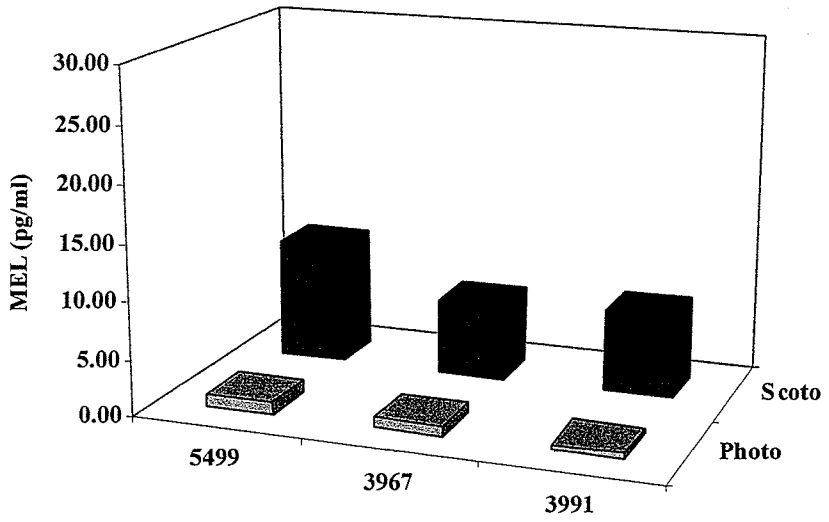


Figure A3.3. MH+HPS MEL concentration for photoperiod and scotoperiod during (a) 148D and (b) DB42ndE.

(a)



(b)

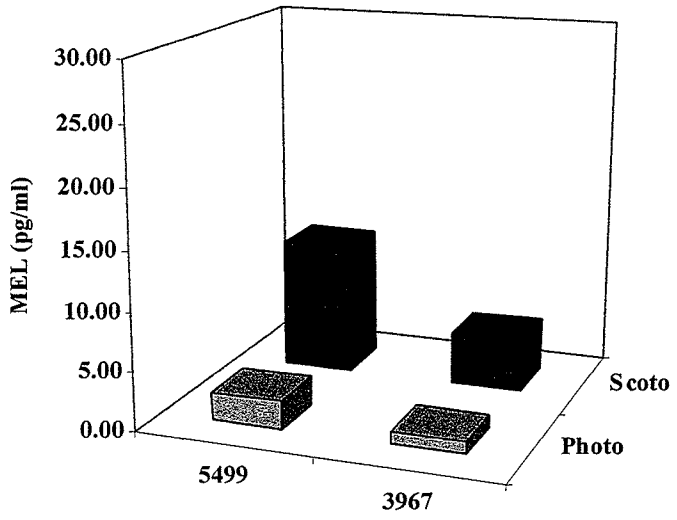
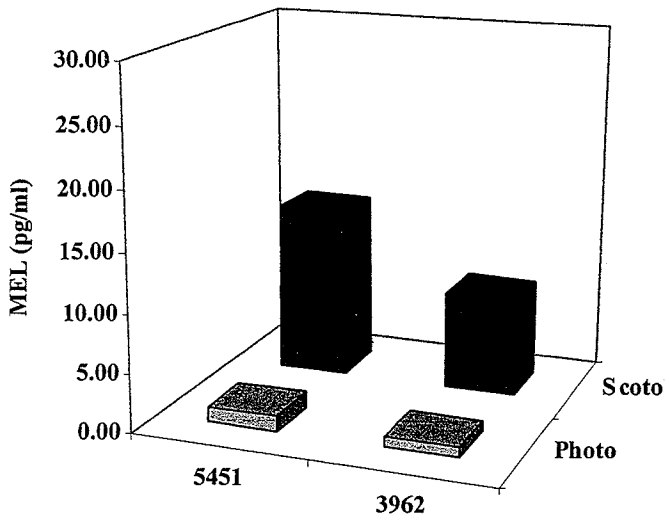


Figure A3.4. T8 MEL concentration for photoperiod and scotoperiod during (a) 148D and (b) DB42ndE.

(a)



(b)

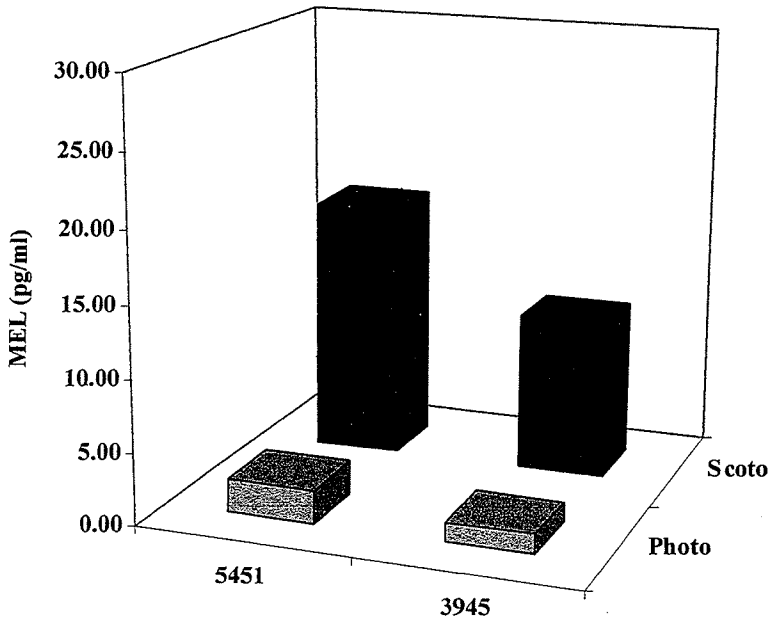


Table A3.1. Contrast significance for treatment by period interactions for DB42ndE MEL.

DB42ndE Treatment Contrast	P-Value
HPS vs MH	0.0243
(HPS vs MH) vs MH+HPS	0.1387
HPS vs MH+HPS	0.0333
HPS vs T8	0.3791
MH vs MH+HPS	0.5086
MH vs T8	0.0186
MH+HPS vs T8	0.0161
MH vs no MH	0.0016

Table A3.2. Trial by period interactions for 148D, DB42ndE, and combined sample days.

MEL (pg/ml)	Period	Trial 1	Trial 2	P-Value
148D	Photo	1.9 ± 0.7	0.5 ± 0.7	0.8175
	Scoto	10.8 ± 0.5	9.1 ± 0.5	
DB42ndE	Photo	1.5 ± 0.9[a]	0.9 ± 0.8[b]	0.0011
	Scoto	12.8 ± 0.6	7.9 ± 0.6	
Combined Sample Days	Photo	1.7 ± 1.3	1.0 ± 1.2	0.5892
	Scoto	11.5 ± 0.8	8.3 ± 0.8	

values are LS mean ± SEM

[a, b] significant difference at P<0.0500