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ABSTRACT

THE EFFECTS OF PHOTOPERIOD ON GROWTH,
BODY COMPOSITION AND SERUM HORMONES IN HOLSTEIN HEIFERS.

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Compared with heifers given 8 h of cool-white fluorescent light (CW-L) and 16 h of dark (D), weight gain increased 7, 5 and 3% in heifers given Vita-Lite® 16L:8D, CW-16L:8D or CW-6L:8D:2L:8D, respectively. Numbers of eating events were greater in heifers given CW-16L:8D compared with CW-8L:16D. Photoperiod did not affect clearance rate, secretion rate or half-life of growth hormone (GH) in serum or feed intake.

In a second experiment, relative to CW-16L:8D, excretion of 3-methylhistidine was greater in prepubertal heifers but not postpubertal heifers given CW-8L:16D. Photoperiod did not affect growth, body composition or prolactin in serum of prepubertal heifers. Average daily gain and percentages of fat in 9-10-11 rib sections were increased, and prolactin in serum and percentages of protein in rib sections were decreased in postpubertal heifers given CW-8L:16D compared with CW-16L:8D.

Photoperiod did not influence cortisol or GH in serum or feed intake in prepubertal or postpubertal heifers.

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Introduction

Increasing costs of animal agriculture and demands for quality foods which animals provide, requires further increases in efficiency of production to maintain profitability and meet demands. Integrated management involving nutrition, genetics, reproduction and disease prevention has enhanced efficiency of animal agriculture. The environment animals are exposed to affects production. For example, in cattle either extremely hot or cold temperatures adversely affect daily weight gain (Milligan and Christison, 1974; Morrison, 1983). In addition, length of daily light exposure affects average daily weight gain in sheep (Forbes et al., 1975, 1979a), cattle (Peters et al., 1978, 1980) and deer (Budde, 1983). As more animals are raised within confinement housing management of the environment becomes a more important segment of integrated management.

Confinement of animals makes management of daily photoperiod a relatively easy task. However, the most efficient and consistent scheme of daily light exposure has yet to be established. The overall objective of this thesis was to investigate management of photoperiod to maximize growth rates in cattle.

More specifically, two experiments utilizing Holstein heifers were conducted. A first experiment was designed to examine the growth response in heifers exposed to one of three different lighting schedules and two different light sources. A second experiment was designed to examine the effect of photoperiod on growth rate and body composition in prepubertal

and postpubertal heifers. Certain hormones were quantified to study hormonal response during photoperiod-induced changes in growth.

Review of Literature

Tissue Development

Animal growth is a primary goal for meat production. Growth has been defined as a correlated increase in the mass of the body in definite intervals of time, in a way characteristic of the species (Schloss, 1911) or more simply defined as an increase in size (Widdowson, 1980). The increase in body size or mass is often expressed in terms of weight gain or increases in height or length. Measuring growth in this manner fails to reveal developmental or compositional changes that may accompany or dictate changes in weight or size. In order to maximize efficiency of production and manipulate these developmental changes to produce the most desirable carcass, an understanding of the development of the major body tissues is important. This section of the review will describe development of skeletal muscle and adipose tissue and the patterns of growth of these tissues.

Skeletal muscle. Mature skeletal muscle is the net result of cell proliferation, cell differentiation, protein synthesis and protein degradation. During the growth phase synthesis of muscle proteins exceeds degradation to result in a net accretion of muscle protein. Accretion continues until, at maturity, the rate of protein synthesis is equal to the rate of protein breakdown (Winick and Noble, 1965).

Myogenic cells are derived from mesoderm. Early in the gastrula stage, mesenchymal cells are recruited into the myogenic lineage (Holtzer, 1970) and these cells eventually differentiate into presumptive myoblasts. The primary distinguishing characteristic of early myogenic cells is that they differentiate into myoblasts (Pryzblyski and Blumberg, 1966). Presumptive myoblasts are capable of proliferation, but cannot produce myofibrillar proteins of the mature muscle cell (Okazaki and Holtzer, 1965). Presumptive myoblasts mature into myoblasts.

Myoblasts are mononucleated cells that produce myofibrillar proteins of the mature muscle cell and have the capacity to fuse but they cannot divide (Okazaki and Holtzer, 1965). Myoblasts fuse to form multinucleated myotubes. Nuclei migrate from the interior of myotubes to the periphery to form myofibers (Pryzblyski and Blumberg, 1966). Similar to the mononucleated myoblast, nuclei of the multinucleated myofiber cannot synthesize deoxyribonucleic acid (DNA) and cannot divide (Stockdale and Holtzer, 1961). Even though myofibrillar nuclei cannot divide, Enesco and Puddy (1964) reported increased [^{3}H]thymidine incorporation into myofibers and Winick and Noble (1965) reported increased DNA in myofibers. These increases in nuclei division and subsequent incorporation were not due to previously differentiated myofibrillar nuclei, but due to division and incorporation of satellite cells (MacConnachie et al., 1964).

Satellite cells, small mononucleated cells lying just outside the plasmalemma of muscle fibers were first described by Mauro (1961) and confirmed by Muir et al. (1965). Satellite cells form mitotic structures and incorporate [^{3}H]thymidine, indicating they have the ability to divide (Shafiq et al., 1968; Moss and Leblond, 1970). Satellite cells are committed to the myogenic cell lineage (Young et al., 1979) and are incorported into

myofibers (Moss and Leblond, 1971). Satellite cells are the source of postnatal increases in the number of nuclei per myofiber (Church, 1969; Stromer et al., 1974).

Muscle size increases with age and is the result of postembryonic splitting of myofibrils and subsequent accretion of muscle protein within myofibers (Goldspink, 1972).

Adipose tissue. The primary function of adipose tissue is to maintain a stable energy supply. Adipose tissue provides protection for internal organs, insulation against heat loss, and enhances the flavor and juiciness of animal products. Allen et al. (1976) estimated that 75% of fat an animal produces is necessary to for metabolic processes. However, that leaves 25% of fat produced as wasted product. This excessive fat accretion in the carcass increases production costs and reduces profits (Leat and Cox, 1980).

Adipose tissue develops from mesoderm and is associated with loose connective tissue (Ham, 1969). Embryonic primitive fat cells are first identified by the accumulation of small, discrete fat droplets within the cytoplasm (Wasserman, 1965). By the time fat cells can be distinguished from other mesenchymal cells, they can no longer proliferate (Simon, 1965). Lipid accumulation continues and lipid droplets begin to fuse as the fat cell matures into an adipoblast and then into a embryonic preadipocyte (Simon, 1965). Bell (1909) was first to report the presence of preadipocytes and he concluded that the preadipocyte had progressed in the adipocyte cell lineage to near terminal differentiation. Adipocytes are distinguished from preadipocytes by the location of the nucleus. Nuclei in preadipocytes lie central in the cell whereas they have been displaced by lipid accumulation to the periphery in mature adipocytes (Simon, 1965). Postnatal adipocytes

continue to accumulate lipid and increase in volume.

Patterns of development. Postnatal growth of an animal follows a sigmoidal curve (Brody, 1945). Animals begin to grow at a slow rate, progress through a period of accelerated growth and finally gain plateaus as they near maturity. Muscle and adipose accretion follow a similar pattern, although fat accretion may not plateau with advancing age (Searle et al., 1972; Bergen, 1974). Muscle develops and matures earlier than adipose tissue (Pálsson, 1955). As a result, fat becomes proportionally greater in weight with advancing age (Searle et al., 1972). Among different depots within a tissue there is also differential development. For example, perirenal fat depots fill with lipid at an earlier age than intramuscular fat.

Therefore, to improve efficiency of animal production and to produce higher quality products, increased protein accretion in skeletal muscle coupled with control of lipid accretion is necessary.

Influence of Photoperiod

Animal growth and body composition is correlated with concentrations of hormones. For example, intact male cattle have elevated concentrations of testosterone in serum and are larger and leaner when compared with castrated cattle (Gailbraith and Topps, 1981). Season of the year and daily light exposure also affect serum concentrations of some hormones and these changes are correlated with changes in growth rate and body composition. This section of the review will describe effects of season and more specifically effects of photoperiod on concentrations of some metabolic hormones, growth and body composition in domestic animals and deer. There is no evidence photoperiod affects insulin or thyroxine secretion (Forbes et al., 1979b;

Leining et al., 1980); thus, these hormones will not be included in the review, nor will I discuss the effects of photoperiod on reproduction or reproductive hormones.

Prolactin. Prolactin is considered to be an anabolic hormone (McAtee and Trenkle, 1971), and of the metabolic hormones studied in cattle, serum concentrations of prolactin are the most responsive to changes in season, temperature and length of daily light exposure (Tucker, 1982).

Elevated concentrations of prolactin in serum have been associated with spring and summer and depressed concentrations have been observed during autumn and winter in cattle (Koprowski and Tucker, 1973a), sheep (Ravault, 1976; Munro et al., 1980; Bosc et al., 1982), goats (Buttle, 1974), deer (Mirarchi et al., 1978) and wild but not domestic pigs (Ravault et al., 1982). In these experiments, animals were exposed to natural uncontrolled seasonal conditions; therefore, the specific factor(s) that regulate the changes in prolactin could not be determined.

In heifers exposed to a controlled daily photoperiod, a rise in ambient temperature caused a rapid increase in concentrations of prolactin, while a drop in temperature had the opposite effect (Wettemann and Tucker, 1974).

In addition to changes in temperature, length of daily light exposure changes with season with most day light occurring in summer and the least in winter in all but equitorial latitudes. The seasonal changes in photoperiod are less variable year to year than seasonal changes in temperature year to year and therefore photoperiod would be a more consistent signal of seasonal change than temperature (Hendricks, 1956). To assess the effects of photoperiod on serum prolactin, independent of changes in temperature, Bourne and Tucker (1975) maintained bull calves at constant temperature

but varied daily light exposure in two experiments. In the first experiment animals were conditioned to a daily photoperiod of 16 h of light and 8 h of dark (16L:8D) for 2 weeks. During a 12-week period the amount of light was decreased from 16 h to 8 h per day. Blood was collected weekly. The second experiment was the reciprocal of the first; bulls were conditioned to 8L:16D for 2 weeks, then exposed to gradual increases in light up to 16 h during a 12-week period. At constant temperatures, decreasing daily light exposure caused an 86% decline in concentrations of prolactin, while increasing light caused a 300% increase. Therefore, independent of temperature, changes in day length alter concentrations of prolactin, but the speed of response was measured in weeks, whereas temperature-induced changes occurred within hours. Increased prolactin with long day length has also been reported in sheep (Pelletier, 1973) and deer (Brown et al., 1979). The sluggishness of the photoperiod-induced response in sheep was similar to that in cattle (Pelletier, 1973; Lincoln et al., 1978). Additional studies in sheep (Forbes et al., 1975, 1979b; Fitzgerald et al., 1982), cattle (Peters and Tucker, 1978; Peters et al., 1981) and deer (Abbott et al., 1984) confirmed results that exposure to a photoperiod of 16L:8D increased serum concentrations of prolactin when compared with animals exposed to less than 12 h of light per day. The prolactin response to 16L:8D occurs regardless of the spectral properties of light. For example, prolactin increased when the light was from red, blue, Vita-Lite® or cool-white fluorescent light, mercury vapor or high-pressure sodium lamps or incandescent bulbs (Leining et al., 1979; Stanisiewski et al., 1984a).

Increasing light exposure to 20 h per day does not increase concentrations of prolactin over that obtained with 16 h of light per day (Leining et al., 1979). In addition, Leining et al., (1979) gradually increased

daily light exposure from 8 h to 24 h of continuous light in prepubertal bulls. Average prolactin in sera increased as light exposure was increased. However, within 1 week of exposure to continuous light, average prolactin decreased to concentrations similar to that in animals exposed to 8 h of light. However, 4-year old ewes exposed to 24 h of continuous light had intermediate concentrations of prolactin relative to ewes exposed to 8 h or 16 h of light (Kennaway et al., 1983). Therefore, 24 h of continuous light is not as effective as 16L:8D, and a period of darkness in each 24 h period is required for maximum concentrations of prolactin.

Light does not need to be present in a continuous 16 h block to stimulate prolactin secretion. Similar serum concentrations of prolactin to 16L:8D can be achieved when 7 or 8 h of light is coupled with a pulse of light at a precise time during the dark. For example, Ravault and Ortavant (1977) exposed ewes to 16L:8D per day or 7 h of light plus a 1 h block of light 7,11,14,17 or 20 h after the initiation of the 7 h block of light. When the 1 h block of light was given 17 h after dawn concentrations of prolactin in ewes were similar to concentrations of prolactin in ewes exposed to 16L:8D but the response was reduced when the block of light was given 7,11,14 or 20 h after dawn. Additional work in sheep has shown 7L:9D:1L:7D or 7L:10D:1L:6D increased concentrations of prolactin in serum as effectively as 16L:8D when compared with concentrations in sheep exposed to 8L:16D (Thimonier et al., 1978; Schanbacher and Crouse, 1981; Brinklow and Forbes 1984a,b).

A 2 h block of light 8 h after a 6 h block of light (6L:8D:2L:8D) increased concentrations of prolactin in prepubertal bulls, equivalent to that observed in 16L:8D. However, a 2 h period of light 14 h after a 6 h block of light (6L:14D:2L:2D) was much less effective (Petitclerc et al., 1983a). Further

evidence in sheep suggests that the photosensitive phase is 9 to 10 h after dusk not 16 h after dawn (Terqui et al., 1984).

Petitclerc et al. (1983b) reported that the eye is essential for long daylengths to increase prolactin. Blinded animals exposed to 16L:8D had similar concentrations of prolactin compared with blind animals exposed to 8L:16D. Blocking a neural pathway from the eye to the pineal gland by superior ganglionectomy reduced the photoperiod-induced response in sheep (Lincoln et al., 1982) and goats (Buttle, 1977). Moreover, pinealectomy blocks photoperiod-induced prolactin release in ewes and wethers (Brown and Forbes, 1980; Brinklow and Forbes, 1984b) and reduces the effect in rams (Barrell and Lapwood, 1979) and prepubertal bulls (Petitclerc et al., 1983b).

Growth hormone. Growth hormone (GH) is considered to be one of the primary hormones responsible for animal growth and development (Bates et al., 1964). Secretion of GH is less responsive to season and photoperiod than secretion of prolactin.

Concentrations of GH in serum were unresponsive to changes in ambient temperature (Tucker and Wettemann, 1976). Neither increasing temperature from 21° to 32°C or decreasing temperature from 21° to 4.5°C caused a significant change in concentrations of GH. However, pituitary tissue in culture that was removed from rats exposed to near freezing temperatures had increased GH secretion (Yamato et. al., 1972).

In cattle, length of daily light exposure has little effect on concentrations of GH. Peters and Tucker (1978) exposed dairy heifers to 16L:8D or to natural winter day lengths and collected blood twice per week. Serum GH averaged 8.0 ± .7 and 7.7 ± .4 ng/ml, respectively. In a similar

designed experiment during summer (16L:8D vs natural summer day length) concentrations of GH were not affected by photoperiod (Peters and Tucker, 1978). These data were confirmed in heifers (Peters et al., 1981) and lactating sows (Kraeling et al., 1983). There were no significant changes in average concentrations of GH in prepubertal bulls conditioned to 8L:16D and subsequently switched to 16L:8D or 20L:4D; however, the variation of GH around the mean was significantly greater in animals exposed to increased daily light (Leining et al., 1980). In these experiments, blood samples were collected infrequently (twice per week) which can affect estimates of variation in average hormone concentrations (Hart et al., 1981).

In contrast to the evidence in sheep, cattle and pigs, there is a photoperiod-induced increase in GH in goats (Terqui et al., 1984). Non-pregnant goats that were induced to lactate were exposed to 8.5 or 15.5 h of light per day. Concentrations of GH were greater in the goats exposed to longer durations of daily light (Terqui et al., 1984).

Brown et al. (1979) failed to show seasonal influences on GH in red deer stags. However, increased GH from December to April and reduced GH at the summer solstice have been reported in white-tailed deer (Bubenik et al., 1975). These changes in GH have been correlated with antler regrowth. Bahnak et al. (1981) reported peak concentrations of GH in late spring, early summer and minimum GH in late autumn, early winter. This apparent seasonal effect on GH may be confounded with stage of gestation and stage of lactation, both of which influence concentrations of GH (Koprowski and Tucker, 1973b).

Glucocorticoids. Increased secretion of glucocorticoids are correlated with reduced average daily weight gain in cattle (Purchas et al., 1971).

Reported effects of season on serum concentrations of total glucocorticoids or cortisol are conflicting.

Serum glucocorticoids were decreased 29 to 58% with exposure to 15.7,16 or 20 h of light per day in prepubertal bulls previously exposed to 8 h of light per day. Red or blue supplemental light or high or low intensity (540 and 22 lux) light did not affect the decline in glucocorticoids (Leining et al., 1980). In contrast, concentrations of glucocorticoids were not different in dairy heifers (Peters et al., 1980) or in lactating cows (Peters et al., 1981) exposed to supplemental light or exposed to less than 12 h of daily light. Blood samples in these experiments were taken infrequently and therefore peaks of glucocorticoid secretions may have been missed which would affect mean concentrations. There is also a diurnal rhythm of cortisol secretion (Thun et al., 1981) which complicates interpretation when blood is collected infrequently.

Young sheep were exposed to 8 h of light per day on a skeleton photoperiod (7L:10D:1L:6D), which mimics long day length. Blood samples were collected every 20 min for 24 h after 38 days of light treatment. Average concentrations of cortisol were lower in sheep exposed to the skeleton photoperiod compared with sheep exposed to 8 h of light per day (Brinklow and Forbes, 1984a). Differences in average cortisol values were due to changes in peak height, not number of peaks. There was no consistent diurnal rhythm of the cortisol peaks (Brinklow and Forbes, 1984a). The same experimental design was utilized with 3- and 10-month old lambs (Brinklow and Forbes, 1984b). Similar to the first experiment, skeleton photoperiods reduced concentrations of cortisol in 3-month old lambs compared with lambs exposed to 8L:16D. Pinealectomy reduced the decline in serum cortisol (Brinklow and Forbes, 1984b). In contrast, no effect of photoperiod was

observed on cortisol in 10-month old lambs.

Additional experiments with young lambs (Kennaway et al., 1981), adult rams (Lincoln et al., 1982) and white-tailed deer (Bubenik et al., 1975) failed to reveal an effect of long day length on concentrations of glucocorticoids. However, long day length (16L:8D) compared with short day length (8L:16D) increased serum glucocorticoids in lactating sows (Kraeling et al., 1983) and young pigs (Barnett et al., 1981).

Conflicting effects of day length on glucocorticoids may be the result of infrequent blood sampling, age or species of animal utilized, or time of feeding relative to bleeding. Brinklow and Forbes (1984b) suggested the effects of photoperiod on glucocorticoids may be limited to young animals, even though Kennaway et al. (1981) failed to observe an effect in 13-week old lambs. Feeding has been reported to entrain concentrations of glucocorticoids in rats (Krieger and Hauser, 1978) and sheep (Lincoln et al., 1982).

Body growth and body composition. Forbes et al. (1975) reported increased average daily weight gains in ewes exposed to 16L:8D compared with ewes exposed to 8L:16D. Increased average daily weight gains in response to long day lengths occurred in ewes fed a restricted diet (70g·kg live weight $^{-.75}$·day) or in ewes fed ad libitum. Significant differences in weight gain were not observed until animals were on photoperiod treatments for 8 to 12 weeks (Forbes et al., 1979a). Increased average daily weight gain in animals exposed to increased day length compared with animals exposed to short day length has been reported also in rams and wethers (Schanbacher and Crouse, 1980, 1981; Brown and Forbes, 1980). In contrast, Hoersch et al. (1961), Fitzgerald et al. (1982) and Eisemann et al. (1984a) failed to observe

a beneficial effect of supplemental light on average daily weight gain in ewes and wethers.

Exposure to 16L:8D, compared with 8L:16D, increased carcass weight in sheep (Forbes et al., 1975, 1979a; Schanbacher and Crouse, 1980, 1981). In a few experiments, the carcasses of sheep exposed to 16L:8D had more muscle mass than sheep exposed to 8L:16D (Forbes et al., 1979a; Schanbacher et al., 1982). In addition, Jones et al. (1982) reported a tendency for reduced fat in 11 to 13 rib sections, caul and mesenteric fat depots and fat depth over the rib-eye in sheep exposed to long day length (20L:4D) compared with sheep exposed to natural short day length (< 12 h light per day). In contrast, others have reported no difference in fat of animals exposed to long or short days in the entire carcass, 11 to 13 rib section, kidney or pelvic fat, backfat thickness or quality or yield grades (Eisemann et al., 1984b; Forbes et al., 1975, 1979a; Schanbacher and Crouse, 1980, 1981).

Photoperiodic-induced increases in body and carcass weight in lambs were not affected by environmental temperature (5, 18 and 31°C) (Schanbacher et al., 1982) but tended to be reduced by pinealectomy (Brown and Forbes, 1980).

Continuous light exposure reduced average daily weight gain in sheep compared with sheep exposed to natural day length (Moose and Ross, 1962; Hulet et al., 1968) or 8L:16D (Hoersch et al., 1961). Light does not have to be given in a continuous block of 16 h to stimulate average daily weight gain. Sheep exposed to a 1 h period of light in the critical period 9 to 10 h after a 7 h block of light (7L:9D:1L:7D or 7L:10D:1L:6D) have increased growth rates compared with sheep exposed to 8L:16D and similar growth rates to sheep exposed to 16L:8D (Schanbacher and Crouse, 1981; Brinklow and Forbes, 1982). If sheep are not exposed to light during this critical period,

no advantage in average daily weight gain is observed (Hackett and Hillers, 1979).

Similar to sheep, cattle exposed to 16 h of light per day had increased heart girths and body weights in animals exposed to natural winter day lengths (Peters et al., 1978; Sorensen, 1984) or 8L:16D (Peters et al., 1980; Petitclerc et al., 1983c). No increased weight gain was observed in animals exposed to 16L:8D compared with animals exposed to natural summer day length (Peters et al., 1978). Increased body weights are not due solely to increased feed intake. Photoperiod induced gains persist when feed intake is restricted and equal amounts of dry matter are offered across photoperiod treatments (Petitclerc et al., 1983c). In agreement with observations in sheep (Forbes et al., 1979a), restricted fed cattle exposed to 16L:8D had a larger percentage increase in body weight than cattle fed ad libitum (Petitclerc et al., 1983c). Daily gain in the carcass and protein content of the 9 to 11 rib section increased with exposure to 16L:8D (Petitclerc et al., 1984).

In contrast, no beneficial effects of long day length compared with short day length were observed in heifers (Hansen et al., 1983), young bulls (10 to 14 days old) or steers (Roche and Boland, 1980). Tucker et al. (1984) also failed to observe a growth response to supplemental light in Holstein steers. This may indicate that in cattle, unlike sheep, the anabolic response to photoperiod may be gonad dependent.

Continuous night lighting increased average daily weight gain in feed lot cattle (Robertson and Lipper 1964; Lipper et al., 1971). Others have confirmed these results, but only in cattle fed low energy diets (Boren et al., 1965; Smith et al., 1964). Parsons et al. (1964) reported no effect of night lighting on weight gain but observed increased feed efficiency. In contrast, heifers exposed to continuous light gained significantly less weight

than heifers exposed to 16L:8D and no better than heifers exposed to natural winter day lengths (Peters et al., 1980).

In contrast to sheep and cattle, shortening of day length stimulated weight gain in white-tailed deer fawns (Verme and Ozoga, 1980). This was confirmed under controlled photoperiods (Budde, 1983, Abbott et al., 1984). Carcass weights were increased in fawns exposed to shortening day lengths (Verme and Ozoga, 1980) or 8L:16D (Abbott et al., 1984). In addition, the percentage of fat in the carcass and abdominal fat were substantially increased in fawns exposed to short day lengths. Therefore, increased weight gain in deer exposed to short day lengths is primarily increased fat deposition.

There is no good evidence that supplemental lighting is beneficial to growth in pigs (Dufour and Bernard, 1968; Hacker et al., 1979; Mahone et al., 1979; Hoagland and Diekman, 1982; Diekman and Hoagland, 1983).

Supplemental lighting improves body and carcass gains and may improve carcass quality (increased protein content) in sheep and cattle. Short days stimulate weight gain and fat deposition in deer.

Materials and Methods

Experiment 1

Introduction. Vita-Lite® (Duro-Test Corporation, North Bergen, NJ) is a fluorescent light source that emits wavelengths of light in a pattern more similar to natural sunlight than cool-white fluorescent light (Wurtman and Weisel, 1969). Exposure to Vita-Lite fluorescent light has been reported to increase calcium absorption in men (57 to 80 years old) compared with men exposed to cool-white fluorescent light (Neer et al., 1971). In cattle, increases in prolactin were similar in prepubertal bulls exposed to 16 h of Vita-Lite fluorescent or 16 h of cool-white fluorescent lights (Stanisiewski et al., 1984a). Stimulatory effects of photoperiod on milk production were also similar between the two fluorescent light sources, although there was a tendency for Vita-Lite to be superior (Stanisiewski et al., 1984b). The first objective of this experiment was to compare body weights in prepubertal heifers exposed to 16 h of Vita-lite or 8 or 16 h of cool-white fluorescent light per day.

Effects of 16L:8D on prolactin secretion and growth rate in sheep can be mimicked by skeleton photoperiods when 7 h of light is coupled with a 1 h block of light in the photosensitive phase, 10 or 11 h later (Ravault and Ortavant, 1977; Thimonier et al., 1978; Schanbacher and Crouse, 1981; Brinklow and Forbes, 1984a,b). In cattle, the photosensitive phase for prolactin secretion is 14 to 16 h after dawn (Petitclerc et al., 1983a). The

second objective of this study was to compare body weight gain in prepubertal heifers exposed to a skeleton photoperiod of 6L:8D:2L:6D or to photoperiods of 8L:16D or 16L:8D.

Photoperiod has little effect on mean serum concentrations of GH in cattle (Peters et al., 1981; Leining et al., 1980). However, average serum concentrations are a function of metabolic secretion and clearance rates; thus average hormone concentrations, although not different, may be a result of differences in secretion or clearance rate (Hart et al., 1980). The final objective of this experiment was to compare the effects of 8 or 16 h of light per day on metabolic clearance and secretion rates and half-life of GH in serum of Holstein heifers.

Animals and management. Sixty-four prepubertal Holstein heifers, approximately 3-months of age (average body weight 102 kg), were blocked by body weight into four groups of 16. Heifers were housed unrestrained in separate light-controlled pens and no supplemental heat was provided. Each group was assigned to one of four photoperiod treatments; 16 h of cool-white fluorescent light:8D, 8 h of cool-white fluorescent light:16D, 16 h of Vita-Lite fluorescent light:8D or 6L:8D:2L:6D (source of L = cool-white fluorescent light). Lights came on at 0700 h each day in all pens. Light intensity, measured at approximate eye level of the heifers averaged 230 lux in each pen. Photoperiod treatments began on October 31 and continued for 112 days.

All groups received the same complete mixed diet, fed ad libitum. The diet was a mixture of corn silage, alfalfa haylage, high moisture ear corn and a 40% protein supplement, formulated for heifers to gain approximately .9 kg/day. Fresh feed was offered daily at 0800 h and group

feed refusals were recorded each day. In addition to feed intake, eating patterns of heifers exposed to 8 or 16 h of cool-white fluorescent light per day were monitered. Eating patterns were determined by counting numbers of animals eating at 10-min intervals for 30 h beginning at 0700 h on day 110 of photoperiod treatment. Approximately once per month, animals were deprived of water for 16 h and weighed.

Clearance rate, secretion rate, half-life of GH. Metabolic clearance (CR) and secretion rates (SR) for GH were determined by a steady-state, constant infusion method (Tait, 1963). Calculations of CR, SR and half-life ($t_{\frac{1}{2}}$) were as follows: CR (ml/min) = GH infusion rate (ng/min) [serum GH (ng/ml) at steady state minus preinfusion concentrations of serum GH (ng/ml)]; SR (ng/ml) = CR (ml/min) X preinfusion concentrations of serum GH (ng/ml); $t_{\frac{1}{2}}$ (min) = ln 2 (slope X ln10). Slope was calculated from a linear regression equation generated from log GH concentrations (ng/ml) versus time (35 min) postinfusion of GH (Akers et al., 1980).

To determine if infusion rate affected CR, SR and $t_{\frac{1}{2}}$ estimates of endogenous GH, two Holstein heifers were infused with either 2 or 4 mg/h of GH (NIH-b18) for 4 h. Infusion rates were reversed between the heifers the next day.

At the conclusion of the growth trial, eight heifers exposed to 8L(cool-white fluorescent):16D and their corresponding block mates exposed to 16L (cool-white fluorescent):8D were infused with 1.5 mg/h of GH (NIH-b18) for 4 h. Four heifers (two heifers exposed to 8L:16D and two heifers exposed to 16L:8D) were infused on each of four days.

On the morning of infusion, heifers were fitted with polyvinyl cannulas in both jugular veins; one side for blood sampling and the other for infusion

of GH. Preinfusion concentrations of GH were determined in sera collected at 10-min intervals for 30 min prior to initiation of infusion. GH was infused at a steady rate using a constant infusion pump (Harvard Apparatus Co., Cambridge, MA). Blood was collected every 15 min during infusion. GH assayed in sera collected during the last hour of infusion was used to estimate concentrations of GH at steady state. Blood was collected every 5 min for 60 min after infusion was terminated. GH assayed in sera collected during the first 35 min following infusion of GH was used to calculate $t_{\frac{1}{2}}$ of GH.

GH for infusion was prepared in a sterile solution containing 2.1% $NaHCO_3$, 2.6% $NaCO_3$, .9% NaCl and .1% bovine serum albumin (BSA) at a pH of 8.0. Before infusion, cannulae were flushed with .1% BSA in .9% NaCl to minimize absorption of GH to the polyvinyl.

Blood was stored at 20°C for 2 to 6 h, then stored overnight at 4°C. The following afternoon, serum was obtained by centrifugation at 2000 x g for 30 min. Sera were decanted and stored at -20°C until assayed for GH, as previously described by Purchas et al. (1970). Bovine GH (NIH-b12) was used as reference standard.

Statistical analysis. Body weights were analyzed by split-plot analysis of variance (Gill and Hafs, 1971). Differences between treatment means of body weight were compared by the Bonferroni test procedure of non-orthogonal contrasts (Gill, 1978). Average body weights of animals on each treatment at each period were compared by Dunnett's procedure (Gill, 1978). Treatment means of CR, SR and $t_{\frac{1}{2}}$ of GH in serum were compared by the two treatment t-test (Gill, 1978).

Experiment 2

Introduction. Exposure to 16 h of light per day increased growth in cattle 10 to 17% (Peters et al., 1978, 1980). There is some evidence that age or size of animals when first exposed to increased day length may affect subsequent growth. For example, Reynolds and Roche (1982) reported a stimulatory effect of supplemental light on growth in weanling heifers, but not in finishing heifers. In addition, small heifers (112 kg at start of treatment) exposed to supplemental light increased body growth approximately 9%, but photoperiod had no effect on body growth in larger heifers (140 or 170 kg at start of treatment) (Tucker et al., 1984). However, the onset of puberty may be confounded with initiation of light treatment and the growth response in these experiments (Reynolds and Roche, 1982; Tucker et al., 1984). Supplemental light tends to hasten onset of puberty in dairy heifers (Petitclerc et al., 1983c). The first objective of this trial was to compare the effects of 8 and 16 h of light per day on weight gain in Holstein heifers which would remain prepubertal for the duration of the treatment period and in Holstein heifers that were postpubertal prior to the treatment period.

Exposure to long day lengths increased carcass weights, muscle mass and protein content with slight or no change in fat content compared with animals exposed to short day lengths (Forbes et al., 1975, 1979a; Schanbacher and Crouse, 1980, 1981; Petitclerc et al., 1984). Increased muscle protein mass is a function of muscle protein turnover and may be the result of decreased myofibrillar protein degradation (Allen et al., 1979). Urinary excretion of 3-methylhistidine (3MeHis) is a quantitative estimate of myofibrillar protein degradation in cattle (Harris and Milne, 1981; McCarthy

et al., 1983). Urinary excretion of creatinine is an estimate of muscle mass (Waterlow, 1969). The second objective of this study was to compare the effects of 8 or 16 h of light per day on body composition and urinary 3MeHis and creatinine excretion in prepubertal and postpubertal heifers.

In addition, serum hormone concentrations of prolactin, GH and cortisol were quantified to determine hormonal response during photoperiod-induced increases in growth and protein accretion.

Animals and management. Forty-two prepubertal Holstein heifers (approximately 2-months of age) and 42 postpubertal Holstein heifers (approximately 10-months of age) were utilized in the trial. Ten heifers from each age group were randomly selected for pretreatment slaughter (20 animals total). The remaining 32 heifers in each age group were paired by body weight (average body weight; prepubertal heifers, 84 kg; postpubertal heifers, 300 kg) and assigned to photoperiod treatments of 8L:16D or 16L:8D (16 prepubertal heifers per photoperiod treatment; 16 postpubertal heifers per photoperiod treatment).

Heifers in the postpubertal group were postpubertal prior to initiation of photoperiod treatments as determined by monitoring serum progesterone (Convey et al., 1977). Concentrations of progesterone greater than 1 ng/ml were used to indicate presence of a functional corpus luteum and therefore onset of estrous cyclicity and puberty. Beginning at 205 kg body weight a biweekly blood sample was taken from prepubertal heifers and assayed for progesterone to determine onset of estrous cyclicity. At slaughter, ovaries from prepubertal heifers were visually examined for corpora lutea as well. All but four of the prepubertal heifers remained acyclic through the entire treatment period.

Animals were housed and fed similar to heifers in Experiment 1, however the ration in this experiment was formulated for prepubertal heifers to gain 1 kg/day. This diet was fed, ad libitum, to all prepubertal and postpubertal heifers. Light treatments began on October 9 and continued for 142 days. Animals were weighed approximately every 2 weeks.

Slaughter and body composition. Slaughter of 10 prepubertal and 10 postpubertal heifers began 24 days prior to initiation of photoperiod treatment. Three animals from one age group and two from the other age group were slaughtered on each of four different days (total of 5 animals slaughtered per day). Ten heifers and their pair-mates were selected for slaughter at the conclusion of the growth trial (40 animals total). One heifer from each of three treatment groups and two heifers from the fourth treatment group were slaughtered on each of 8 different days (total of 5 animals slaughtered per day).

Fourteen hours prior to slaughter animals were weighed and transported to the abattoir. Heifers were housed overnight without feed or water. Animals were stunned and then killed by exsanguination. Carcass and cannon bone length and weights of the carcass, semitendinosus muscle, cannon bone, kidney and omental fat and other carcass characteristics were obtained on the day of slaughter. Each carcass was washed with water and stored overnight at 0°C. The following morning, semimembranosus and quadriceps muscles, and tibia and femur bones were removed from the right leg and weighed. Length of tibia and femur, and loin eye area and fat depth at the 12th rib were also obtained. The 9-10-11 rib section from both sides of the carcass were dissected according to the method of Hankins and Howe (1946). Rib sections were weighed, deboned, ground and subsampled for

analysis of lipid, water and protein content. Fat was determined on dried samples by ether extraction and protein was determined on fresh sample macro-Kjeldahl procedures (AOAC, 1965).

Blood collection and hormone assays. On day 94 of photoperiod treatment the five heaviest heifers of the 10 animals designated for slaughter from each of the four treatment groups (20 animals total) were moved to stanchions. Photoperiod treatments, rations and feeding schedules were identical to those of animals that remained in pens. Animals were fitted with a polyvinyl cannula in the jugular vein. The next day, beginning at 0600 h blood samples were taken from each animal every 30 min for 26 h. Blood was stored at 20°C for 6 to 8 h and then stored overnight at 4°C. The next day, sera were obtained by centrifugation at 2000 x g for 30 min. Sera were decanted and stored at -20°C until assayed for concentrations of prolactin (Koprowski and Tucker, 1971), GH (Purchas et al., 1970) and cortisol (Appendix 1). Bovine prolactin (NIH-b3), GH (NIH-b12) and cortisol (Sigma Chemical Co, St Louis, MO) were used as reference standards.

Urine collection. Prior to initiation of photoperiod treatments the 40 heifers selected for slaughter at the conclusion of the growth trial were transferred to individual stanchions. One-half of these animals were transferred 28 days prior to photoperiod treatment and the other one-half were transferred 21 days prior to photoperiod treatment. A total of 5 prepubertal and 9 postpubertal heifers assigned to each photoperiod group were fitted with indwelling Foley catheters (C.R. Bard Inc., Murray Hill, NJ). Catheters were placed through the urethra and into the bladder of each heifer. Catheter size utilized in prepubertal heifers was 16 French,

30cc balloon; postpubertal heifers were fitted with 20 French, 30 cc balloon catheters. Catheters were maintained in each heifer for 7 days and urine collected continuously the last 4 days. Urine was collected in 5 gallon containers by connecting polyethylene tubing (Tygon®, Norton, Akron, OH) to the catheter. Approximately 100 ml of 6 N hydrochloric acid were added to each collection container. Urine was collected and weighed each day at 1630 h. Urine was mixed and 120 ml of acidified urine were saved from each animal and stored at -20°C until analysis.

After exposure to photoperiod treatment these same heifers with four additional prepubertal heifers from each photoperiod treatment were subjected to the same urine collection regimen as the first groups. Twenty heifers (five prepubertal, 8L:16D; five prepubertal, 16L:8D; five postpubertal, 8L:16D; five postpubertal, 16L:8D) began the urine collection regimen after 96 days on photoperiod treatment. The remaining 16 heifers began the urine collection regimen after 103 days on photoperiod treatment. Photoperiod treatment, ration and feeding schedule of urine-collected animals were identical to heifers that remained in pens.

3-Methylhistidine analysis. One percent of the urine collected on each of the four days of the pretreatment collection period and each of the four days of the final collection period was composited for each heifer. Eight ml of composited urine were deproteinized with .8 ml 50% sulfosalicyclic acid at 90°C for 30 min and then centrifuged at 2000 x g for 30 min. Urine samples were filtered through a metricel (Gelman Sciences Inc., Ann Arbor, MI) .2 micron membrane filter. One ml of 1 M pyridine was added to 4 ml of filtered urine and applied to a column (1.5 x 7.5 cm) which contained the cation exchange resin, Dowex 50w-x8, 200-400 mesh (Sigma Chemical

Co., St. Louis, MO). Prior to application of the sample, 30 ml of distilled water and 30 ml of .2 M pyridine were passed through the column to desalt and reequilibrate the column. The 3MeHis fraction was eluted with 120 ml of 1 M pyridine. Remaining amino acids were removed with an additional 130 ml of 1 M pyridine.

The 3MeHis fraction collected was evaporated to dryness, dissolved in 5 ml of .01 N HCL and filtered through a metricel .2 micron membrane filter. L-α-amino-guanidinopropionic acid (GPA, 100 nM, Pierce Chemical Co., Rockford, IL) was used as standard. Samples were analyzed by ion exchange chromotagraphy and amino acids eluted with a sequence of four lithium citrate buffers (I. .25 N Li citrate, pH 2.67; II. .45 N Li citrate, pH 3.2; III. 1.0 N Li citrate, pH 4.17; IV 1.2 N Li pH 5.05; Pierce Chemical Co., Rockford, IL) as mobile phase and post column derivitization with ninhydrin (McCarthy et al., 1983).

Calculation of 3MeHis excretion per day was as follows:

I. 3MeHis (nmoles/ml urine) =

$$\left[\frac{\text{Area of 3MeHis in sample } (cm^2)}{\text{GPA in standard (nmoles)}} \times \frac{\text{GPA in standard (nmoles)}}{\text{Area of 3MeHis standard } (cm^2)}\right] \left[\frac{\text{GPA in sample (nmoles)}}{\text{Area of GPA in sample } (cm^2)} \times \frac{\text{Area of GPA in standard } (cm^2)}{\text{GPA in standard (nmoles)}}\right]$$

$$\left[\frac{\text{Sample size (ml)}}{\text{Sample size + internal standard added (ml)}}\right]$$

II. 3MeHis (nmoles/day) = 3MeHis (nmoles/ml urine) X urine produced (ml/day)

Creatinine analysis. Urinary creatinine was determined using Sigma kit number 555-A (Sigma Chemical Co., St. Louis, MO) based on the Jaffé reaction (Sigma, 1982).

Statistical analysis. Differences between photoperiod treatment means, within each age group (prepubertal or postpubertal) were compared. Average daily weight gain between consecutive weigh periods and average daily weight gain from the beginning of treatment to any weigh period were analyzed by paired t-test (Gill, 1978). Differences in body composition and other carcass characteristics between photoperiods or between final and pretreatment slaughter, were compared by analysis of variance (Gill, 1978). Carcass weight and days on experiment were used, individually, as covariates in the analysis of body composition and carcass characteristics at final slaughter. Treatment means of urinary excretion of 3MeHis, creatinine, 3MeHis/kg body weight, creatinine/kg body weight and ratio of 3MeHis/creatinine were compared by paired t-test in postpubertal heifers and by analysis of variance in prepubertal heifers. To minimize heterogenous variance, concentrations of serum hormones were transformed to natural logarithms for analysis. Room temperature at the time of each blood sample was used as a covariate. Concentrations of hormone were compared by split-plot analysis of variance (Gill and Hafs, 1971).

RESULTS

Experiment 1

Weight gain. Body weight of all heifers averaged 102 kg at the start of photoperiod treatment. Weight increased to 206, 210, 212 and 214 kg after 112 days on photoperiod treatments of 8L:16D (cool-white fluorescent light); 6L:8D:2L:8D (cool-white fluorescent light); 16L:8D (cool-white fluorescent light); and 16L:8D (Vita-Lite fluorescent light), respectively (Figure 1). There was no significant effect of photoperiod on average daily weight gain ($P<.10$). However, heifers exposed to 16 h of Vita-Lite:8D weighed more ($P<.05$) on day 112 of treatment compared with heifers exposed to 8 h of cool-white fluorescent light:16D.

Feed intake and eating patterns. Animals within a treatment were fed as a group. Daily dry matter intake per pen averaged 79.8, 80.3, 82.4 and 79.0 kg in animals exposed to 16L:8D (cool-white fluorescent light); 6L:8D:2L:8D (cool-white fluorescent light); 16L:8D (Vita-Lite fluorescent light); and 8L:16D (cool-white fluorescent light), respectively (Figure 2).

The effects of photoperiod on eating patterns are presented in figure 3. Animals given 16L:8D had more eating events than heifers on 8L:16D (444 vs 396) in the first 24 h of the observation period, but there was no apparent difference in feed intake. Heifers exposed to 16L:8D had 96% of their eating events in the light period of the day, whereas heifers exposed to 8L:16D had

Figure 1. Changes in body weight of Holstein heifers in response to photoperiod. Each point represents the mean of 16 animals. Pooled standard error was 3.8 kg.

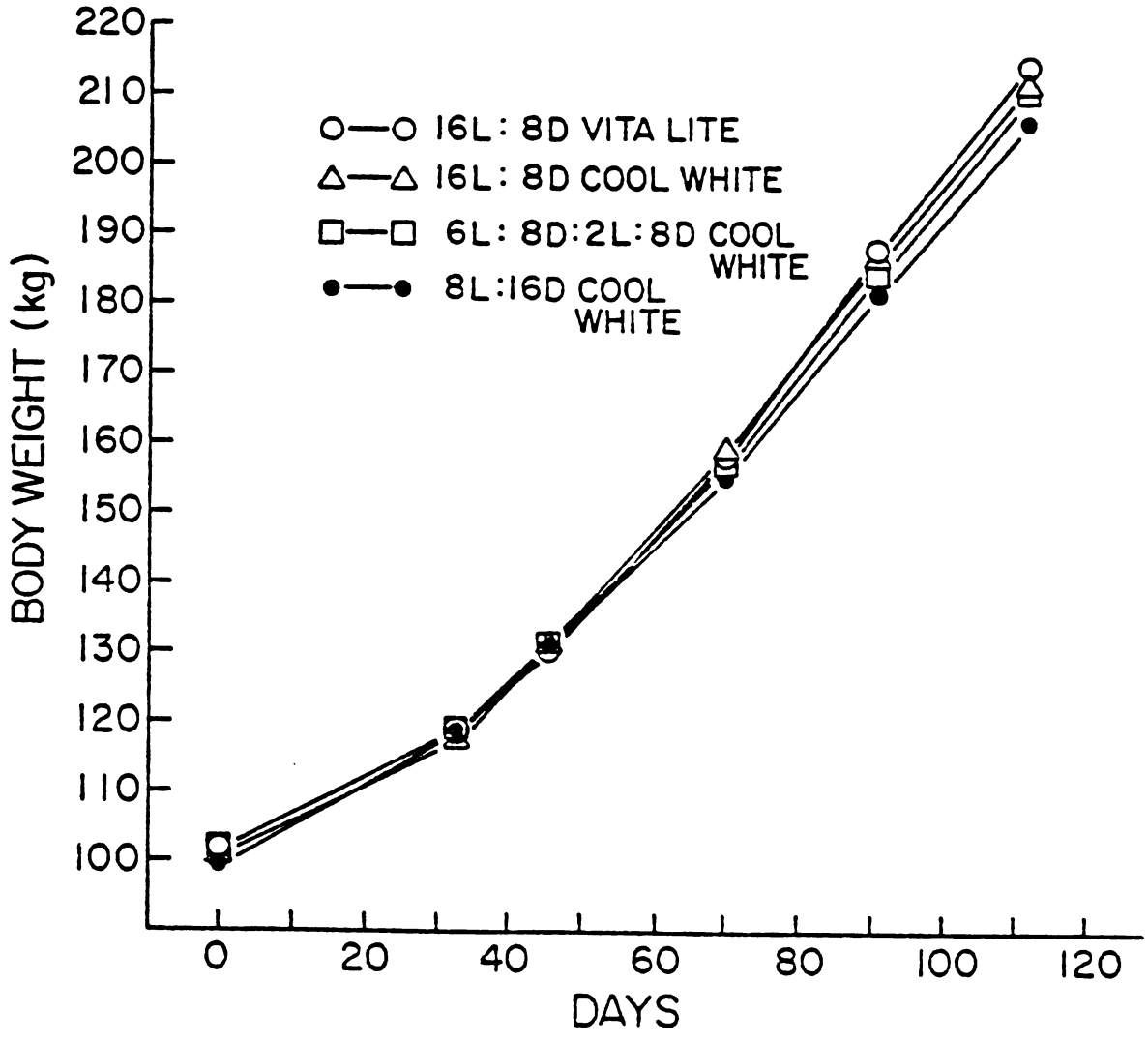

220
210
200
190
180
170
160
150
140
130
120
110
100
BODY WEIGHT (kg)
O—O 16L: 8D VITA LITE
△—△ 16L: 8D COOL WHITE
□—□ 6L: 8D:2L:8D COOL WHITE
●—● 8L:16D COOL WHITE
0
20
40
60
80
100
120
DAYS

Figure 2. Dry matter intake of Holstein heifers exposed to 16L:8D (cool-white fluorescent light; ▲); 16L:8D (Vita-Lite fluorescent light; ○); 6L:8D:2L:8D (cool-white fluorescent light; □); and 8L:16D (cool-white fluorescent light; ●); 16 heifers per treatment.

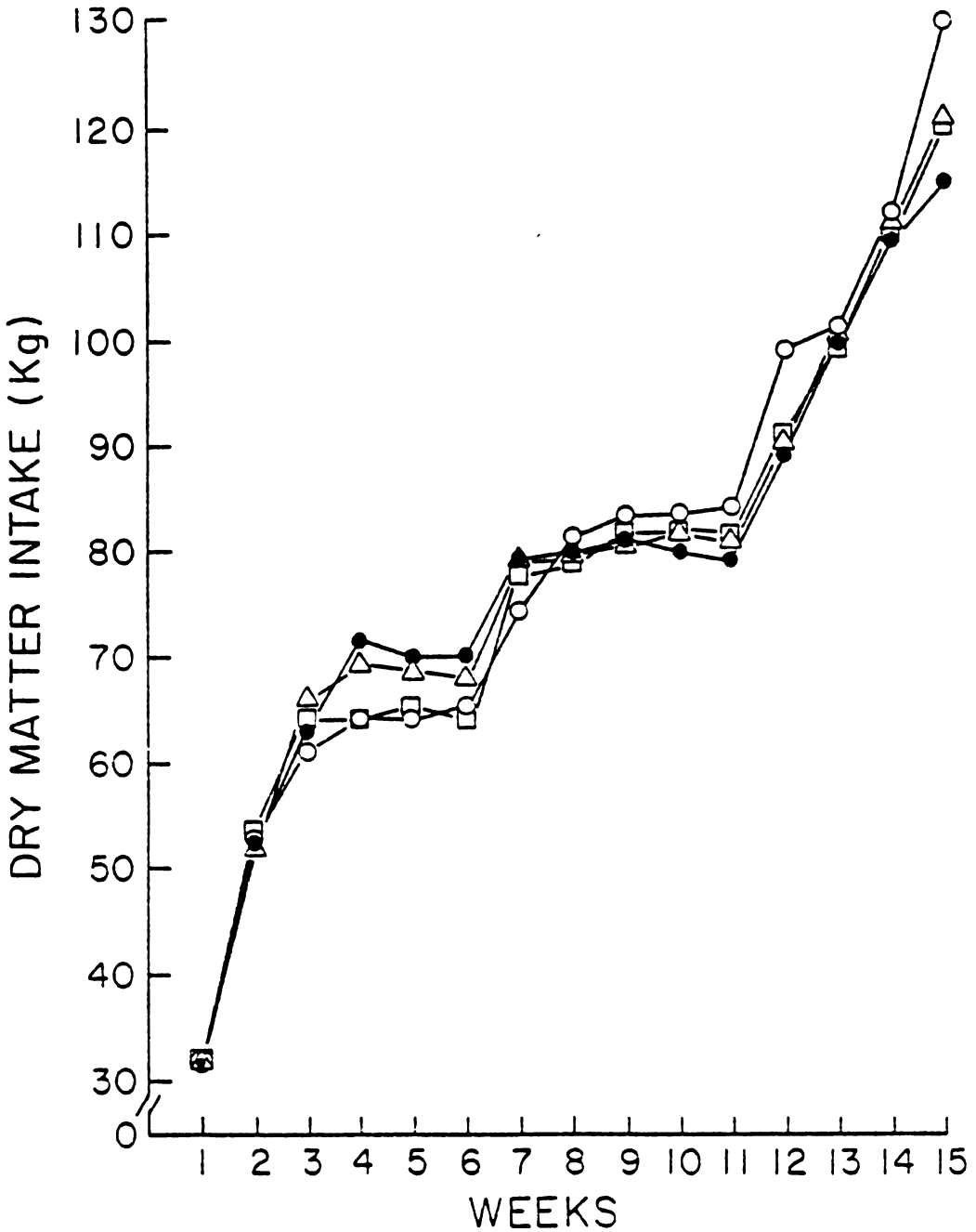
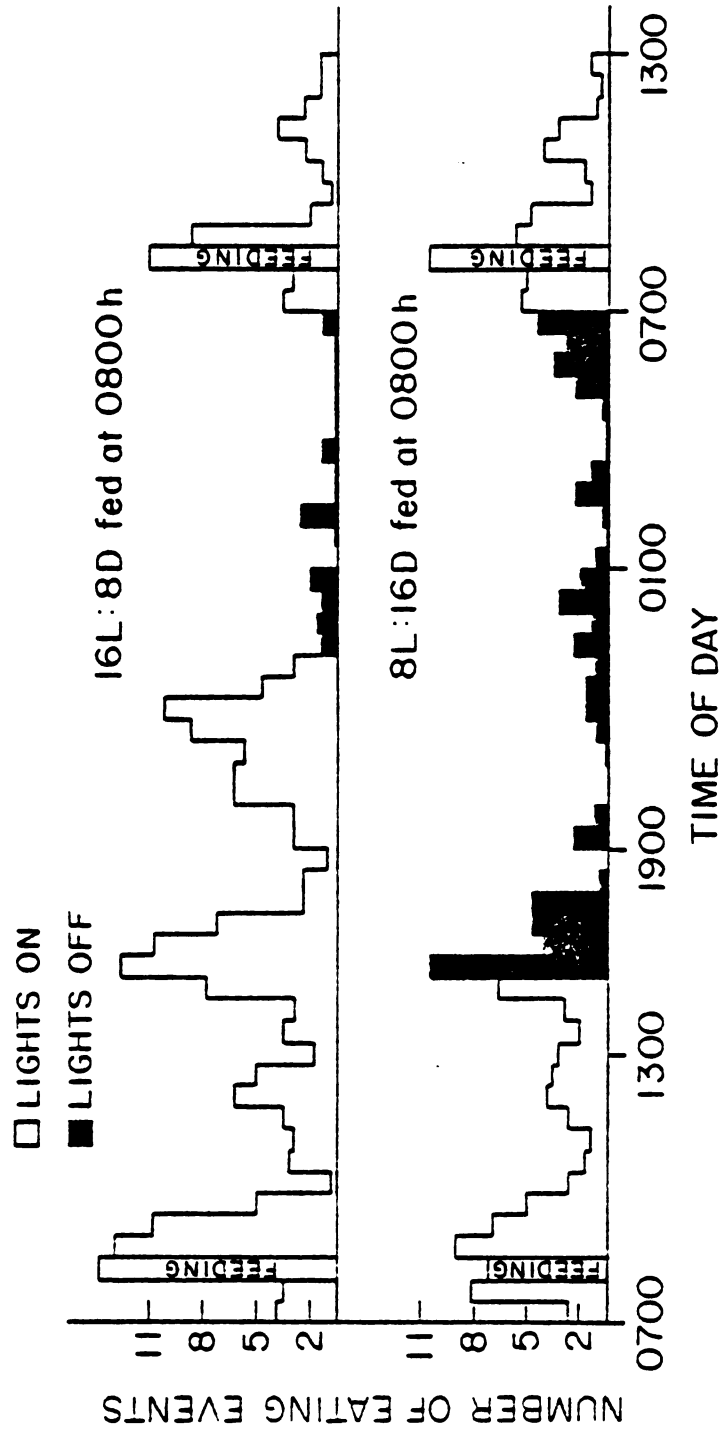

DRY MATTER INTAKE (Kg)
130
120
110
100
90
80
70
60
50
40
30
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
WEEKS

Figure 3. Eating events of Holstein heifers exposed to 16L:8D (cool-white fluorescent light) or 8L:16D (cool-white fluorescent light). Eating events were determined by counting numbers of animals eating at 10-min intervals for a period of 30 h on day 110 of photoperiod treatment. Each bar represents a 30-min average of 10-min observations; 16 heifers per treatment.


LIGHTS ON
LIGHTS OFF
16L:8D fed at 0800h
8L:16D fed at 0800h
FEEDING
FEEDING
11
8
5
2
11
8
5
2
0700
1300
1900
0100
0700
1300
TIME OF DAY
NUMBER OF EATING EVENTS

more eating activity in the dark than animals on 16L:8D, especially in the 2 h period just after lights out and the 2 h period just prior to lights on (Figure 3). In addition heifers in both groups (3 of 4 cases) increased eating activity when fresh feed was offered, even though heifers had ample feed available at all times (Figure 3).

Secretion rate, clearance rate and half-life of GH in serum. Concentrations of GH in serum averaged 6.2 ng/ml in heifers prior to infusion of 2 or 4 mg GH/h (Table 1). Steady state of GH in serum was reached in 90 to 120 min at concentrations of 56.5 and 101.8 ng/ml in heifers infused with 2 or 4 mg GH/h, respectively. CR, SR and $t_{\frac{1}{2}}$ of GH were not different (P>.10) in heifers infused with 2 or 4 mg GH/h (Table 1).

Concentrations of GH in serum prior to infusion of 1.5 mg GH/h were 5.3 and 4.7 ng/ml in heifers exposed to 8 or 16 h of cool-white fluorescent light, respectively (Table 1). Steady state of GH was attained in 90 to 120 min at concentrations of 44.6 and 45.1 ng/ml in animals exposed to 8L:16D and 16L:8D, respectively (Figure 4). Photoperiod had no effect (P>.10) on CR, SR or $t_{\frac{1}{2}}$ of GH in serum (Table 1).

Experiment 2

Weight gain. Body weight of prepubertal heifers averaged 84 kg at the start of photoperiod treatments. Final body weight on day 142 (and average daily weight gain for the 142 days) was 231 kg (1.03 kg/day) and 235 kg (1.06 kg/day) in prepubertal heifers exposed to 8L:16D and 16L:8D, respectively (Figure 5). Average daily weight gain from the beginning of light treatment to any weigh period or average daily weight gain between any two consecutive weigh

Table 1. Clearance rates (CR), secretion rates (SR) and half-life ($t_{\frac{1}{2}}$) of growth hormone (GH) in Holstein heifers infused with GH (NIH-b18)

	Natural[a]	Natural[a]	16L:8D	8L:16D
Infusion rate (mg/h)	2	4	1.5	1.5
Number of animals	2	2	8	8
Preinfusion [GH] (ng/ml)	6.2 ±.4	6.2 ±.1	4.7±1.0	5.3 ±.9
CR (ml/min)	663 ± 6	697 ± 73	620± 46	636 ± 50
SR (μg/min)	4.1 ±.2	4.3 ±.4	3.0±.6	3.8 ±.7
$t_{\frac{1}{2}}$ (min)	3.4±.1	3.5 ±.4	3.6±.3	3.7 ±.2

[a] Natural photoperiod on date of infusion was approximately 11L:13D.

Figure 4. Concentrations of GH in sera of Holstein heifers infused with 1.5 mg/h of NIH-b18 GH for 4 h and exposed to 8 (●) or 16 (▲) h of light per day. Each point is the mean of 7 or 8 samples. Standard error prior to infusion was .9 ng/ml and 4.3 ng/ml at steady state (90 to 240 min).

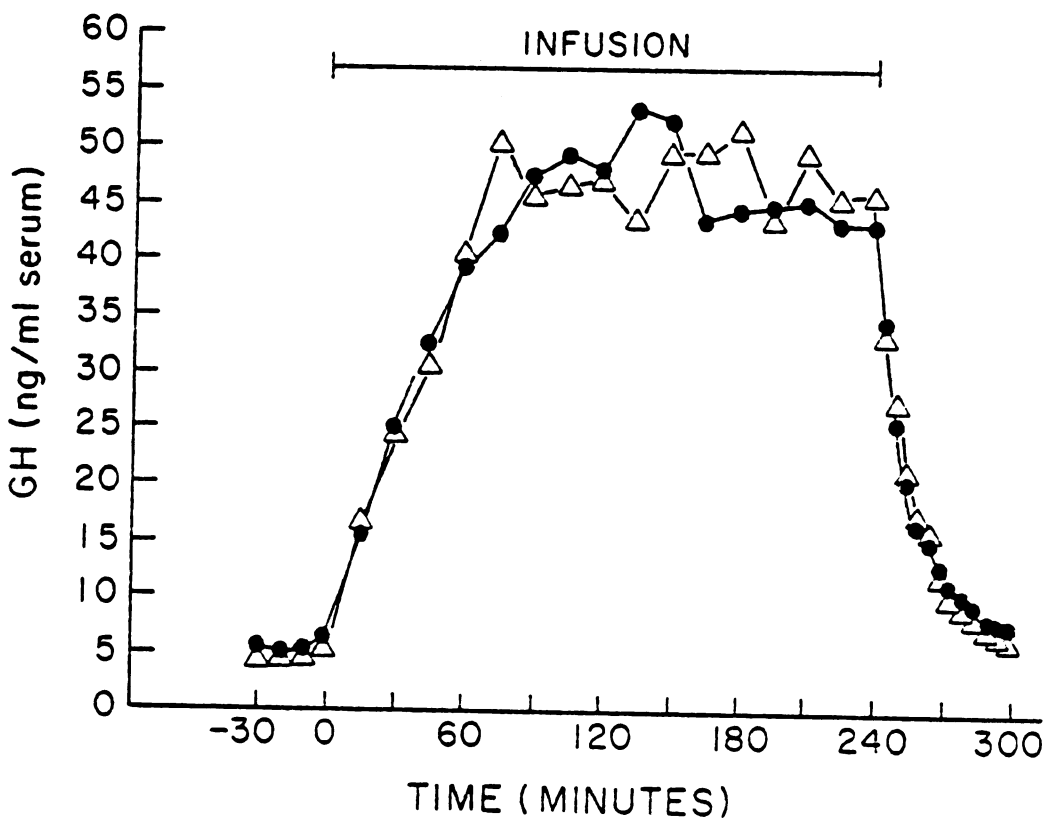
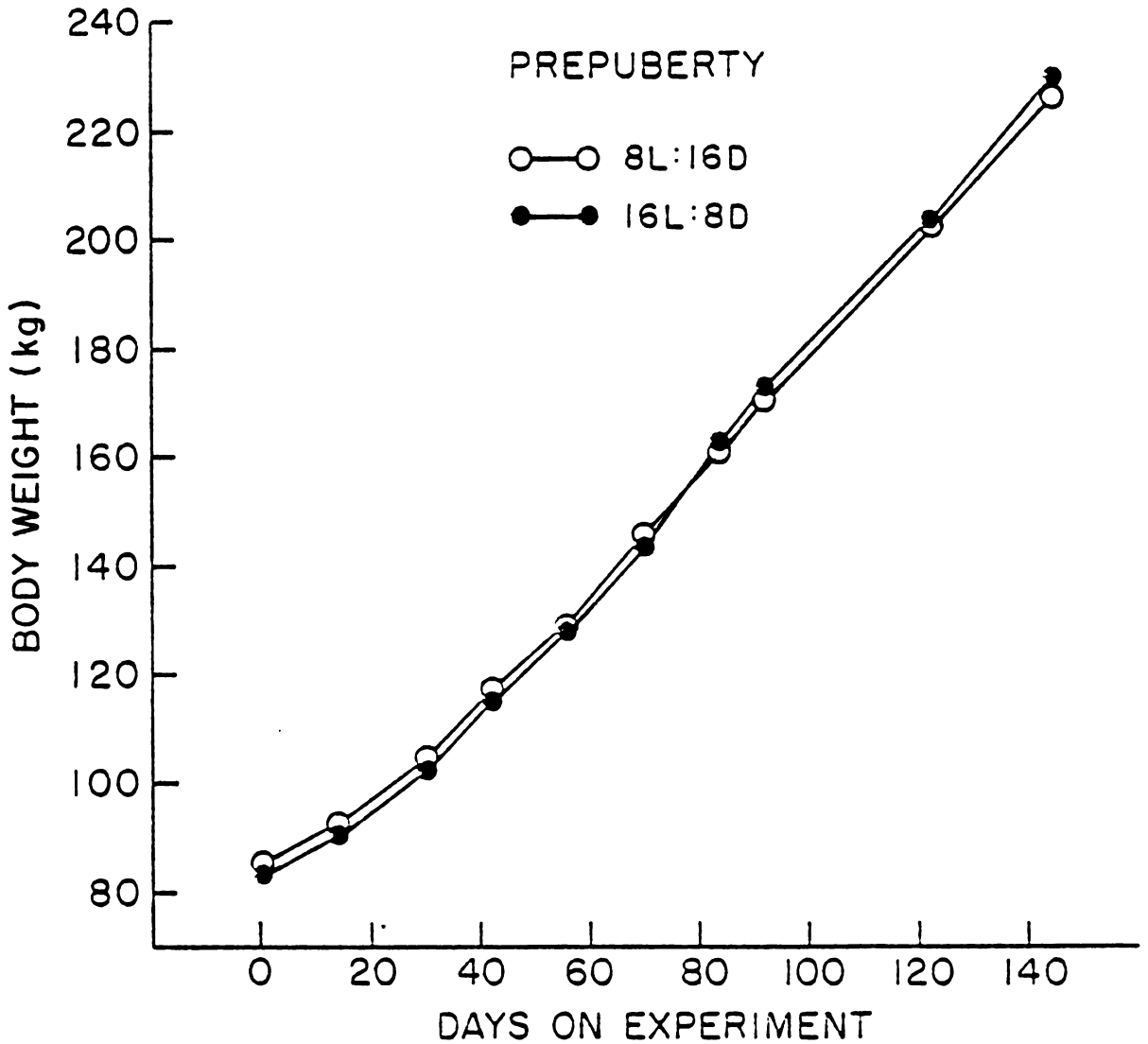

INFUSION
GH (ng/ml serum)
60
55
50
45
40
35
30
25
20
15
10
5
0
-30
0
60
120
180
240
300
TIME (MINUTES)

Figure 5. Changes in body weight of prepubertal Holstein heifers in response to photoperiod. Each point represents the mean of 14 to 16 animals. Pooled standard error was 5.8 kg.


PREPUBERTY
8L:16D
16L:8D
BODY WEIGHT (kg)
240
220
200
180
160
140
120
100
80
0
20
40
60
80
100
120
140
DAYS ON EXPERIMENT

periods in prepubertal heifers were not different (P>.10) between photoperiod treatments.

Body weight of postpubertal heifers averaged 300 kg at the start of light treatment. Average body weight of postpubertal heifers exposed to 8L:16D or 16L:8D increased to 476 and 453 kg, respectively on day 142 of treatment (Figure 6). Average daily weight gain was greater in postpubertal heifers exposed to 8L:16D compared with postpubertal heifers exposed to 16L:8D between consecutive weigh days on days 40 and 56 (P<.01); 56 and 70 (P<.001); and 70 and 84 (P<.08). Postpubertal heifers exposed to 8L:16D had greater average daily weight gain, calculated from the start of light treatment to day 56 (P<.05); 70 (P<.001); 84 (P<.02); 92 (P<.002); 123 (P<.02); and 142 (P<.01) than postpubertal heifers exposed to 16L:8D.

Feed intake. Animals within a treatment group were fed as a group. Daily dry matter intake per pen averaged 72 and 72 kg in prepubertal heifers and 138 and 136 kg in postpubertal heifers exposed to 8L:16D or 16L:8D, respectively (Figure 7).

Carcass composition. Percentages of water, protein and fat in 9-10-11 rib sections (Figure 8 A,B,C) averaged 74.0, 19.5 and 5.1, respectively in prepubertal heifers slaughtered prior to light treatment. Percentage of water and protein in 9-10-11 rib section decreased (P<.05) and fat increased (P<.001) in prepubertal heifers slaughtered at the conclusion of the experiment compared with prepubertal heifers slaughtered prior to photoperiod treatments (Figure 8 A,B,C). Exposure to 8 or 16 h of light did not affect (P>.25) percentage of water, fat and protein in 9-10-11 rib sections of prepubertal heifers. All other carcass characteristics of prepubertal heifers were larger in animals that were

Figure 6. Changes in body weight of postpubertal Holstein heifers in response to photoperiod. Each point represents the mean of 14 to 16 animals. Pooled standard error was 8.2 kg.

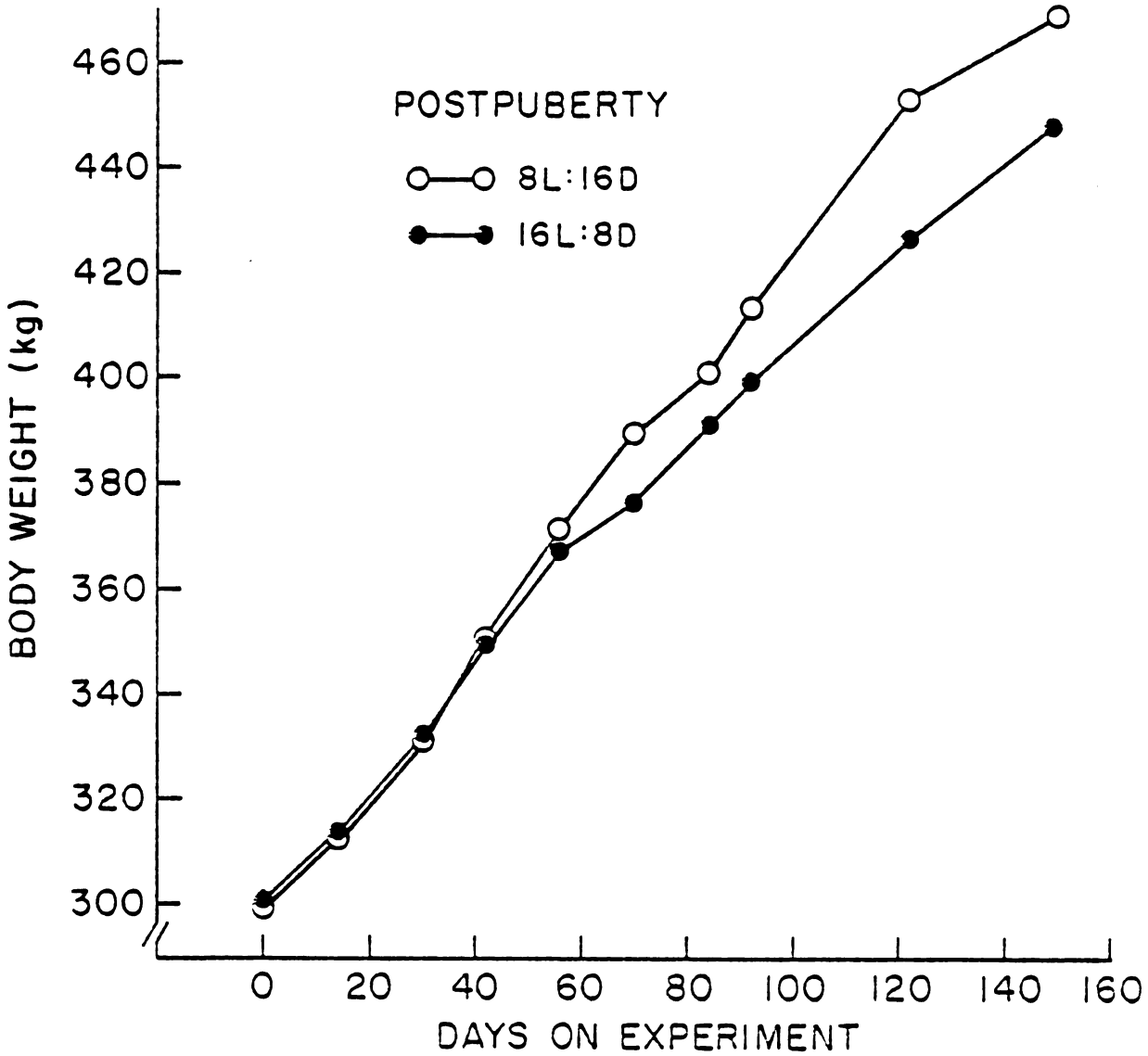

POSTPUBERTY
8L:16D
16L:8D
BODY WEIGHT (kg)
460
440
420
400
380
360
340
320
300
0
20
40
60
80
100
120
140
160
DAYS ON EXPERIMENT

Figure 7. Dry matter intake of prepubertal (----) and postpubertal (——) Holstein heifers exposed to 8L:16D (○) and 16L:8D (●); 16 heifers per treatment.

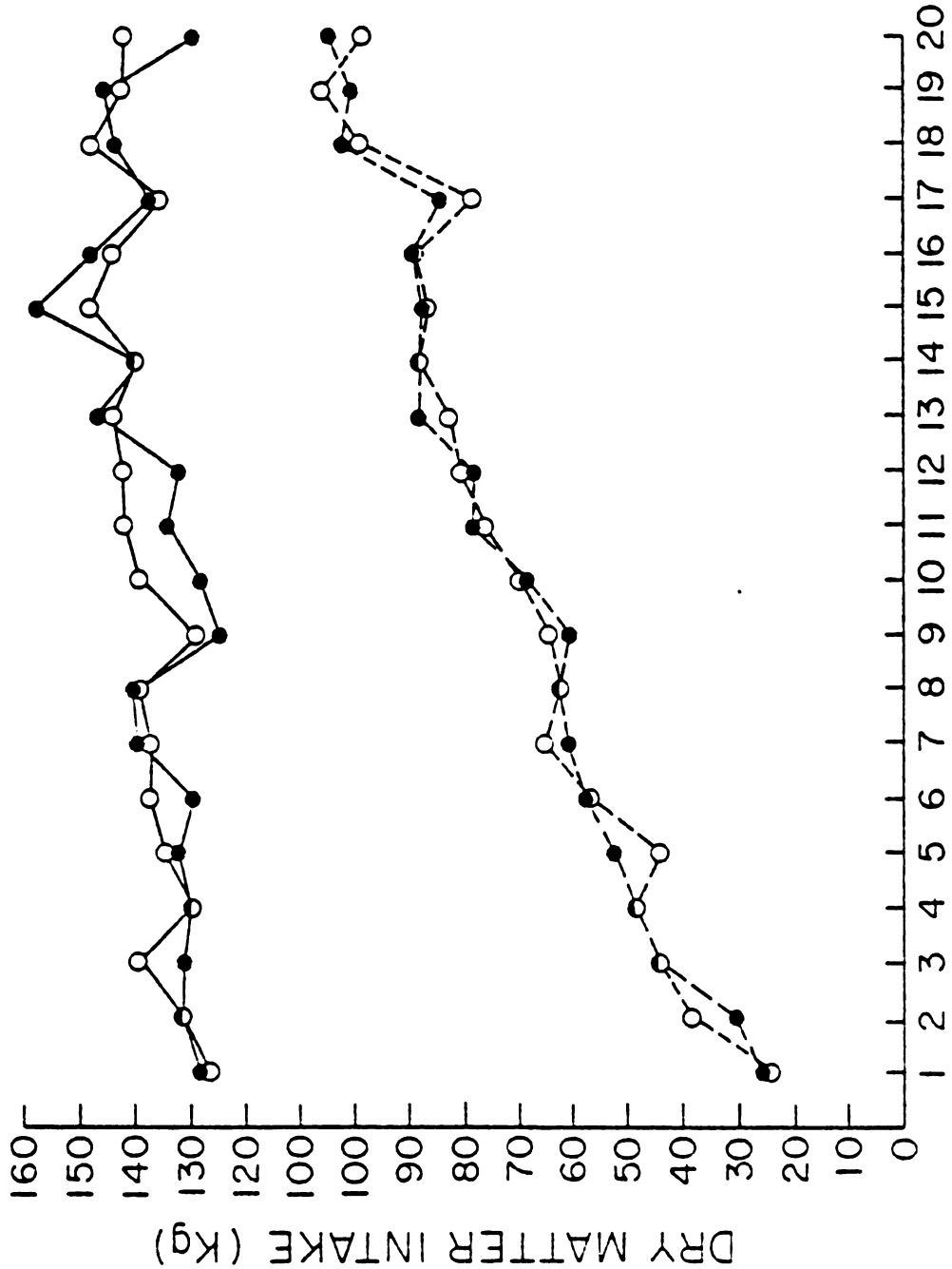

DRY MATTER INTAKE (kg)
160
150
140
130
120
100
100
90
80
70
60
50
40
30
20
10
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
WEEKS

Figure 8. Composition of 9-10-11 rib sections of prepubertal Holstein heifers prior to photoperiod treatment and after exposure to 8L:16D or 16L:8D. Each bar is the mean of 10 animals A. Percentage of water. B. Percentage of protein. C. Percentage of fat.

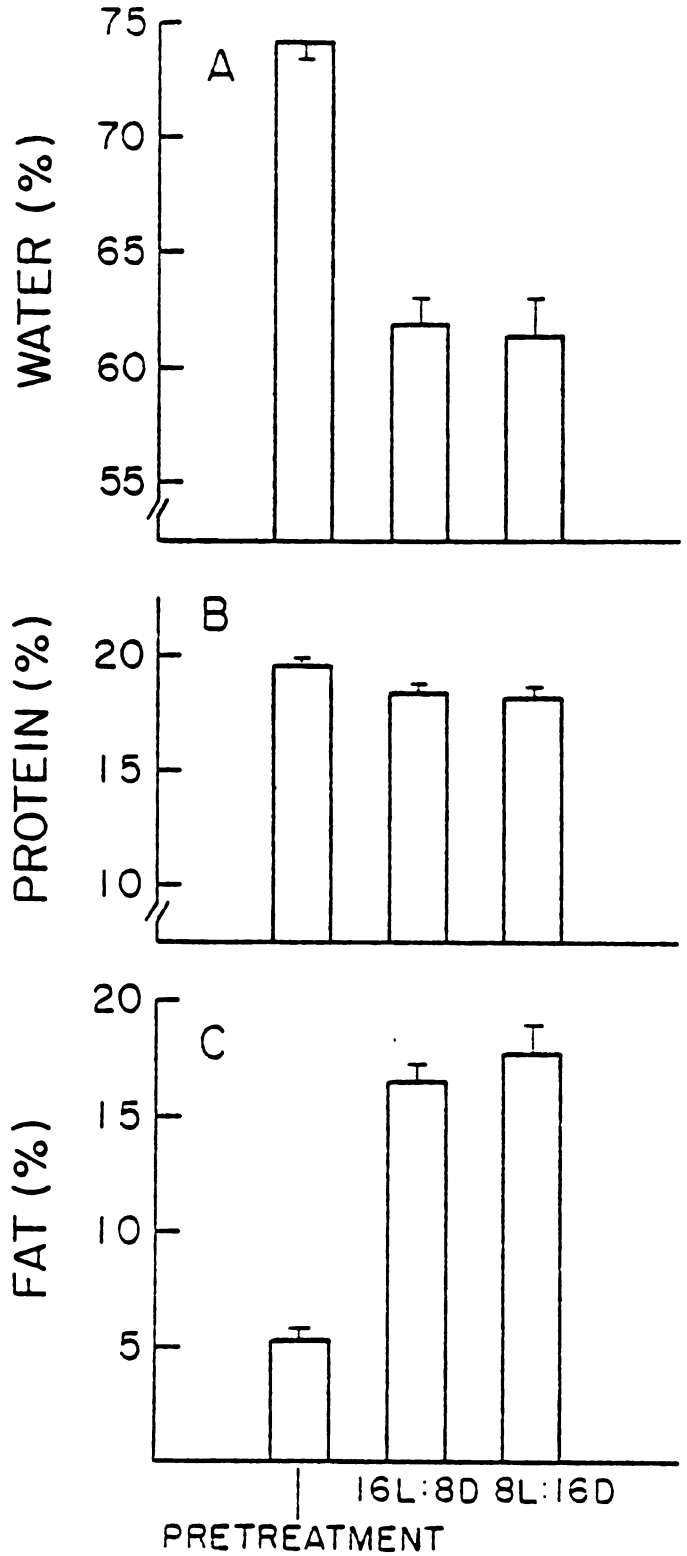

75
70
65
60
55
WATER (%)
A
20
15
10
PROTEIN (%)
B
20
15
10
5
FAT (%)
C
16L:8D
8L:16D
PRETREATMENT

Table 2. Carcass characteristics of prepubertal Holstein heifers slaughtered prior to photoperiod treatment or after exposure to 8L:16D or 16L:8D.

	Pretreatment[a]	SE	8L:16D	16L:8D	SE[b]
Number of animals	10		10	10	
Carcass weight, kg	35.0[c]	1.6	116.8[d]	112.2[d]	5.5
Carcass length, cm	72.4[c]	1.0	104.3[d]	103.4[d]	.8
Kidney fat, g	337.1[c]	35.9	3004.2[e]	2690.2[e]	262.5
Omental fat, g	249.0[c]	26.7	3300.0[d]	3186.4[d]	305.9
Fat depth at 12 rib, mm	.1[c]		1.7[e]	1.8[e]	.2
Loin eye area at 12 rib, cm^2	21.4[c]	.9	42.6[e]	42.8[e]	.9
Semimembranosus muscle, g	886.5[c]	44.2	2652.1[e]	2591.9[e]	87.1
Semitendinosus muscle, g	264.0[c]	14.6	889.0[e]	858.2[e]	30.4
Quadriceps muscle, g	784.6[c]	33.6	2363.2[e]	2243.4[e]	66.4
Hide, kg	4.9[c]	.2	16.1[e]	15.7[e]	1.8
Rumen contents, kg	6.2[c]	.4	15.6[e]	13.7[f]	.7

a Heifers slaughtered prior to treatment were exposed to natural autumn photoperiods of approximately 12L:12D at time of slaughter.

b Pooled standard error.

Entries with different superscripts in a row differ.

c vs d,f; $P<.001$.
c vs e; $P<.01$.
e vs f; $P<.10$.

Table 2 (cont.)

	Pretreatment[a]	SE	8L:16D	16L:8D	SE[b]
Femur weight, g	590.5[c]	24.7	1300.7[d]	1267.1[d]	33.2
Femur length, cm	24.8[c]	.3	33.1[d]	32.8[d]	.3
Tibia weight, g	388.9[c]	16.3	852.7[d]	822.7[d]	20.5
Tibia length, cm	24.7[c]	.4	31.6[d]	31.3[d]	.2
Cannon bone weight, g	165.4[c]	8.6	300.4[g]	292.5[g]	5.3
Cannon bone length, cm	17.4[c]	.3	20.8[g]	20.3[g]	.3
Thyroid, g	12.6[c]	.9	21.9[g]	21.2[g]	1.5
Spleen, g	190.5[c]	11.8	535.5[g]	574.8[g]	82.1
Heart, g	339.7[c]	13.7	1040.2[d]	1011.8[d]	36.5
Liver, g	1374.6[c]	58.6	4065.0[d]	3888.9[d]	123.0
Adrenals, g	5.0[c]	.2	13.0[d]	13.0[d]	.6
Kidneys, g	299.4[c]	12.0	886.2[e]	789.4[e]	36.7

a Heifers slaughtered prior to treatment were exposed to natural autumn photoperiods of approximately 12L:12D at time of slaughter.

b Pooled standard error.

Entries with different superscripts in a row differ.

c vs d; $P<.001$.
c vs g; $P<.05$.

slalughtered at the end of photoperiod treatment compared with heifers slaughtered prior to light treatment (Table 2). Photoperiod did not affect any of these measurements except rumen content; heifers exposed to short days tended ($P<.10$) to have greater rumen contents than heifers on long days.

Percentages of water, protein and fat in 9-10-11 rib sections (Figure 9 A,B,C) averaged 61.4, 17.6 and 19.4 respectively in postpubertal heifers slaughtered prior to photoperiod treatments. Rib sections of postpubertal heifers slaughtered at the conclusion of the trial had decreased ($P<.02$) percentages of water and protein and increased ($P<.001$) percentage of fat compared with postpubertal heifers slaughtered prior to photoperiod treatment (Figure 9 A,B,C). There was no effect of photoperiod ($P>.25$) on percentage of water in rib sections. However, exposure to 8L:16D decreased ($P=.07$) percentage of protein and increased ($P=.06$) percentage of fat in rib sections of postpubertal heifers compared with rib sections of postpubertal heifers exposed to 16L:8D.

All other carcass characteristics of postpubertal heifers, except rumen contents were larger in animals slaughtered at the conclusion of the trial than in animals slaughtered prior to light treatment (Table 3). There was no difference ($P>.20$) in amount of rumen contents between the two slaughter periods. Photoperiods of 8L:16D increased weight and length of the femur, tibia and cannon bones in postpubertal heifers, compared with postpubertal heifers exposed to 16L:8D (Table 3). However, heifers on 16L:8D had heavier thyroids than heifers on 8L:16D. Photoperiod did not affect other carcass characteristics measured in postpubertal heifers (Table 3). Covariates of carcass weight and days on experiment were not significant ($P>.10$) and therefore were not included in the analysis.

Based on regression equations developed by Hankins and Howe (1946) and Garrett and Hinman (1969), percentages of fat and protein in the carcass and

Figure 9. Composition of 9-10-11 rib sections of postpubertal Holstein heifers prior to photoperiod treatment and after exposure to 8L:16D or 16L:8D. Each bar is the mean of 10 animals. A. Percentage of water. B. Percentage of protein. C. Percentage of fat.

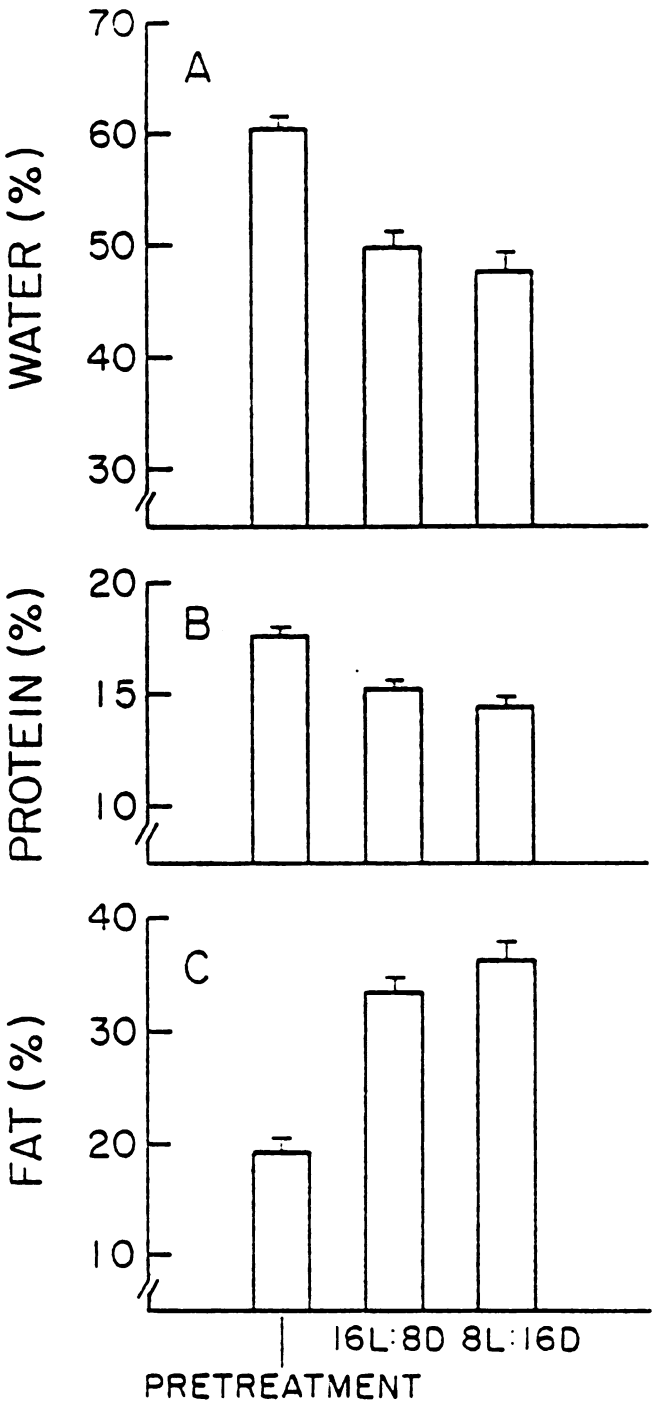

70
60
50
40
30
A
WATER (%)
20
15
10
B
PROTEIN (%)
40
30
20
10
C
FAT (%)
16L:8D
8L:16D
PRETREATMENT

Table 3. Carcass characteristic of postpubertal Holstein heifers slaughtered prior to photoperiod treatment or after exposure to 8L:16D or 16L:8D.

	Pretreatment[a]	SE	8L:16D	16L:8D	SE[b]
Number of animals	10		10	10	
Carcass weight, kg	123.9[c]	3.0	240.2[d]	230.1[d]	5.5
Carcass length, cm	109.7[c]	1.1	125.1[d]	124.5[d]	.7
Kidney fat, g	3875.4[c]	369.0	13,171.2[d]	11,473.1[d]	1114.0
Omental fat, g	4168.6[c]	221.1	2370.1[d]	2287.6[d]	549.0
Fat depth at 12 rib, mm	1.0[c]	.2	7.2[d]	6.0[d]	1.9
Loin eye area at 12 rib, cm^2	47.6[c]	2.0	67.3[e]	66.2[e]	2.2
Semimembranosus muscle, g	3023.7[c]	91.4	4762.6[d]	4627.5[d]	94.5
Semitendinosus muscle, g	1031.4[c]	36.9	1723.1[d]	1594.4[d]	91.3
Quadriceps muscle, g	2686.8[c]	61.1	4134.3[d]	3943.2[d]	194.9
Hide, kg	19.7[c]	.6	29.1[d]	27.6[d]	1.2
Rumen contents, kg	22.5	1.7	24.7	22.8	2.1

a Heifers slaughtered prior to treatment were exposed to natural autumn photoperiod of approximately 12L:12D at time of slaughter.

b Pooled standard error.

Entries with different superscripts in a row differ.

c vs d; $P<.001$.
c vs e; $P<.01$.

Table 3 (cont.)

	Pretreatment[a]	SE	8L:16D	16L:8D	SE[b]
Femur weight, g	1443.6[c]	32.0	4134.3[d]	3943.2[f]	33.84
Femur length, cm	34.6[c]	.3	39.6[d]	38.8[f]	.4
Tibia weight, g	935.5[c]	19.8	1277.0[d]	1222.6[f]	16.9
Tibia length, cm	33.9[c]	.2	37.1[d]	36.0[f]	.2
Cannon bone weight, g	339.1[h]	8.5	429.2[d]	408.7[f]	4.9
Cannon bone length, cm	21.1[c]	.2	22.7[d]	22.1[f]	.1
Thyroid, g	21.3[d]	2.2	26.6[f]	29.0[g]	1.1
Spleen, g	555.9[c]	21.5	803.9[d]	822.9[d]	27.4
Heart, g	1024.7[c]	2.6	1594.2[e]	1681.7[e]	93.5
Liver, g	4271.8[c]	94.0	5986.6[d]	5977.1[d]	92.1
Adrenals, g	12.7[c]	.5	18.2[d]	17.6[d]	.5
Kidneys, g	724.9[d]	23.4	1082.0[f]	1142.2[f]	50.4

a Heifers slaughtered prior to treatment were exposed to natural autumn photoperiods of approximately 12L:12D at time of slaughter.

b Pooled standard error.

Entries with different superscripts in a row differ.

c vs d,f; $P<.001$.
c vs e; $P<.01$.
d vs f,g; $P<.05$.
f vs g; $P<.05$.
h vs d,f; $P<.02$.

empty body (live weight minus gut fill) were calculated from 9-10-11 rib analysis (Table 4). In addition, total fat and protein in the carcass and empty body were calculated (Table 4).

Percentage of protein decreased and percentage of fat increased in the carcass and empty body between pretreatment and postreatment slaughter in prepubertal and postpubertal heifers (Table 4). Similar to 9-10-11 rib analysis, percentages of fat and protein and total fat and protein in the carcass and empty body of prepubertal heifers were not influenced by photoperiod ($P>.25$). Postpubertal heifers exposed to 16L:8D had increased percentage of protein ($P=.07$) and decreased percentage of fat ($P=.06$) in both carcass and empty body (Table 4). However, total protein in carcass or empty body of postpubertal heifers did not differ between photoperiods ($P>.10$), whereas postpubertal heifers exposed to 8L:16D had greater total amounts of fat ($P<.05$) than heifers exposed to 16L:8D (Table 4).

Serum prolactin, GH and cortisol. Concentrations of prolactin, GH and cortisol on day 95 of treatment averaged 11.1, 4.0 and 6.7 ng/ml and 11.7, 3.7 and 6.0 ng/ml in prepubertal heifers exposed to 8 or 16 h of light (Figure 10A, 11A, 12A) and 8.9, 1.9 and 8.4 ng/ml and 9.8, 2.0 and 7.5 ng/ml in postpubertal heifers exposed to 8 or 16 h of light (Figure 10B, 11B, 12B), respectively. There was no effect of photoperiod ($P>.25$) on concentrations of GH and cortisol in prepubertal and postpubertal heifers nor was there an effect ($P>.25$) of photoperiod on concentrations of prolactin in prepubertal heifers. However, photoperiods of 16L:8D tended to increase ($P<.10$) prolactin compared with 8L:16D in postpubertal heifers. Covariate of temperature at the time of bleeding was significant in the analysis of prolactin ($P<.05$) but not in the analysis of GH and cortisol ($P>.10$). Therefore, the covariate was included in the analysis

Table 4. Calculations[a] of carcass and empty body fat and protein in prepubertal and postpubertal Holstein heifers exposed to 8 or 16 h of light per day.

	Prepubertal				Postpubertal			
	Pretreatment[b]	8L:16D	16L:8D	SE[c]	Pretreatment[b]	8L:16D	16L:8D	SE[c]
Carcass fat, %	6.7[d]	17.6[e]	16.8[e]	2.2	17.8[d]	30.9[e]	28.6[f]	1.1
Carcass fat, kg	2.4[d]	21.2[e]	19.0[e]	2.1	24.1[d]	74.5[e]	66.0[g]	2.8
Carcass protein, %	18.9[d]	18.0[e]	18.2[e]	.4	17.6[d]	15.8[e]	16.0[h]	.1
Carcass protein, kg	6.6[d]	20.9[e]	20.4[e]	.9	23.4[d]	37.3[e]	36.8[e]	.8
Empty body fat, %	5.6[d]	15.6[e]	14.9[e]	2.1	15.8[d]	27.9[e]	25.8[f]	1.0
Empty body fat, kg	3.4[d]	27.4[e]	26.0[e]	2.9	31.5[d]	97.9[e]	84.3[g]	3.6
Empty body protein, %	19.1[d]	18.4[e]	18.6[e]	.3	18.1[d]	16.5[e]	16.9[h]	.2
Empty body protein, kg	11.7[d]	31.7[e]	29.0[e]	1.3	35.1[d]	56.2[e]	55.2[e]	1.0

a Calculations based on regression equations of Hankins and Howe (1946) and Garrett and Hinman (1969).

b Heifers slaughtered prior to treatment were exposed to natural autumn photoperiods of approximately 12L:12D at time of slaughter.

c Pooled standard error.

Entries with different superscripts in a row differ.

d vs e,f,g,h; $P<.01$.
e vs f; $P=.06$.
e vs g; $P<.05$.
e vs h; $P=.07$.

Figure 10. Concentrations of prolactin in sera collected from Holstein heifers after 95 days of exposure to 8L:16D (○) or 16L:8D (●). Each point represents the mean of 4 to 5 samples. Data presented are nontransformed means.

A. Prepubertal heifers, pooled standard error = .8 ng/ml.

B. Postpubertal heifers, pooled standard error = .6 ng/ml.

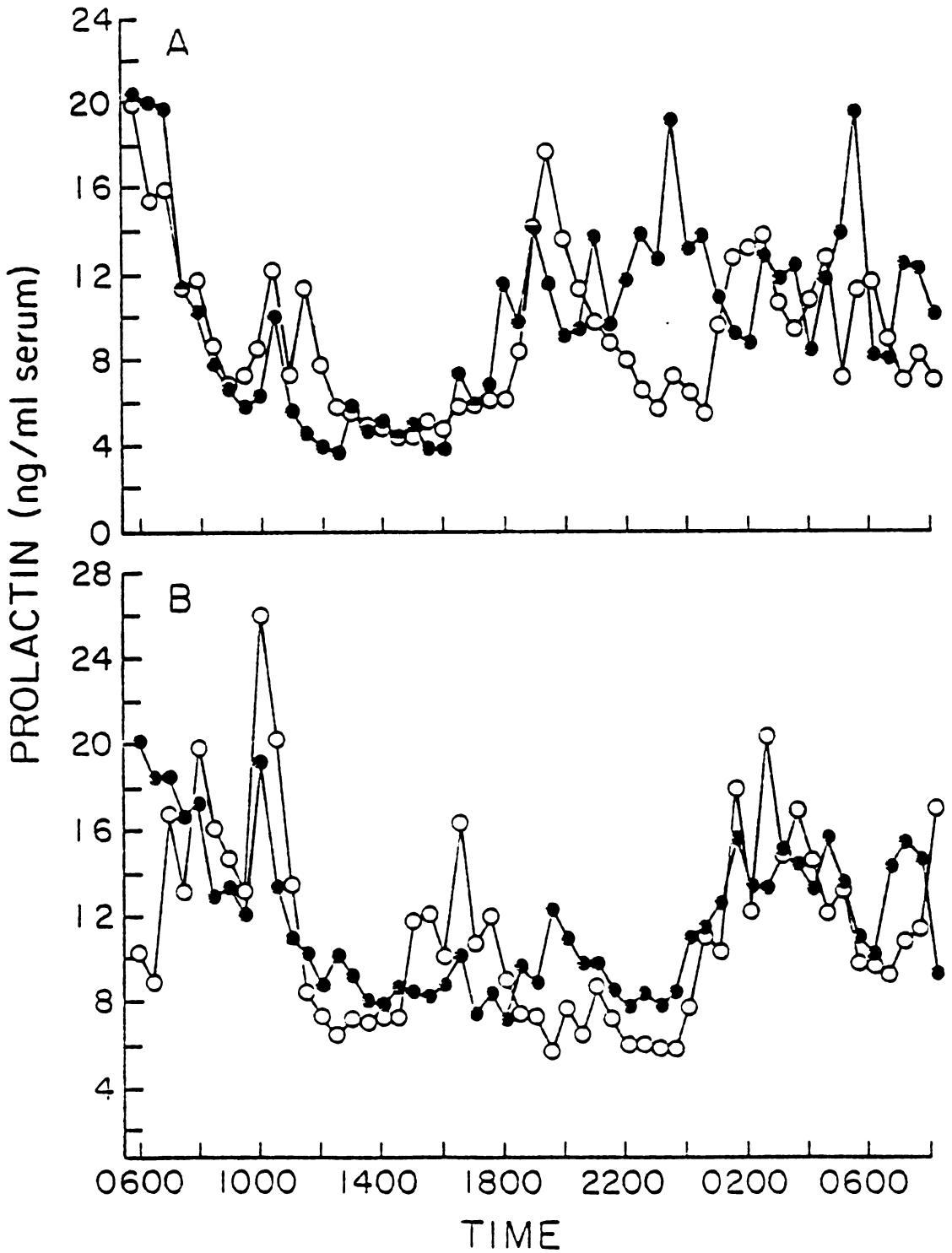

A
B
PROLACTIN (ng/ml serum)
0
4
8
12
16
20
24
28
0600
1000
1400
1800
2200
0200
0600
TIME

Figure 11. Concentrations of GH in sera collected from Holstein heifers after 95 days of exposure to 8L:16D (○) or 16L:8D (●). Each point represents the mean of 4 to 5 samples. Data presented are nontransformed means.

A. Prepubertal heifers, pooled standard error = .3 ng/ml.

B. Postpubertal heifers, pooled standard error = .1 ng/ml.

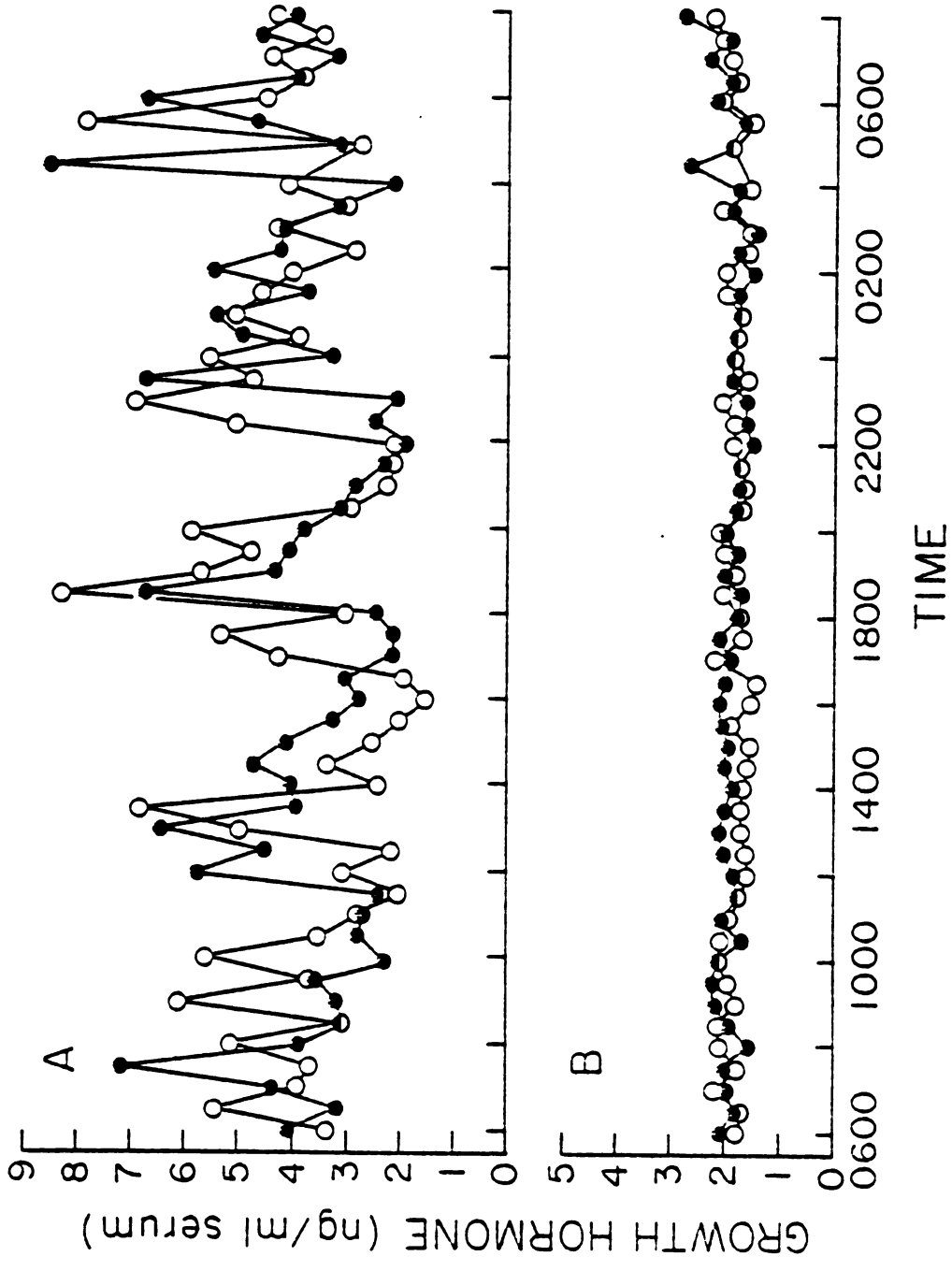
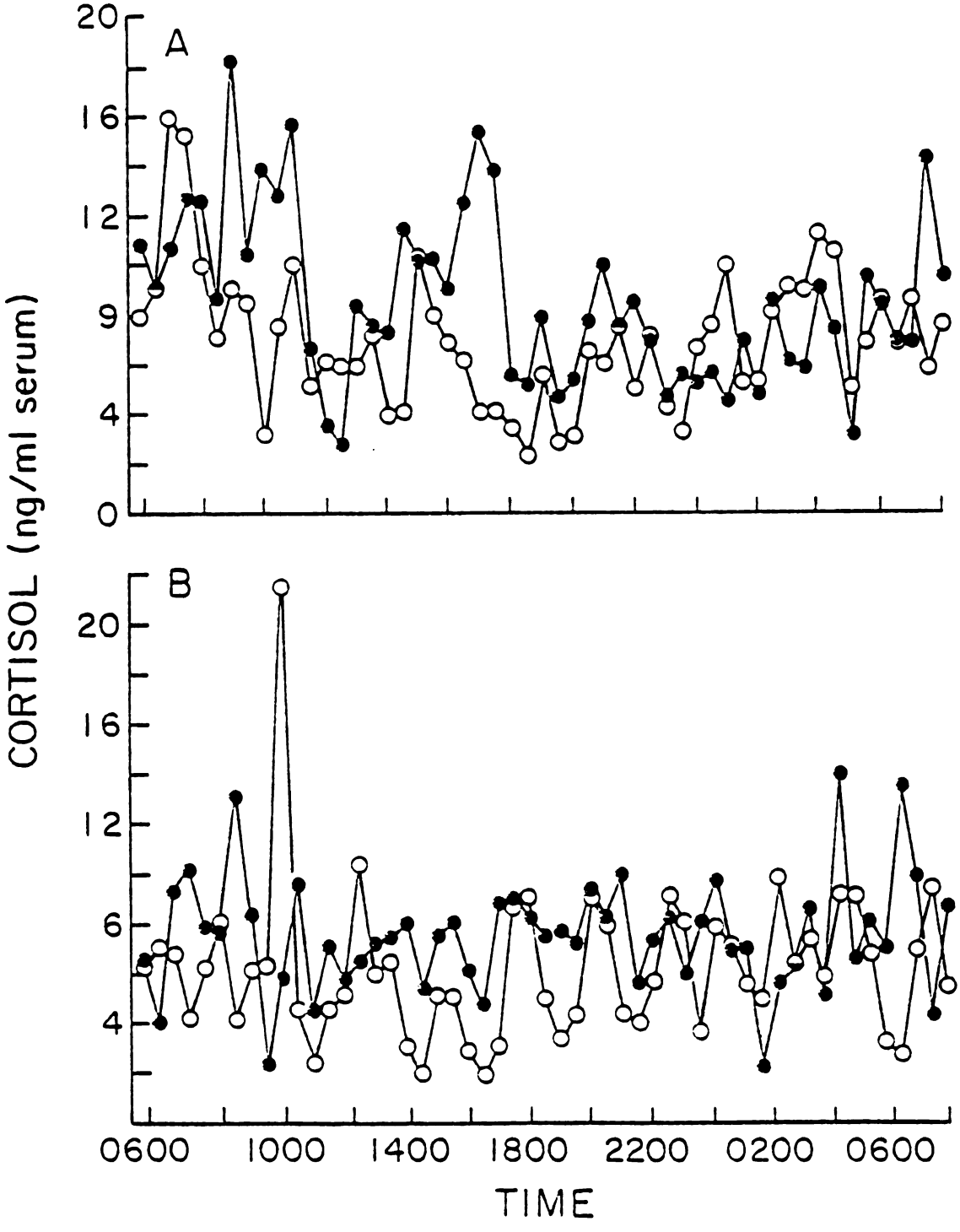

A
B
GROWTH HORMONE (ng/ml serum)
TIME
0600
1000
1400
1800
2200
0200
0600

Figure 12. Concentrations of cortisol in sera collected from Holstein heifers after 95 days of exposure to 8L:16D (○) or 16L:8D (●). Each point represents the mean of 4 to 5 samples. Data presented are nontransformed means.

A. Prepubertal heifers, pooled standard error = .7 ng/ml.

B. Postpubertal heifers, pooled standard error = .6 ng/ml.


A
B
20
16
12
9
6
4
0
CORTISOL (ng/ml serum)
0600
1000
1400
1800
2200
0200
0600
TIME

of prolactin but not GH or cortisol. Mean concentrations of GH and variation around the mean were greater (P<.01) in prepubertal heifers than postpubertal heifers. There were no differences (P>.25) in prepubertal and postpubertal heifers of mean concentrations or variation around the mean of prolactin or cortisol.

3-methylhistidine and creatinine. Photoperiods of 16L:8D, compared with 8L:16D, reduced daily urinary excretion of 3MeHis, creatinine, 3MeHis/kg body weight and ratios of 3MeHis/creatinine but not creatinine/kg in prepubertal heifers (Table 5). Urine collected from prepubertal heifers prior to photoperiod treatment had less 3MeHis and creatinine; intermediate ratios of 3MeHis/creatinine; and similar creatine/kg body weight ratios than urine collected after 99 days of treatment. Urine collected from prepubertal heifers after 99 days of exposure to 8L:16D contained greater concentrations of 3MeHis/kg body weight than urine collected from prepubertal heifers prior to light treatment. However, there was no difference in concentrations of 3MeHis/kg body weight in urine collected from heifers prior to photoperiod treatment and after 99 days of exposure to 16L:8D (Table 5).

Urinary excretion of 3MeHis and creatinine in postpubertal heifers increased in urine collected after day 99 of photoperiod treatment compared with urine collected prior to treatment (Table 6). Urine collection (pretreatment versus postreatment) did not affect (P>.10) excretion of 3MeHis/kg body weight, creatinine/kg body weight or ratios of 3MeHis/creatinine in postpubertal heifers. Photoperiod did not influence (P>.10) any measurements of urinary excretion of 3MeHis and creatinine in postpubertal heifers (Table 6). Postpubertal heifers had greater (P<.01) 3MeHis and creatinine excretion than prepubertal heifers. However, 3MeHis/kg body weight, creatinine/kg body weight and ratios of

3MeHis/creatinine were not different (P>.10) between prepubertal and postpubertal heifers.

Table 5. Urinary 3-methylhistidine (3MeHis) and creatinine from pretreatment and postreatment urine collections of prepubertal Holstein heifers exposed to 8L:16D or 16L:8D.

	Pretreatment			Postreatment		
	natural[a]	natural[b]	SE[d]	8L:16D[c]	16L:8D[c]	SE[d]
Number of animals	5	5		9	9	
3MeHis (nmole/day)	.5[e]	.4[e]	.1	1.5[f]	1.0[g]	.2
Creatinine (g/day)	4.5[e]	3.6[e]	.5	12.7[f]	11.3[g]	.3
3MeHis (μmole)/kg body weight	5.5[f]	5.0[f]	.7	7.8[g]	5.2[f,h]	1.2
Creatinine (mg)/kg body weight	51.0[e]	44.0[e]	7.1	64.3[e]	57.1[e]	7.8
3MeHis/Creatinine (nmoles/g)	105.5[e]	115.4[e]	20.3	124.3[f]	92.7[g]	7.6

a Animals were preassigned to treatment group of 8L:16D but were actually exposed to natural autumn photoperiod of approximately 12L:12D at the time of pretreatment urine collection.

b Animals were preassigned to treatment group of 16L:8D but were actually exposed to natural autumn photoperiod of approximately 12L:12D at the time of pretreatment urine collection.

c Collection of urine from postreatment animals began after 99 days of exposure to photoperiod treatment.

d Pooled standard error.

Entries with different superscripts in a row differ.

e vs f,g; P<.01.
f vs g; P<.01.
g vs h; P<.07.

Table 6. Urinary 3-methylhistidine (3MeHis) and creatinine from pretreatment and postreatment urine collections of postpubertal Holstein heifers exposed to 8L:16D or 16L:8D.

	Pretreatment			Postreatment		
	natural[a]	natural[b]	SE[d]	8L:16D[c]	16L:8D[c]	SE[d]
Number of animals	9	9		9	9	
3MeHis (mmole/day)	1.8[e]	1.5[e]	.1	2.3[f]	2.2[f]	.1
Creatinine (g/day)	14.0[e]	13.7[e]	.7	22.3[g]	22.3[g]	.8
3MeHis (μmole)/kg body weight	6.1	5.4	.4	5.3	5.4	.2
Creatinine (mg)/kg body weight	48.6	47.0	1.3	52.3	54.1	1.7
3MeHis/Creatinine (nmoles/g)	130.8	106.1	15.2	102.8	101.9	5.4

a Animals were preassigned to treatment group of 8L:16D but were actually exposed to natural autumn photoperiod of approximately 12L:12D at the time of pretreatment urine collection.

b Animals were preassigned to treatment group of 16L:8D but were actually exposed to natural autumn photoperiod of approximately 12L:12D at the time of pretreatment urine collection.

c Collection of urine from postreatment animals began after 99 days of exposure to photoperiod treatment.

d Pooled standard error.

Entries with different superscripts in a row differ.

e vs f; $P<.05$.
e vs g; $P<.01$.

Discussion

In contrast to previously reported data in cattle (Peters et al., 1978, 1980; Petitclerc et al., 1983c), there were no significant differences in body weight gain in heifers in Experiment 1 or prepubertal heifers in Experiment 2 exposed to 8 or 16 h of light per day. Experimental conditions of the present studies and conditions in previous work were not identical. These differences may account for the contrasting results. For example, lights in the current study were abruptly turned on and off at dawn and dusk. Peters et al. (1978, 1980) compared growth rate of heifers exposed to natural winter photoperiods with natural winter photoperiods plus supplemental light. In both treatments, natural transitions of light intensity occurred at dawn and dusk (Peters et al., 1978, 1980). Changes in daily activity cycles of deer mice are entrained faster and to a wider range of day lengths when gradual transitions of light intensity at dawn and dusk are utilized compared with abrupt changes in light intensity (Kavanau, 1962). Gradual transitions of light intensity at dawn and dusk may also be a more potent cue for day length than abrupt changes in light intensity in the photoperiod-induced growth response of cattle. For example, Holstein heifers exposed to 16L:8D with gradual transitions of light intensity at dawn and dusk (30 min from dark to maximum light intensity, dawn; 30 min from maximum light intensity to dark, dusk) gained 15% more weight than animals exposed to 8L:16D with gradual transitions of light intensity. In addition, heifers exposed to 16L:8D with gradual transitions gained 6

and 3% more weight than heifers exposed to 8L:16D and 16L:8D with abrupt changes in light intensity (S.A. Zinn, W.J. Enright, L.T. Chapin and H.A. Tucker, unpublished observations). Therefore, increases in average daily weight gain to supplemental light are larger when gradual transitions in light intensity at dawn and dusk are utilized compared with abrupt changes in lgiht. The increase in weight gain with gradual transitions of light may partially account for the difference between the growth responses in Experiment 1 and the prepubertal heifers in Experiment 2 and previous work in cattle.

Photoperiod prior to treatment also influences the response to the subsequent photoperiod (Moore-Ede et al., 1982). For example, mice previously exposed to light-dark cycles that differed by 8 h and then exposed to identical photoperiods required 100 days of exposure to the new photoperiod before activity cycles in the two groups of mice were similar (Pittendrigh and Daan, 1976). Photoperiod prior to treatment may also influence the growth response to supplemental light in cattle. Petitclerc et al. (1983c) reported an increase in growth in heifers exposed to 16L:8D compared with animals exposed to 8L:16D. However, all heifers were exposed to 16L:8D for 8 weeks prior to onset of photoperiod treatment. Animals in the present studies were exposed to natural autumn photoperiods of approximately 12L:12D prior to treatment. Perhaps prior exposure to 12L:12D may not be as effective for the photoperiod-induced growth response as prior exposure to 16L:8D.

Age of prepubertal heifers at the start of the trial and duration of photoperiod exposure also influences the growth response to photoperiod. Sorensen (1984) reported that 28-day old bulls required 140 days of exposure to long days to show a significant weight advantage compared with young

bulls exposed to short days. Prepubertal heifers (Experiment 1) were exposed to photoperiod treatment for 112 days and prepubertal heifers (Experiment 2) were exposed to photoperiod treatment for 142 days. At the end of both trials, an advantage in weight (sigificant, Experiment 1; non-significant, Experiment 2) in prepubertal heifers exposed to 16:8D compared with 8L:16D was observed. Differences in weight gain due to photoperiod over the entire treatment period may have become significant had both trials continued longer.

Urine collection inhibited weight gains in prepubertal heifers (Experiment 2) and this may have reduced any advantage in growth rate with exposure to photoperiod. Heifers fitted with Foley catheters gained less weight during the last 35 days of the growth trial than their non-collected pen-mates. Kidneys removed at slaughter from prepubertal heifers that were catheterized had visible signs of infection and damage.

Photoperiods of 8L:16D increased weight gain compared with 16L:8D in postpubertal heifers, a response similar to that observed in white-tailed deer (Budde, 1983; Abbott et al., 1984). In contrast, only long-day photoperiods have been reported to increase weight gains in sheep (Forbes et al., 1975, 1979a; Schanbacher and Crouse, 1980, 1981) and cattle (Peters et al., 1978, 1980; Petitclerc et al., 1983c; Sorensen, 1984). Sheep and cattle in previous photoperiod studies were prepubertal at the start of the trial. Postpubertal animals may respond to photoperiod differently than prepubertal animals. Prepubertal and postpuberal hamsters exposed to identical photoperiods display different activity cycles (Moore-Ede et al., 1982). Age and reproductive status of heifers at the start of the trial may account for the different growth response to photoperiod between postpubertal heifers in the current study and previously reported responses

in sheep and cattle.

Similar to average daily weight gain, there was no effect of photoperiod on carcass composition in prepubertal heifers. Duration of photoperiod treatment may have been too short to observe a significant response. If photoperiod treatment had continued until a significant response in weight gain had occurred, a difference in carcass composition may have been observed as well.

Postpubertal heifers exposed to 8L:16D had a greater percentage of fat in the 9-10-11 rib sections than heifers exposed to 16L:8D, an effect also observed in white-tailed deer (Abbott et al., 1984). From the data of Petitclerc et al. (1984) it appears that pubertal heifers exposed to 8L:16D had a 10 to 15% increase, although non-significant, in percentage of fat in rib sections than heifers given 16L:8D. In addition to the effect of photoperiod on fat, postpubertal heifers given in the current study long-day photoperiods, compared with short days, had an increased percentage of protein in rib sections. These data confirm the results of Petitclerc et al. (1984).

Total carcass fat and total empty body fat were greater in postpubertal heifers exposed to 8L:16D than heifers on 16L:8D. However, photoperiod did not affect total carcass protein or total empty body protein in postpubertal heifers. Therefore, in the current study, the additional weight gain of postpubertal heifers exposed to short days versus long days was primarily additional gain of fat, not protein. Similarly in white-tailed deer, accretion of fat, not protein, accounted for photoperiod-induced weight gain (Abbott et al., 1984). Exposure to short days partitions nutrients towards fat accretion in heifers gaining more than 1.0 kg/day. The increase in fat production accounts for the increased weight gain in postpubertal heifers

in the current study exposed to 8L:16D.

The effect of short day length on fat accretion may not have been observed in prepubertal heifers because of their body weight at the conclusion of the growth trial. Prepubertal heifers gained more than 1.0 kg/day but averaged less than 240 kg total body weight at the conclusion of the growth trial and accumulation of large amounts of fat in cattle does not begin until cattle weigh approximately 300 kg (Searle et al., 1972).

As average daily weight gain approaches .7 to 1.0 kg/day in cattle, daily protein accretion plateaus and no additional daily protein accretion is observed when daily body weight gain surpasses 1.0 kg/day (Byers, 1980). Prepubertal and postpubertal heifers on both photoperiod treatments gained more than 1.0 kg/day. Thus, heifers in both 8L:16D and 16L:8D photoperiod treatment groups may have surpassed their physiological limit of daily protein accretion. As a result photoperiod would not be expected to affect protein accumulation.

Total urinary excretion per day of 3MeHis and 3MeHis per kg body weight were similar, as was the direction of change over time in these measurements to previously reported values in cattle of similar body weight (Nishizawa et al., 1979; Harris and Milne, 1981; McCarthy et al., 1983). Total urinary excretion of creatinine per day and creatinine per kg body weight were greater in the present study than values reported by McCarthy et al. (1983) and Benner (1983). Cattle used by McCarthy et al. (1983) were small and large frame beef steers at different stages of maturity. These authors reported an increase in excretion of creatinine per kg body weight associated with larger frame size or earlier stage of maturity. Benner (1983) also used large frame beef cattle. Therefore, frame size or stage of maturity affects creatinine excretion. Cattle in the present

study were Holstein heifers and differences in breed, frame size and stage of maturity between beef and dairy animals may account for the greater creatinine excretion in dairy heifers in the current study. Ratios of 3MeHis/creatinine in the current study were similar to ratios reported by Gopinath and Kitts (1982). Ratios of 3MeHis/creatinine in the present study were lower than ratios in beef cattle reported by McCarthy et al. (1983) and Benner (1983) because of the greater excretion of creatinine in heifers in the current study.

Based on creatinine excretion, prepubertal heifers exposed to 16L:8D compared with 8L:16D had reduced muscle mass. However, no significant differences in muscle weight were observed. Creatinine is produced in the metabolism of creatine in muscle. Reduction of excretion of creatinine in prepubertal heifers exposed to long day length may be the result of a decrease in metabolism of creatine and not a decrease in muscle mass.

Prepubertal heifers exposed to 16L:8D had reduced protein degradation compared with 8L:16D. However, photoperiod did not affect protein accretion. There are several possible explanations for this. First, reduction in protein degradation may precede increases in protein accretion. If prepubertal heifers were maintained on photoperiod treatments for a longer duration a significant effect of photoperiod on protein accretion may have been observed. Secondly, protein synthesis may have been reduced by a similar magnitude as protein degradation. This would decrease protein turnover and should increase efficiency of protein accretion (Garrett and Johnson, 1983) without an effect on total protein accretion. Reduction of protein turnover in heifers exposed to 16L:8D compared with 8L:16D could be the cause of increased feed efficiency in cattle exposed to long-day photoperiods (Peters et al., 1980; Petitclerc et al., 1983c).

A third alternative is that the effect of photoperiod on 3MeHis excretion is not on muscle protein degradation but on non-muscle protein. Controversy still exists regarding the contribution of non-skeletal muscle sources of 3MeHis (McCarthy et al., 1983). Muscle has the largest store of 3MeHis, but because of the slow turnover rate of muscle proteins the contribution of skeletal muscle to total urinary 3MeHis is reported to be less than 60% (Wassner and Li, 1982) and may be as low as 25 to 41% (Millward et al., 1980; Bates and Millward, 1981). In contrast, several other studies have reported at least 75% of urinary 3MeHis originates from skeletal muscle in rabbits (Harris, 1981), rats (Nagaswa and Funabiki, 1981), cattle (Nishizawa et al., 1979) and a human (Afting et al., 1981). This still leaves up to 25% of 3MeHis originating from non-muscle sources. Therefore, reduction in 3MeHis excretion and 3MeHis per kg body weight in prepubertal heifers exposed to 16L:8D may reflect a reduction in protein degradation in non-muscle sources and not an influence of photoperiod on muscle protein degradation. This would explain the effect of photoperiod on 3MeHis excretion without an affect on protein content in the carcass.

Photoperiod did not affect urinary excretion of 3MeHis or creatinine in postpubertal heifers. These data would be expected since there was no effect of photoperiod on total protein or muscle mass in postpubertal heifers.

Photoperiod did not affect average dry matter intake in Experiment 1 or 2. Therefore, the increased weight gain observed in postpubertal heifers in the current study on 8L:16D was associated with increased feed efficiency, not increased feed intake. In contrast, cattle exposed to 16L:8D and fed ad libitum had increased weight gain and required more feed than cattle on 8L:16D (Peters et al., 1980; Petitclerc et al., 1983c). However, these

gains were associated with higher percentage of protein in 9-10-11 rib sections (Petitclerc et al., 1984), whereas in the present study the added weight gain, due to photoperiod, was primarily fat. Fat accretion is more efficient than protein accretion (Garrett and Johnson, 1983) and this may account for increased fat accretion without requiring increased feed intake in postpubertal heifers in the current study.

Heifers exposed to 16L:8D had 96% of their eating activity in the lighted period, whereas heifers exposed to 8L:16D had 50% of their eating activity in the lighted period. Heifers exposed to 8L:16D had 75 and 25% of their eating activity in the first 16 h and last 8 h of the day, respectively, compared with 96 and 4% for the same time periods in heifers exposed to 16L:8D. Therefore, heifers exposed to 8L:16D had more eating activity in the dark, especially 2 h after lights off and 2 h prior to lights on. This indicates heifers exposed to short day photoperiods do not concentrate all of their eating activity in the lighted period. Both groups increased eating activity when fresh feed was presented. Schanbacher and Crouse (1981) showed that in comparison to sheep given 16L:8D, sheep exposed to 8L:16D had more eating activity in the dark period, including increased activity prior to lights on. In contrast, Eisemann et al. (1984a) reported feed consumption was not different when both groups were in the dark and sheep exposed to 8L:16D concentrated feed consumption into the lighted period.

Differences in eating patterns may be a cause for the anabolic effects of long-day photoperiods (Schanbacher and Crouse, 1981; Eisemann et al., 1984a). However, Tanida et al. (1984) failed to observe a correlation between eating patterns of dairy cows exposed to different photoperiods and milk production. In addition, Zinn et al. (1983) reported that influence of time

of feeding on eating patterns may affect growth rate more than photoperiod-associated changes in eating patterns.

Eating patterns may account for increased rumen contents in prepubertal heifers exposed to 8L:16D. Heifers were transferred to the abattoir 2 to 3 h after lights out in the 8L:16D treatment group, which corresponds to the end of a period of increased eating activity in the short-day photoperiod group. Therefore, heifers exposed to 8L:16D would likely have greater rumen content compared with animals on 16L:8D that did not have the increased eating activity prior to arrival at the abattoir. Differences in eating patterns between photoperiods may also explain the gut-fill response in sheep (Forbes et al., 1979a, 1981).

Photoperiod did not affect organ weights, except thyroids, in prepubertal or postpubertal heifers. These data confirm observations in sheep (Eisemann et al., 1984b). Photoperiod does not affect thyroid hormone secretion in bulls (Leining et al., 1980) or sheep (Forbes et al., 1979b) and therefore, there is no immediate explanation for the effect of photoperiod on thyroid weight in postpubertal heifers.

Compared with heifers on 16L:8D, tibia, femur and cannon bones were longer and heavier in postpubertal heifers given 8L:16D. To meet the stresses applied to the skeleton by increased body mass, bone growth increases as an animal matures (Trenkle and Marple, 1983). Since 8L:16D increased body weight in postpubertal heifers more than 16L:8D, the stress from the additional body mass may have induced the additional bone growth.

Growth hormone is considered one of the principal anabolic hormones regulating the growth process (Davis et al., 1984). However, GH is probably not directly involved in photoperiod-induced increments in growth. In agreement with previous work in cattle (Leining et al., 1980; Peters and

Tucker, 1981; Peters et al., 1981; Petitclerc et al., 1983c), photoperiod did not affect serum concentrations of GH in the current studies. Moreover, photoperiod did not affect CR, SR or $t_{\frac{1}{2}}$ of GH in serum. However, mean concentrations of GH and variation around mean GH were lower in postpubertal heifers than prepubertal heifers. CR and SR of GH in heifers in the present study were lower than values reported for lactating and non-lactating dairy cows (Yousef et al., 1969; Bourne et al., 1977) but similar to CR and SR in beef heifers and steers of comparable body weight (Trenkle, 1971; Trenkle and Topel, 1978).

Half-life of GH in serum was shorter than values previously reported for cattle (Yousef et al., 1969; Trenkle, 1971, 1976, 1977; Trenkle and Topel, 1978). To estimate serum kinetics of a compound the infusate should be in equilibrium with all compartments of the body (Tait, 1963). Single-injection methods were utilized to determine $t_{\frac{1}{2}}$ in previous work (Yousef et al., 1969; Trenkle 1971, 1976, 1977; Trenkle and Topel, 1978), whereas the current study utilized a constant infusion method. Distribution of exogenous GH among all body compartments may be different between infusion and injection techniques and differences in distribution could result in different estimates of $t_{\frac{1}{2}}$. Preinjection concentrations of a hormone can also affect kinetic estimates of that hormone in serum (Tait, 1963). Preinjection concentrations of GH were higher in previous studies with cattle (Yousef, 1969; Trenkle, 1971, 1976, 1977; Trenkle and Topel, 1978) than in the current study. Lower preinjection concentrations of GH are associated with shorter $t_{\frac{1}{2}}$ of GH (Trenkle, 1971). Disappearance of GH follows a two-compartment model; a fast component (0 to 20 min postinjection) with a $t_{\frac{1}{2}}$ of 3.4 to 7.9 min and represents GH clearance from the blood and a slow component with a $t_{\frac{1}{2}}$ of 22.0 to 34.1 min.

Estimates of $t_{\frac{1}{2}}$ in the present study were calculated from concentrations of GH 0 to 35 min postinfusion. The estimate of $t_{\frac{1}{2}}$ in the present study may represent only the fast component, which would account for the different values of $t_{\frac{1}{2}}$ between the current study and previous work.

Concentrations of GH in postpubertal heifers were lower than in prepubertal heifers and confirms previous work in cattle (Armstrong and Hansel, 1956; Tucker et al., 1974; McCarthy et al., 1979) and pigs (Siers and Swiger, 1971). The decrease in GH is associated more with increased size than increased age (Siers and Swiger, 1971). In agreement with data in bulls (McCarthy et al., 1979), variation around mean concentrations of GH was greater in prepubertal heifers than in postpubertal heifers. Reduction in variation around mean concentrations is associated with decreased number and amplitude of secretory spikes (McCarthy et al., 1979).

There was no effect of photoperiod on serum concentrations of cortisol in prepubertal or postpubertal heifers. The influence of photoperiod on cortisol in cattle in the current study supports similar observations made in dairy heifers (Peters et al., 1980), lactating cows (Peters et al., 1981), sheep (Kennaway et al., 1981; Lincoln et al., 1981; Brinklow and Forbes, 1984b) and deer (Bubenik et al., 1975) but conflicts with data in young bulls (Leining et al., 1980) and young sheep (Brinklow and Forbes, 1984a,b).

Actions of glucocorticoids are generally catabolic (Goldberg et al., 1980) and reductions in concentration of glucocorticoids may be associated with the photoperiod-induced growth response (Tucker et al., 1984). Serum glucocorticoids are positively correlated with fat in the carcass (Trenkle and Topel, 1978) and negatively correlated with daily weight gain (Purchas et al., 1971). In addition, injections or implants of cortisone acetate increase

percentage of fat in sheep (Spurlock and Clegg, 1962; Ellington et al., 1967) and cattle (Carroll et al., 1963). Bleeding of heifers in the present study occurred on day 95 of treatment. This was before a photoperiod-induced growth response was observed in prepubertal heifers and after a difference was observed in postpubertal heifers. In order to analyze further the role of glucocorticoids in the photoperiod-induced growth response, blood samples for glucocorticoid analysis should be taken at or near the time photoperiod-induced changes in growth occur. In addition, blood samples should be taken for at least 24 h, at frequent intervals (every 15 to 30 min), to account for diurnal variation in glucocorticoids.

Concentrations of prolactin were not affected by photoperiod in prepubertal heifers and there was only a tendency for prolactin to be increased in postpubertal heifers exposed to 16L:8D compared with animals given 8L:16D. This response of prolactin to 16L:8D versus 8L:16D is smaller than increases normally associated with long day lengths in cattle (Bourne and Tucker, 1975; Peters et al., 1978; Leining et al., 1980; Petitclerc et al., 1983a,c; Stanisiewski et al., 1984a). However, ambient temperatures during blood sampling averaged 6°C and were below 1°C for 25% of the sampling period. Cold temperatures decrease serum prolactin (Wettemann and Tucker, 1974) and suppress photoperiod-induced increments in serum prolactin (Peters and Tucker, 1978). Therefore, cold temperatures during bleeding in the current study probably masked the effects of photoperiod on serum prolactin.

Although there was no effect of photoperiod on prolactin in the present study, prolactin is responsive to long day lengths, suggesting a role for prolactin in photoperiod-induced changes in growth. However, evidence for a role in growth is equivocal. Prolactin, injected into rats, increased

weight gain and body length compared with uninjected rats (Cargill-Thompson and Crean, 1963; Bates et al., 1964; Thorngren and Hansson, 1974). Somatomedin activity, which may mediate growth responses (Daughaday, 1982), is increased by prolactin (Francis and Hill, 1975; Holder and Wallis, 1976; Hill et al., 1977). In addition, infusion of prolactin increased nitrogen retention in sheep (Brinklow and Forbes, 1983) and active immunization against prolactin reduced growth in sheep (Ohlsen at al., 1981). However, sheep injected with prolactin and exposed to 8L:16D did not improve body weight gain compared with uninjected sheep exposed to 8L:16D (Eisemann et al., 1984a). In addition, Brown et al. (1976) and Ravault et al. (1977) reduced prolactin concentrations with 2-bromo-α-ergocryptine (CB-154), an inhibitor of prolactin, without changing growth rate in sheep. In contrast, sheep exposed to 16L:8D and injected with CB-154 gained less weight than uninjected sheep exposed to 16L:8D (Eisemann et al., 1984a). Photoperiods of 16L:8D stimulated prolactin secretion in sheep without causing changes in weight gain (Brinklow and Forbes, 1984c).

In conclusion, photoperiods of 8L:16D compared with 16L:8D increased growth rate in postpubertal heifers. However, the additional weight was primarily fat. In addition, prepubertal heifers exposed to long day lengths had reduced protein degradation compared with heifers given short day lengths. However, photoperiod did not affect overall growth rate or body composition in prepubertal heifers.

Summary and Conclusions

The effects of photoperiod on growth rate, body composition and serum hormones in Holstein heifers were studied in two experiments. Heifers were housed unrestrained in one of four light-controlled pens and no supplemental heat was provided. Sixteen animals were housed in each pen and comprised a treatment group. Lights came on at 0700 h each day and light intensity throughout each pen, measured at approximate eye level of the heifers, averaged 230 lux. Heifers received a total mixed diet, fed ad libitum and fresh feed was offered daily at 0800 h with group refusals recorded each day.

A first experiment was designed to compare growth rates of prepubertal Holstein heifers exposed to photoperiods of 8L(cool-white fluorescent light):16D, 16L(cool-white fluorescent light):8D, 16L(Vita-Lite):8D or 6L:8D:2L:8D. Sixteen heifers (average body weight 102 kg) were assigned to each treatment. In addition, eating patterns and CR, SR and $t_{\frac{1}{2}}$ of GH in serum were determined in heifers exposed to 8 or 16 h of cool-white fluorescent light per day. After 112 days of treatment, differences due to photoperiod on average daily body weight gain were not significant ($P>.10$). However, heifers exposed to 16 h of Vita-Lite:8D weighed more ($P<.05$) on day 112 of treatment compared with heifers exposed to 8L(cool-white fluorescent light):16D. Animals exposed to 16L:8D had more eating events than heifers given 8L:16D, but there was no difference in feed intake between photoperiod treatments groups. Heifers exposed to 8L:16D had more of their eating activity in the dark period than heifers exposed to 16L:8D, especially in the 2 h period just after lights out and

the 2 h period just prior to lights on. Photoperiod had no effect (P>.10) on CR, SR or $t_{\frac{1}{2}}$ of GH in serum.

A second study was designed to compare the effects of 8 and 16 h of light per day on weight gain, body composition and serum hormones in Holstein heifers which remained prepubertal for the duration of the trial and in Holstein heifers that were postpubertal at the start of the trial. In addition, urinary excretion of 3-MeHis was quantified as an estimate of muscle protein degradation. Forty-two prepubertal heifers (2-months of age, 102 kg average body weight) and 42 postpubertal heifers (10-months of age, 300 kg average body weight) were utilized in the trial. Ten animals from each puberty group were randomly selected for pretreatment slaughter. The remaining 32 heifers in each age group were paired by body weight and assigned to photoperiod treatments of 8 or 16 h of light per day. After an average of 139 days on treatment ten heifers from each treatment group were slaughtered (40 animals total). Percentages of fat, water and protein were quantified in 9-10-11 rib sections. On day 94 of treatment, blood was collected from 5 animals from each treatment and assayed for prolactin, GH and cortisol. After 96 days on treatment urine was collected for 4 consecutive days from heifers designated for slaughter and assayed for 3-MeHis. In prepubertal heifers given 8 or 16 h of light per day there were no differences due to photoperiod in average daily weight gain, feed intake, fat percentage or protein percentage. There was also no effect of photoperiod on concentrations of prolactin, GH or cortisol in serum of prepubertal heifers. However, excretion of urinary 3-MeHis was reduced in prepubertal heifers exposed to 16L:8D compared with 8L:16D. Postpubertal heifers exposed to 8L:16D had a greater rate of weight gain than heifers exposed to 16L:8D. In addition, heifers exposed to short days had increased percentage of fat in rib sections (P=.06) and increased total fat accretion in the carcass (P=.06). Compared with

postpubertal heifers exposed to short days, heifers given 16L:8D had an increased percentage of protein in rib sections (P=.07) but there was no effect of photoperiod on total protein accretion in the carcass. In addition, photoperiod did not affect concentrations of GH or cortisol in serum of postpubertal heifers. However, postpubertal heifers exposed to 16L:8D tended to have higher serum concentrations of prolactin (P<.10).

In conclusion, in these experiments duration of daily light affected growth rate of postpubertal but not prepubertal Holstein hiefers. Photoperiod did not affect body composition of prepubertal heifers. However, photoperiods of 16L:8D reduced protein degradation in prepubertal heifers. The additional weight gain in postpubertal heifers exposed to short days was primarily fat accretion. Any effect of photoperiod on protein accretion was masked in both prepubertal and postpubertal heifers because average daily weight gain in these animals was greater than 1 kg/day. There was no effect of photoperiod on feed intake, but photoperiod did affect eating patterns. CR, SR, $t\frac{1}{2}$ and serum concentrations of GH were not affected by photoperiod and therefore, GH is probably not directly involved in photoperiod-induced changes in growth. The role of prolactin and cortisol in photoperiod-induced changes requires further investigation. Size and pubertal status of livestock and duration and method of light exposure must be taken into account when utilizing photoperiod to improve livestock efficiency.

APPENDIX

Appendix 1

Validation of Radioimmunoassay of cortisol

Materials and Methods

Dilutions of antiserum, hormone and serum. Rabbit antiserum (F3-314) raised against a conjugate of cortisol-3-oxime bovine serum albumin was obtained from Endocrine Sciences (Tarzana, CA) as a lyophilized powder. The lyophilized antiserum was reconstituted to its original concentration of 1:20 with double distilled, deionized water. Subsequent dilution to 1:400 was made with borate buffer (.05 M, pH 8.0) containing .25% bovine serum albumin (BB-BSA). Further antiserum, dilutions to 1:2400, 1:3200, 1:4000 and 1:4800 were made with 1:400 normal rabbit serum in phosphate-buffered saline (.01 M, pH 7.0)-disodium ethylenediaminetetraacetate (.05 M).

A solution of [1,2,6,7 ^{3}H]cortisol (80 Ci/mMole, New England Nuclear, Boston, MA) was diluted in methanol to approximately 3400 cpm/µl. A total of 350 µl of diluted [^{3}H]cortisol was added directly to 20 ml of the diluted antiserum. A stock solution of unlabeled cortisol (Sigma Chemical Co., St. Louis, MO), 10 µg cortisol/ml methanol, was diluted with BB-BSA to provide standards of 1500, 1000, 750, 500, 375, 250, 187.5, 62.5, 31.25 and 15.625 pg cortisol/100 µl.

All serum samples were diluted with BB-BSA and then heated at 60°C for 30 min to denature endogenous corticoid binding globulin (Daughaday et al., 1962).

Routine assays. Routine assays were carried out within 9 ml glass scintillation vials (13 x 51 mm). One hundred microliters of diluted sera with unknown concentrations of cortisol or known standard concentrations of cortisol were added to the vials. Immediately thereafter, 200 µl of the antiserum-[^{3}H]cortisol (12,000 cpm) mixture were added to each vial, for a total aqueous volume of 300 µl. Vials were shaken 30 to 50 times manually, incubated 15 to 18 h at 0 to 5°C and then 6 ml of a toluene-based scintillation fluid (3a20, Research Products International Corp., Elk Grove, IL) were added. The 3a20 contains only toluene and primary (PPO) and secondary (bis-MSB) fluors and therefore when combined with aqueous BB-BSA, two phases, one organic and one aqueous, formed (Neame and Homewood, 1974). After 30 h of incubation at 0 to 5°C, counting of [^{3}H]cortisol was conducted in a refrigerated spectrometer (Model LS 3130, Nuclear Chicago Corp., Des Plains, IL).

Initial testing. Vials containing a total aqueous volume of 300 µl of BB-BSA with [^{3}H]cortisol (12,000 cpm/vial) and F3-314 antiserum (diluted to 1:2400, 1:2800, 1:3200, 1:4000 or 1:4800) plus 6 ml of 3a20 were used to assess the effect of dilution of antiserum on maximal binding and sensitivity in the assay. These vials were counted without any other method to separate unbound from bound [^{3}H]cortisol.

Standard curves and recovery. Standard cortisol concentrations of 0, 15.625, 31.25, 125, 187.5, 250, 375, 500, 750, 1000, and 1500 pg/100 µl BB-BSA were assayed in duplicate. Standard curves were derived by fitting a polynomial regression equation including linear, quadratic and cubic terms, to a plot of $\log_{10}$ cpm versus $\log_{10}$ cortisol concentration. The goodness of fit (r^2) was assessed by the percent of variation accounted for in $\log_{10}$ cpm. Two unknown

serum samples (replicated 15 times) were supplemented with either 250 or 500 pg of cortisol to assess recovery of mass.

Specificity, parallelism and accuracy. The degree of cross-reactivity of the F3-314 antiserum was tested with 11 other steroids. One hundred microliters of two concentrations (1000 and 2000 pg/100 µl BB-BSA) of each steroid were assayed for cross-reactivity with the F3-314 antiserum.

Parallelism was tested by assaying two serum samples diluted in BB-BSA to 1:2, 1:5, 1:9, 1:11, 1:17, 1:23, 1:29 and 1:35.

Twenty serum samples were assayed for total glucocorticoids by competitive binding to dog plasma (Smith et al., 1972) and assayed for cortisol by the current method.

The intra-assay coefficient of variation in cortisol concentrations was calculated from five serum samples replicated 10 times in one assay. The inter-assay coefficient of variation was calculated from the same five serum samples assayed in quadruplicate in 10 different assays.

Temperature effect. The effect of temperature on the equilibrium of [^{3}H]cortisol between the aqueous and organic phases was evaluated. Vials contained [^{3}H]cortisol and F3-314 antiserum in 300 µl of BB-BSA and 6 ml of 3a20. These samples were assayed according to the current method except that during the last hour of incubation, vials were incubated at either 0, 5, 10, 15, 20, 25, 30, 40, 50 or 60°C (three vials per temperature). At the end of the hour a 5 ml aliquant of the organic phase was taken from each vial to prevent further change in equilibrium. Each aliquant was immediately counted at 0 to 5°C. Aliquants were then returned to their original vials, reincubated for 6 h at 0 to 5°C and recounted at 0 to 5°C.

Results

Initial testing. In vials containing [^{3}H]cortisol, F3-314 antiserum and 3a20 scintillation fluid, the quantity of unbound [^{3}H]cortisol detected in the organic phase varied inversely with concentrations of antiserum. That is, as concentrations of antiserum present in a vial increased, less unbound [^{3}H]cortisol was detected in the organic phase (Figure 13).

Standard curves and recovery. The goodness of fit of standard curves had coefficients of variation ranging from .980 to .999. The standard curve in Figure 14 was produced at an antiserum dilution of 1:4000. Percent binding was calculated from the equation:

$$\left[\frac{([^3\text{H}]\text{cortisol added minus unbound } [^3\text{H}]\text{cortisol})}{[^3\text{H}]\text{cortisol added}}\right]$$

The range in binding of [^{3}H]cortisol was from 69% for the zero cortisol standard to 8% for the highest cortisol standard (1500 pg/100 µl), with an r^2 of .999. Recoveries from serum supplemented with 250 or 500 pg cortisol averaged 97.5% (SD = 7.8).

Specificity, parallelism and accuracy. The degree of cross-reaction with 11 other steroids (Table 7) indicated that only cortisone and prednisone had greater than 3% cross-reactivity with the F3-314 antiserum. These results parallel the cross-reactivity data provided by Endocrine Sciences for the antiserum.

Some problems with lack of parallelism were encountered when serum samples were only diluted to 1:2 in BB-BSA. However, when serum was diluted to 1:9 or greater the parallelism problem disappeared (Figure 15). When serum

Figure 13. The effect of antibody concentration on the quantity of unbound [^{3}H]cortisol in the organic phase. Each vial contained [^{3}H]cortisol (12,000 cpm) and antiserum (at indicated dilutions) in 300 µl of BB-BSA (aqueous phase) plus 6 ml of 3a20 scintillation fluid (organic phase), without any other means to separate unbound and bound hormone. Vials were incubated 30 h and counted at 0 to 5°C.

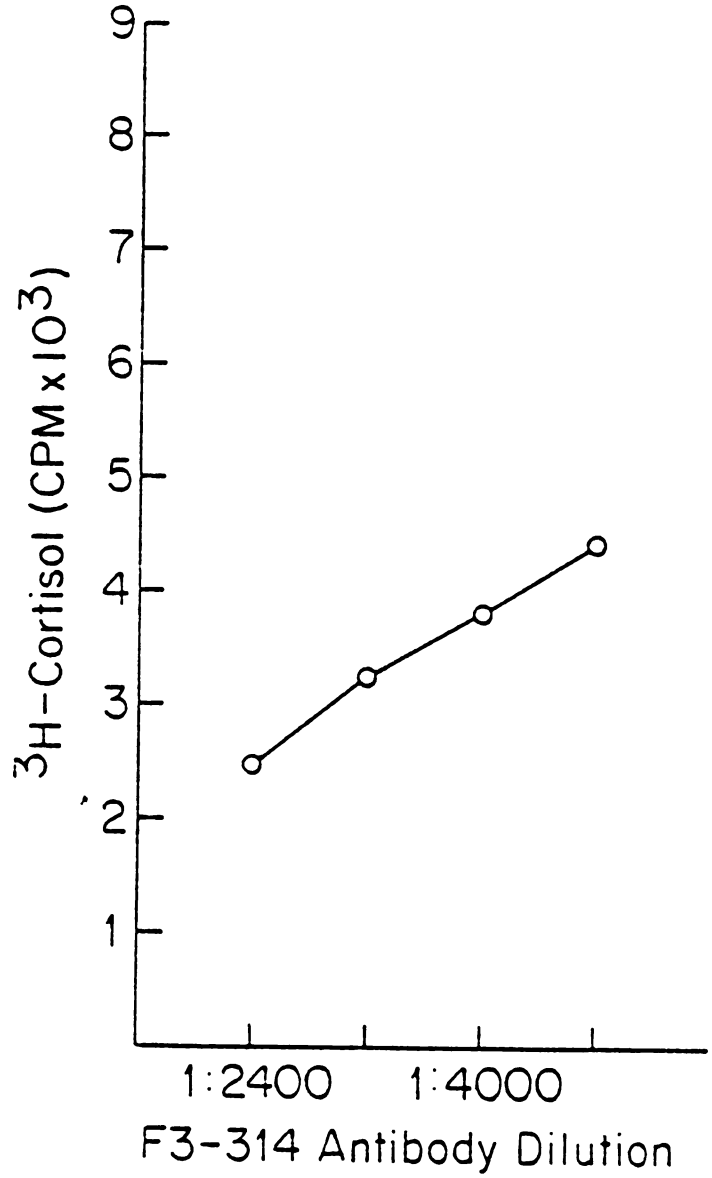

^{3}H-Cortisol (CPM x 10^3)
9
8
7
6
5
4
3
2
1
1:2400
1:4000
F3-314 Antibody Dilution

Figure 14. A standard curve for cortisol. Each vial contained [^{3}H]cortisol (12,000 cpm), F3-314 antiserum (1:4000) and standard cortisol (at indicated concentrations) in 300µl of BB-BSA plus 6 ml of 3a20 scintillation fluid.

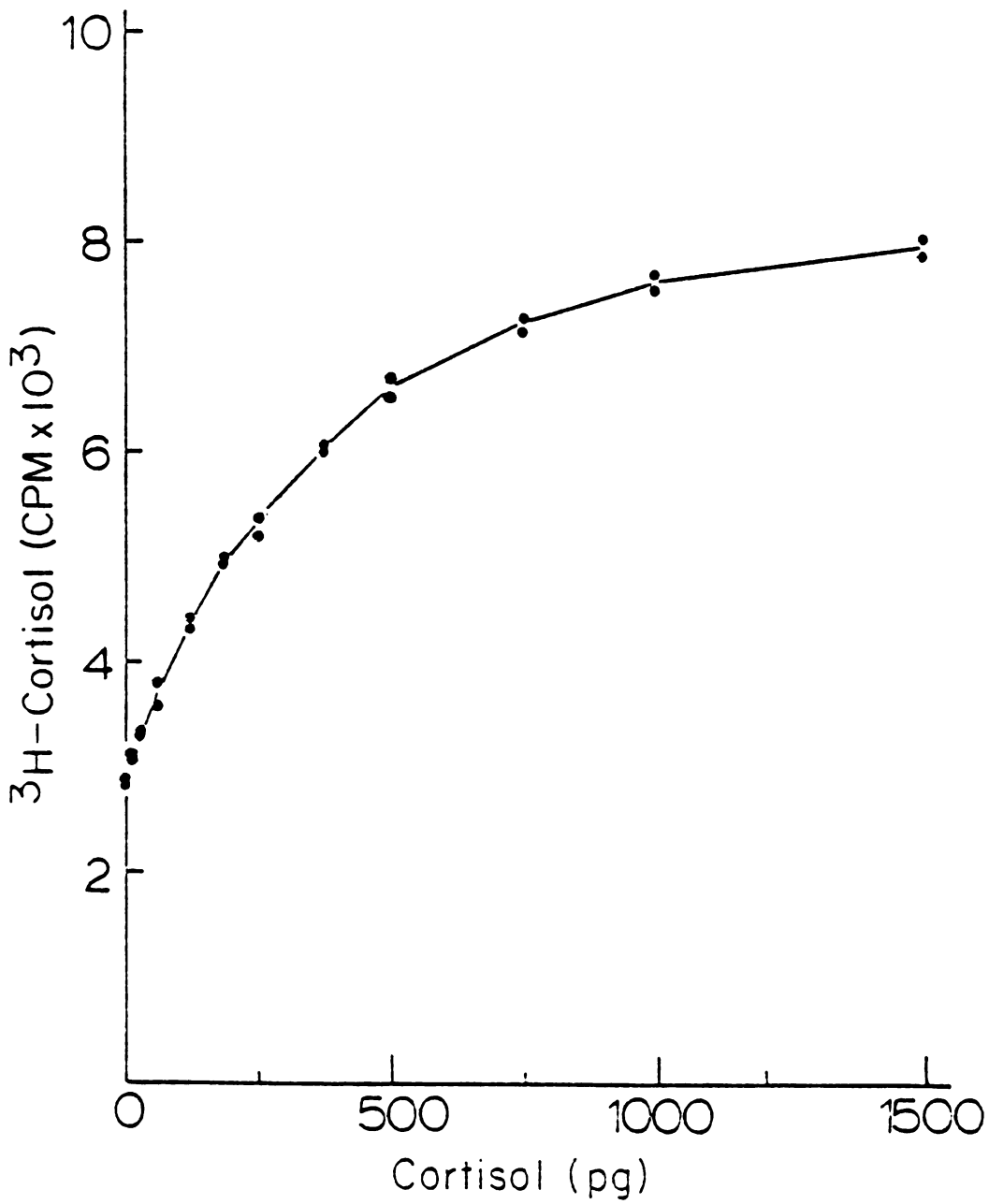
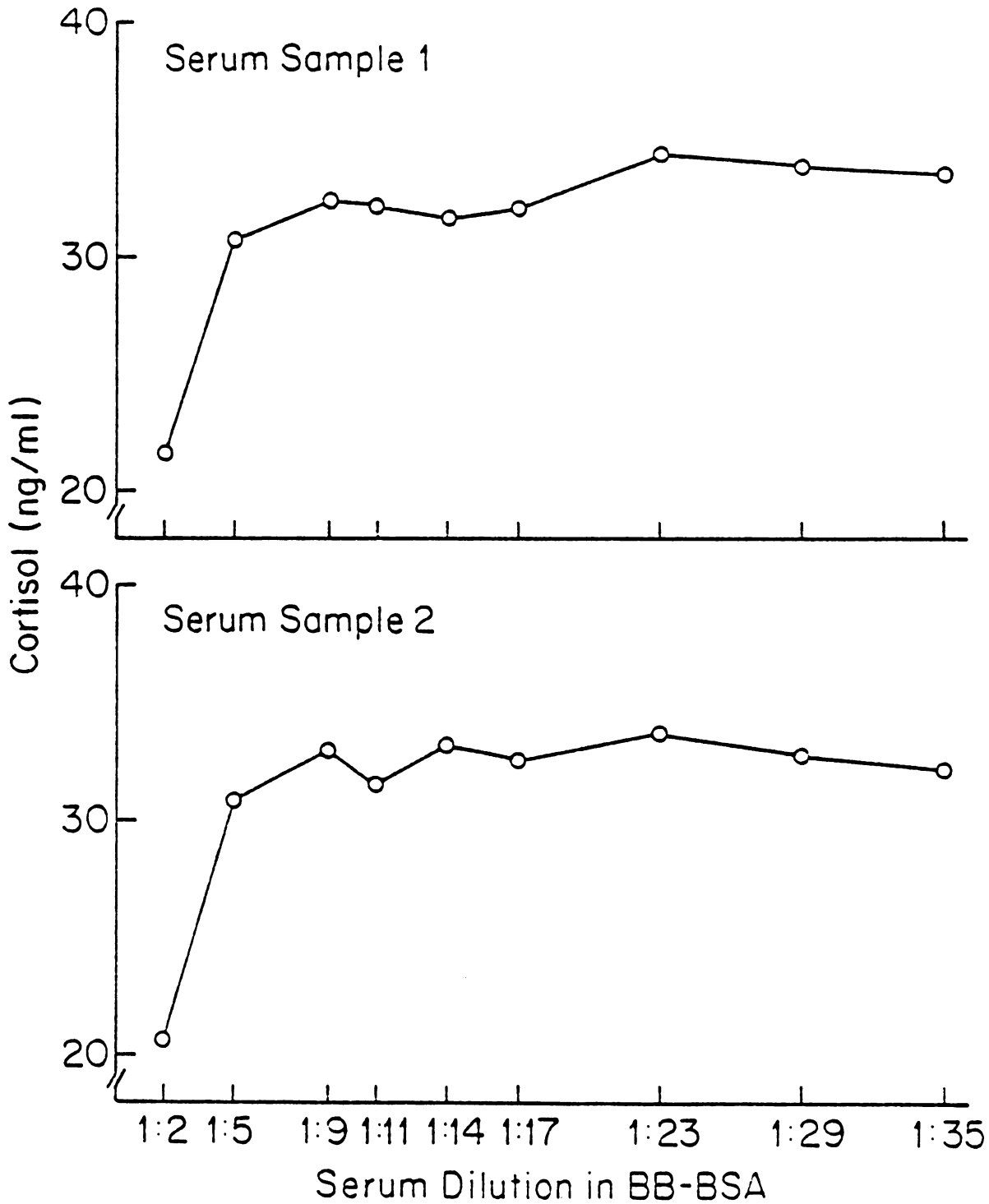

10
8
6
4
2
^{3}H-Cortisol (CPM x 10^3)
0
500
1000
1500
Cortisol (pg)

Table 7. Percent cross-reaction of various steroids with antiserum F3-314.

Compound	Percent cross-reactivity
Corticosterone	3
Deoxycorticosterone	< 1
d-Aldosterone	< 1
17-Hydroxy-progesterone	< 1
Testosterone	< 1
Progesterone	< 1
17β-Estradiol	< 1
11-Deoxy 17-hydroxy corticosterone	< 1
Estriol	< 1
Prednisone	12
Cortisone	16

Figure 15. The effect of serum dilution on parallelism. Two serum samples were diluted in BB-BSA as indicated. Each point represents the mean of three values corrected for dilution.


Serum Sample 1
Serum Sample 2
40
30
20
Cortisol (ng/ml)
1:2 1:5 1:9 1:11 1:14 1:17 1:23 1:29 1:35
Serum Dilution in BB-BSA

is diluted 1:9 in BB-BSA the assay detects concentrations of cortisol ranging from 1.4 ± .3 to 135 ± 8.1 ng/ml of serum.

Serum concentrations of glucocorticoids determined by competitive binding assay (Smith et al., 1972) were highly correlated (r = .90) with cortisol concentrations measured by the current procedure (Table 8). Willett and Erb (1972) reported a similar correlation (r = .85) between concentrations of cortisol and total glucocorticoids in bovine serum. Concentrations of cortisol represent 30 to 60% of total glucocorticoids in bovine serum (Willet and Erb, 1972; Gaverick et al., 1971). Cortisol in bovine serum, measured by the current method, averaged 41% of total glucocorticoids, which lies within this range.

Intra-assay coefficient of variation in five serum samples was 8% (n = 10 replicates/serum sample) and inter-assay coefficient of variation for the same serum samples in 10 assays was 13% (n = 4 replicates · serum sample $^{-1}$· assay^{-1}).

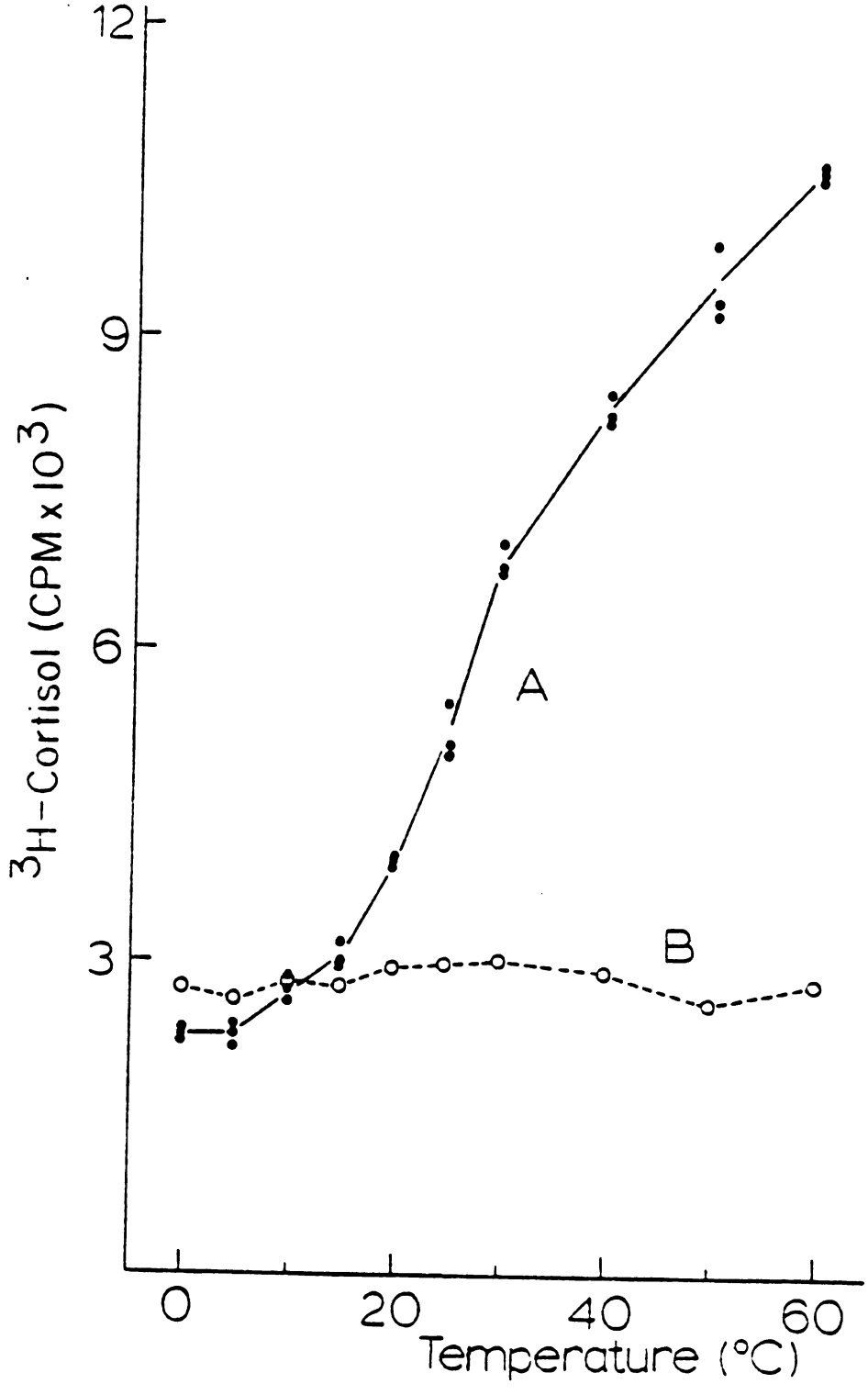
Temperature effect. As temperatures increased from 10 to 60°C, the quantity of [^{3}H]cortisol detected in the organic phase increased (Figure 16, curve A). When the aliquant was returned to its original vial, reincubated at 0 to 5°C for 6 h and recounted at 0 to 5°C, the [^{3}H]cortisol which had passed into the organic phase at high temperatures returned to the aqueous phase (Figure 16, curve B). Because of this effect of temperature, the assay should be incubated and counted at temperatures below 10°C. Alternatively, if a refrigerated spectrometer is unavailable, the assay should be incubated at temperatures below 10°C, followed by decanting a portion of the organic phase to another scintillation vial and counting at ambient temperature. Separation of the organic from the aqueous phase before counting prevents the repartitioning of [^{3}H]cortisol that occurs with temperatures above 10°C.

Table 8. Comparison of serum samples assayed with a competitive binding assay and the current radioimmunoassay method.

Sample	Competitive binding assay (total glucocorticoids, ng/ml)	Current method (cortisol, ng/ml)
1	24.9	11.5
2	52.6	26.0
3	6.3	2.9
4	66.9	25.6
5	7.7	3.4
6	10.4	4.7
7	8.0	4.2
8	10.8	4.8
9	8.3	4.7
10	5.9	1.6
11	48.9	18.4
12	8.3	2.1
13	6.5	2.5
14	8.0	2.7
15	25.7	15.8
16	8.8	3.9
17	8.6	8.7
18	10.2	17.6
19	6.8	5.1
20	37.4	22.9

Correlation coefficient = .90.

Figure 16. The effect of temperature on the quantity of [^{3}H]cortisol in the organic phase. Each vial contained [^{3}H]cortisol (12,000 cpm) and F3-314 antiserum (1:4000) in 300 µl of BB-BSA plus 3a20 scintillation fluid (6 ml). Vials were counted at 0 to 5°C. Curve A: Vials were incubated for 1 h at the indicated temperature. A 5 ml aliquant of the organic phase was taken and counted. Curve B: Each aliquant was returned to its original vial, reincubated for 6 h at 0 to 5°C and recounted. Each point is the mean of three values.


12
9
6
3
^{3}H-Cortisol (CPM x 10^3)
A
B
0
20
40
60
Temperature (°C)

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