bial products that are cost-effective and can be adapted to fit the technology of modern agriculture represents a significant challenge. Considerable work must be done in such areas as developing a highly concentrated inoculum with a relatively long shelf life and in a form that can be applied commercially. Also, health-related data for state and federal registration must be obtained depending on the claimed mode of action of the product. Ironically, a major nuisance at the marketplace may be how to distinguish an efficacious microbial product from the many dubious microbial elixirs that have been sold to farmers since the beginning of the century. None of the dozens of the microbial concoctions sold as soil catalysts, activators, and soil builders has proved effective, and the success that some have had in marketing products of no known value does not argue well that the truth will ultimately prevail.

The development of efficacious microbial products will depend on the cooperative efforts of bacterial ecologists, plant pathologists, physiologists, biochemists, and genetic engineers. Such a team could cooperate, for example, on the determination of the important key characters and systems which enable a microorganism to successfully compete in a particular ecological niche. With PGPR, once the specific metabolite in excluding a deleterious microorganism is known, research can begin on the regulatory mechanisms affecting its production, followed by genetic manipulation of key biochemical processes. As in industrial microbiology, it should be possible to obtain highly productive strains. The competitive ability of epiphytic bacteria to colonize roots and their capacity to exclude deleterious microorganisms from the root surface also could be greatly improved through genetic engineering to enable them to tolerate great moisture stress or to produce a wider array of metabolites that would affect a greater spectrum of deleterious microorganisms. Characters that allow rhizobacteria to proliferate on roots should be examined with particular care; they are the key to using other beneficial bacteria as root colonizers.

References and Notes

1. P. J. Shipton, in *Soil-Borne Plant Pathogens*, B. Schippers and W. Gams, Eds. (Academic Press, London, 1979), pp. 201–206.
2. ———, *Eur. Mediterr. Plant Prot. Organ. Bull.* **5**, 363 (1975).
3. C. Alabouvette, F. Rouxel, J. Louvet, in *Soil-Borne Plant Pathogens*, B. Schippers and W. Gams, Eds. (Academic Press, London, 1979), pp. 166–168.
4. S. Wilhelm and A. O. Paulus, *Plant Dis. Rep.* **64**, 264 (1980); ———, R. C. Storkan, J. E. Sagen, *Phytopathology* **51**, 744 (1961).
5. T. A. Toussoun, in *Biology and Control of Soil-Borne Plant Pathogens*, G. W. Bruehl, Ed. (American Phytopathological Society, St. Paul, Minn., 1975), pp. 145–151.
6. F. M. Scher and R. Baker, *Phytopathology* **70**, 412 (1980); *ibid.*, in press.
7. S. Liu and R. Baker, *ibid.* **70**, 404 (1980).
8. F. N. Martin and J. G. Hancock, *ibid.* **71**, 893 (Abstr.) (1981).
9. P. Broadbent, K. F. Baker, Y. Waterworth, *Aust. J. Biol. Sci.* **24**, 925 (1971).
10. K. F. Baker and R. J. Cook, *Biological Control of Plant Pathogens* (Freeman, San Francisco, 1974); J. D. Menzies, *Phytopathology* **49**, 648 (1959); P. J. Shipton, R. J. Cook, J. W. Sitton, *ibid.* **63**, 511 (1973).
11. G. C. Papavizas and R. D. Lumsden, *Annu. Rev. Phytopathol.* **18**, 389 (1981).
12. D. Hornby, in *Soil-Borne Plant Pathogens*, B. Schippers and W. Gams, Eds. (Academic Press, London, 1979), pp. 133–156.
13. T. J. Burr and M. N. Schroth, *Phytopathology* **68**, 1377 (1978).
14. M. N. Schroth and J. G. Hancock, *Annu. Rev. Microbiol.* **35**, 453 (1981).
15. J. W. Kloepper and M. N. Schroth, in *Proceedings of the IVth International Conference on Plant Pathogenic Bacteria II*, Angers, France, 27 August to 2 September 1978, edited by Station de Pathologie Végétale et Phytobactériologie (Gibert-Clarey, Tours, France, 1978), pp. 879–882.
16. R. Y. Stanier, N. J. Palleroni, M. Doudoroff, *J. Gen. Microbiol.* **43**, 159 (1966).
17. J. W. Kloepper, M. N. Schroth, T. D. Miller, *Phytopathology* **70**, 1078 (1980).
18. T. V. Suslow and M. N. Schroth, *ibid.* **72**, 199 (1982).
19. J. W. Kloepper and M. N. Schroth, *ibid.* **71**, 590 (1981).
20. ———, *ibid.*, p. 1020.
21. T. V. Suslou and M. M. Schroth, *ibid.* **72**, 111 (1982).
22. R. M. Jackson and M. E. Brown, *Ann. Inst. Pasteur Paris* **3** (Suppl.), 102 (1966).
23. M. E. Brown, *Annu. Rev. Phytopathol.* **12**, 181 (1974).
24. J. W. Kloepper and M. N. Schroth, *Phytopathology* **71**, 642 (1981).
25. T. V. Suslou and M. M. Schroth, *ibid.*, in press.
26. R. W. Leukel, *J. Agric. Res.* **77**, 201 (1948); R. A. Steinberg, *Plant Physiol.* **27**, 302 (1952).
27. T. Leisinger and R. Margraff, *Microbiol. Rev.* **43**, 422 (1979).
28. J. B. Neilands, *Adv. Chem. Ser. No. 162* (1977), p. 3.
29. J. W. Kloepper, J. Leong, M. Teintze, M. N. Schroth, *Nature (London)* **286**, 885 (1980).
30. R. J. Cook and A. D. Rovira, *Soil Biol. Biochem.* **8**, 269 (1976).
31. R. W. Smiley, *ibid.* **10**, 169 (1978).
32. J. W. Kloepper, J. Leong, M. Teintz, M. N. Schroth, *Curr. Microbiol.* **4**, 317 (1980).
33. D. M. Weller and R. J. Cook, *Phytopathology*, in press.
34. M. Teintze, M. B. Hossain, C. L. Barnes, J. Leong, D. van der Helm, *Biochemistry* **20**, 446 (1980).
35. M. Teintze and J. Leong, *ibid.*, p. 6457.
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Controlled Photoperiodic Environments for Food Animals

H. Allen Tucker and Robert K. Ringer

In many areas of the world, climate limits the productivity as well as the survival of livestock. Consequently, man has provided shelter to animals in an effort to improve their productivity. Since the end of World War II, animals in the more developed countries have been housed in increasingly closer confinement and their environments have been more regulated. This shift toward more closely controlled environments has contributed to increased productivity. This in turn has reduced production costs and, in proportion to income, has lowered costs of food from animals.

Seasonal variation in the reproductive activity of several domestic species used for food has been known for centuries. Of the variables associated with the environment, it is now recognized that photoperiod is the primary cue regulating seasonal reproduction. Modern production methods often expose animals to photoperiods substantially different in intensity and duration from those of natural photoperiods. Manipulation of photoperiod has been practiced commercially for more than 60 years to control the onset of egg production and to stimulate egg laying and regulate body growth in chickens. Results of recent research suggest that photoperiod may be manipulated to stimulate reproduction and body growth, increase milk production and the efficiency of feed utilization, and hasten puberty in several domestic species. In this article we focus on the role of photoperiod in the regulation of these traits.

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Summary. Recent studies suggest that control of daily light exposure regulates reproduction, stimulates body growth, and increases the efficiency of feed utilization and egg and milk yields in several domestic species used for food. Hormones mediate photoperiod-induced changes in these traits. Photoperiod manipulation is a promising method of increasing food production from domestic animals.

Reproduction in Seasonally Breeding Domestic Mammals

Sheep and goats exhibit marked seasonal variations in sexual activity. In many breeds of sheep, estrous cyclicity in females (ewes) and sexual aggressiveness, testis size, and sperm production in males (rams) are minimal from spring until late summer. Maximal sexual activity occurs in autumn, coincident with the decreasing duration of daily light (*1*). This seasonal pattern of reproduction, coupled with the duration of gestation, ensures that lambs are born in the spring. Shelters provided in modern intensive sheep-farming operations may permit development of methods to overcome seasonal anestrus and thereby distribute lamb production throughout the year.

Seasonal breeding activity in ewes is controlled by a complicated interplay of stimulation and inhibition between luteinizing hormone (LH) secreted from the anterior pituitary and estradiol and progesterone secreted from the ovary (*2*). During the breeding season, estrus and ovulation recur at 16- to 17-day intervals in ewes. Before ovulation, concentrations of LH in serum are suppressed by high concentrations of progesterone secreted from the corpus luteum. About 4 days before ovulation the corpus luteum regresses, resulting in a sharp decline in progesterone secretion. The decrease in progesterone allows basal secretion of LH to increase over the next 2 to 3 days; this stimulates secretion of estradiol from the developing ovarian follicle. By the time of behavioral estrus, secretion of estradiol has been of sufficient duration and magnitude to induce a massive surge in the LH concentration, causing ovulation approximately 24 hours after estrus. Thus, during the breeding season estradiol enhances the secretion of LH (Fig. 1). As the duration of daily light increases during late winter, estradiol begins to inhibit the initial rise in LH secretion in the preovulatory period (Fig. 1). Thus, during anestrus estradiol has an inhibitory effect on LH secretion, the preovulatory surge of LH does not occur, ovulation fails, and nonpregnant ewes become anestrous. As the length of daylight decreases during summer the hypothalamopituitary system, responsible for tonic secretion of LH, once more becomes less sensitive to the inhibitory effects of estradiol (*2*). A rapid, pulsatile pattern of LH secretion develops, causing increased secretion of estradiol which in turn initiates the first preovulatory surge of LH and the first estrus and ovulation of the season in mature ewes (Fig. 1). The specific signals in the brain which translate shifts in the duration of daily light into seasonal shifts in the responsiveness of LH to estradiol remain to be determined.

Shortening the daily light exposure during the normal period of seasonal anestrus induces ovulation and estrus. Although some breeds of sheep in anestrus return to estrus within 24 days of shortening the period of daily light (*3*), in other breeds more than 100 days are required (*1*). Administration of 6-methyl-17-acetoxyprogesterone (a progestin) and pregnant mare serum gonadotropin (PMSG) to ewes during anestrus without altering the environment induces estrus and ovulation, but conception rates are low (*4*). However, by reducing daily light, controlling ambient temperatures, and administering a sequence of progesterone (for 14 days), PMSG (on day 15), and estradiol (on day 16), it is possible to breed the ewes within 24 hours after the estradiol administration (*5*). Conception rates average 80 percent in these ewes when bred at the first estrus. Hence, with controlled environments and hormone administration it is possible to distribute the lamb crop throughout the year.

Shortening the duration of daily light may also initiate the first ovulation in pubertal ewe lambs (*6*). As in mature ewes, decreased sensitivty of the hypothalamopituitary system to the inhibitory effects of estradiol on LH secretion initiates the first ovulation and estrus (puberty) in ewe lambs (*2*, *6*). Breeding of ewe lambs at an earlier age would shorten the generation interval and reduce the costs of rearing. This would lead to greater efficiency in the sheep industry.

Rams also show marked seasonal variation in reproductive activity. Under normal temperate-zone climatic conditions, mating activity in autumn is about twice that in later winter and spring in rams cohabited with ewes artificially induced into estrus (*7*). Decreasing duration of daily light increases testis size, spermatogenesis, and secretion of LH, follicle-stimulating hormone (FSH), and testosterone (*8*). Increased secretion of testosterone stimulates the accessory sex glands and causes aggressive sexual behavior (*9*). The photoperiod entrains the reproductive cycle (*10*). For example, the cycle of testis development and testosterone secretion waxed and waned coincidently in two groups of rams maintained between 4 and 20 months of age under photoperiods with 16 hours of light and 8 of darkness or 8 hours of light and 16 of darkness (*11*). Both groups of rams were, however, approximately 4 months

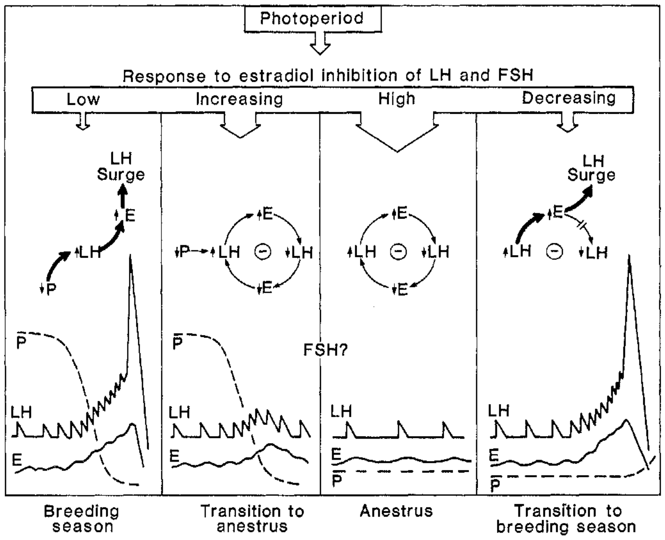

Fig. 1. Proposed model for photoperiodic control of seasonal breeding in ewes. During the breeding season a decrease in progesterone increases pulsatile releases of LH, which in turn increase estradiol, causing the preovulatory surge of LH (far left panel). As daily light increases during late winter, pulsatile releases of LH become more sensitive to the inhibitory action of estradiol, which prevents continuation of the rise in LH secretion. Ovulation does not occur and anestrus begins. As daily light decreases in late summer, the inhibitory effects of estradiol disappear, and estradiol again drives the preovulatory surge of LH (far right panel). [From Karsh and Foster (*2*)]

out of phase with a group of control rams exposed to the natural photoperiod. These cycles are probably of endogenous origin, but are entrained by the photoperiod.

Analogous to the mechanism in ewes whereby the photoperiod affects sensitivity of the hypothalamopituitary system to estradiol inhibition of LH secretion (*2*), in rams decreasing duration of daily light decreases the inhibitory effects of testosterone on LH secretion (*12*). These effects are reversed as the duration of daily light increases, and sexual activity of the rams ceases. Secretions from the pineal gland also must constitute part of the mechanism whereby photoperiod effects are mediated. For example, in rams pinealectomy or removal of sympathetic innervation of the pineal abolishes photoperiod-induced changes in sexual aggressiveness, testis weight, seminal volume, number of spermatozoa, and secretion of LH, FSH, and testosterone (*13*). Long-term cyclical changes in the reproductive system persist even though these animals are no longer affected by the photoperiod. This constitutes additional evidence that the seasonal reproductive cycle may be an endogenous rhythm or may respond to a climatic variable other than photoperiod. Indeed, reducing the environmental temperatures from an average maximum of 26° to 31°C in uncontrolled conditions to a controlled 7° to 9°C increases sperm motility 68 percent (*14*). Lambing of ewes bred to rams exposed to the uncontrolled environment averaged 13 percent, whereas in ewes bred to rams maintained at the lower temperatures lambing averaged 50 percent.

Shortening the duration of light exposure during the nonbreeding season stimulates sperm production in rams, leading to an increased lambing rate in ewes artificially induced to ovulate (*15*). Thus, exposure of sheep to shortened periods of light is likely to be a useful means for distributing the lamb crop throughout the year.

Reproduction in Chickens

In the natural environment of the temperate zones, chickens exhibit annual variations in reproductive activity. However, through maintenance of a continuous feed supply and manipulation of the photoperiod, reproductive activity in chickens can be sustained throughout the year (*16*). Specifically, photoperiod regulation is used to control the onset of egg production and to maintain sustained egg production in commercial flocks. The reproductive capacity of egg-laying strains is approaching an average of 260 eggs per bird annually. Without management of the photoperiod there would be marked seasonal variations in the price and availability of eggs and broilers.

The periodicity of light influences egg laying by domestic chickens through two processes, an annual cycle and a circadian rhythm. The two modes interact but their mechanisms of action differ. The annual cycle delineates the onset and termination of egg production, and light is the sole signal. In the circadian rhythm, the daily light-dark cycle is normally the most important cue in setting the time of oviposition, but when exposure to light is continuous, temperature or noise may determine the time of oviposition.

The eyes in chickens are not necessary for photoperiodic responses, since light, especially rays of long wavelength, can penetrate the skull of birds (*17*). Wavelength affects development of the reproductive system, with near-red light (575 to 650 nanometers) being more stimulatory than other wavelengths (*18*). Beads of luminous paint implanted into specific areas of the hypothalamus have caused testicular growth in Japanese quail (*19*) similar to that in light-stimulated animals. Extraretinal light receptors have been postulated for chickens (*20*).

If chickens are raised under a constantly repeating photoperiod following hatching, egg laying will commence at approximately 5 months of age regardless of whether daily exposure to light is short (6 hours) or long (22 hours). Chickens reared under short days are delayed slightly in sexual maturity (appearance of the first egg). Rearing of chickens under a progressively increasing or decreasing period of daily light markedly hastens or delays, respectively, the onset of sexual maturity. Delayed sexual maturity normally increases the weight of eggs subsequently laid. Changes in gonadotropin secretion mediate the effects of photoperiod. For example, LH concentrations in plasma of chickens reared under a constant short- or long-day photoperiod are comparable, whereas shifting from short to long daily light or vice versa increases or decreases, respectively, LH concentrations in plasma (*16*).

Once sexual maturity has been attained, the number of eggs laid per unit of time responds to increases or decreases in daily light more than to a constant number of hours of light per day. For example, annual egg production rates nearly 60 percent of maximum have been achieved in chickens kept in darkness beginning at 1 week of age (*21*). Greater egg production can be sustained by exposure to 6 to 10 hours of light per day (*16*). Progressively longer days maximize the egg-laying rate, while progressively shorter days reduce egg produc-

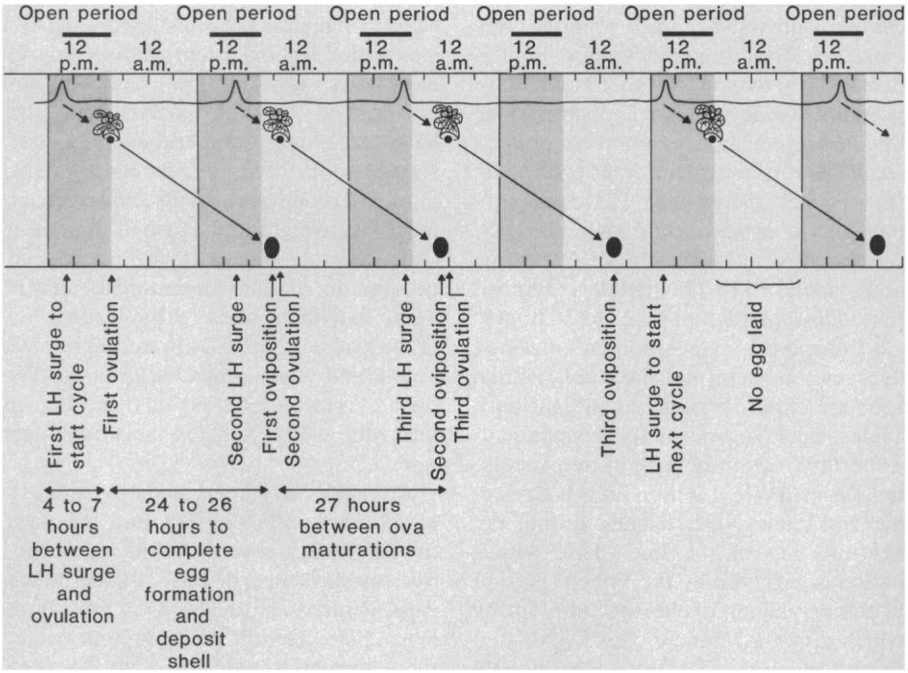

Fig. 2. Ovulatory response in chickens. Typically, chickens lay their eggs in a sequence of several days, pause for one or more days, and then start the cycle again. Pictured is a 3-day sequence of egg laying followed by a 1-day pause. An LH surge, which occurs only during the open period, ovulates a mature follicle and starts egg formation. The open period is entrained by light-dark cycles and does not shift. In contrast, the LH surge, ovulation, and oviposition shift each day; therefore, the open period and the LH surge are not always synchronized.

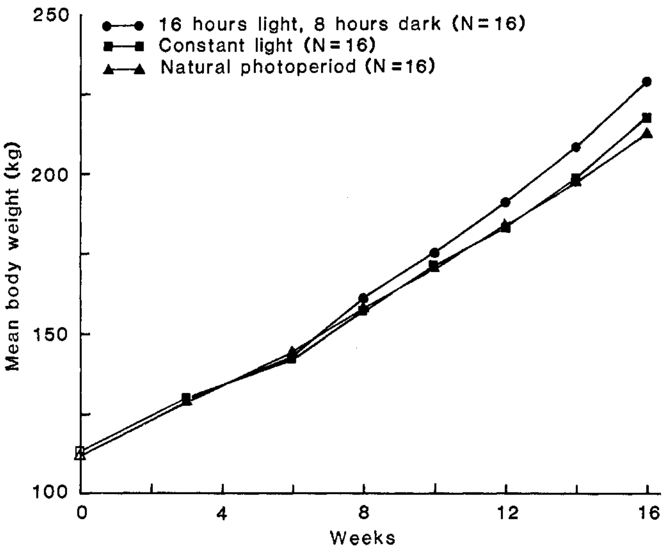

Fig. 3. Body growth of Holstein heifers exposed to different photoperiods between 11 November and 10 March. The slope of the line for exposure to 16 hours of light and 8 hours of darkness differs significantly from the slope of the line for constant light ($P < .05$, Bonferroni test) and from the slope of the line for natural photoperiod ($P < .02$). Standard errors did not exceed 6 kilograms at week 16. The length of natural daylight varied from 9 to 12 hours. [From Peters *et al.* (*37*)]

tion. The latter response is more pronounced than the former. In general, exposing chickens to incrementally increasing periods of daily light beyond 17 hours in duration yields no further increase in the number of eggs laid. The minimum intensity of lighting required for maximum egg production is between 2 and 10 lux (*22*).

After chickens become sexually mature, their ovarian follicles develop in response to increased secretion of LH and FSH. Follicular maturation follows an established course. Normally, one follicle reaches maturity approximately every 27 hours (Fig. 2). The largest follicle, as it grows, becomes progressively more sensitive to gonadotropin stimulation and will ovulate (rupture and release yolk) in response to an LH surge. The follicle secretes large amounts of progesterone as it nears maturity. It is possible that a small increase in LH causes an increase in progesterone secretion (*23*) which, in turn, results in an LH surge and ovulation 4 to 7 hours later. Oviposition follows ovulation by 24 to 26 hours.

In chickens, oviposition is entrained by cycles of light and dark (*24*). When hens are exposed to circadian light-dark cycles, egg laying normally is confined to a specific fraction of each cycle. That is, most eggs are laid within an 8-hour segment of each cycle, usually during the light portion of the day. This 8-hour period is triggered by the "open" period of the egg-laying cycle, the time during which a mature follicle can be ovulated by an LH surge. The first LH surge in a sequence of egg laying occurs early in the open period (Fig. 2). The follicular maturation period (27 hours) is out of phase with the 24-hour light-dark cycle. Since the open period is an approximately 8-hour segment of the light-dark cycle, once this period ends ovulation is prevented and the chicken pauses for one or more days and then starts the sequence anew. Time of occurrence of the open period is determined in chickens by the onset of darkness (*25*). If there is a shift in the lighting schedule, chickens require several days to entrain to the new cycle. The use of absolute darkness for entrainment is unnecessary, provided the bright portion is ten times more intense than the dim (*26*).

Since there is a lag in the endocrine control of ovulation resulting in oviposition at 27-hour intervals on average, the effects of ahemeral cycles (light-dark cycles with durations other than 24 hours) have been studied (*21, 27*). Hens do not lay eggs at intervals shorter than the light-dark cycle. Ahemeral cycles longer than 24 hours lower the rate of egg laying in highly productive flocks, but increase mean egg weight and eggshell thickness (*27*). The rate of egg laying is reduced because in modern commercial strains many individual hens within a flock lay at intervals closer to 24 hours than to 27 hours, and when subjected to 26- or 27-hour cycles they adjust to these longer intervals, which reduces their ovulation rate (*21*).

Attempts have been made, by the use of light-dark cycles of less than 24 hours, to increase egg production through selection and breeding of birds that produce eggs at intervals of less than 24 hours. When placed on the usual 24-hour cycle, these strains should produce one egg each day. Selection for strains responsive to ahemeral cycles less than 24 hours, however, has not yielded significantly better results than selection based on the 24-hour-day cycles.

Reproduction in Mammals That Breed Throughout the Year

Domesticated cattle and swine ovulate and conceive throughout the seasons. This may reflect the intense selection of these animals by man to provide a year-round supply of food. Nevertheless, the rate of conception in cattle is reduced slightly in winter at northern latitudes and in summer at latitudes closer to the equator (*28*). Reduced fertility in hotter climates in summer is associated with increased temperatures. The effects of seasonal variation in photoperiod on reproduction in cattle and swine are minimal compared to those in sheep and fowl. For example, Sweetman (*29*) found that exposure of dairy cows to 14 hours of light per day in the winter in Alaska resulted in a conception rate of 54 percent, whereas the conception rate for control cows given less than 8 hours of light daily was 49 percent (*29*). Neither 8 nor 16 hours of light per day affect the magnitude, duration, or timing of preovulatory surges in LH and FSH or the length of the estrous cycle in postpubertal dairy heifers (*30*). Furthermore, LH secretion is not affected in prepubertal bulls subjected to increasing or decreasing photoperiods (*31*). This relative lack of effect of photoperiod on reproduction fits well with the fact that cattle are nonseasonal breeders.

For the reasons previously described for ewe lambs, shortening the time to puberty and breeding could lead to more efficient production of food from domestic mammals. Progress in this area is promising for cattle. For example, in a study by Roy *et al.*, heifers born in seasons of increasing daylight reached puberty about 2 months earlier than heifers born during seasons of decreasing daylight (*32*). This hastening of puberty occurred independent of the rations fed, which were similar across seasons. Furthermore, heifers exposed to 16 hours of light per day reach puberty 1 month earlier and weigh 13 kilograms less than heifers exposed to 8 hours of light per day (*33*).

The effects of photoperiod on puberty in female pigs are controversial. Recent evidence suggests that rearing young sows in the dark (1.5 hours of light per day) delays puberty (*34*). Puberty in sows provided 18 hours of light per day does not arrive sooner than puberty in sows exposed to 9 to 11 hours of natural light per day. However, puberty in young male pigs exposed to 15 hours of illumination does arrive sooner than puberty in those exposed to increasing or decreasing natural photoperiods (*35*).

Body Growth

Average daily weight gains are 10 to 15 percent greater in heifers exposed to 16 hours of light per day than in heifers subjected to 9 to 12 hours of light in autumn and winter in Michigan (Fig. 3) (*36*). Continuous lighting does not increase the rate of body weight gain (*37*). Moreover, heifers exposed to 16 hours of light per day eat more and are more efficient in converting feed into body mass than heifers given less than 12 hours of light per day (*37*). Even when feed intakes are restricted, increased rates of weight gain persist in animals exposed daily to 16 hours of light (*33*). The extra mass is not fat; the percentage of protein in carcasses of heifers exposed to 16 hours of light was higher than that in controls. To date, castrated male cattle (steers) exposed to long-day photoperiods have not grown faster than steers under short-day photoperiods (*38*), suggesting that the gonads are essential to photoperiod-induced growth in cattle.

Castrated male sheep (*39*) as well as intact male and female sheep grow faster in response to a 16-hour period of daily light than controls given 8 hours of light daily (*40, 41*). It is not essential to supply 16 hours of continuous light each day. For example, a photoperiod of 7 hours of light, 9 hours of darkness, 1 hour of light, and 7 hours of darkness was as effective as 16 hours of light and 8 hours of darkness in stimulating growth (*42*). These data support the hypothesis that physiological responses occur if light coincides with an endogenous daily rhythm in photosensitivity (*43*).

One report suggests that a portion of the increased daily weight gains in sheep in response to long days is associated with increased filling of the gut (*44*). In several other studies the longer periods of daily illumination led to increased carcass weight at slaughter (*40–42*). Fat and protein percentages in sheep carcasses are not markedly affected by photoperiod (*41, 42, 44*). The growth response to long days may be limited to ruminants. Neither male nor female swine grow faster in response to various photoperiods (*34, 35*).

The mechanism whereby photoperiod controls growth in sheep and cattle has not been elucidated, but the anterior pituitary hormone prolactin could be involved. Of all the hormones measured in cattle, prolactin is most affected by a changing photoperiod. Gradually reducing the daily light exposure from 16 to 8 hours decreases prolactin concentrations from 57 to 8 nanograms per milliliter of serum (*45*); gradually increasing the light exposure from 8 to 16 hours increases prolactin secretion. Similarly, in sheep 16-hour days increase serum prolactin and 8-hour days reduce it (*46*). After an abrupt alteration in photoperiod, the first detectable change in prolactin secretion does not occur for at least 1 week, and to achieve maximal concentrations requires 5 to 8 weeks (*47*). Induction of increased growth rates also requires several weeks (*36, 37*).

Prolactin is anabolic in some systems (*48*). When sheep are immunized against prolactin, thereby minimizing prolactin concentrations in the blood, body growth rates are decreased (*49*); in contrast, suppression of prolactin secretion with an ergot alkaloid fails to affect body growth in ram lambs (*50*). The effects of suppressing prolactin with ergots on the growth of animals exposed to long or short days has not been reported. Pinealectomy abolishes photoperiod-induced changes in prolactin secretion in lambs and blocks the effect of photoperiod on weight gains (*51*).

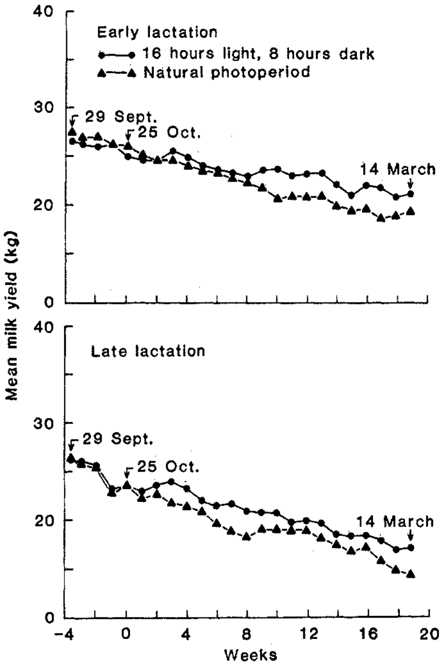

Fig. 4. Milk production by Holstein cows. Between 29 September and 24 October 24 cows in early lactation (37 to 74 days postpartum) and 18 cows in late lactation (94 to 204 days postpartum) were exposed to natural photoperiods of approximately 12 hours of light per day and standardized dietary conditions. From 25 October to 14 March, 12 cows in early lactation and 9 in late lactation were exposed to a natural photoperiod (9 to 12 hours of light daily) and 12 cows in early lactation and 9 cows in late lactation were exposed daily to fluorescent lighting between 0300 and 1900 hours, superimposed on a natural photoperiod. Pooled standard errors of average milk yields of cows in early and late lactation were 1.1 and 1.5 kilograms, respectively. Mean daily milk yields under the 16-hour photoperiod for cows in early and late lactation were 6 and 7 percent greater, respectively, than yields for cows exposed only to the natural photoperiod. [From Peters *et al.* (*57*)]

There is evidence that prolactin may not mediate photoperiod-induced increments in growth. For example, low ambient temperatures block the ability of 16-hour periods of daily light to increase prolactin concentrations in serum of heifers (*52*), yet the increased weight gains associated with 16-hour days persist in Michigan in winter (*36, 37*). Caution in the interpretation of these data is warranted because neither ambient temperature nor prolactin were monitored continuously. Unrecorded sporadic increases in temperature could have allowed secretion of prolactin to increase temporarily in response to the 16-hour periods of light. Whether temporary increases in secretion of prolactin are sufficient to permit increased body growth has not been determined. It was reported recently that photoperiod affects patterns of feed intake in lambs (*42*). This may explain a portion of the change in growth rates and feed intakes in response to photoperiod, but it does not clarify the nature of the endogenous signal.

Chickens are diurnal species; the greatest feeding activity occurs soon after dawn and about 5 hours before darkness (*53*). The rhythm of eating activity influences the nutritional and physiological well-being of chickens. Continuous light results in greater weight gains than light-dark sequences or total darkness (*54*). Under continuous light, feed intake occurs uniformly around the clock, supplying a constant flow of nutrients to the tissues (*55*). Use of low intensities (0.8 to 1.5 lux) of continuous light results in faster growth in broilers than high intensities (6.5 to 21.5 lux) of continuous light. Wavelength influences growth of chicks, with a continuous green light (545 nanometers) giving the fastest growth (*20*). Low-intensity or red lights are commonly used to reduce cannibalism in poultry production.

Milk Yields

When cows are exposed to 16 hours of light per day during autumn and winter, milk yields increase 6 to 13 percent (*36, 56*). The increases in production are similar for cows in early and late lactation (Fig. 4). Photoperiod does not affect the percentage of fat in milk (*57*). Lactating cows subjected to increased lighting consume sufficient amounts of extra feed to account for the increased milk yield (*57*).

It has been speculated that prolactin is involved in the mediation of milk yield responses to photoperiod (*36*). Indeed, prolactin is essential for maximum synthesis of milk in the immediate postpartum period and is needed for several key biochemical steps involved in milk synthesis (*58*). Exposure of lactating cows to 16 hours of light per day stimulates prolactin secretion except when temperatures approach freezing (*57*). Since milk yields do not decrease during exposure to cold (*57*), it has been impossible to directly associate increased secretion of prolactin with increased milk production in response to 16-hour days.

Conclusion

Manipulation of the daily light-dark cycle markedly affects reproduction, growth, and lactation in domestic food animals. Shortening the duration of daily light induces onset of estrus and ovulation in seasonally breeding species such as sheep, whereas a progressively increasing duration of daily light is required for maximal ovulation rates in chickens. Ovulation in nonseasonally breeding species such as cattle is not affected by photoperiod. Shortening the duration of light exposure in sheep and lengthening the duration of light exposure in cattle hasten the onset of puberty. Many of the effects of photoperiod on reproduction are mediated through interactions of estradiol and LH secretion in sheep or progesterone and LH secretion in chickens. Maximal growth rates in chickens are achieved with continuous light, whereas in sheep and cattle exposure to 16 hours of light daily is optimal for stimulating body growth rates and milk yields. Of all the hormones measured in sheep and cattle, prolactin is the most responsive to changes in photoperiod, but it has not yet been shown whether this hormone is involved directly in the mechanism whereby photoperiod affects growth rates and milk yields. We conclude that control of light is a promising method for increasing food production from domestic animals.

References and Notes

1. N. T. M. Yeates, *J. Agric. Sci.* **39**, 1 (1949).
2. F. J. Karsch and D. L. Foster, in *Environmental Factors in Mammal Reproduction*, D. Gilmore and B. Cook, Eds. (Macmillan, London, 1981), p. 30.
3. B. D. Schanbacher, *J. Anim. Sci.* **50**, 293 (1980).
4. M. A. Brunner, W. Hansel, D. E. Hogue, *ibid.* **23**, 32 (1964).
5. G. J. Mears, G. R. Van Petten, W. H. Harris, J. U. Bell, F. L. Lorscheider, *J. Reprod. Fertil.* **57**, 461 (1979).
6. D. L. Foster, *Biol. Reprod.* **25**, 85 (1981).
7. B. D. Schanbacher and D. D. Lunstra, *J. Anim. Sci.* **43**, 644 (1976).
8. J. Pelletier and R. Ortavant, *Acta Endocrinol. (Copenhagen)* **78**, 435 (1975); G. A. Lincoln, M. J. Peet, R. A. Cunningham *J. Endocrinol.* **72**, 337 (1977); B. D. Schanbacher and J. J. Ford, *Biol. Reprod.* **20**, 719 (1979).
9. D. Amir and R. Volcani, *J. Agric. Sci.* **64**, 115 (1965); J. Lees, *Nature (London)* **207**, 221 (1965).
10. G. A. Lincoln and W. Davidson, *J. Reprod. Fertil.* **49**, 267 (1977).
11. C. M. Howles, G. M. Webster, N. B. Haynes, *ibid.* **60**, 437 (1980).
12. J. Pelletier and R. Ortavant, *Acta Endocrinol. (Copenhagen)* **78**, 442 (1975).
13. G. K. Barrel and K. R. Lapwood, *Neuroendocrinology* **27**, 216 (1978); G. A. Lincoln, *J. Endocrinol.* **82**, 135 (1979).
14. R. H. Dutt and E. C. Simpson, *J. Anim. Sci.* **16**, 136 (1957).
15. B. D. Schanbacher, *ibid.* **49**, 927 (1979).
16. T. R. Morris, in *Animal Reproduction* (Beltsville Symposia in Agricultural Research, 3), H. W. Hawk, Ed. (Allanheld, Osmum, Montclair, N.J., 1979), p. 307.
17. J. Benoit, *Ann. N.Y. Acad. Sci.* **117**, 204 (1964).
18. D. C. Foss, L. B. Carew, E. L. Arnold, *Poult. Sci.* **51**, 1922 (1972).
19. J. Oliver and J. D. Bayle, *J. Physiol. (Paris)* **72**, 627 (1976).
20. M. Menaker, *Biol. Reprod.* **4**, 295 (1971).
21. W. H. Foster, in *Egg Formation and Production*, B. M. Freeman and P. E. Lake, Eds. (British Poultry Science, Edinburgh, Scotland, 1972), p. 161.
22. T. R. Morris and V. M. Owen, paper presented at the 13th World Poultry Congress, Kiev, Soviet Union (1966).
23. P. C. Lague, A. van Tienhoven, F. J. Cunningham, *Biol. Reprod.* **12**, 590 (1975); B. K. Follett and D. T. Davies, in *Animal Reproduction* (Beltsville Symposia in Agricultural Research, 3), H. W. Hawk, Ed. (Allanheld, Osmum, Montclair, N.J., 1979), p. 323.
24. B. M. Bhatti and T. Morris, *Br. Poult. Sci.* **19**, 333 (1978).
25. ———, *ibid.*, p. 365.
26. T. Morris and B. M. Bhatti, *ibid.*, p. 341.
27. T. Morris, *ibid.*, p. 207.
28. H. A. Tucker and W. D. Oxender, *Prog. Reprod. Biol.* **5**, 155 (1980).
29. W. J. Sweetman, *J. Dairy Sci.* **33**, 391 (1950).
30. R. A. Rzepkowski *et al.*, *J. Anim. Sci.*, in press.
31. R. A. Bourne and H. A. Tucker, *Endocrinology* **97**, 473 (1975).
32. J. H. B. Roy, C. M. Gillies, M. W. Perfitt, I. J. F. Stobo, *Anim. Prod.* **31**, 13 (1980).
33. D. Petitclerc, L. T. Chapin, R. S. Emery, H. A. Tucker, paper presented at the 73rd Annual Meeting of the American Society of Animal Science, Raleigh, N.C. (1981); H. A. Tucker, unpublished observations.
34. B. N. Ntunde, R. R. Hacker, G. J. King, *J. Anim. Sci.* **48**, 1401 (1979).
35. J. P. Mahone, T. Berger, E. D. Clegg, W. L. Singleton, *ibid.*, p. 1159; T. Berger, J. P. Mahone, G. S. Svoboda, K. W. Metz, E. D. Clegg, *ibid.* **51**, 672 (1980).
36. R. R. Peters, L. T. Chapin, K. B. Leining, H. A. Tucker, *Science* **199**, 911 (1978).
37. R. R. Peters, L. T. Chapin, R. S. Emery, H. A. Tucker, *J. Anim. Sci.* **51**, 1148 (1980).
38. J. F. Roche and M. P. Boland, *Ir. J. Agric. Res.* **19**, 85 (1980).
39. J. M. Forbes, P. M. Driver, A. A. El Shahat, T. G. Boag, C. G. Scanes, *J. Endocrinol.* **64**, 549 (1975).
40. J. M. Forbes, A. A. El Shahat, R. Jones, J. G. S. Duncan, T. G. Boaz, *Anim. Prod.* **29**, 33 (1979).
41. B. D. Schanbacher and J. D. Crouse, *J. Anim. Sci.* **51**, 943 (1980).
42. ———, *Am. J. Physiol.* **241**, E1 (1981).
43. E. Bunning, *Cold Spring Harbor Symp. Quant. Biol.* **25**, 249 (1960).
44. J. M. Forbes, W. R. Brown, A. G. M. Al-Banna, R. Jones, *Anim. Prod.* **32**, 23 (1981).
45. R. A. Bourne and H. A. Tucker, *Endocrinology* **97**, 473 (1975).
46. J. P. Ravault and R. Ortavant, *Ann. Biol. Anim. Biochim. Biophys.* **17**, 459 (1977).
47. K. B. Leining, R. A. Bourne, H. A. Tucker, *Endocrinology* **104**, 289 (1979).
48. C. S. Nicoll, *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **39**, 2563 (1980).
49. D. L. Ohlson, L. J. Spicer, S. L. Davis, *J. Anim. Sci.* **52**, 1350 (1981).
50. J. P. Ravault, M. Courot, D. Garnier, J. Pelletier, M. Terqui, *Biol. Reprod.* **17**, 192 (1977).
51. W. B. Brown and J. M. Forbes, *J. Endocrinol.* **84**, 91 (1980).
52. R. R. Peters and H. A. Tucker, *Endocrinology* **103**, 229 (1978).
53. P. D. Ballard and H. V. Biellier, *Poult. Sci.* **48**, 1781 (1969).
54. W. D. Weaver and P. B. Siegel, *ibid.* **47**, 1148 (1968).
55. R. L. Squibb and G. H. Collier, *ibid.* **58**, 641 (1979).
56. N. Bodurov, *Vet. Med. Nauki* **16**, 58 (1979).
57. R. R. Peters, L. T. Chapin, R. S. Emery, H. A. Tucker, *J. Dairy Sci.* **64**, 1671 (1981).
58. R. M. Akers, D. E. Bauman, A. V. Capuco, G. T. Goodman, H. A. Tucker, *Endocrinology* **109**, 23 (1981).
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