Hormones and Behavior **42,** 13–20 (2002)
doi:10.1006/hbeh.2002.1797

Short Days and Exogenous Melatonin Increase Aggression of Male Syrian Hamsters (*Mesocricetus auratus*)

Aaron M. Jasnow,* Kim L. Huhman,* Timothy J. Bartness,*,† and Gregory E. Demas‡,[1]

*Center for Behavioral Neuroscience, *Department of Psychology and †Department of Biology, Georgia State University, Atlanta, Georgia 30303; and ‡Center for the Integrative Study of Animal Behavior, Department of Biology, Indiana University, Bloomington, Indiana 47405*

Received July 20, 2001; revised September 24, 2001; accepted December 14, 2001

Many nontropical rodent species rely on photoperiod as a primary cue to coordinate seasonally appropriate changes in physiology and behavior. Among these changes, some species of rodents demonstrate increased aggression in short, "winter-like" compared with long "summer-like" day lengths. The precise neuroendocrine mechanisms mediating changes in aggression, however, remain largely unknown. The goal of the present study was to examine the effects of photoperiod and exogenous melatonin on resident-intruder aggression in male Syrian hamsters (*Mesocricetus auratus*). In Experiment 1, male Syrian hamsters were housed in long (LD 14:10) or short (LD 10:14) days for 10 weeks. In Experiment 2, hamsters were housed in long days and half of the animals were given daily subcutaneous melatonin injections (15 μg/day in 0.1 ml saline) 2 h before lights out for 10 consecutive days to simulate a short-day pattern of melatonin secretion, while the remaining animals received injections of the vehicle alone. Animals in both experiments were then tested using a resident-intruder model of aggression and the number of attacks, duration of attacks, and latency to initial attack were recorded. In Experiment 1, short-day hamsters underwent gonadal regression and displayed increased aggression compared with long-day animals. In Experiment 2, melatonin treatment also increased aggression compared with control hamsters without affecting circulating testosterone. Collectively, the results of the present study demonstrate that exposure to short days or short day-like patterns of melatonin increase aggression in male Syrian hamsters. In addition, these results suggest that photoperiodic changes in aggression provide an important, ecologically relevant model with which to study the neuroendocrine mechanisms underlying aggression in rodents.

Key Words: **seasonal; photoperiod; pineal; agonistic; testosterone; dominance.**

Individuals of many nontropical rodent species undergo a variety of physiological and behavioral responses across the seasons of the year (reviewed in Bronson and Heideman, 1994; Nelson, Badura, and Goldman, 1990). Although a variety of environmental factors (e.g., ambient temperature, humidity, food availability) also fluctuate on a seasonal basis, photoperiod (day length) appears to be the primary environmental cue used by most nontropical mammalian species to coordinate behavioral and physiological responses with the optimal time of year (Bronson, 1989). Animals maintained in short "winter-like" days (i.e., <12 h of light / day) undergo a variety of physiological and behavioral changes, including regression of the reproductive system, as well as changes in body mass, pelage, thermoregulation, and general activity (reviewed in Bartness, Bradley, Hastings, Bittman, and Goldman, 1993). These physiological and behavioral changes in response to changes in photoperiod are mediated by a multisynaptic pathway that conveys photic information received by the retina to the pineal gland and results in changes in the pattern of secretion of the pineal indolamine hormone, melatonin. Interruption of this pathway at any point or removal of the pineal gland blocks physiological and behavioral responses to short day lengths (Elliot and Goldman, 1981; Tamarkin, Baird, and Almeida, 1985).

[1] To whom correspondence should be addressed at Department of Biology, Indiana University, 1001 East 3rd Street, Bloomington, IN 47405. Fax: (812) 855-6705. E-mail: gdemas@bio.indiana.edu.

0018-506X/02 $35.00

The majority of studies examining photoperiodic changes in mammalian physiology and behavior have focused primarily on changes in reproduction and energy balance (reviewed in Bronson and Heideman, 1994; Bartness and Wade, 1985). Several studies, however, have demonstrated photoperiodic changes in aggressive behavior in both male and female rodents (Badura and Nunez, 1989; Fleming, Philips, Rydall, and Levesque, 1988; Garrett and Campbell, 1980; Jasnow, Huhman, Bartness, and Demas, 2000). For example, male Syrian hamsters maintained in short days for 8 weeks undergo gonadal regression and subsequent increases in aggression compared with long-day hamsters, despite basal serum concentrations of testosterone (Garrett and Campbell, 1980). Typically, serum testosterone is reduced to ~0.5 ng/ml under short-day, nonbreeding conditions (Turek, Elliot, Alvis, and Menaker, 1975). Interestingly, prolonged maintenance in short days (>15 weeks) triggers gonadal recrudescence and the short-day increases in aggressive behavior largely disappear, returning to long-day levels of aggression by 21 weeks (Garrett and Campbell, 1980). This return to long-day levels of aggression is likely due the development of an insensitivity of target tissue to melatonin, given that the meltonin pattern, per se, does not change after gonadal recrudescence has occurred. Consistent with this idea, changes in melatonin sensitivity apparently explain the reproductive photoperiodic refractoriness that develops after prolonged exposure to short days (Freeman and Zucker, 2001). It is important, however, to note that the Garrett and Campbell study described above used a short-day photoperiod of light:dark (LD) 6:18. This photoperiod is an extremely short photoperiod and one that Syrian hamsters would not experience in their natural environment. It remains unknown how male Syrian hamsters would respond to a more natural decrease in photoperiod. Recently, a similar pattern of aggression was reported in Siberian hamsters (*Phodopus sungorus*), using more natural photoperiodic manipulations. Specifically, male Siberian hamsters housed in short days (LD 8:16) for 10 weeks were more aggressive than long-day (LD 16:8) housed hamsters; after 20 weeks of short days, however, gonadal recrudescence occurred and aggression was reduced to long-day levels (Jasnow *et al.*, 2000). Surprisingly, treatment of short-day hamsters with exogenous testosterone, comparable to long-day titers, reduced aggression to that of long-day animals (Jasnow *et al.*, 2000). Similar patterns of aggression have been observed in female Syrian hamsters (Fleming *et al.*, 1988). In addition, pinealectomy eliminates the short-day increase in aggression in female hamsters, whereas exogenous melatonin treatment augments aggression in long-day animals (Fleming *et al.*, 1988), suggesting an important role for melatonin in mediating photoperiodic changes in aggression in female hamsters.

In support of the hypothesis that pineal melatonin mediates aggression in some rodent species, male house mice treated daily with exogenous melatonin for 5 consecutive days display significant increases in territorial aggression compared with control mice treated with saline (Paterson and Vickers, 1981). In addition, pinealectomy suppresses territorial aggression in mice (Paterson and Vickers, 1981), consistent with the results reported for female Syrian hamsters. These latter results are particularly intriguing given that house mice have traditionally been assumed to be photoperiodically nonresponsive (Nelson, 1990). Despite the results discussed above, the precise hormonal mechanisms underlying short-day increases in aggression in rodents remain largely unknown. Although melatonin has been implicated as a possible mediator of short-day increases in aggression, at least in female Syrian hamsters, the precise role of melatonin in mediating photoperiodic changes in aggression in male rodents is unknown. The goal of the present study was to evaluate the effects of photoperiod and exogenous melatonin on aggression in male Syrian hamsters. In Experiment 1 we tested the hypothesis that Syrian hamsters housed in short days show increased aggression compared with long-day animals. In Experiment 2, we tested the hypothesis that short-day patterns of melatonin will increase aggression independent of changes in gonadal steroid hormones. Specifically, long-day animals were treated with exogenous melatonin to mimic short-day patterns of melatonin secretion for a period not sufficient to trigger gonadal regression.

MATERIALS AND METHODS

Animals and Housing Conditions

Adult (>60 days of age) Syrian hamsters (*Mesocricetus auratus*) were obtained from a commercial supplier (Charles River Laboratories) and were housed individually in polypropylene cages (40 × 20 × 20 cm) in colony rooms with a 24 h LD 14:10 cycle (lights off 1200 h EST). Temperature was kept constant at 20 ± 2°C and relative humidity was maintained at 50 ± 5. Food (Purina Rat Chow) and tap water were available

ad libitum throughout the experiment. Additional animals were used as nonaggressive intruders during behavioral testing and were group-housed (5 animals per cage) to keep aggression to a minimum (Brain, 1972). These animals were approximately the same weight as long-day experimental animals (~150 g) and were the same age as experimental animals and were housed under long-day conditions. All animals were treated in accordance with the Georgia State University Institutional Animal Care and Use Committee.

Experiment 1: Effects of Photoperiod on Aggression

Twenty-four hamsters were used in Experiment 1. At the start of the experiment, all animals were housed in a colony room with a long-day (LD 14:10) photoperiod. After 1 week, a random subset of animals (n = 14) was selected and transferred to a colony room with a short-day (LD 10:14) photoperiod. The remaining animals (n = 10) were maintained in long days for the duration of the experiment. Mean body masses was determined for each group after the experimental assignment to ensure that the groups did not differ in body mass prior to the experiment. Animals were kept in their respective photoperiod for 10 weeks. At this time, animals were tested using a resident–intruder model of aggression by introducing a nonaggressive intruder into the home cage of an experimental animal for 5 min. Behavioral testing was conducted during the first 2 h of the dark phase to control for circadian rhythmicity of behavior. Intruder males were dye-marked on the tail for the purposes of identification and each animal was used only once during the test period to control for any experience-related changes in the behavior of these animals. Attack latency, number of attacks, and the total duration of attacks were recorded by two observers blind to experimental conditions. An "attack" is a combination of chasing and biting, and was operationally defined as a rapid approach of the resident animal toward an intruder in either a sideways or upright offensive posture in an attempt to bite the intruder (Huhman, Bunnel, Mougey, and Meyerhoff, 1990). After behavioral testing, animals were killed by lethal injection of sodium pentobarbital. Paired testes were removed, cleaned of fat and connective tissue, and weighed to the nearest 0.1 mg.

Experiment 2: Effects of Exogenous Melatonin on Aggression

Twenty-five hamsters were used in Experiment 2. All animals were maintained in long days as described in Experiment 1 for the duration of the experiment. A subset of the animals (n = 13) were randomly selected and received daily subcutaneous injections (0.1 ml/animal) of melatonin [15 μg/day (Sigma Chemical, Saint Louis, MO) dissolved in a 1:10 ethanol:saline solution (Stetson and Tay, 1985) for 10 days, while the remaining animals (n = 12) received injections of the vehicle alone. All injections were administered 2 h before lights out. The melatonin injection protocol that was used in Experiment 2 was chosen for several reasons. First, the precise timing of melatonin injections (i.e., 2 h before lights off) has been shown to extend the normal long-day pattern of endogenous melatonin secretion; the resulting extended pattern of melatonin is interpreted by hamsters as a short day (Stetson and Tay, 1985). Thus, the pattern of melatonin generated in experimental animals, rather than being artificial or supraphysiological, accurately reflects typical short-day patterns. Second, melatonin treatment was administered on a relatively short-term basis (i.e., 10 days); this period was chosen because it is not sufficiently prolonged to trigger gonadal regression and unlike maintenance in short days, leaves gonadal steroid concentrations unaffected. This allows the effects of exogenous melatonin to be tested directly, without subsequent changes in steroid hormones. Following 10 days of injections, resident–intruder aggression was assessed as in Experiment 1. Reproductive condition was determined by weighing the testes at necropsy as described in Experiment 1 and measuring serum testosterone concentrations as described below.

Blood Collection and Testosterone Assay

Blood samples were not taken from animals in Experiment 1 because the blood sampling procedure could be perceived as stressful and thus affect subsequent behavior. In addition, many previous studies have confirmed the significant reduction in serum testosterone concentrations in short-day rodents. In Experiment 2, animals were brought into the surgery room individually 24 h before testing (between 1000 and 1200 h EST), lightly anesthetized with methoxyflurane vapors (Metofane, Mundelein, IL), blood samples (~500 μl) were drawn from the retroorbital sinus, and animals were returned to their respective housing conditions. Blood samples were allowed to clot for 1 h,

the clots were removed, and the samples were centrifuged (at 4°C) for 60 min at 2500 rpm. Serum aliquots were aspirated and stored in sealable polypropylene microcentrifuge tubes at −80°C until assayed. Blood serum testosterone concentrations were determined in a single radioimmunoassay (RIA) from a commercially prepared kit (Diagnostic Systems Laboratories, Webster, TX). This assay was validated for use with Syrian hamsters by the Neuroendocrinology Core Facility at the Yerkes Regional Primate Center. The antiserum used was highly specific for testosterone; cross-reactivity with other steroid hormones was <0.01%. Intraassay variability was <10% for all samples.

Statistical Analyses

The data from all dependent measures from Experiments 1 and 2 were analyzed using separate independent, two-tailed Student *t*-tests (Sigma Stat, Jandel Scientific, San Rafael, CA). Differences between group means were considered statistically significant if $P < 0.05$. In Experiment 1, one short-day hamster was reproductively nonresponsive (i.e., paired testes mass was >2.5 g) and, thus, this animal was removed from the subsequent data analyses.

RESULTS

Experiment 1: Short-Day Hamsters Increased Aggression Compared with Long-Day Animals

In Experiment 1, hamsters maintained in short days had significantly increased body mass (143.85 ± 4.86 g in long days and 169.85 ± 4.50 g in short days) ($t(22) = 3.77$, $P < 0.05$) and significantly reduced paired testes masses (3.76 ± 0.08 g in long days and 0.37 ± 0.04 g in short days) ($t(22) = 3.39$, $P < 0.05$) compared with long-day animals.

Short-day hamsters displayed an increased number of attacks compared with long-day housed animals ($t(22) = 2.69$, $P < 0.05$) (Fig. 1a). Short-day hamsters also displayed an increased duration of attacks ($t(22) = 2.08$, $P < 0.05$) (Fig. 1b). Short- and long-day hamsters did not differ in the latency to the initial attack ($P > 0.05$) (Fig. 1c).

Experiment 2: Melatonin-Treated Hamsters Increased Aggression Compared with Control Animals

In Experiment 2, there were no significant differences between melatonin- and vehicle-treated hamsters in either body mass (122.62 ± 1.65 g in long days and 128.17 ± 2.58 g in short days), paired testes mass (3.21 ± 0.08 g in long days and 3.27 ± 0.08 g in short days), or serum testosterone concentrations (2.78 ± 0.35 ng/ml in long days and 3.02 ± 0.25 ng/ml in short days) ($P > 0.05$ in all cases).

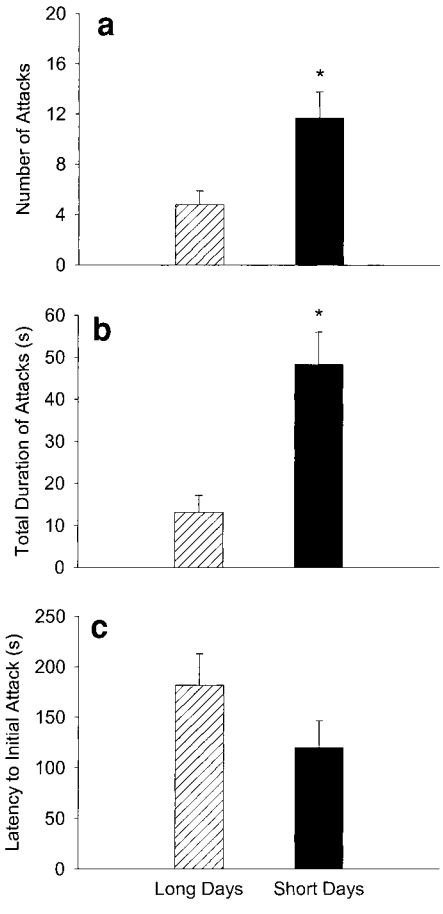

FIG. 1. Mean (± SEM) number of attacks (a), total duration of attacks (b), and latency to initial attack (c) of hamsters housed in either long ($n = 10$) or short ($n = 13$) days for 10 weeks. *Significant differences between pairwise means, $P < 0.05$.

Melatonin-treated hamsters displayed a significantly larger number of attacks ($t(24) = 2.59$, $P < 0.05$) (Fig. 2a) and a shorter latency to initial attack compared with vehicle-treated animals ($t(24) = 2.79$, $P < 0.05$) (Fig. 2c). There was no significant difference in the total duration of attacks between melatonin-treated and control animals ($P > 0.05$) (Fig. 2b).

DISCUSSION

The results of the present study demonstrate that exogenous melatonin increases aggression in male

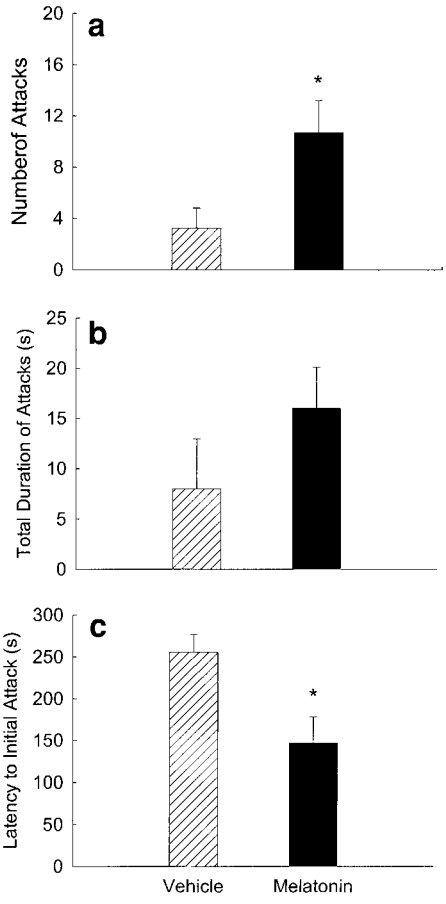

FIG. 2. Mean (± SEM) number of attacks (a), total duration of attacks (b), and latency to initial attack (c) of hamsters treated with melatonin ($n = 13$) or vehicle ($n = 12$). *Significant differences between pairwise means, $P < 0.05$.

Syrian hamsters and that increased aggression is not correlated with changes in testosterone because testosterone levels did not differ between melatonin-treated and vehicle-treated animals. The increased aggression observed in the present study also was not due to differences in body mass between melatonin- and vehicle-treated animals. In addition, these results confirm a previous study demonstrating short-day increases in aggression in male Syrian hamsters. In Experiment 1, short-day hamsters displayed gonadal regression and presumably reduced circulating testosterone concentrations, although testosterone was not measured directly. In addition, short-day hamsters displayed increased aggression compared with long-day animals. In Experiment 2, long-day animals treated with exogenous melatonin to mimic short-day patterns of the hormone also displayed increased aggression compared with vehicle-treated animals. This effect occurred without changes in gonad size or circulating testosterone concentrations. Taken together, the results of the present study suggest that short-day increases in aggression may be due, in part, to increased melatonin without concomitant changes in circulating testosterone concentrations.

These results are consistent with previously published studies demonstrating photoperiodic changes in aggression in male Syrian and Siberian hamsters (Garrett and Campbell, 1980; Jasnow *et al.*, 2000), as well as previous reports of increased aggression in short days in female hamsters (Fleming *et al.*, 1988; Badura and Nunez, 1989). Interestingly, Syrian hamsters have a reversed sexual dimorphism in aggressive behavior relative to other commonly studied rodent species (e.g., rats and mice), with females typically displaying significantly more aggression than their male counterparts except when estrogen concentrations are elevated (e.g., during estrus) (Ciacco, Lisk, and Rueter, 1979; Marques and Valenstein, 1977). Consistent with this pattern, female Syrian hamsters maintained in short days (which reduce circulating estradiol concentrations) display significantly more aggression compared with long-day housed animals (Badura and Nunez, 1989; Fleming *et al.*, 1988). Interestingly, ovariectomy of long-day animals does not result in short-day levels of aggression, suggesting that photoperiodic effects on aggression are not mediated by gonadal steroid hormones in female Syrian hamsters (Fleming *et al.*, 1988). In contrast, pinealectomy eliminates the short-day increase in aggression in these animals, whereas exogenous melatonin treatment augments aggression in long-day animals (Fleming *et al.*, 1988). Taken together, the present data and previous data suggest an important role for melatonin in mediating photoperiodic changes in aggression in male and female hamsters.

The present results are also in accordance with findings of increased aggression during the nonbreeding season of certain avian species when testosterone levels are basal (e.g., Logan and Wingfield, 1990; Wingfield, 1994). Importantly, these studies were conducted in the field, further supporting the ecological relevance of the present findings. It is important to note that, although the present data suggest that changes in endogenous melatonin may play a role in short-day changes in aggression, this hypothesis was not tested directly in the present study. In principle, a more direct test of this hypothesis could be conducted by disrupting the actions of melatonin in short-day housed hamsters and determining if short-day increases in aggression are maintained. Unfortunately,

this approach is not possible because melatonin is the primary signal of short days; disruption of this signal, therefore, renders the animals physiologically "blind" to short days.

One possible explanation for the present results is that maintenance in short days (and subsequent increases in the duration of melatonin secretion) leads to changes in the hypothalamic–pituitary–adrenal (HPA) axis and changes in the HPA axis subsequently mediate changes in aggression. In support of this hypothesis, changes in both the size and function of the adrenal gland are associated with changes in aggression. For example, house mice treated with melatonin display increased territorial aggression but decreased adrenal masses compared with saline-treated animals (Paterson and Vickers, 1981). The increases in aggression seen in short days, however, could be blocked by either adrenalectomy or acute reductions in glucocorticoids via injections of aminoglutethimide, a chemical that reduces glucocorticoid secretion by disrupting ACTH release (Paterson and Vickers, 1981). The results of the study described above suggest that the effects of exogenous melatonin on aggression are mediated by its effects on the adrenal gland and, specifically, adrenal cortical glucocorticoids. It is important, however, to note that the effect of glucocorticoids on aggression reported in this study was in response to aggression elicited by exogenous melatonin treatment rather than short days. Although the results of the present study in Syrian hamsters suggests that short-day increases in aggression are mediated by melatonin, the precise role of adrenal hormones in mediating photoperiodic changes in aggression has not been tested.

Another possible mechanism underlying increased aggression in short-day or melatonin-treated hamsters is via changes in the hormone vasopressin released from the posterior pituitary. Vasopressin has been implicated in mediating aggression in several rodent species (Winslow, Hastings, Carter, Harbaugh, and Insel, 1993; Goodson, 1998; Stribley and Carter, 1999; Bester-Meredith, Young, and Marler, 1999). For example, injections of vasopressin into the anterior hypothalamus stimulate aggression, while vasopressin antagonists injected into the same brain region inhibit aggression in Syrian hamsters (Ferris and Potegal, 1989; Potegal and Ferris, 1989; Ferris, Melloni, Koppel, Perry, Fuller, and Delville, 1997). The effects of photoperiod on vasopressin in the central nervous system, however, are less clear. Some studies have reported increased vasopressin immunoreactivity in short-day housed animals (Juszczak, Luciano, Bozena, Steger, Fadden, and Bartke, 1997; Bittman, Jetton, Villaba, and DeVries 1996; Duncan, Cheng, and Heller, 1995), while others have reported no differences or decreases in vasopressin immunoreactivity in hamsters exposed to short days (Albers, Rowland, and Ferris, 1991; Duncan, 1998). Siberian and Syrian hamsters, however, display some differences in the distribution of vasopressin neurons and these differences may explain some of the differences described above (Shi and Bartness, 2000).

The present results demonstrating increased aggression in melatonin-treated hamsters compared with vehicle-treated control animals are particularly intriguing given that melatonin has been shown to exert a tranquilizing or sedative effect in some species (Romijn, 1978), and this hormone is administered clinically to induce sleep (Arendt, 1995). One interesting hypothesis is that melatonin exerts differential physiological and behavioral effects in diurnal versus nocturnal species. For example, nocturnal animals (e.g., Syrian hamsters) are more active during the dark phase (i.e., night) when circulating melatonin concentrations are at their peak, compared with the light phase (i.e., day) when melatonin release is at its daily nadir. In addition, overall activity of nocturnal rodents is increased in short compared with long days (Hastings, Walker, and Herbert, 1987). Thus, increased aggression in short-day Syrian hamsters appears to be consistent with the increase in general activity in nocturnal rodents. If the hypothesis that melatonin exerts differential effects on nocturnal compared with diurnal animals is correct, then aggression should be reduced in diurnal rodent species (e.g., Nile grass rats [*Arvicanthus niloticus*], degus [*Octodon degus*]) treated with exogenous melatonin. Although this idea is intriguing, further research is needed to test it.

The adaptive significance of short-day increases in aggression was not examined in the present study. Increased aggression during the short days of winter, however, likely confers an evolutionary advantage to animals at a time when food availability is low and competition for limited resources is relatively high. Specifically, dominant animals may be more successful in obtaining or protecting limited resources compared with subordinate animals and, thus, may have higher reproductive success relative to subordinate animals. Regardless of the adaptive significance of photoperiodic changes in aggression, the present results suggest that short-day increases in aggressive behavior are due, at least in part, to increases in the duration of melatonin secretion in short compared with long days. In addition, the short-day increases in

aggression, unlike the more commonly studied reproductive aggression, appear to be independent of circulating gonadal steroid hormones (i.e., testosterone). These results suggest that the traditional idea that testosterone causes aggression is too simplistic and that a variety of neuroendocrine factors may interact to regulate aggression in mammals. Collectively, these results suggest that photoperiodic changes in aggression provide an important, ecologically relevant model with which to study the neuroendocrine mechanisms underlying aggressive behavior in rodents.

ACKNOWLEDGMENTS

The authors acknowledge Debbie Drazen and Stephanie Plunkett for providing helpful comments on the manuscript, David Marshall and Charlie Smeeten for expert animal care, and Susie Lackey at the Neuroendocrine Core Facility of Yerkes Regional Primate Center for running the testosterone RIA. This research was supported by USPHS Grants NS 34896 to K.L.H., DK 35254 to T.J.B., and NS 10596 to G.E.D. This material is based on work supported, in part, by the STC program of the NSF under agreement IBN-9876754.

REFERENCES

Albers, H. E., Rowland, C. M., and Ferris, C. F. (1991). Arginine–vasopressin immunoreactivity is not altered by photoperiod or gonadal hormones in the Syrian hamster (*Mesocricetus auratus*). *Brain Res.* **539,** 137–142.

Arendt, J. (1995). *Melatonin and the Mammalian Pineal Gland.* Chapman & Hall, New York.

Badura, L. L., and Nunez, A. A. (1989). Photoperiodic modulation of sexual and aggressive behavior in female golden hamsters (*Mesocricetus auratus*): Role of the pineal gland. *Horm. Behav.* **23,** 27–42.

Bartness, T. J., Bradley, J., Hastings, M. H., Bittman, E. L., and Goldman, B. D. (1993). The timed infusion paradigm for melatonin delivery: What has it taught us about the melatonin signal, its reception, and the photoperiodic control of seasonal responses? *J. Pineal Res.* **15,** 161–190.

Bartness T. J., and Wade, G. N. (1985). Photoperiodic control of seasonal body weight cycles in hamsters. *Neurosci. Biobehav. Rev.* **9,** 599–612.

Bester-Meredith, J. K., Young, L. J., and Marler, C. A. (1999). Species differences in paternal behavior and aggression in *Peromyscus* and their associations with vasopressin immunoreactivity and receptors. *Horm. Behav.* **36,** 25–38.

Bittman E. L., Jetton A. E., Villalba C., and DeVries G. J. (1996). Effects of photoperiod and androgen on pituitary function and neuropeptide staining in Siberian hamsters. *Am. J. Physiol.* **271,** R64–R72.

Brain, P. F. (1972). Effects of isolation / grouping on endocrine and fighting in male and female golden hamsters (*Mesocricetus auratus*). *Behav. Biol.* **7,** 349–357.

Bronson, F. H. (1989). *Mammalian Reproductive Biology*. Univ. of Chicago Press, Chicago.

Bronson, F. H., and Heideman, P. D. (1994). Seasonal regulation of reproduction in mammals. In E. Knobil and J. D. Neill (Eds.), *The Physiology of Reproduction*, Vol. 2, 2nd ed., pp. 541–584, Raven Press, New York.

Ciacco, L. A., Lisk, R. D., and Rueter, L. A. (1979). Prelordotic behavior in the hamster: A hormonally modulated transition from aggression to sexual receptivity. *J. Comp. Physiol. Psychol.* **93,** 771–780.

Duncan, M. J. (1998). Photoperiodic regulation of hypothalamic neuropeptide messenger RNA expression: Effect of pinealectomy and neuroanatomical location. *Brain Res. Mol. Brain Res.* **57,** 142–148.

Duncan, M. J., Cheng X., and Heller K. S. (1995). Photoperiodic exposure and time of day modulate the expression of arginine vasopressin mRNA and vasoactive intestinal peptide mRNA in the suprachiasmatic nuclei of Siberian hamsters. *Brain Res. Mol. Brain Res.* **32,** 181–186.

Elliot, A. S. and Goldman, B. D. (1981). Seasonal reproduction: Photoperiodism and biological clocks. In N.T. Adler (Ed.), *Neuroendocrinology of Reproduction: Physiology and Behavior*, pp. 377–423. Plenum, New York.

Ferris, C. F., Melloni, R. H., Jr., Koppel, G., Perry, K. W., Fuller, R. W., and Delville, Y. (1997). Vasopressin / serotonin interactions in the anterior hypothalamus control aggressive behavior in golden hamsters. *J. Neurosci.* **17,** 4331–4340.

Ferris, C. F., and Potegal, M. (1989). Vasopressin receptor blockade in the anterior hypothalamus suppresses aggression in Syrian hamsters. *Physiol. Behav.* **44,** 235–239.

Fleming, A. S., Phillips, A., Rydall, A., and Levesque, L. (1988). Effects of photoperiod, the pineal gland and the gonads on agonistic behavior in female golden hamsters (*Mesocricetus auratus*). *Physiol. Behav.* **44,** 227–234.

Freeman, D. A., and Zucker, I. (2001). Refractoriness to melatonin occurs independently at multiple brain sites in Siberian hamsters. *Proc. Natl. Acad. Sci. USA* **98,** 6447–6452.

Garrett, J. W., and Campbell, C. S. (1980). Changes in social behavior of the male golden hamster accompanying photoperiodic changes in reproduction. *Horm. Behav.* **14,** 303–319.

Goodson, J. L. (1998). Vasotocin and vasoactive intestinal polypeptide modulate aggression in a territorial songbird, the violet-eared waxbill (Estrildidae: *Uraeginthus granatina*). *Gen. Comp. Endocrinol.* **111,** 233–244.

Hastings, M. H., Walker A. P., and Herbert J. (1987). Effect of asymmetrical reductions of photoperiod on pineal melatonin, locomotor activity and gonadal condition of male Syrian hamsters. *J. Endocrinol.* **114,** 221–229.

Huhman, K. L., Bunnell, B. N., Mougey, E. H., and Meyerhoff, J. L. (1990). Effects of social conflict on POMC-derived peptides and glucocorticoids in male golden hamsters. *Physiol. Behav.* **47,** 949–956.

Jasnow, A. M., Huhman, K. L., Bartness, T. J., and Demas, G. E. (2000). Short-day increases in aggression are inversely related to circulating testosterone concentrations in male Siberian hamsters (*Phodopus sungorus*). *Horm. Behav.* **38,** 102–110.

Juszczak, M., Luciano, D., Bozena, S., Steger, R. W., Fadden, C., and Bartke, A. (1997). Neurohypophyseal vasopressin in the Syrian hamster: Response to short photoperiod, pinealectomy, melatonin treatment, or osmotic stimulation. *Brain Res. Bull.* **42,** 221–225.

Logan, C. A., and Wingfield, J. C. (1990). Autumnal territorial aggression is independent of plasma testosterone in mockingbirds. *Horm. Behav.* **24,** 568–581.

Marques, D. M., and Valenstein, E. S. (1977). Individual differences

in aggressiveness of female hamsters: Response to intact and castrated males and to females. *Anim. Behav.* **25,** 131–139.

Nelson, R. J. (1990). Photoperiodic responsiveness in house mice. *Physiol. Behav.* **48,** 403–408.

Nelson, R. J., Badura, L. L., and Goldman, B. D. (1990). Mechanisms of seasonal cycles of behavior. *Annu. Rev. Psychol.* **41,** 8–109.

Paterson, A. T., and Vickers, C. (1981). Melatonin and the adrenal cortex: Relationship to territorial aggression in mice. *Physiol. Behav.* **27,** 983–987.

Potegal, M., and Ferris, C. F. (1989). Intraspecific aggression in male hamsters is inhibited by intrahypothalamic vasopressin-receptor antagonist. *Aggress. Behav.* **15,** 311–320.

Romijn, H. J. (1978). The pineal, a tranquilizing organ? *Life Sci.* **23,** 2257–2273.

Shi, H., and Bartness, T. J. (2000). Catecholaminergic enzymes, vasopressin and oxytocin distribution in Siberian hamster brain. *Brain Res. Bull.* **53,** 833–843.

Stetson, M. H., and Tay D. E. (1985). Time course of sensitivity of golden hamsters to melatonin injections throughout the day. *Biol. Reprod.* **29,** 432–438.

Stribley, J. M., and Carter, C. S. (1999). Developmental exposure to vasopressin increases aggression in adult prairie voles. *Proc. Natl. Acad. Sci. USA.* **96,** 12601–12604.

Tamarkin L, Baird C. J., and Almeida O. F. (1985). Melatonin: A coordinating signal for mammalian reproduction? *Science* **227,** 714–20.

Turek, F. W., Elliot, J., Alvis, J., and Menaker, M. (1975). Effects of prolonged exposure to nonstimulatory photoperiods on the activty of the neuroendocrine–testicular axis of golden hamsters. *Biol. Reprod.* **13,** 475–481.

Wingfield, J. C. (1994). Regulation of territorial behavior in the sedentary song sparrow, *Melospiza melodia morphna. Horm. Behav.* **28,** 1–15.

Winslow, J. T., Hastings, N., Carter, C. S., Harbaugh, C. R., and Insel, T. R. (1993). A role for central vasopressin in pair bonding in monogamous prairie voles. *Nature* **365,** 545–548.