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Effects of light pollution on photoperiod-driven seasonality

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Abstract

Changes to photoperiod (day length) occur in anticipation of seasonal environmental changes, altering physiology and behavior to maximize fitness. In order for photoperiod to be useful as a predictive factor of temperature or food availability, day and night must be distinct. The increasing prevalence of exposure to artificial light at night (ALAN) in both field and laboratory settings disrupts photoperiodic time measurement and may block development of appropriate seasonal adaptations. Here, we review the effects of ALAN as a disruptor of photoperiodic time measurement and season-specific adaptations, including reproduction, metabolism, immune function, and thermoregulation.

Keywords

Light at night; Photoperiod; Seasonality; Circadian rhythm disruption; Reproduction; Immune function; Metabolism; Thermoregulation

1. Introduction

Most contributions to this special issue focus on the mechanisms underlying seasonal interactions among hormones, brain, and behavior. For the vast majority of vertebrate animals living outside of the tropics, seasonality is determined by monitoring the annual cycle of changing photoperiod (day length) (Stevenson et al., 2017). Whether day lengths are above or below a critical threshold allows individuals to precisely determine time of year and evoke appropriate seasonal adaptations (Stevenson et al., 2017). However, in order for individuals to assess photoperiod, a clear delineation between day and night is critical. In both laboratory and field conditions, significant exposure to artificial light at night (ALAN) is increasing; importantly, ALAN can derange the assessment of photoperiod and impair seasonally appropriate adaptations.

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Declaration of competing interest

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Although spatial adaptations are typically considered in discussions of fitness, temporal adaptations are also critical for successful reproduction and survival. For example, photoperiodic information is critical for timing the onset and termination of seasonal adaptations that promote reproduction and survival, the two pillars of fitness. Instead of day length, however, most vertebrates assess time of year by monitoring night length, encoded by the nightly duration of melatonin secretion (Goldman, 2001). Melatonin is secreted only during the night in both nocturnal and diurnal vertebrates (Pévet, 2003; Smale and Nunez, 2009). Elevated melatonin concentrations (amplitude) broadly communicate night-time within the body, whereas the duration of elevated melatonin (frequency) indicates the length of night and thus time of year. Thus, melatonin serves as both biological clock and calendar (Reiter, 1993).

Breeding seasons are probably the most salient annual cycles among animals (Dawson et al., 2001; Prendergast et al., 2002). Outside of the tropics, offspring are typically produced during spring or summer when food is most abundant, and other environmental factors are optimal for survival (Nelson, 2004). The energetic bottleneck resulting from increased thermoregulatory demands when food availability is scarce makes winter a particularly difficult time to breed and survive. These energetic constraints drive photoperiod-mediated seasonal adaptations including adjustments in metabolism, adiposity, immune function, thermoregulation, and reproduction (Walton et al., 2011; Weil et al., 2015; Owen and Moore, 2008; Tavolaro et al., 2014; Heldmaier et al., 1989a). Below, we discuss the role of light in setting the circadian clocks and the role of circadian information on photoperiodic responses in vertebrate systems, then, the effects of ALAN in the regulation of these functions.

Assessment of photoperiod requires functional endogenous circadian time keepers (Hastings and Herzog, 2004). The circadian clock in mammals is located in the suprachiasmatic nuclei (SCN) of the hypothalamus (Stephan and Zucker, 1972). The circadian (~24 h) clocks are set to precisely 24 h by exposure to daylight early in the day. Light enters the eyes of mammals and stimulates the retinas, activating intrinsically photosensitive retinal ganglion cells (ipRGCs) that contain the photopigment melanopsin; melanopsin is maximally responsive to short wavelength light (blue), and less responsive to long wavelength light (red) (Hattar et al., 2002). IpRGCs are not image-forming receptors (Gooley et al., 2001). Light information is relayed to the master circadian clock in the SCN, by the monosynaptic retinohypothalamic tract (RHT) (Moore and Eichler, 1972; Stephan and Zucker, 1972). The SCN communicates temporal information to secondary oscillators in the brain, including the pineal gland, pituitary gland, and other brain regions; these brain regions relay temporal information to the rest of the body. In contrast to mammals (Nelson and Zucker, 1981), aves contain additional circadian oscillators and reside in the retina, pineal gland, and hypothalamus (Singh et al., 2013). These independent oscillators communicate and comprise the central circadian pacemaker in the avian circadian system that coordinates with the external environment (Saldanha et al., 1994; Singh et al., 2013; Trivedi et al., 2017). Importantly, having entrained circadian rhythms is critical for endogenous assessment of day length.

The molecular function of the circadian clock is driven by interlocking transcription-translation feedback loops comprising transcriptional activators and repressors (reviewed in (Mohawk et al., 2012; Reppert and Weaver, 2002). Within the cell nucleus, circadian

locomotor output cycles kaput (CLOCK) and brain and muscle arnt-like protein 1 (BMAL1) form heterodimers that induce expression of period (*Per1, Per2*, and Per3) and cryptochrome (*Cry1* and *Cry2*) through *E*-box enhancers (Gekakis et al., 1998; Hogenesch et al., 1998; Jin et al., 1999). Period (PER) and cryptochrome (CRY) proteins accumulate in the cytoplasm throughout the circadian day, and as levels rise, PER and CRY form a heterodimer complex that translocates back to the nucleus to repress their own transcription (Patke et al., 2020). This process requires ~24 h to complete a cycle. Other regulatory loops influence the molecular mechanisms underlying circadian clocks in addition to the primary feedback loop: e.g., the CLOCK:BMAL1 heterodimer also activates transcription of retinoic acid- related orphan nuclear receptors, *Rev-erbα* and *Rora*, which have feedback effects primarily on *Bmal1* (Patke et al., 2020). Exposure to dim ALAN alters both the amplitude and phase of peak expression of key clock genes leading to dysregulation of critical physiological function (e.g., Fonken et al., 2013c; Fonken and Nelson, 2014).

The central clock in the SCN is responsible for encoding photoperiodic regulation by regulating the synthesis and release of pineal melatonin (Hastings and Herzog, 2004; Cassone, 1990; Gwinner et al., 1997). This process is dependent on differential day length, determined by the duration of melatonin secretion into the blood and cerebrospinal fluid, which is responsible for monitoring night length (Reiter et al., 2014). Short days (long nights) are encoded by extended durations of melatonin secretion, and conversely, reduced durations of elevated melatonin secretion encode long days (short nights). Melatonin signals to several target regions of the brain responsible for altering the phase of central and peripheral circadian clocks through alterations in circadian clock gene expression (Pevet and Challet, 2011). This includes the pars tuberalis (PT) of the pituitary stalk in mammals, which governs photoperiodic regulation of seasonal reproduction. Upon activation, the PT initiates a hormonal cascade response, resulting in thyroid 3 hormone interacting with hypothalamic peptides controlling the release of gonadotropins from the adenohypophysis, which, in time, lead to seasonal changes in gonadal size and function (see section on Reproductive Function), as well as alterations in body mass (Barrett et al., 2007; Pevet and Challet, 2011).

Annual photoperiodic changes follow a consistent and predictable pattern. However, rapid changes to the natural environment including nighttime illumination from recent urbanization activity can alter specific photoperiod and seasonal adaptations necessary for optimal function (Hölker et al., 2010; Nelson, 1987). The synchronization of photoperiodic time measurement and the circadian clock rely on a distinct differentiation between day and night, and exposure to ALAN can disrupt circadian clock gene expression and alter melatonin secretion, affecting photoperiod-regulated behavior and physiology (Navara and Nelson, 2007; Dominoni et al., 2016; Dominoni and Nelson, 2018). Next, this review will discuss the effects of ALAN-induced photoperiodic disruption and its impact on physiological function.

2. Reproduction

ALAN disrupts circadian rhythms and photoperiodic responses (Grubisic et al., 2019). Abundant data demonstrate harmful effects caused by altering the natural photoperiod (Dominoni et al., 2020; Sanders et al., 2020). For example, ALAN exposure of great tits

(*Parus major*) and European blackbirds (*Turdus merula*) advanced the timing of vernal gonadal growth and reproductive function (Dominoni and Nelson, 2018; Dominoni et al., 2013). Similar changes in reproduction and mating have been reported for fruit flies (*Drosophila melanogaster)* (McLay et al., 2017); ALAN prolonged courting behavior and changed oviposition patterns.

A 5-year study on two populations of free-living tammar wallabys (*Macropus eugenii*) exposed to different levels of ALAN, revealed that light exposure at levels common in urban environments blunts seasonal changes to ambient light cues, suppresses melatonin concentrations, and delays births (Robert et al., 2015).

Because laboratory animals are commonly exposed to significant ALAN, it is important to investigate its role on seasonally-mediated physiology and behavior (Emmer et al., 2018; Russart and RJ, 2018). ALAN alters short-day regulation of reproduction in male Siberian hamsters (*Phodopus sungorus*) (Ikeno et al., 2014a). Exposure to dim ALAN (5 lx) reduced nocturnal locomotor activity and changed expression of genes implicated in photoperiod responses including, *Mel-1a melatonin receptor, Eyes absent 3, thyroid stimulating hormone receptor, gonadotropin-releasing hormone*, and *gonadotropin-inhibitory hormone*. Additionally, these changes were associated with altered circadian clock gene expression (*Per1*), and altered gonadal mass, sperm numbers, pelage color, and pelage density (Ikeno et al., 2014b).

Nocturnal mouse lemurs (*Microcebus murinus*) typically use long day lengths to time reproduction in their native Madagascar. Male mouse lemurs that were reproductive quiescent during the short days of winter, were exposed to 50 lx of white light for 5 weeks. Indirect melatonin assays indicated suppression of this hormone. Testes size and function underwent recrudescence comparable to breeding, suggesting that moderate light pollution (a single street lamp) is capable of disrupting seasonal breeding in this species (le Tallec et al., 2016).

3. Immune function

Optimal immune function is critical for survival; indeed, immune function is often considered a proxy for survival costs in life history approaches (Nelson, 2004). Immune function requires significant energetic investment for ideal function and protection against disease, and these costs must often be balanced against reproductive costs to optimize fitness. Thus, energetic tradeoffs have often evolved to balance seasonal challenges, resulting in season-specific investments (Nelson and Demas, 1996). Variation in immune function and defense across seasons has been previously reviewed (Nelson, 2004); photoperiodic changes across seasons contribute to survival (Weil et al., 2015). The short days (long nights) of winter are often associated with enhanced innate immune responses in field studies; in small mammals and birds, this short-day enhanced investment in immune function occurs when reproductive costs are minimal (Nelson and Demas, 1996; Demas and Nelson, 1998; Nelson, 2004). This strategy allows investment in survival mechanisms such as immune function and thermoregulation when food resources are most limited (Navara and Nelson, 2007). Short day conditions in laboratory-based studies are associated

with enhanced immune function when other factors are constant (Demas and Nelson, 1998); presumably this tactic combats seasonal variance in disease incidence and severity (Stevenson and Prendergast, 2015).

ALAN from cities and recent urbanization results in significant sky glow that invades previously dark nights. Light pollution can block assessment of photoperiodic response and blur trade-offs that evolved to enhance fitness (de Jong et al., 2015). Many studies evaluating the role of ALAN on immune function have used laboratory rodents, and the vast majority indicate that ALAN has detrimental consequences. Dim and ecologically relevant levels of ALAN impaired daily circulations of monocytes and T-cells, along with a reduction in blood monocytes and Cd68 (Okuliarova et al., 2021). Dim ALAN also impairs cell-mediated immune responses in nocturnal rodents with robust pineal melatonin rhythms, including Siberian hamsters (*P. sungorus*) (Bedrosian et al., 2011) and rats (*Rattus norvegicus*) (Oishi et al., 2006). ALAN also alters immune function in diurnal Nile grass rats (*Arvicanthis niloticus*). In this study, male Nile grass rats increased bactericidal capacity, pinnal swelling in a cell-mediated delayed-type hypersensitivity test, and upregulated antibody production post keyhole lymphocyte hemocyanin (KLH) (Fonken et al., 2012). Notably, studies performed in nocturnal mice without robust pineal melatonin rhythms have demonstrated that exposure to ALAN induces increased proinflammatory cytokines and enhanced inflammatory response to LPS (Fonken et al., 2013a). Exposure to ALAN resulted in a greater pinnal swelling in a delayed type hypersensitivity reaction in short day hamsters relative to long day animals. Other results of this study indicated that ALAN interfered with short day phenotype development which can negatively impair photoperiodic reproductive responses, survival, and fitness (Aubrecht et al., 2014).

In addition to mammals, reproductively photoperiodic organisms such as aves also rely on day length to regulate adaptive physiological and behavioral responses. In contrast to mammals (Nelson and Zucker, 1981), however, birds assess lighting conditions via extra-retinal and extra-pineal photoreceptors (Saldanha et al., 1994). Aves undergo seasonal physiological preparation for long-distance migration which requires significant energetic investment that can suppress immune function (Eikenaar and Hegemann, 2016; Owen and Moore, 2008). Thus, the effects of ALAN and other disruptions to natural photoperiodic response can be especially pronounced. ALAN detrimentally affects immune function in wild Japanese quail (*Coturnix japonica*) (Moore and Siopes, 2000), cockerels (*Gallus gallus domesticus*) (Kirby and Froman, 1991), and captive bred Australian budgerigars (*Melanopsittacus budgerigar*). Birds maintained under natural outdoor short day conditions supplemented with ALAN (200 lx), displayed increased duration and severity of infections (Malek and Haim, 2019). A study evaluating the effects of ALAN on resident and migrant dark eyed juncos (*Junco hyemalis*) reported increased total leukocyte concentrations across the experiment, with heightened inflammation during the beginning and end of the study (early spring, late fall) (Becker et al., 2020). Additionally, this study investigated rates of infection and reported increased parasitemia, hemosporidian intensity, and chronic infections in birds exposed to ALAN, with a pronounced effect in resident juncos. Chronic exposure to ALAN also increased bactericidal activity and immunity at varying time points for males compared to females in developing king quails (*Excalfactoria chinensis*) (Saini et al., 2019). A study of wild great tit nestlings (*Parus major*) that are urban/rural avian species, reported

that 3 lx of ALAN decreased melatonin concentrations, as well as decreased haptoglobin and nitric oxide levels, both of which are markers for innate immune function (Ziegler et al., 2021). Together, these studies highlight and demonstrate the detrimental impact of ALAN on immune function by altering natural photoperiodic response.

4. Metabolism

Male C57Bl/6 J mice exposed to constant light (LL) conditions elevate food intake and decreased energy expenditure compared to their counterparts with dark nights, although weight gain preceded the change in energy uptake and expenditure (Coomans et al., 2013a). Mice fed a high fat diet (HFD) while in LL, elevated weight gain more rapidly in response to ALAN than in response to the HFD. Exposure to LL dramatically reduced the circadian rhythm in insulin sensitivity (~50% less), which correlated with loss (~56%) in SCN rhythmic output. These data are consistent with prior studies in which bilateral SCN lesions of male C57Bl/6 J mice impaired hepatic insulin sensitivity and elevated fat mass gain (Coomans et al., 2013b). Together these data demonstrate that disturbing endogenous SCN function, and consequently, circadian rhythms, significantly impairs metabolic homeostasis in mice.

Studies in rats suggest that, in common with mice, physiological outputs related to metabolism including resting insulin and glucose levels, elevated lipid composition, as well as disrupted melatonin and corticosterone rhythms are altered with changed photoperiods (reviewed in (Rumanova et al., 2020). In contrast to mice studies, studies in rats infrequently report elevations in body mass in response to exposure to ALAN (Rumanova et al., 2020). For instance, Long Evans rats in LL with ad libitum access to food, do not increase total weight, despite increased visceral adiposity and elevated feed efficiency, compared to rats exposed to either a standard light-dark (LD) cycle or constant dim ALAN (Wideman and Murphy, 2009). Male Wistar rats exposed to LL that were fed during a restricted 3 h period were able to rescue activity rhythms, as well as *Per2* expression in the SCN (Lamont et al., 2005). Although similar results have been described, this was the first study to report that restricting diet to the inactive phase could reestablish rhythmicity. This study, however, did not report changes in body mass, caloric intake, or other metabolic and/or hormonal changes, that are negatively affected by mistimed eating (during the inactive phase), in mice (Fonken et al., 2010; Hatori et al., 2012). Rat clock gene data contrast murine data (Sudo et al., 2003) where mice lost *mPER2* rhythmicity in the SCN under ALAN. Although these results establish a molecular modulatory relationship between feeding and the SCN in rats, they do not consider the direct effects on metabolism and homeostasis. Melatonin may also play a role in mediating the differing effects of ALAN on metabolic parameters between mice and rats, as rats used in the studies display robust pineal melatonin rhythms that are not present in mice.

Swiss Webster mice housed in a 14:10 light cycle for 6 weeks, and exposed to dim white ALAN (5 lx) increased body mass and reduced glucose processing without changing total daily home cage activity or caloric intake (Fonken et al., 2010). These mice shifted the time of day during which they consumed daily calories to their typically inactive phase (day). Another study examining photoperiodic effects on lipid metabolism reported that

short-day rats (SD; LD 4:20) increased serum triglycerides, lipoprotein, and leptin levels, as well as increased body weight, fat to weight ratio, and hepatic TG levels (an indicator of lipid metabolism) compared to long-day (20:4 light cycle) rats. Nonetheless, the effects of disrupted metabolic rhythms by ALAN exposure may not be permanent. Return to dark nights restores impaired glucose metabolism following prolonged exposure to illuminated nights (Fonken et al., 2013b). Together, these data suggest that obscuring or abolishing photoperiodic cues disrupts circadian rhythms, consequently altering metabolic processing in rodents.

In addition to chronic photoperiodic disruption, short term studies investigating acute changes to metabolic parameters have demonstrated that ALAN alters factors contributing to mass gain (Borniger et al., 2014). As previously observed (Fonken et al., 2010), mice gained significantly more mass despite similar locomotor activity levels (and rhythms) and total food intake between groups. This study further characterized metabolic effects by identifying an overall reduction in whole body energy expenditure, increased carbohydrate over fat oxidation, and altered temperature circadian rhythms, all of which contribute to the increase in mass gain. Further, circadian fluctuations in body temperature can entrain peripheral rhythms (Brown et al., 2002; Buhr et al., 2010) and feed back to the SCN, further misaligning the synchrony between central and peripheral oscillators. Indeed, these data in conjunction with prior observations that ALAN shifts the time of food intake, suggest a relation between these metabolic outputs, and which may be partially uncoupled from the SCN (Damiola et al., 2000), although locomotor activity rhythms persist.

We have emphasized how photoreception initiates a signaling pathway that relays photoperiodic information to the brain, and through pineal melatonin release, provides day length information. The humoral cascade initiated by melatonin synchronizes central and peripheral oscillators to the light signal, and establishes circadian and circannual rhythms. However, synchronization in reproduction, body mass, activity pattern and body temperature to photoperiod was achieved in pinealectomized European hamsters (*Cricetus cricetus*) (Monecke et al., 2013). If pinealectomy is performed in hamsters of this species during long days, then ~70% of the animals are able to entrain to the light cycle independent of melatonin signaling. Moreover, other studies have established similar results in pinealectomized golden-mantled ground squirrels *(Spermophilus lateralis)* with respect to body weight and reproductive state (Hiebert et al., 2000), as well as in pinealectomized female wolves (*Canis lupus*) with respect to the progesterone peak associated with estrus (Asa et al., 1987). These data suggest that, at least in these species, photoperiod-driven physiological functions can occur independent of melatonin signaling. Alternatively, because some of these studies (Asa et al., 1987; Hiebert et al., 2000) were conducted outdoors, it is possible that other factors typically associated with changes in photoperiod, such as temperature, may have played a role in signaling physiological changes that typically accompany changes in day length.

5. Thermoregulation and body temperature

Many organisms display photoperiod-regulated fluctuations of body temperature and thermogenic function to match seasonal variations in energetic needs and output (Heldmaier

et al., 1989b). In some organisms, these fluctuations are also matched with seasonal variations in fur composition and feather replacement. Photoperiodic alterations in thermogenesis and body temperature are necessary for efficient energy consumption during periods of low resource availability and low environmental temperature (Lovegrove, 2005). Individuals of some species use short days to generate physiological cues needed to begin hibernation (Hut et al., 2014).

Annual changes in thermogenesis may be deranged by ALAN if clear photoperiodic cues are not available. Numerous studies have demonstrated negative effects of ALAN exposure both within and across seasons. Social voles (*Microtus socialis)* housed in LD 8:16 and exposed to 15-min light pulses of fluorescent white light every 4 h during the dark phase (Haim et al., 2005) reduced thermogenesis at ambient room temperatures of 15 °C and reduced non-shivering thermogenesis. Furthermore, ALAN also impaired thermogenesis in response to noradrenaline injections (Haim et al., 2005). Similarly, social voles exposed to 15-min light pulses of 450 lx during the dark phase of LD 8:16 light-dark cycles reduced core body temperature during the light phase and reduced energy expenditure across the day (Zubidat et al., 2007).

Similar effects of ALAN on body temperature and short-day photoperiodic thermogenic responses have been observed in other species. For example, kangaroo rats (*Dipodomys ingens*) housed in short photoperiods reduced thermogenesis in response to noradrenaline injections when exposed to single 15-min light pulses in the middle of the dark phase (Gettinger and Ralph, 1985). Photoperiodic Indian weaver birds (*Ploceus philippinus*) housed under 2 lx of ALAN increased body temperatures during the light phase (Kumar et al., 2018). Although this study did not directly measure the effects of ALAN on photoperiodic variations in body temperature or thermogenesis, these results indicate the potential for ALAN to disrupt seasonal physiology in this species. Further research is needed to elucidate these potential effects.

Continued research will be needed to understand the consequences of ALAN on seasonal alterations in thermogenesis and the implications this may have for individual survival among species. ALAN may alter seasonal thermogenic variations because of impaired ability of the SCN to properly coordinate time-of-day cues (Bumgarner and Nelson, 2021). Alternatively, ALAN may alter thermogenesis through the disruption of thyroid hormone secretion. Thyroid hormones are under the control of circadian and seasonal rhythms, and they play key roles in the regulation of thermogenesis (Hazlerigg and Loudon, 2008; Ouyang et al., 2018). Siberian hamsters (*P. sungorus*) exposed to 5 lx of ALAN increased thyroid stimulating hormone receptor gene expression in the hypothalamus and pars tuberalis (Ikeno et al., 2014a). The results of this study suggest one potential mechanism by which ALAN may alter thermogenesis and body temperature regulation.

6. Fur and feather molting

Seasonal variations of avian feather molting (Lesher and Kendeigh, 1941; Holmgren and Hedenström, 1995) and fur composition are observed in response to varying environmental temperatures. European black birds (*Turdus merula merula*) were exposed to ALAN and

molting was assessed across 2 years (Dominoni et al., 2013). During the first year, ALAN advanced the onset of molting, but did not advance the completion of molting. Several birds exposed to ALAN in this study failed to complete molting during the first year of analysis. During the second year, molting was examined at a single timepoint to assess the pattern of feather turnover. ALAN was observed to alter the pattern of molting, such that molting was skewed towards outer feathers rather than progressing from inner feathers outward, as in control animals. As mentioned by the authors, these effects may either be due to a consequence of impaired photoperiodic signaling or stress (Dominoni et al., 2013). Indeed, ALAN is observed to alter feather glucocorticoid concentrations in blue tit nestlings (Dominoni et al., 2021). Another study examining the effect of ALAN exposure across stimulated photoperiods confirmed similar seasonal advances in molting (Singh et al., 2021).

Siberian hamsters exhibit seasonal alterations in fur color and density as a result of varying photoperiods. Thus, ALAN was hypothesized to alter fur color scores and density of animals exposed to 5 lx of ALAN for 4–8 weeks (Ikeno et al., 2014b). Indeed, ALAN exposure leads to darkened fur color, shifting the pelage towards a summer phenotype. However, ALAN did not alter fur density.

Together, these three studies indicate the potential for ALAN to alter seasonal variations in molting and fur characteristics. In birds that depend on new seasonal feathers and other species that depend on seasonal fur alterations either to facilitate hibernation or evade predators, ALAN may have serious implications on survival and environmental adaptation. However, continued research is necessary to characterize further and elucidate the effects of ALAN on seasonal variations in fur and molting.

7. Conclusions

Taken together, ALAN may disrupt photoperiodic time measurement and derail the onset and offset of seasonal adaptations of physiology, morphology, and behavior. The role of melatonin as a photoperiodic mediator and its role in influencing seasonal changes in the brain has been well characterized; ALAN can perturb this response, along with the distinct delineation between light required for day length or time-of-day assessment. This review has identified potentially harmful effects of disrupting assessment of natural photoperiods by disrupting circadian rhythms via ALAN. We have highlighted the role of ALAN in several physiological systems important for fitness, including reproduction, immune function, metabolism, thermoregulation, and molting. Animals have evolved with exquisitely precise timing mechanisms that are hijacked by the increasing exposure to ALAN. ALAN should be a pressing ecological concern, as well as a concern for animal welfare in vivaria that allow ALAN exposure. We conclude that ALAN can disrupt photoperiodic responses, negatively impact fitness, and impair laboratory studies.

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