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ARTIFICIAL LIGHT AT NIGHT PROLONGS JUVENILE DEVELOPMENT TIME IN THE BLACK FIELD CRICKET, *TELEOGRYLLUS COMMODUS*

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ABSTRACT

A growing body of evidence exists to support a detrimental effect of the presence of artificial light at night (ALAN) on life-history and fitness traits. However, few studies simultaneously investigate multiple traits and the life stages at which changes manifest. We experimentally manipulated ALAN intensities, within those found in the natural environment, to explore the consequences for growth, survival and reproductive success of the field cricket, *Teleogryllus commodus*. We reared crickets from egg to adult under a daily light-cycle consisting of 12 hr bright daylight (2600 lux) followed by either 12 hr darkness (0 lux) or dim-light environments (1, 10 or 100 lux). We found egg hatch, adult survival and reproductive measures were largely comparable for all treatments. However, juvenile development time (number of days from egg to adult) was on average 10 days (14%) longer and adults were also larger when crickets were exposed to any light at night (1 lux, 10 lux or 100 lux). Our data demonstrate that chronic lifetime exposure to ALAN can modulate the timing of life-history events and may disrupt phenology to a similar extent as other abiotic factors.

Key words: light pollution, invertebrate, development, phenology, urbanisation

HIGHLIGHTS

1. The detrimental effect of the artificial light at night (ALAN) is increasingly documented for life history traits
2. We reared black field crickets under one of four ALAN intensities (0 lux, 1 lux, 10 lux, 100 lux) and monitored growth, survival and reproductive success.
3. The presence of ALAN increased juvenile development time, resulted in larger adults but had limited effect on subsequent reproduction or survival.

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1. INTRODUCTION

The presence of artificial light at night (ALAN) has dramatically altered the nocturnal environment resulting in night-time light levels in urban areas that are three to 100-fold brighter than that of a full moon (approximately 0.3 lux) (Bennie, Duffy, Davies, Correa-Cano, & Gaston, 2015; Falchi et al., 2016; Gaston, Bennie, Davies, & Hopkins, 2013; Gaston, Visser, & Holker, 2015). The ecological consequences of chronic exposure to even relatively low intensity ALAN, if consistently present, includes major disruption to the behavioural and physiological processes of individuals (Bennie, Davies, Cruse, & Gaston, 2016; Bennie et al., 2015; Gaston et al., 2015; Longcore & Rich, 2004; Navara & Nelson, 2007; Rich & Longcore, 2006). ALAN has become one of the most rapidly growing forms of environmental pollution (Hölker, Wolter, Perkin, & Tockner, 2010). Thus, understanding how species are responding to this rapid shift in the nocturnal environment and the degree to which the presence of ALAN affects individual fitness traits is of key concern.

One of the effects of artificially illuminating the night sky is that it masks natural day-night changes in light intensity. Behaviourally, the presence of artificial night lighting is negatively associated with changes in the movement and migration patterns of a diversity of animals, including birds, fish, turtles, bats and insects (see, Rich & Longcore, 2006). Moreover, direct effects on the foraging capacity (Bird, Branch, & Miller, 2004; Freeman, 1981; Polak, Korine, Yair, & Holderied, 2011; Rotics, Dayan, & Kronfeld-Schor, 2011; Santos et al., 2010; Titulaer, Spoelstra, Lange, & Visser, 2012), and an increased risk of death, due to disorientation (Merkel & Johansen, 2011; Rodriguez et al., 2012;

Witherington, 1992) are reported for both nocturnal and diurnal vertebrates. The presence of ALAN also has reproductive consequences: in European passerines, urban populations not only commence singing earlier in the day but they also tend to have a more protracted breeding season compared to their rural counterparts (Dominoni, Quetting & Partecke, 2013; Kempenaers, Borgstrom, Loes, Schlicht, & Valcu, 2010; Partecke, Van Hof, & Gwinner, 2004). Most recently, such changes have been linked to potentially adaptive shifts in behaviour (Altermatt & Ebert, 2016).

Accumulating evidence highlights the physiological implications of ALAN for key life-history traits (Dominoni, Borniger, & Nelson, 2016). In fish, the presence of constant artificial light during early embryonic development results in species-specific variation in hatch rates and swim bladder inflation (Bruning, Holker, & Wolter, 2011). Similarly, in *Drosophila jambulina*, synchronicity of adult eclosion is negatively related to both the presence and intensity of nocturnal illumination (Thakurdas et al., 2009) and in the cricket *Gryllus bimaculatus* egg hatch is under circadian control (Itoh & Sumi, 2000). While in the moth *Mamestra brassicae*, males (but not females) reared from the second instar under dim green or white lights at night spent less time in pupal diapause than moths reared with no light at night (van Geffen, van Grunsven, Ruijven, Berendse, & Veenendaal, 2014). ALAN also potentially masks seasonal variation in light. This is critical for any species whose reproduction is seasonally controlled. For example, a five-year study on the Tammar wallaby (*Macropus eugenii*), a species highly dependent on changes in day length with respect to its reproductive events, determined that individuals living in dimly lit areas delayed the birth of their offspring by one month compared to those living in darker areas (Robert, Lesku, Partecke, & Chambers, 2015). Collectively, these studies highlight the potential for ALAN to create trophic mismatches that may lead to resource depletion and reductions in offspring growth rates (Raap, Casasole, Costantini, et al., 2016;

Robert et al., 2015). However, whether ecologically relevant levels of light directly affect egg and sperm production in such species is largely undocumented. Indirect evidence, using male and female *Drosophila,* suggest that clock-gene deficient individuals (who therefore lack a normal circadian rhythm) produce less sperm and lay fewer eggs, respectively (Beaver et al., 2002). Hence, ALAN is likely to be associated with changes in gamete maturation (Dominoni et al., 2013; Dominoni, Quetting, & Partecke, 2013). Furthermore, current research on the effect of ALAN on life history traits typically explores individual traits in relative isolation; to our knowledge few studies have explicitly tested the effect of variation in the intensity of night lighting across multiple traits using an invertebrate model.

When considering the effects of ALAN on life history traits, there are potentially several mechanisms by which changes in the duration or intensity of ALAN may result in perturbed behavioural and physiological responses. The presence of ALAN may extend the temporal diurnal or crepuscular photic niche or conversely, may contract (or potentially eliminate) the true nocturnal temporal space. Theoretically, species that are exclusively active in one of these time-periods are likely most obviously affected, but species that occupy both the diurnal and nocturnal environments may also be vulnerable. Such species are less likely to be photophobic; however, the presence of extended periods of light may drive behavioural adaptation and have physiological consequences (Santos et al., 2010). Here, we investigated experimentally the effect of lifelong exposure to different ALAN intensities (0, 1, 10 or 100 lux) on the growth, survival and reproductive success using an invertebrate model, the Australian black field cricket, *Teleogryllus commodus*. The black field cricket is largely nocturnal with life history and mating strategies evolved to be optimal during darkness (Loher, 1962, 1974, 1979). Male courtship song commences prior to sunset but continues long into the night and it is therefore a species that is potentially exposed to ALAN, where it is present (Loher, 1962; Sokolove, 1975). Naturally, *T. commodus* is a native inhabitant of heath and grassland landscapes (where ALAN is typically absent) but, critically for the current study, it also inhabits urban and agricultural environments (where ALAN is present) (Robinson, 2005). Previous research in this species suggests that the presence of constant bright illumination reduces adult immune function (Durrant et al., 2015) and some aspects of mating (Botha, Jones, & Hopkins, 2017) but whether ALAN affects other life history traits is untested. Here, we assessed the effects of variation in the intensity of ALAN on egg hatch, juvenile development, adult morphology, survival and reproductive output in *Teleogryllus commodus*.

2. METHODS

Experimental crickets were sourced from a 10th generation laboratory-adapted population of founders captured from a dark location in Victoria, Australia (37.56238 S, 145.31920 E). Stock population crickets (approximately 1000 per generation) were maintained under standard conditions (Durrant et al., 2015) and held in a climate-controlled laboratory under a 12 hr light: 12 hr dark lighting regimen.

2.1 Experimental ALAN treatments and rearing conditions

To investigate the effect of artificial light at night on the life history of *T. commodus*, we maintained experimental individuals from eggs to adults under comparable simulated daylight conditions (2600 lux, equivalent to a cloudy day, 6800K) for 12 hours followed by one of four ALAN treatments (0 lux, 1 lux, 10 lux or 100 lux, all at 5900K) for a further 12 hours. The ALAN treatments were chosen to fall within the range of conditions currently present in and around urban areas, where 100 lux is acknowledged as an extreme but is an intensity present in a heavily urbanised city centre where crickets are present (TMJ personal observations). To create the different ALAN treatments, we used retrofitted Westinghouse incubators (model number WRM4300WB-R) set at a constant 28°C and lit by cool-white LED strip lighting on the front panels (World of Thought; Melbourne, Australia). Individual incubator effects were eliminated by alternating the light regimen in each incubator thrice weekly, and swapping the crickets accordingly, ensuring each incubator contributed equally to each regimen, but crickets were always maintained on their designated light regimen. Each cricket was maintained individually throughout the experiment but was rotated within each incubator, so that each experimental individual experienced a range of positions. It should also be noted that while crickets were isolated in an incubator in terms of light regimen, incubators were not sound proofed and thus no cricket was acoustically isolated from one another regardless of their light treatment or incubator.

2.2. Juvenile development and survival

2.2.1 Egg hatch

To explore whether responses to variation in ALAN were consistent across all families, we allocated offspring from known families equally to each of the ALAN treatments. To create known family groups, we paired 60 virgin adult females with virgin adult males from the stock population (24 ± 3 days post the final juvenile moult to the adult stage). After 24 hrs, we removed the male and provided each female with a sand pad for oviposition (standard 60 ml Petri dish base filled with 1 cm deep sand). The sand pad was replaced with a fresh sand pad every two days for a maximum of 10 days (five sand pads per female) or until she had laid at least 100 eggs. Any female that laid less than 100 eggs during this time-period was discarded (n = 47 females); a random sample of 100 eggs from the remaining 13 females were divided equally between the four ALAN treatments (n = 25 eggs per family per treatment), transferred to moistened cotton wool pads (Swisspers, Australia) in transparent round 100 ml containers and then placed in their designated ALAN treatment. Eggs were initially maintained in family groups of comparable age (designated by the date when laid) at densities of between 3 and 25 per container (dependent on the number laid during the given two-day oviposition period). Eggs were checked daily for four weeks or until all nymphs had hatched.

2.2.2 Juvenile development and survival

Within 24 hr of hatching, each first instar cricket nymph was transferred to an individual transparent rearing container (70 x 70 x 40 mm) which contained a folded 10 x 30 mm cardboard shelter, *ad libitum* water and dried cat food (Friskies Senior; Rhodes, Australia). We monitored each nymph daily until death or until after its final juvenile moult to an adult. The time taken for a hatched individual to reach the adult stage of the lifecycle (herein defined as juvenile development time) or the time to death during the juvenile phase of the lifecycle was recorded.

2.3 Adult morphology and survival

Adult crickets were sexed within a day of completing their final moult and then transferred individually to larger transparent containers (150 x 90 x 50 mm) containing a piece of egg carton for shelter and *ad libitum* water and dried cat food (as above). Adults were similarly checked daily until death or until the completion of the experiment (33 ± 1 days after the final moult) at which point they were euthanised. A small haemolymph sample was taken from each cricket at 3, 17 and 31 ± 1 days after the final moult (following, Durrant et al., 2015), which equated to an additional stressor at the adult phase of the lifecycle (notably imposed on all individuals). Haemolymph samples were assayed for immune function but these data are not reported here (Durrant J unpublished data). All adult crickets were treated in the same manner and all surviving crickets were terminated 33 ± 1 days after the final moult. Body weight (to the nearest mg) was taken prior to mating (see section 2.4) and, following death, both back leg femurs were removed; taped to a glass slide and then digitally photographed with a Canon EOS 60D (Tokyo, Japan) mounted at x 10 magnification on an Olympus SZX7 stereomicroscope (Tokyo, Japan). Femur length was determined using image J (Version 1.48V, NIH; Maryland, USA). As femur size is fixed in adult crickets, the average length of the two femurs was used as an index of body size (Danielson-Francois, Fetterer, & Smallwood, 2002; Jones, Durrant, Michaelides, & Green, 2015; Mousseau & Roff, 1989). We used the scaled mass index as a measure of body condition, as it provides a better indicator of the relative size of body components than residuals from a standard least square regression of body weight and length (Peig & Green, 2009).

2.4 Reproductive output

To assess the reproductive output of individuals in each ALAN treatment, at 21 ± 3 days after the final moult both male and female crickets were paired to virgin stock population individuals (reared under a normal 12 hr light: 12 hr dark cycle) of similar age and provided a 45-minute period to mate (for details on the experimental protocol and results, see Botha *et al.* 2017). If this first mating did not result in the successful transfer of a spermatophore, the stock individual was replaced and the experimental cricket was provided a second opportunity. If the experimental cricket failed to mate after these two opportunities, they were considered *unmated* and either they (for females) or their stock population partner (for males) were discarded from further analyses of fecundity (individuals discarded in 0 lux: n = 14/23 females, 5/29 males; 1 lux: n = 9/27 females, 7/37 males; 10 lux: n = 10/29 females, 9/39 males; 100 lux: n = 8/44 females, 4/45 males; Botha et al 2017).

2.4.1 Number of eggs laid

To assess whether variation in ALAN treatment affected egg production, mated experimental females and stock females mated to experimental males were provided with a sand pad for oviposition (see above) for a total of seven days. After this period, the sand pad was removed and the total number of eggs counted as a measure of reproductive output.

2.4.2 Sperm quality

To assess whether variation in ALAN treatment affected sperm quality (measured as sperm viability), males were provided a second mating opportunity (as above) with a virgin stock-female two days following their initial mating trial. If this second mating was successful, the spermatophore was removed from the female (within one minute of a male attaching it to the female's genitalia), transferred to an Eppendorf tube (1.5 ml) containing 80 µl Beadle saline solution (128.3 mM NaCl, 4.7 mM KCl, and 23 mM $CaCl_2$) and left for 10 mins. After this time, the spermatophore was removed and the solution mixed using a pipette to ensure homogeneity of sperm within the solution. This protocol maximised sperm effusion whilst avoiding sperm damage caused by the common forceful spermatophore rupturing technique (Gress & Kelly, 2011).

To determine sperm viability we used the standard live-dead sperm assay containing SYBER-14 dye and propidium iodide (Invitrogen Molecular Probes, U.S.A) with a protocol adapted from Gress and Kelly (2011) and García-González and Simmons (2005). A 5 µl aliquot of the sperm solution was transferred to 10 µl of diluted 1 mM SYBR-14 dye and left for ten minutes on a glass slide in darkness. After 10 minutes, 4 µl of 2.4 mM propidium iodide was added to the sample, and left in darkness for a further 10 minutes. A cover slip was then added and samples were viewed using a fluorescent microscope (Nikon Eclipse Ti-U, Japan) under a blue excitation filter (λ=470) and at x 100 magnification. Viability was assessed electronically using the NIS-Elements (Br 3.0) software. Both live and dead images were taken at four different areas of the slide (where sperm was present) and an average proportion live sperm calculated for each sample, with a minimum of 200 sperm assessed per slide. Samples with less than 200 sperm were discarded from the analysis.

2.5 Statistical analyses

We used JMP 12.1.0 (SAS Institute, NC, USA) for all analyses except variation in the adult survival and proportion of viable sperm which were assessed using a multi-level approach in MLwiN Version 3.00 (Centre for Multilevel Modelling, University of Bristol) (Rasbash et al., 2000). We used two-level models with ID and family as the first and second levels, respectively and assumed a binomial error distribution with logit link function. The cause of early (first and second instar) juvenile death was not always possible to determine, however the number of individuals whose death was undetermined was comparable across all ALAN treatments (0 lux = 81/269, 1 lux = 72/278, 10 lux = 70/280, 100 lux = 63/287; $\chi_3^2 = 4.93$, $P = 0.18$). To ensure that these differences did not affect our results, for juvenile survival, we ran two models: the first included all juveniles, the second we discarded individuals where the death was undetermined. Both models were qualitatively similar and thus only the second model is presented (see, Table 1B). Due to the experimental design, adults were euthanised at 33 days (see above) at which point 80% of females and 83% of males were still alive. Variation in adult survival was therefore assessed using probability models (probability of surviving to the end of the experiment) rather than with a formal survival analysis incorporating censoring. Each model was reduced using hierarchical removal of all terms with a significance of $P > 0.1$ (except our designated light regimen). Once the minimum adequate model was obtained, excluded terms ($P > 0.10$) were reintroduced back to confirm the model fit did not improve with their inclusion. Maximal models included, where biologically appropriate, the fixed factors ALAN treatment, days until hatch, juvenile development time, sex, femur length, mating status, body condition and whether or not they survived to the end of the experiment, as well as appropriate interactions between these terms. Family was included as a random term. Unless otherwise stated, significant interactions were assessed using post-hoc planned contrast tests; data presented are untransformed means ± SE and all tests were two-tailed with a significance level of $P < 0.05$.

3. RESULTS

3.1 Juvenile development and survival

3.1.1 Egg hatch

The number of eggs hatched (out of each family set of 25; N = 13 families) was comparable for all ALAN treatments (mean number ± standard error of eggs hatched under 0 lux = 17.5 ± 1.35, 1 lux = 17.5 ± 1.10, 10 lux = 18.1 ± 1.30, and 100 lux = 17.9 ± 1.26; P = 0.31; Table 1A). The random term *family* explained 79.5% of the observed variation (P = 0.02).

3.1.2. Juvenile survival

All 13 families produced at least one adult cricket, but there was significant variation between the four ALAN treatments (Table 1B; P = 0.04). *Post-hoc* analyses revealed that 100 lux juveniles were more likely to reach the adult stage than 0 lux crickets (Figure 1). The random term *family* explained 40.4% of the observed variation (P = 0.08).

3.1.3. Juvenile development time

For the subset of crickets that completed their final moult, the time from oviposition through to the adult stage of the life cycle (Table 1C) varied significantly with ALAN treatment (P < 0.0001). *Post-hoc* analyses revealed that crickets exposed to light at night (1 lux, 10 lux and 100 lux treatments) took 10 days longer to reach the adult phase of the lifecycle compared to 0 lux crickets (Figure 2A), with no difference in development time between any other groups. There was also a significant sex difference: juvenile development time for females was two days shorter (64.55 ± 0.85 days, n = 112) compared to males (66.44 ± 0.63 days, n = 137; P = 0.001). The random term *family* explained 24.9%

of the observed variation (P = 0.07).

3.2 Adult morphology and survival

3.2.1. Femur length and body condition

Adult femur length (Table 1D) varied between treatments: crickets exposed to light at night (1 lux, 10 lux and 100 lux treatments) were significantly larger than 0 lux crickets ($P < 0.0001$; Figure 2B). There was a positive relationship between femur length and juvenile development time ($\beta \pm SE = 0.03 \pm 0.005$; $P < 0.0001$), and females had larger femurs (11.46 ± 0.07 mm, n = 112) compared to males (11.28 ± 0.06 mm, n = 137; $P < 0.0001$). The random term *family* explained 11.8% of the observed variation (P = 0.08).

Body condition (measured as the scaled mass index) at the time of mating was comparable across the four ALAN treatments for females but not males (Table 1E and 1F). Planned comparisons revealed 0 lux treatment males had a higher mass scaled index than 10 and 100 lux males (both $P < 0.05$) and tended to have a higher index compared to 1 lux males. Female body condition was also weakly negatively related to leg length (P = 0.07). The random term *family* explained 21.4% (female, P = 0.10) and 33.5% (male, P = 0.04) of the variation respectively.

3.2.2. Adult female survival

The probability that a female survived to the end of the experiment (Table 1G) was unrelated to her ALAN treatment (P = 0.09). None of the variation in the model was explained by the random term *family* (P = 1.0).

3.2.3. Adult male survival

The probability of a male surviving to the end of the experiment (Table 1H) was unrelated to light treatment (P = 0.73). The random term *family* did not explain significant levels of variation (P = 0.29).

3.3 Reproductive output

3.3.1. Egg number

There were no significant difference across the ALAN treatments in the number of eggs laid per day by either treatment females mated to stock males (0 lux = 11.27 ± 4.60, n = 13; 1 lux = 11.78 ± 4.07, n = 15; 10 lux = 19.22 ± 4.98, n = 14; 100 lux = 20.56 ± 3.63, n = 23; P = 0.18; Table 1I) or the number of eggs laid by stock females mated to treatment males (0 lux = 17.89 ± 3.43, n = 19; 1 lux = 11.73 ± 2.86, n = 16; 10 lux = 13.74 ± 15.30, n = 19; 100 lux = 12.85 ± 2.75, n = 24; P = 0.35; Table 1J). The random term family explained 12.08% (treatment females; P = 0.42) and 12.63% (treatment males; P = 0.49) of the variation respectively.

3.3.2. Sperm viability

The proportion of viable sperm transferred during a male's second mating attempt was comparable across the four ALAN treatments (median [interquartile range] proportion of viable sperm transferred by 0 lux males = 0.26 [0.15-0.35], n = 15; 1 lux = 0.16 [0.07-0.29], n = 13; 10 lux = 0.15 [0.11-0.33], n = 12; 100 lux = 0.25 [0.16-0.39], n = 11; P = 0.97; Table 1K). The random term *family* explained significant levels of variation; P < 0.001).

4. DISCUSSION

Our experiment yielded three key findings related to the presence of artificial light at night. First, the presence of any ALAN (1, 10 or 100 lux) resulted in a consistent 10 day (equating to approximately 14%) increase in the time taken for crickets to complete the juvenile phase of their development compared to crickets reared in the absence of light at night (0 lux crickets). Second, crickets reared under artificial light at night were significantly larger than crickets under 0 lux, even when accounting for variation in juvenile development time. Third, despite the significant effect of ALAN on juvenile development and resulting adult size, its presence had limited effect on adult survival or reproductive output. Our experiment also revealed substantial variation in adult body size. Combined our data highlight the potential for artificial light at night to disrupt the physiology and development of organisms and alter the timing of life history events, potentially disrupting phenology in much the same way as other recent abiotic or climatic shifts (Neil & Wu, 2006; Visser, Both, & Lambrechts, 2004).

Shifts in the timing of juvenile development linked to the presence of artificial light at night are demonstrated for invertebrates (Thakurdas et al., 2009; van Geffen et al., 2014) and vertebrates (Bruning et al., 2011; de Jong et al., 2015) but the mechanisms under-pinning these differences are rarely tested. We suggest that two types of physiological responses may contribute to the observed variation: disruption of the hormonal system around appetite control and growth, as well as disruption of perception of day-length and its implicit seasonality. Initially, exposure to ALAN may interfere with key hormones involved in appetite, satiety and growth rate (such as ghrelin, leptin, insulin, insulin-like growth factor (IGF), growth hormone and melatonin). Ghrelin stimulates appetite and potentially lead to increased weight gain in an environment where food is available *ad libitum* (Mataruga et al., 2012; Peric-Mataruga et al., 2009; Peric-Mataruga et al., 2015). The indolamine melatonin, in particular, is a powerful chemical driver of circadian rhythm, whose production is reduced in the presence of light at night (Pandi-Perumal et al., 2006; Poeggeler, 1993; Reiter, Poeggeler, Tan, Chen, & Manchester, 1993; Reiter, Tan, Osuna, & Gitto, 2000; Tan et al., 2010; Tan, Manchester, Terron, Flores, & Reiter, 2007; Vivienroels, Pevet, Beck, & Fevremontange, 1984). The complex interacting effects between melatonin and ghrelin, (i.e. lower melatonin concentration from a reduced dark phase is unable to suppress ghrelin expression and elevated appetite, as well as normal metabolism and energy expenditure from food) possibly explain the increased weight gain in crickets reared under ALAN (Kirsz, Szczesna, Borsuk, & Zieba, 2017; Kirsz & Zieba, 2012). This interaction is supported experimentally in numerous vertebrate species, by removal of the pineal gland resulting in obesity, as well as administration of melatonin reversing weight gain (Kirsz et al., 2017; Kirsz & Zieba, 2012). Reduced melatonin concentrations, through decreased dark phase, also explain the slower growth of juvenile crickets exposed to light at night (even the lowest intensity of 1 lux). The actions of melatonin are mediated through its receptor that are widely distributed in the body in many tissues (Slominski, Reiter, Schlabritz-Loutsevitch, Ostrom, & Slominski, 2012). This allows melatonin to regulate growth and development via multiple mechanisms. For example, melatonin is known to induce IGF (Picinato et al., 2008), a potent cellular mitogen required for growth and development, as well as modulating growth hormone secretion (Recabarren, Lobos, Robinson, Orellana, & Parilo, 2000). Melatonin is a thyroid antagonist thought to have a role in metamorphosis, as reported in amphibian studies (Wright, 2002). However, our findings are in contrast to comparable studies, exploring the effect of dim night lighting on offspring growth that found reduced juvenile to adult development times and reduced body size for ALAN individuals (Raap, Casasole, Costantini, et al., 2016; van Geffen et al., 2014). One potential explanation for the observed variation in development is that the 0-lux condition with its 12 hour dark period may have triggered a short day-length response in *T. commodus*, a species with demonstrated facultative diapause (Hogan, 1965, 1967). While this is yet to be demonstrated in *T. commodus*, in the closely related *T. emma*, juveniles developed faster with shorter day-lengths (< 13 hours light) compared to longer day-lengths (14 to 16 hours of light) and adult body weight was positively related to the juvenile period (Kim, Hong, Seol, & Kim, 2008).

We did not measure melatonin in this study, however chronic exposure to ecologically relevant levels of ALAN can reduce melatonin concentrations (Brüning, Hölker, Franke, Preuer, & Kloas, 2015; Evans, Elliott, & Gorman, 2007) and in *T. commodus* the presence of constant light negatively affects both melatonin concentrations and immune function (Durrant et al., 2015; Jones et al., 2015). Moreover, the loss of melatonin in species exposed to ALAN is implicated in a range of downstream negative fitness effects including shifts in circadian rhythm, impaired growth and compromised immune function (Brüning et al., 2015; Calvo, Gonzalez-Yanes, & Maldonado, 2013; Cuesta, Cerezuela, Esteban, & Meseguer, 2008; Navara & Nelson, 2007; Pohanka, 2013; Reiter et al., 2009;

Reiter et al., 2003; Srinivasan et al., 2005). Furthermore, direct links between the presence of ALAN, melatonin and sexual maturation are demonstrated (Asher, Archer, Ward, Scott, & Littlejohn, 2011; Dominoni et al., 2013; Dominoni, Goymann, Helm, & Partecke, 2013; Le Tallec, Théry, & Perret, 2016; Robert et al., 2015) and it is increasingly clear that even dim night lighting (1 lux and below) can affect circulating levels of melatonin (Bruning et al., 2011; Dominoni, Goymann, et al., 2013; Durrant et al., 2015). The adaptive benefit of a longer development period may be reduced in a natural more malign and likely colder environment, where a protracted juvenile phase of the lifecycle and resulting larger individuals may increase predation risk within the population (Dixon & Baker, 1988) thus reducing species fitness.

Given the large developmental difference observed between ALAN and 0 lux crickets, and the likely physiological mechanism underpinning it, the lack of a subsequent effect on reproduction or adult survival is perhaps surprising, although comparable with other studies where it has been explicitly investigated (de Jong et al., 2015; van Geffen et al., 2014). The lack of a detectable difference in the proportion of eggs hatching after their initial allocation to the ALAN treatments (c.f. Bruning et al., 2011) likely arose because we used initially stock population individuals and thus sperm and eggs matured and eggs were subsequently laid under a natural light:dark cycle prior to transferring to the ALAN treatments. Moreover, our protocol artificially selected only clutches with equal to or more than 100 eggs and thus more sensitive females/families were potentially discarded at this initial phase. Moreover, the nature of our subsequent experimental protocol, which promoted extreme selection throughout the entire juvenile phase of the lifecycle (compared with studies that have focussed on either one stage or a partial stage of the life-history, Altermatt & Ebert, 2016; Dominoni et al., 2013; Raap, Casasole, Pinxten, & Eens, 2016; Robert et al., 2015; van Geffen et al., 2014) again likely resulted in only the fittest juveniles surviving to the adult phase of the lifecycle. Our experimental design does not permit us to determine the relative importance of ALAN at the early and late juvenile phases of the lifecycle but given that these phases differ in the nature of resource investment – the former typically involves rapid growth and somatic investment, whereas the latter must allocate resources to reproductive development also (Imms, 1925) – our data perhaps hint at high susceptibility of the earliest instars.

5. CONCLUSION

Our results concur with previous studies that find even extremely low levels of ALAN affect key life-history traits (Bruning et al., 2011; de Jong et al., 2015; Dominoni et al., 2013). Variation in the timing of development and sexual maturation, due to the presence of ALAN, has broad ecological implications (Dominoni et al., 2013; Hölker et al., 2010; Neil & Wu, 2006; Visser et al., 2004), which may lead to a mismatch within populations in terms of the reproductive window (de Jong et al., 2015; Robert et al., 2015; van Geffen et al., 2014). This is particularly important for species with multiple generations within a breeding season, such as *T. commodus*. What is perhaps most challenging is that species clearly respond differently to urbanisation but we have a limited understanding of the capability of most species to adapt to this significant environmental change (Evans, Hatchwell, Parnell, & Gaston, 2010; Swaddle et al., 2015). Assessing the amount of natural genetic variation is a first step, although further research is needed to identify if species in their natural habitats are actually adapting to this rapid environmental shift.

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Table 1. Generalised mixed models exploring the effect of ALAN treatment (0 lux, 1 lux, 10 lux or 100 lux) on (A) the probability of egg hatch, (B) juvenile survival, (C) juvenile development time, (D) adult size, (E) adult scaled mass index at mating, (F) adult female survival, (G) adult male survival, (H) number of eggs laid per day by treatment females (I) number of eggs laid per day by stock females, and the (J) proportion live sperm.

Model Parameters	**β ± SE**	**Statistic**	**P Value**
(A) Number of eggs hatching			
ALAN treatment		$F_{3,36} = 1.23$	0.31
(B) Juvenile survival			
ALAN treatment		$F_{3,36} = 2.99$	0.04
(C) Juvenile development time			
ALAN treatment		$F_{3,233.9} = 33.8$	<0.0001
Sex		$F_{1,233.9} = 10.8$	0.001
(D) Adult size (femur length)			
ALAN treatment		$F_{3,227} = 9.27$	<0.0001
Sex		$F_{1,227.8} = 14.1$	0.0002
Days until adult from hatch	0.03 ± 0.005	$F_{1,224.4} = 36.2$	<0.0001
(E) Adult female scaled mass index at mating			
ALAN treatment		$F_{3,98.6} = 0.56$	0.56
Leg length (mm)	-0.01 ± 0.008	$F_{3,103.6} = 3.46$	0.07
(F) Adult male scaled mass index at mating			

ALAN treatment	$F_{3,\ 116.1} = 3.56$	0.02
(G) Adult female survival		
Light treatment	$\chi^2_{3,132} = 49$	0.09
(H) Adult male survival		
Light treatment	$\chi^2_{3,109} = 1.32$	0.73
(I) Number of eggs laid per day (treatment females mated to stock males)		
ALAN treatment	$F_{3,\ 58.08} = 1.71$	0.18
(J) Number of eggs laid per day (stock females mated to treatment males)		
ALAN treatment	$F_{3,\ 71.72} = 1.11$	0.35
(K) Proportion live sperm		
ALAN treatment	$\chi^2_{3} = 0.24$	0.97

FIGURE 1A

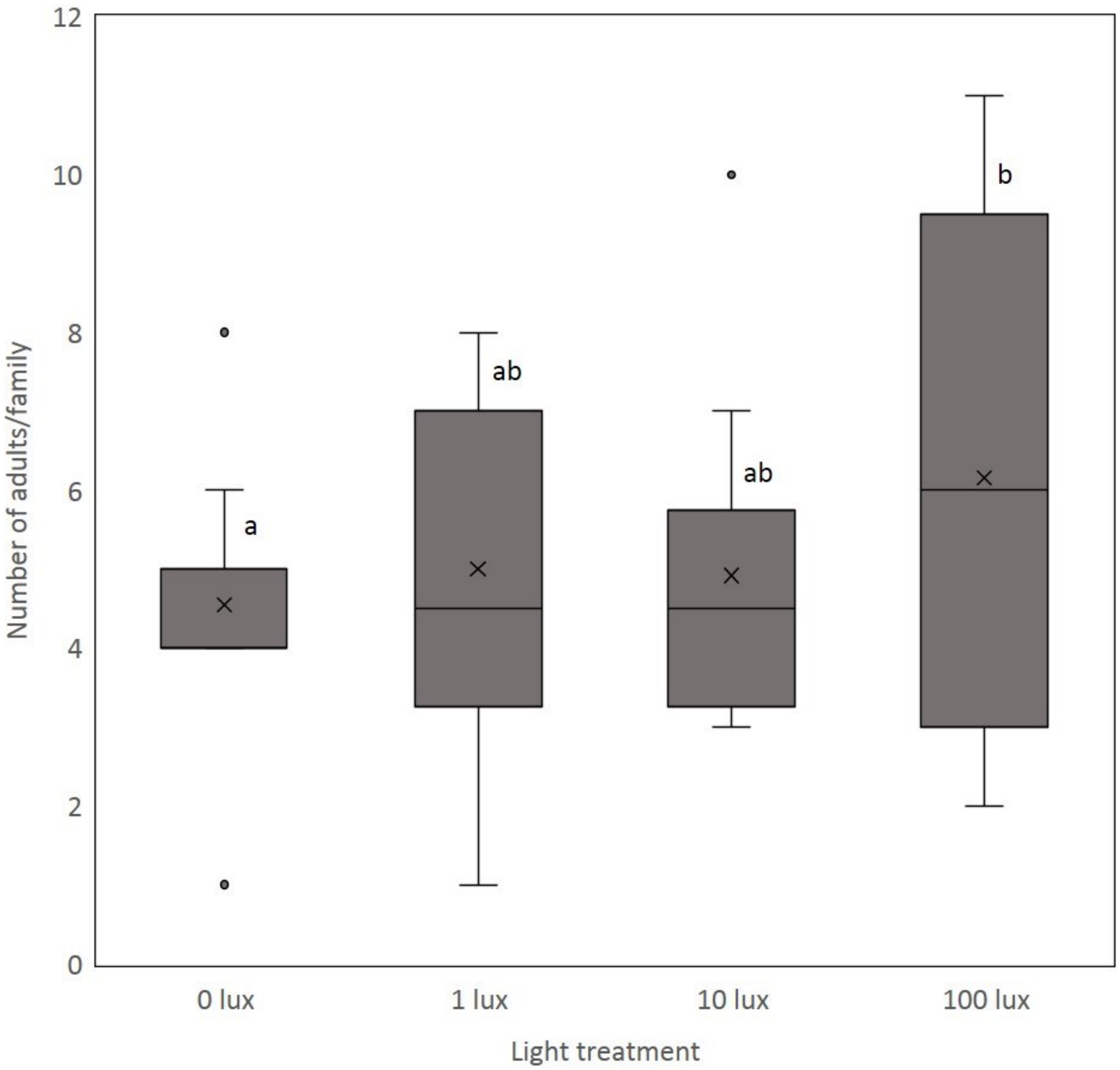
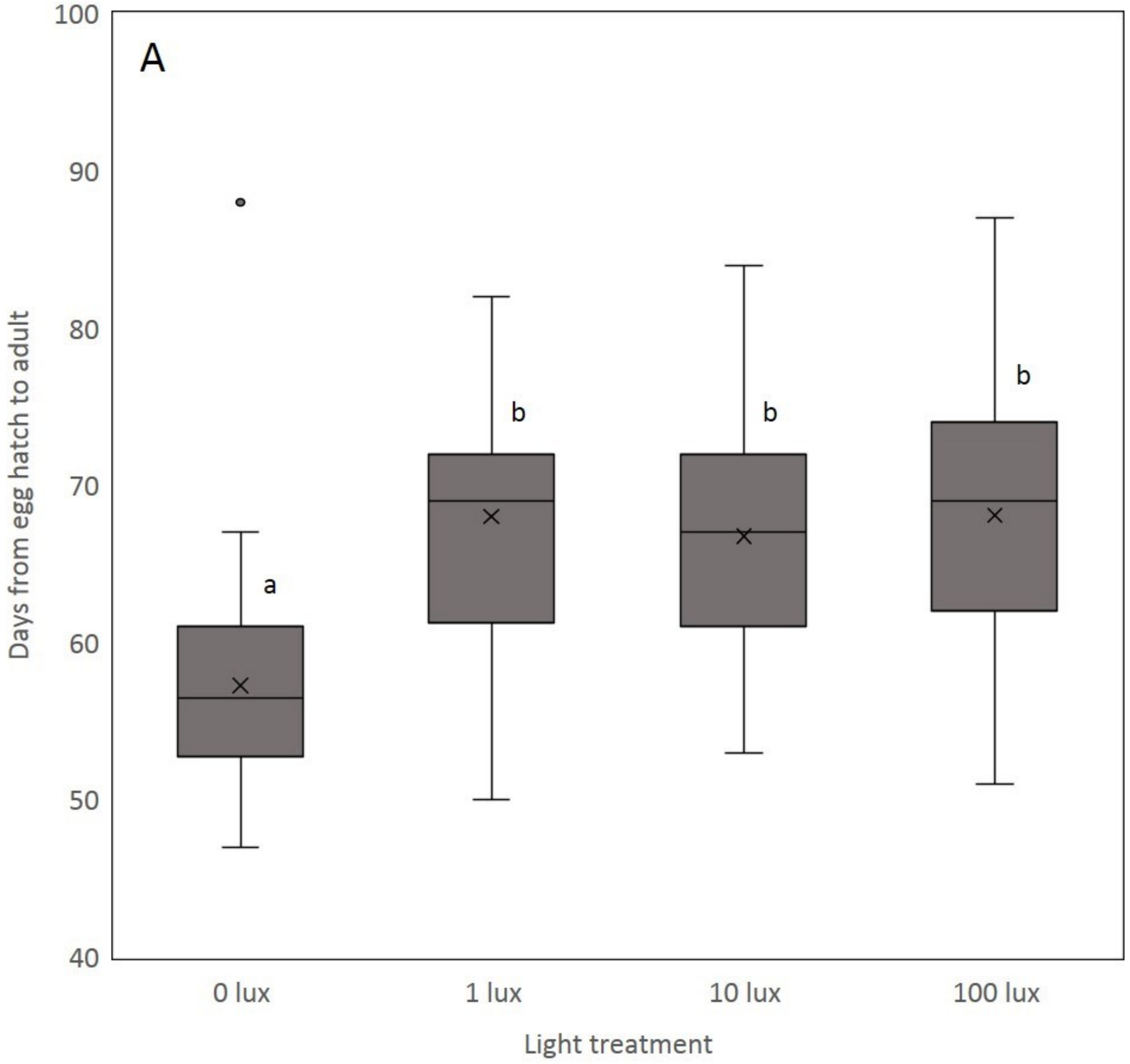


Figure 1. The median proportion of juveniles within each family surviving to complete the final moult for each of the four ALAN treatments (*n* = 13 families per light treatment). Box plots denote median and interquartile ranges, whiskers denote the lower 5th and upper 95th percentiles and the x denotes the mean; different letters denote significant ($P < 0.05$) differences between treatments.

FIGURE 2A


A
Days from egg hatch to adult
100
90
80
70
60
50
40
a
b
b
b
0 lux
1 lux
10 lux
100 lux
Light treatment

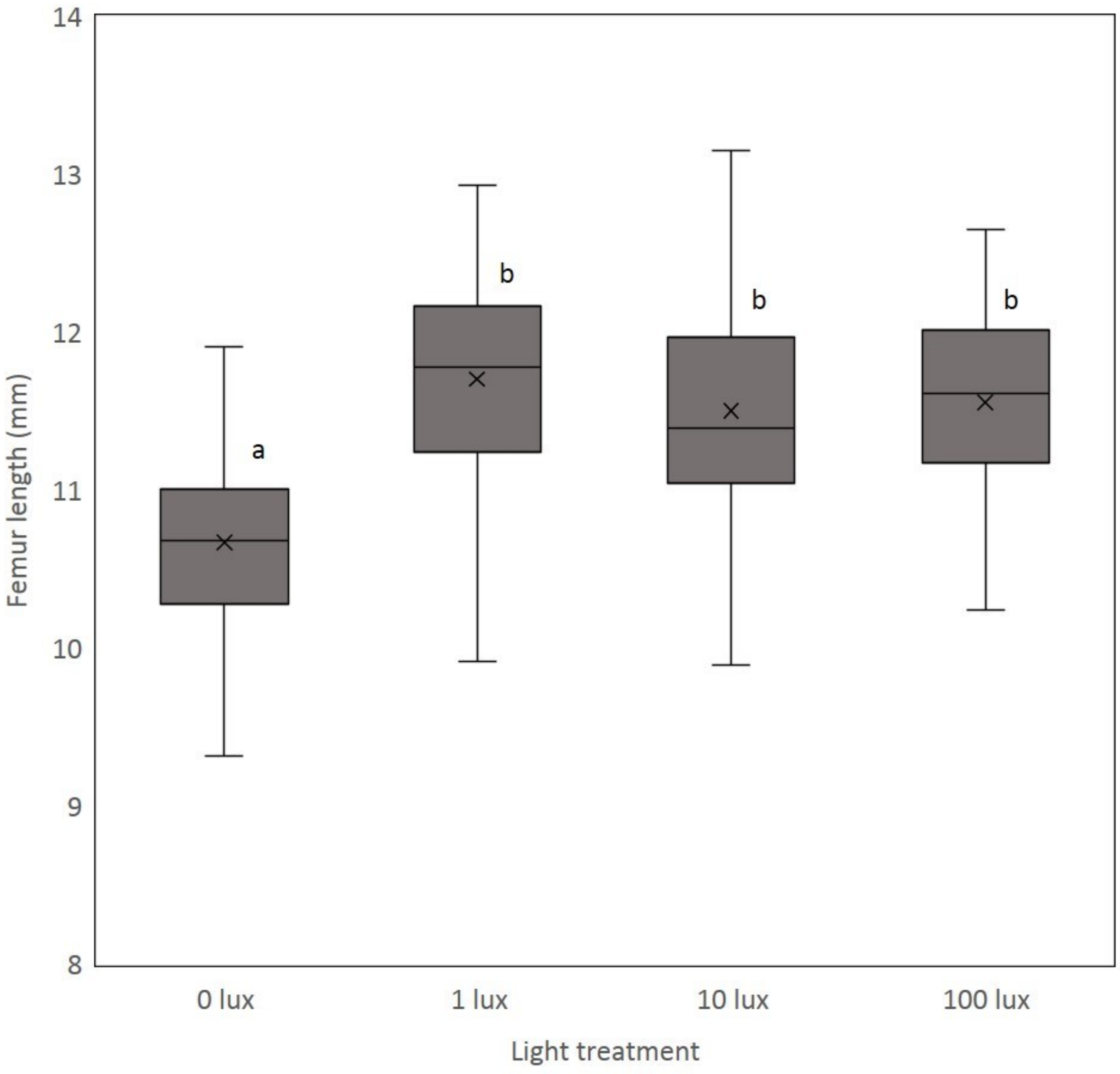


Figure 2. Median (**A**) juvenile development time (days) for crickets reared under 0 lux (n = 50), 1 lux (n = 60), 10 lux (n = 59), 100 lux (n = 80); and (**B**) femur length for crickets reared under 0 lux (n = 50), 1 lux (n = 62), 10 lux (n = 60), 100 lux (n = 84). Box plots denote median and interquartile ranges, whiskers denote the lower 5th and upper 95th percentiles and the x denotes the mean; different letters denote significant ($P < 0.05$) differences between treatments.