Frontiers in Neuroendocrinology 37 (2015) 52–64

Contents lists available at ScienceDirect

Frontiers in Neuroendocrinology

journal homepage: www.elsevier.com/locate/yfrne

Review

Annual gonadal cycles in birds: Modeling the effects of photoperiod on seasonal changes in GnRH-1 secretion

Alistair Dawson

Centre for Ecology & Hydrology, Bush Estate, Penicuik, Midlothian EH26 0QB, UK

ARTICLE INFO

Article history:
Available online 4 September 2014

Keywords:
Birds
GnRH1
Seasonality
Photoperiodism
Photorefractoriness
Starling
Quail

ABSTRACT

This paper reviews current knowledge of photoperiod control of GnRH-1 secretion and proposes a model in which two processes act together to regulate GnRH1 secretion. Photo-induction controls GnRH1 secretion and is directly related to prevailing photoperiod. Photo-inhibition, a longer term process, acts through GnRH1 synthesis. It progresses each day during daylight hours, but reverses during darkness. Thus, photo-inhibition gradually increases when photoperiods exceed 12 h, and reverses under shorter photoperiods. GnRH1 secretion on any particular day is the net result of these two processes acting in tandem. The only difference between species is their sensitivity to photo-inhibition. This can potentially explain differences in timing and duration of breeding seasons between species, why some species become absolutely photorefractory and others relatively photorefractory, why breeding seasons end at the same time at different latitudes within species, and why experimental protocols sometimes produce results that appear counter to what happens naturally.

1. Introduction

1.1. Photoperiodism

It is nearly 9 decades since Rowan (1925) first demonstrated that increasing daylength is the major cue used by birds to initiate gonadal maturation in spring. Strictly, it was not clear until later that light that was directly responsible, rather than light influencing the length of the bird's daily duration of activity (Bissonnette, 1931). Subsequently, the effect of photoperiod (p) on testicular maturation was quantified by transferring birds from a short photoperiod to various longer photoperiods and assessing the initial rate of increase in testicular mass (k) where k is log10 increase in testicular mass per day. In general, the rate of maturation was found to be proportional to photoperiod between photoperiods of 8 h of light:16 h of darkness (8L:16D) and 18L:6D day (Farner and Wilson, 1957; Follett and Maung, 1978). [Hereafter, photoperiod will just be referred to as the hours of light, e.g. 8L.] A similar relationship was found for ovarian growth in females (Farner et al., 1966).

Photoperiodic responses are dependent on an interaction between endogenous circadian clocks (Ball and Balthazart, 2003; Yasuo et al., 2003; Follett et al., 1992; Brandstätter, 2003; Brandstätter et al., 2001) and encephalic photoreceptors. Unlike mammals, birds do not use melatonin to relay photoperiodic information; they use photoperiodic information directly through photoreceptors located within the mediobasal hypothalamus (Benoit, 1964; McMillan et al., 1975; Oliver and Baylé, 1982; Saldanha et al., 1995; Saldanha et al., 2001). A variety of opsins have been suggested as the photopigment involved (Davies et al., 2012; Nakane et al., 2010; Wang and Wingfield, 2011). The magnitude of the photoperiodic response does not depend on an hour glass model whereby the response is related to the total number of hours of light; rather that it is dependent upon when light is perceived in relation to circadian time (Follett et al., 1992; Bűnning, 1960; Hamner, 1960; Hamner, 1963; Juss et al., 1995; Kumar et al., 1996; Menaker, 1971; Kumar et al., 2010; Hamner, 1964). The external coincidence model postulates that light has two functions: one to entrain the circadian clock and the other related to photoperiodic time measurement.

1.2. Neuroendocrinology of photostimulation

Photostimulation is essentially the control by photoperiod of the rate of secretion of gonadotropin releasing hormone 1 (GnRH1) from the median eminence. In the context of this paper, one particularly important fact is that the response is effectively immediate; the response to a particular photoperiod in terms of GnRH1 secretion happens on the same day as that photoperiod (Meddle and Follett, 1997; Nicholls et al., 1983). There are several isoforms of GnRH, but it is GnRH1 that controls the endocrine cascade leading

E-mail address: asda@ceh.ac.uk

http://dx.doi.org/10.1016/j.yfrne.2014.08.004
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to gonadal maturation (Sharp et al., 1990). GnRH1 cells are bi-laterally distributed along the third ventricle, and they send long projections to the median eminence where GnRH1 is secreted (Stevenson et al., 2012). Although thyroid hormones have for some time been known to play a key role in the photoperiodic responses (Dawson, 1993), the precise mechanism has only more recently been elucidated. Long photoperiods stimulate synthesis of thyrotropin-stimulating hormone beta (TSH-ß) in the pars tuberalis (Nakao et al., 2008; Stevenson and Ball, 2012), and this changes the ratio of the thyroid hormone enzymes deiodinase type II and type III (DiO_2 and DiO_3) in favor of DiO_2 leading to increased local production of triiodothyronine (T3) Watanabe et al., 2007; Yasuo et al., 2005; Yoshimura et al., 2003. T3 alters the structural arrangement of the GnRH1 terminals at the median eminence; the glial endfeet that ensheath the terminals retract and this allows increased secretion of GnRH1 (Yamamura et al., 2004, 2006).

This neuroendocrine mechanism titrates photoperiod. Under long photoperiods, the DiO_2/DiO_3 ratio increases leading to increased secretion of GnRH1, and under shorter photoperiods this is reversed. Presumably the relationship between photoperiod and the secretion rate of GnRH1 reflects the k/p relationship. Since the neuroendocrine response to an experimental acute increase in photoperiod is immediate, GnRH1 secretion rate at any one time is determined by the ambient photoperiod and the k/p relationship; therefore the secretion rate is proportional to p.

There are some unresolved questions relating to photoperiodic control. Firstly, to what photoperiod are birds responding; is it photoperiod as determined by sunrise to sunset, or do birds include civil twilight? Secondly, is the magnitude of the GnRH1 secretion rate directly related to ambient photoperiod, or does the direction and rate of change in photoperiod have an influence? Many experimental protocols involve an acute change in photoperiod which birds would never naturally experience. Does a particular photoperiod cause a different stimulus as photoperiod is decreasing as opposed to increasing? In other words, does the k/p relationship vary with the direction of change in photoperiod? In theory this could account for why species such as Japanese quail (*Coturnix coturnix*) show gonadal regression during the autumn under photoperiods that would have been gonado-stimulatory during spring (Robinson and Follett, 1982). This paper will argue against this explanation.

1.3. Photorefractoriness

If GnRH1 secretion is simply proportional to photoperiod, then the duration of full gonadal maturity, and hence the length of the breeding season, would always be symmetrical about the summer solstice. For a variety of ecological reasons, this is rarely, if ever, appropriate. Birds need to time their breeding attempts to the period when the food on which the nestlings depend is most abundant. Different species rely on different food supplies, and these will be available at different times and for differing durations. Consequently, breeding seasons of different species vary widely in duration and timing. Secondly, young birds need time to develop somatically and behaviorally sufficiently to survive the ensuing winter. Birds also normally need time to molt after breeding. Breeding seasons tend to be in spring or early summer, rather than later.

Many species of birds show gonadal regression well before the return of short photoperiods during autumn and are said to become photorefractory (Burger, 1949). If experimental birds are moved from a short photoperiod to a long photoperiod, the initial response is gonadal maturation, but later the gonads regress as birds become photorefractory (Burger, 1949; Miller, 1954). In such situations, the timing of the onset of photorefractoriness is inversely proportional to the photoperiod (Burger, 1952; Dawson and Goldsmith, 1983). Short photoperiods, normally during autumn, are required to end the photorefractory period and restore birds' ability to respond to an increase in photoperiod (Farner and Mewaldt, 1955). Photorefractoriness was considered to prevent normal photoperiodic responses, hence the term photorefractoriness. Clearly photorefractory birds are still monitoring photoperiod, because short photoperiods dissipate photorefractoriness, so the term is somewhat misleading. Furthermore, the term has a very different meaning in mammals (see Section 4).

1.4. Neuroendocrinology of photorefractoriness

In contrast to the recent advances in understanding the neuroendocrine changes associated with photostimulation, comparatively little is known about events during the onset of photorefractoriness. The first major advance was the surprising finding that hypothalamic stores of the GnRH1 peptide decrease 100-fold at the onset of photorefractoriness in common starlings (*Sturnus vulgaris*) Dawson et al., 1985, 2002; Foster et al., 1987; Goldsmith et al., 1989. Juvenile starlings develop in a state equivalent to photorefractoriness (Williams et al., 1987; McNaughton et al., 1992) with low hypothalamic GnRH1. This increases during short photoperiods (Dawson and Goldsmith, 1989) in the same way as it does when photorefractory adults are moved to short photoperiods (Dawson et al., 1986). Thus the recovery from photorefractoriness is essentially a repeated puberty. During the recovery from photorefractoriness there is first a measurable increase in GnRH1 in the preoptic area followed by an increase in the median eminence (Dawson and Goldsmith, 1997). At the same time, there is a rapid increase in circulating gonadotropins in gonadectomized birds (Dawson and Goldsmith, 1984). Marked seasonal changes in hypothalamic GnRH1 content have been found in other Passeriforme species e.g. house sparrows (*Passer domesticus*) Hahn and Ball, 1995; Stevenson and MacDougall-Shackleton, 2005, American tree sparrows (*Spizella arborea*) Reinert and Wilson, 1996, house finches (*Carpodacus mexicanus*) (Cho et al., 1998), dark-eyed juncos (*Junco hyemalis*) Deviche et al., 2006; Meddle et al., 2006, and rufous-winged sparrows (*Aimophila carpolis*) (Small et al., 2008). These data led to the following model of seasonality (Dawson et al., 2001). During increasing photoperiods of spring, GnRH1 secretion increases, leading to gonadal maturation. GnRH1 synthesis increases at least sufficiently to compensate for increased secretion because hypothalamic stores increase slightly. During exposure to long photoperiods, some unknown process leads to the onset of photorefractoriness at which point GnRH1 synthesis ceases. The drive on GnRH1 secretion may still be high, because photoperiod is long, but since synthesis has ceased, no GnRH1 is secreted and the gonads regress. GnRH1 synthesis resumes during short photoperiods of autumn. However, because photoperiods are then short, there is little secretion and so little gonadal maturation until increasing photoperiods during the following spring. Thus, photoperiod has two effects. Long photoperiods initially induce increased secretion of GnRH1 and they also later lead to an inhibition of GnRH1 synthesis. In the absence of thyroid hormones, photorefractoriness does not develop (Wieselthier and van Tienhoven, 1972; Woitkewitsch, 1940) and is not maintained (Dawson et al., 1985) suggesting that the inhibition of GnRH1 synthesis is an active thyroid-dependent process. Increased synaptic input to the GnRH1 cell bodies is seen in photorefractory birds (Parry and Goldsmith, 1993).

The absence of an available GnRH1 gene sequence in songbirds prevented confirmation of this model for some time. A major breakthrough came with the cloning of the GnRH1 gene (Stevenson et al., 2009; Stevenson et al., 2013; Ubuka and Bentley, 2009; Ubuka et al., 2009). This resulted in confirmation that GnRH1 mRNA levels in the medial preoptic area decline in parallel with the onset of testicular regression under long photope-

riods to very low values. Conversely, GnRH1 mRNA levels increase as photorefractoriness is terminated during short photoperiods (Stevenson et al., 2012). Thus, photorefractoriness is indeed associated with marked changes in GnRH1 gene expression. This degree of GnRH1 gene plasticity is not seen in mammals and may have evolved in birds because of their restricted breeding seasons (Stevenson et al., 2012).

1.5. Relative photorefractoriness

This description of photorefractoriness is typical of birds whose breeding season ends comparatively early, often while photoperiod is still increasing before the summer solstice. Such species show spontaneous gonadal regression under chronic long experimental photoperiods and do not show renewed gonadal maturation following subsequent transfer to an even longer photoperiod. These species are said to become absolutely photorefractory. Some species have longer breeding seasons and, under experimental conditions, do not show spontaneous gonadal regression during chronic long photoperiods. However, they do show regression when photoperiod decreases, even though the photoperiod that induces regression can be longer than that which stimulated gonadal maturation earlier in spring (Robinson and Follett, 1982; Follett and Pearce-Kelly, 1990). Renewed gonadal maturation can be stimulated at any time by transfer to a long photoperiod. Such species are said to be relatively photorefractory. The classic experimental species showing this is the Japanese quail. A major difference between absolute and relative photorefractoriness is that gonadal regression induced by the latter is not associated with a decrease in hypothalamic GnRH1 peptide (Foster et al., 1988). Changes in reproductive function in quail are primarily controlled at the level of GnRH1 secretion (Stevenson et al., 2012). However, like absolute photorefractoriness, relative photorefractoriness is thyroid-dependent (Follett and Nicholls, 1984; Follett and Potts, 1990). Other species of birds can show elements of both absolute and relative photorefractoriness (Stevenson et al., 2012; Hahn et al., 2004; MacDougall-Shackleton et al., 2001; MacDougall-Shackleton et al., 2006; Marsh et al., 2002; Dawson, 1998). For example, house sparrows kept on constant long photoperiods show spontaneous gonadal regression, but regression can be advanced by a decrease in photoperiod. Absolute and relative photorefractoriness permit different species to end breeding at different times of the year.

1.6. The timing of breeding seasons at different latitudes

Within species, breeding seasons tend to be longer at lower latitudes. Breeding starts earlier at lower latitudes and this can be explained because gonadal maturation starts (and is often nearly complete) before the spring equinox when photoperiod will be longer at lower latitudes (Dawson, 2013). However, within species (both those that show absolute and relative photorefractoriness) gonadal regression occurs at the same time at different latitudes even though birds at higher latitudes will experience longer photoperiods. Similarly, starlings in captivity on photoperiods that simulate annual cycles at 52°N and 9°N also show earlier testicular maturation at 9°N, but the timing of regression is the same for both latitudes even though the 52°N birds experience much longer photoperiods (Dawson, 2007). Yet in birds on constant experimental photoperiods, those on longer photoperiods become photorefractory sooner. The reason for this apparent dichotomy between experimental and real situations is not known.

1.7. Unresolved problems

It will be clear from the introduction above that there are several unresolved questions:

1. What photoperiod do birds use – sunrise to sunset or including civil twilight?
2. Are responses related to prevailing absolute photoperiod or is the rate of change in photoperiod and the direction of change important?
3. Why do breeding seasons end at the same time at different latitudes when birds will have experienced different changes in photoperiod? In experimental birds, the onset of photorefractoriness is inversely proportional to photoperiod.
4. Why do some species need to perceive a decrease in photoperiod to induce gonadal regression (relative photorefractoriness) and can always respond to an increase in photoperiod while others show spontaneous regression under long photoperiods and then show no response to an increase in photoperiod (absolute photorefractoriness).
5. Why does hypothalamic GnRH1 peptide change profoundly during absolute photorefractoriness but not in relative photorefractoriness?

1.8. Aims

There has been the view that birds are either photosensitive, when they are responsive to changes in photoperiod, or photorefractory, when they are insensitive to an increase in photoperiod and that these two states are mutually exclusive. The aim of this paper is to propose a model in which two photo-neuroendocrine processes act in tandem at all times of the year to regulate GnRH1 secretion. The first, photo-induction, is an effect whereby GnRH1 secretion is directly related to the prevailing photoperiod. The second, photo-inhibition, is a longer term process acting through changes in GnRH1 synthesis. Can this model answer any of the unresolved problems? The model will be developed using two species at each end of the seasonality spectrum: starlings which have a short breeding season early in the year and become absolutely photorefractory, and quail, which have a long breeding season lasting until late summer and which show relative photorefractoriness. A wealth of experimental data exists for these species. In the majority of this paper, data and modeling refer to males because more data is available for males. Also, males can achieve near full gonadal maturation through photoperiodic effects alone whereas females need supplementary stimuli for full ovarian maturation leading to ovulation (Ball and Ketterson, 2008). However, the underlying neuroendocrine photoperiodic mechanisms are probably similar between the sexes. For example, seasonal changes in circulating concentrations of gonadadtropins in gonadectomized birds of both sexes are similar suggesting that photoperiodically-induced neuroendocrine changes upstream of the pituitary are the same (Dawson and Goldsmith, 1984). Both male and female starlings show the same dramatic changes in GnRH1 gene expression (Ubuka et al., 2009).

2. Developing the model

2.1. What photoperiod do birds use?

Sunrise and sunset conventionally refer to the times when the upper edge of the disk of the sun is on the horizon. Before sunrise and again after sunset there is twilight, during which there is natural light provided by the upper atmosphere, which does receive direct sunlight and reflects part of it toward the Earth's surface. Civil twilight is defined to begin in the morning, and to end in the evening when the center of the sun is geometrically 6° below the horizon. For example, at 56°N (the latitude of Edinburgh), photoperiod including civil twilight is 1.2 h longer than sunrise to sunset at the equinoxes, and 2.1 h longer at the summer solstice. Birds

measure photoperiod, but which photoperiod do they use, sunrise to sunset or do they include civil twilight? In some photoperiodic studies on birds, it has been assumed to be civil twilight, since this is the duration of "usable" light. I examined this by comparing testicular cycles in starlings under a simulated natural cycle in photoperiod that was either calculated as sunrise to sunset or included twilight, and compared these to another group exposed to natural daylight (Fig. 1). The changes in testis size in the sunrise to sunset group were more closely aligned to natural daylight birds than those under civil twilight. As I shall show later, during the whole year the effects of long photoperiods need to be balanced by the effects of short photoperiods for cycles to last 12 months. Symmetry is required and is provided by sunrise to sunset (mean photoperiod of 12.0 h during the year), rather than civil twilight. If starlings, and some other species, are kept under constant photoperiods of 12L (sunrise to sunset at the equator) they undergo circannual cycles of testis size and molt (Dawson, 1997; Gwinner, 1996; Gwinner, 2003; Rutledge, 1974). If they are kept under a constant photoperiod which includes civil twilight at the equator (about 12 h and 50 min), they perceive this as constant long photoperiods and do not show repeated cycles (Dawson, 2007; Gwinner and Wozniak, 1982). How birds measure sunrise to sunset is unclear. However, it is known that a higher light threshold is required for photostimulation than to entrain circadian rhythms (Menaker and Keatts, 1968), and also the spectral quality of light changes at sunrise, so different photoreceptors may play a role. In starlings held on an 18L photoperiod, but at different low light intensities, there was a graded response in both the rate of testicular maturation and the time to the onset of regression (Bentley et al., 1998).

2.2. Photo-induction

In an effort to understand the physiology underlying photoperiodic control, many studies have used the paradigm of transferring birds from a short to different constant longer photoperiods. The initial rate of gonadal maturation is a function of that longer photoperiod. Specifically, the logarithmic rate of testicular growth (k) has a nearly linear relationship with photoperiod (p) Farner and Wilson, 1957. This k/p relationship has been calculated for a number of species (Follett and Maung, 1978; Farner and Lewis, 1971) and has a linear relationship over a wide range of photoperiods (typically 8L to 16L). Although this clearly demonstrates that the rate of maturation is greater on longer photoperiods, it must be borne in mind when estimating growth rates at different times of year and at different latitudes that this is an entirely unnatural scenario. Firstly, free-living birds will rarely experience an acute change in photoperiod (rapidly migrating birds may be an exception). Secondly, by the time birds naturally experience fairly long photoperiods, they already will have undergone a degree of gonadal maturation and gonadal steroid feedback may tend to inhibit gonadotropin secretion. The k/p relationship therefore may exaggerate growth rate at longer photoperiods.

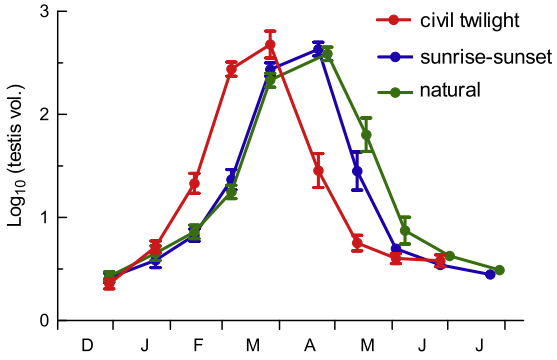

Fig. 1. Do birds use sunrise–sunset or include twilight as the photoperiodic signal? Juvenile male starlings were caught from the wild in August and kept in an indoor aviary with a translucent roof and so exposed to natural daylight. In mid December two groups of birds were moved into controlled-environment chambers (3.5 m × 2.5 m × 2.2 m) provided with artificial light (about 1000 lux) in which photoperiod simulated either natural changes from sunrise to sunset at 56°N (blue line) or included civil twilight at 56°N (red line). Data were obtained from US Navy Observatory Astronomical Applications Department: http://aa.usno.navy.mil/index.php. In each case the period of increasing or decreasing light intensity at the start or end of each day lasted just 1 min. Photoperiod was changed each day in a pre-programmed sequence. These two groups were compared to another group which remained in the indoor aviary exposed to natural daylight (green line). The figure shows changes in testis sizes in the three groups (mean ± SEM, $n = 8$ for each group). Clearly changes in the sunrise to sunset group were more closely aligned to natural daylight birds than those under civil twilight. The latter group showed earlier and more rapid testicular maturation and an earlier onset of regression.

In order to model the effect of photoperiod on photo-induction during natural annual cycles, it is necessary to evaluate the rate of testicular growth (k) at different photoperiods (p) during natural incremental changes in photoperiod rather than following an acute change to that photoperiod from a short photoperiod. This was done by keeping male starlings under simulated natural sunrise to sunset photoperiods from the winter solstice as described above (Fig. 2). The relationship between k and p was a linear regression ($k = (p \times 0.0118) - 0.09$).

In an experimental situation, following an acute increase in photoperiod from a short photoperiod, the initial rate of testicular maturation in starlings is proportional to the new longer photoperiod (Fig. 3). The initial rate of testicular growth in these birds was close to that predicted using k derived from birds under naturally increasing photoperiods. This close agreement suggests that photoperiodic drive at any time is determined by absolute photoperiod at that time and is not affected by the rate of change in photoperiod or photoperiodic history. In starlings transferred from 8L to 13L, the longer photoperiod is perceived as long and eventually induces photorefractoriness. In photorefractory birds transferred from 18L to 13L, the same magnitude of change but in the other direction, 13L is still perceived as long and birds remain photorefractory (Dawson, 1987). This is important – it means that photoperiodic drive is simply dependent on the prevailing photoperiod and irrespective of the rate or direction of change in photoperiod. This means that the relationship between k and p remains the same at different latitudes, where the rate of change in photoperiod differs, and at different times of year, when the direction of change in photoperiod differs. In Japanese quail, which become relatively photorefractory, transfer from a short photoperiod to a moderately long photoperiod induces gonadal maturation, whereas a decrease in photoperiod from a long photoperiod to the same moderately long photoperiod induces regression. This may not be due to photoperiodic history affecting the relationship between k and p, but rather to the development of photoinhibition.

2.3. Photo-inhibition

What are the properties of photorefractoriness, or the photoinhibitory process, that may be useful in developing a hypothesis to explain differences in seasonal cycles between species and within species at different latitudes? Firstly, in general the process does not start until photoperiod exceeds 12L. Birds do not become photorefractory under short photoperiods, and they do not terminate photorefractoriness under long photoperiods. In fact this statement may not be strictly true in all cases and there may be some flexibility. In starlings, and several other species, it has been shown that birds held on constant 12L photoperiods can show repeated cycles of gonadal maturation and regression with a peri-

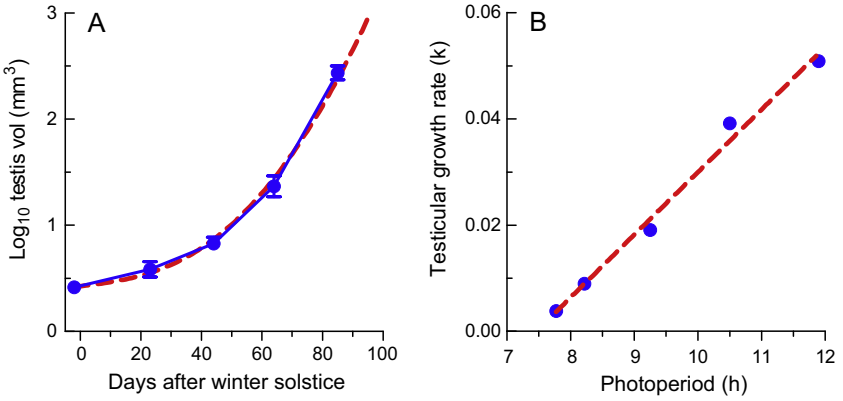

Fig. 2. Estimating *k*. In order to model annual changes in testicular size, it is necessary to evaluate the rate of testicular growth (*k*) at different photoperiods during natural incremental changes in photoperiod rather than following an acute change to that photoperiod from a short photoperiod. Male starlings were kept in a controlled-environment chamber provided with artificial light in which photoperiod simulated natural changes from sunrise to sunset at 56°N from the winter solstice (as described in Fig. 1). Birds were laparotomized to determine testicular volume on five occasions during testicular maturation and during which photoperiod increased from 7.8 h to 11.9 h (A, blue points and line mean ± SE). The rate of testicular growth (*k*) at each point was calculated and plotted against photoperiod at that time (B, blue points). The linear regression of these points was calculated (B, dashed red line). The regression was $k = (p \times 0.0118) - 0.09$ where *p* is photoperiod. Using this regression equation to calculate daily increments in testis size from initial values gives a close fit to actual increases (A, red dashed line).

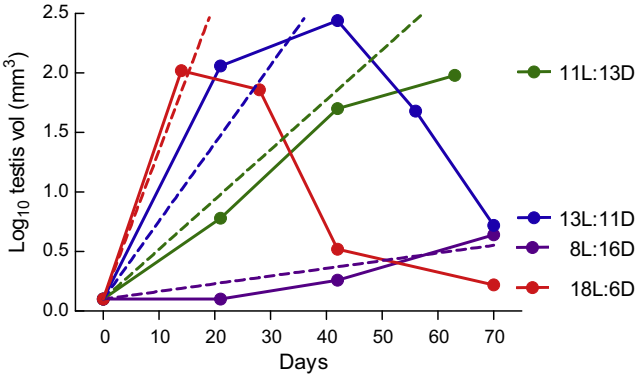

Fig. 3. Changes in testis size in starlings following an acute change in photoperiod from 8L to 18L (red line), 13L (blue line), 11L (green line) or kept on 8L (purple line). These data are re-drawn from Dawson and Goldsmith (1983). The initial rate of testicular maturation was proportional to photoperiod. Birds under the two longer photoperiods showed subsequent testicular regression and the time of regression was inversely proportional to photoperiod. Under 18L, regression began before full testicular maturation was attained. There was no regression under the two shorter photoperiods. The broken lines show predicted rates of maturation using the relationship between the rate of maturation (*k*) and photoperiod (*p*): $k = (p \times 0.0118) - 0.09$, which was derived from birds under naturally increasing photoperiods (Fig. 2) rather than following an acute change in photoperiod. The close agreement suggests that photoperiodic drive art any time is determined by absolute photoperiod at that time and is not affected by the rate of change in photoperiod or photoperiodic history.

odicity that, while it can vary widely between individuals, is often about 12 months – circannual cycles (Dawson, 1997; Gwinner, 1996, 2003; Rutledge, 1974). The periods of maturation and regression can be similar to those under natural seasonal cycles. Presumably 12L is perceived sequentially as a long photoperiod to induce maturation and then photorefractoriness, and then a short photoperiod to terminate photorefractoriness. Starlings held chronically on 11.5L also show repeated cycles, but these comprise long periods of maturity interspersed with short periods of regression (Dawson, 2007). Starlings kept under chronic 11L:13D never become photorefractory, but they may show some progression towards photorefractoriness; after chronic 11L they become photorefractory more rapidly following transfer to a long photoperiod than birds held for only a short time on 11L (unpublished data). Under 12.5L or 13L, starlings remain photorefractory indefinitely (Dawson, 2007; Gwinner and Wozniak, 1982). However, for the purposes of this modeling exercise, I will initially assume that the photo-inhibition process starts only when photoperiod exceeds 12L.

Secondly, the process of photo-inhibition is progressive. Although photo-induction starts immediately a bird perceives an increase in photoperiod, the effects of photo-inhibition do not become apparent until some weeks later. In starlings transferred from 8L to 18L, gonadal regression starts 2–3 weeks later. This is the point at which the photo-inhibitory process exceeds photo-induction (Fig. 3). However, the process of photo-inhibition, which leads to regression, starts before then. In starlings held chronically on 11L, not long enough to induce regression, and transferred to 18L for varying periods before return to 11L, 7 long days is sufficient to induce subsequent regression (Dawson et al., 1985). However, shorter periods of three long days, or even just one long day, pre-dispose birds to become photorefractory sooner on subsequent transfer to long photoperiods. A photo-regime comprising one long photoperiod every 10 days can hold starlings in a permanent semi-photorefractory state (Dawson, 2001). Each long photoperiod causes a degree of photo-inhibition which is reversed during the following 9 short photoperiods. This progressive feature is also true of the reverse procedure – the termination of photorefractoriness. If photorefractory starlings are transferred to short photoperiods, it takes 10 weeks before photosensitivity is fully restored. But there is a partial return to photosensitivity after just 4 weeks and transfer back to long photoperiods between 4 and 10 weeks induces sub-maximal photo-induction and a more rapid return to photorefractoriness (Boulakoud and Goldsmith, 1995; Dawson, 2004). Similarly, the return to photosensitivity from relative photorefractoriness in quail is gradual (Follett and Pearce-Kelly, 1991). After transfer from long photoperiods to 8L to induce regression, there was no response to 13L after one week of short days, a minor response after two weeks, a strong response after three weeks and a full response after five weeks.

Thirdly, the longer the photoperiod (in excess of 12L) the sooner birds become photorefractory. In starlings following transfer to 18L, regression starts after 2–3 weeks, but after about 5 weeks under 13L (Fig. 3). In starlings held chronically on 11L and transferred to 18L for varying periods before return to 11L, 7 long days is sufficient to induce subsequent regression (Dawson et al., 1985), but if transferred to 13L rather than 18L, then 46 days is required (Falk and Gwinner, 1988). Again, the reverse is true for the termi-

nation of photorefractoriness – the shorter the photoperiod below 12L the sooner birds return to photosensitivity (Boulakoud and Goldsmith, 1994; Dawson, 1991). In relatively photorefractory quail transferred to short photoperiods, there is no responses to 13L or 14L after one week, but there is a moderate response to 16L (Follett and Pearce-Kelly, 1991). So in quail the depth of photorefractoriness is dependent upon the previous duration of exposure to long photoperiods and to length of that long photoperiod.

Finally, photo-inhibition progresses independently of photo-induction (photo-induction starts under photoperiods too short to induce any progress towards photo-inhibition).

In conclusion, photo-inhibition is progressive. It starts when photoperiod exceeds 12L and progresses more rapidly under longer photoperiods. This may suggest that birds are summing the number and length of long photoperiods, accumulating the total number of light hours in excess of 12 h, and progressively building up the photo-inhibitory process until it equals the photo-inductive drive, at which point gonadal regression starts and birds become photorefractory. However, it is difficult to conceive a neuroendocrine process that could do this. However, the same effect could be achieved if there was a process which each day progressed towards photo-inhibition during daylight hours and was reversed during darkness. Thus, net progress towards inhibition only progresses when photoperiod exceeds 12L, but progresses more rapidly as photoperiod increases further beyond 12L. The reverse would be true under photoperiods shorter than 12L.

2.4. Modeling seasonal changes in photo-induction and photo-inhibition

So the hypothesis I am suggesting is this: photo-induction and photo-inhibition are two independent processes and the rate of gonadal maturation or regression at any one time is the net difference between these two processes at that time. Photo-induction is proportional to prevailing photoperiod, as has been shown in many studies (the k/p relationship). Photo-inhibition progresses during daylight and is reversed in darkness. So the rate of progression of photo-inhibition is related to the length photoperiod in excess of 12L. The degree of photo-inhibition is a function of photoperiod and time. This hypothesis is developed in Figs. 4 and 5.

2.5. Photo-induction and photo-inhibition under constant experimental photoperiods

Figs. 4 and 5 show how this hypothesis can potentially explain a mechanism by which different species end their breeding seasons at different times of the year. It can also explain why, within species, breeding seasons end at the same time at different latitudes, despite the differences in photoperiod that they would experience. However, there is an apparent anomaly. If birds experiencing different photoperiods at different latitudes show gonadal regression at the same time, why do experimental birds transferred from short photoperiods to different long photoperiods show earlier regression under longer photoperiods. For example, starlings transferred to 18L show regression after 2–3 weeks, whereas birds moved to 13L show regression after 6 weeks (Fig. 3).

Photo-induction is proportional to prevailing photoperiod. It is equivalent to testicular growth rate but only under photoperiods less than 12L. Under longer photoperiods testicular growth rate is also affected by the development of photo-inhibition. Using the equation that the rate of testicular growth $k = (p \times 0.0118) - 0.09$ where p is photoperiod, which was derived from birds under a simulated natural increase in photoperiod (Fig. 2), then in birds transferred from 8L to 13L, k increases from 0.006 to 0.065 and in birds moved to 18L it increases to 0.124, approximately double that under 13L (Fig. 5). Under 13L the cumulative hours in excess of 12 h increases at 1 h per day, whereas under 18L it increases at 6 h per day. In this situation, birds under 13L should show gonadal regression after three times the length of time shown by birds under 18L. This exaggerates the differences between what actually happens between the two groups (Fig. 3). However, as explained in Section 2.3, the photo-induction process does not suddenly start at exactly 12 h – there is a degree of plasticity. Birds under 11L never become photorefractory but do show some progression towards it. Birds under 11.5L do eventually become photorefractory, but then show abnormal repeated testicular cycles. Birds under 12L can show cycles similar to seasonal cycle. If the figure for cumulative hours in excess of 11.5 h is used rather than 12 h, then this predicts that birds under 13L will show testicular regression in somewhat over twice the time taken by birds under 18L, (Fig. 6) which is similar to what actually happens. At 52°, photoperiod increases from 11.5L to 12L in just 7 days, so the consequence of photo-induction gradually starting at 11.5L, rather than at 12L, on seasonal gonadal cycles would be much less significant than on constant experimental photoperiods. Although using hours in excess of 11.5L appears to fit the data from Dawson and Goldsmith (1983), it is interesting to note that in starlings held chronically on 11L and transferred to 18L for varying periods before return to 11L, 7 long days is sufficient to induce subsequent regression (Dawson et al., 1985), which equals $7 \times 6 = 42$ excess hours, but if transferred to 13L rather than 18L, then 46 days is required (Falk and Gwinner, 1988), which equals $46 \times 1 = 46$ excess hours – quite good agreement.

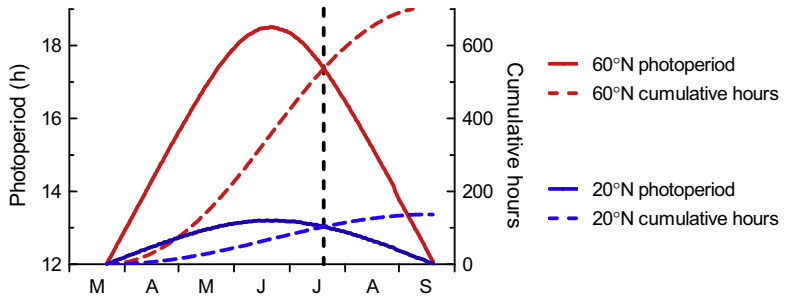

Fig. 4. Photo-induction and photo-inhibition during the year. The solid red line shows photoperiod during the summer months at 60°N, increasing from 12 h at the spring equinox to 18.5 h at the summer solstice. The broken red line shows the cumulative hours in excess of 12 h at 60°N. This is 0 at the equinox, increases slowly initially when photoperiod is just over 12 h, increases at a maximal rate at the summer solstice and then flattens off at about 700 h as photoperiod again decreases below 12 h. The hypothesis is that photo-induction is related to photoperiod and photo-inhibition to the cumulative hours. Net drive on GnRH1 secretion is the difference between the two. Thus testicular regression starts at the time that the two lines cross, after which photo-inhibition exceeds photo-induction. In this example, this happens in July, shown by the broken vertical line. This is typical of many temperate zone species with moderately long breeding seasons such as greenfinches (*Carduelis chloris*) (I have kept greenfinches under simulated natural photoperiods, and testicular regression occurs then). The solid and broken blue lines also show photoperiod and cumulative hours respectively, but in this case at 20°N. The two lines cross at the same time as those for 60°N, suggesting that testicular regression starts at the same time at different latitudes, exactly as appears to happen in free-living birds (Dawson, 2013).

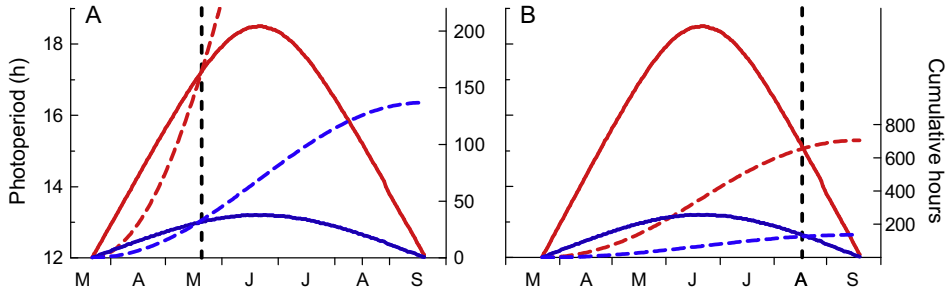

Fig. 5. Species differences in photo-inhibition. Obviously, different species have breeding seasons of different durations, and show gonadal regression at different times of the year. Figures A and B both show exactly the same data as in Fig. 4. The only difference is the scale of the right vertical axis, showing cumulative hours. In A, the scale has been extended. This represents the situation in birds with a high propensity to become photorefractory, species that become absolutely photorefractory and have a breeding season very asymmetrical with respect to photoperiod. In this example, gonadal regression would occur in May, before the summer solstice, as it does in starlings (Dawson, 2013). In contrast in B, the right axis has been shortened, representing species with a low propensity to become photorefractory, species that show relative photorefractoriness and have a breeding season only slightly asymmetrical with respect to photoperiod. In this example, gonadal regression would occur in August, well after the summer solstice but before the equinox. This is the situation in Japanese quail (Robinson and Follett, 1982). In both cases, the time of regression is the same at different latitudes. The only inter-species difference needed for gonadal regression to happen at different times of the year is a difference in the sensitivity to cumulative hours – a difference in the degree of photo-inhibition induced by the same amount of accumulated hours.

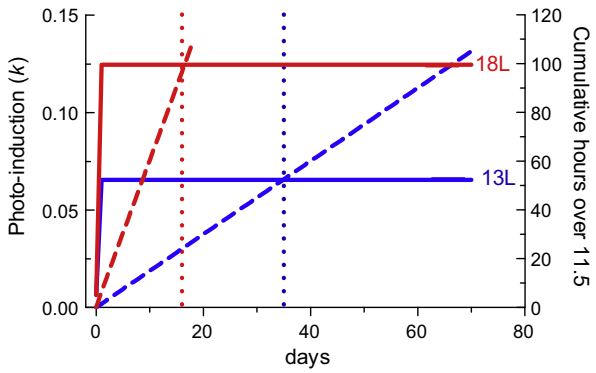

Fig. 6. Photo-induction and photo-inhibition during constant photoperiods. The solid lines represent the increase in photo-induction (k), calculated as $k = (p \times 0.0118) - 0.09$ where p is photoperiod, in birds transferred from 8L to 13L (blue line) or 8L to 18L (red line). The increase in cumulative hours in excess of 11.5 h is shown by the respective broken lines. The lines cross after 35 days under 13L and after 16 days under 18L (shown by the vertical dotted lines).

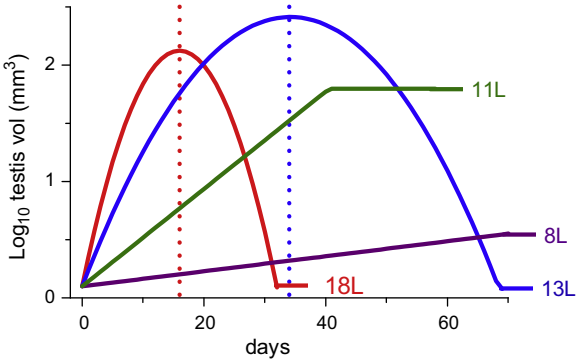

Fig. 7. The modeled changes in testis size predicted by Fig. 6 in starlings transferred on day 0 from a photoperiod of 8L to 18L (red line), 13L (blue line), 11L (green line) or kept on 8L (purple line). The model is based on the difference between photo-induction (equivalent to k), which remains constant because photoperiod remains constant and photo-induction (related to the cumulative hours in excess of 11.5 h). Daily increments in testis size are calculated from a known initial value. It is assumed that under the two shorter photoperiods, testicular growth eventually ceases at a size where the effects of photo-induction are balanced by gonadal steroid feedback. The vertical dotted lines show where photo-induction equals photo-inhibition for the two longer photoperiods and where regression begins. These are the same as in Fig. 6. Beyond these points net drive is negative. This model gives good agreement with what actually happens (Fig. 3). The model predicts that under 18L regression starts before maximal testis size is attained. It also predicts that changes in the two longer photoperiods are symmetrical about the point that regression starts. This is because photo-induction remains constant and photo-inhibition increases at a constant rate. This would not happen under natural seasonal cycles because in that case photoperiod would continuously change.

If the solid lines in Fig. 6 represent photo-induction, which in this case is constant because photoperiod is constant, and the broken lines the build-up of photo-inhibition, then resultant drive leading to an increase in testicular size at any time, the drive on GnRH1 secretion, is equal to the distance between the two lines (from the broken line up to the solid line). So we should be able to predict not just the time of regression but also the pattern of gonadal maturation leading up to, and possibly even beyond, the start of regression. The initial testicular growth rates are simply equivalent to k. For birds remaining on 8L and those transferred to 11L, there is no accumulation of photo-induction, so testis size follows these initial trajectories. But for 13L and 18L we can calculate resultant drive as photo-inhibition accumulates. In Fig. 6, the right vertical scale has been adjusted so that the lines representing photo-induction and photo-inhibition cross at the appropriate times. Since photo-induction and photo-inhibition are equal at those times, equivalence between k and cumulative hours can be calculated. The resultant drive on testicular growth before the lines cross is then quantifiable. In theory, the (negative) difference between the two lines after they cross may also predict the rate of testicular regression. The results of this modeling are shown in Fig. 7.

In the example above, the excess of photo-induction over photo-inhibition equals the rate of increase in testis size, until the two are equal, after which regression ensues. In this model, the rate of regression, when photo-inhibition is greater that photo-induction, is also assumed to be the difference between the two. This gives a good agreement with actual data. But does this have any meaning? The rate of GnRH1 secretion cannot be less than zero. However, when gonadal regression is caused by photorefractoriness, it is associated with other physiological changes. Molt of the feathers occurs then, and the rate of molt is faster under more rapidly decreasing photoperiods (Dawson, 1994). Testicular regression is rapid, and involves apoptosis (Young et al., 2001). Testicular regression can be induced in the absence of photorefractoriness, for example by reducing photoperiod from 11L to 8L (Goldsmith and Nicholls, 1984), but in this situation regression is slow and there is no molt.

2.6. Dissipation of photo-inhibition

Thus far, the model can explain why within species breeding ends at the same time at different latitudes, and how species differ

in the timing of the end of breeding. To extend this further, and be able to model complete annual cycles in testis size, it is necessary to investigate processes during the winter months when photoperiod is less than 12 h. During this period, the inhibitory process which appears to be related to cumulative hours in excess of 12 h must be reversed. What do we know about this process – the termination of photorefractoriness? It is essentially the direct opposite of the development of photorefractoriness. It does not start to terminate until photoperiod is less than 12L, termination is gradual, and the shorter the photoperiod the faster it terminates (see Section 2.3). If development of photo-inhibition is related to cumulative hours in excess of 12 h, then the reverse must be true for dissipation of photo-inhibition. This means that the termination of photorefractoriness may be represented by inverse image of Fig. 5 and this is shown in Fig. 8.

In the same way that regression starts where the two lines cross in Fig. 5 (cumulative hours starts to exceed photoperiod), so maturation should start where the two lines cross in Fig. 8. This does not mean that birds are fully photosensitive at that point, rather that photo-induction is greater than photo-inhibition so net drive on GnRH1 secretion becomes positive. Intriguingly, this predicts that maturation should start in the autumn in starlings, but not until spring for quail, which is true in both cases (Robinson and Follett, 1982; Dawson, 2003).

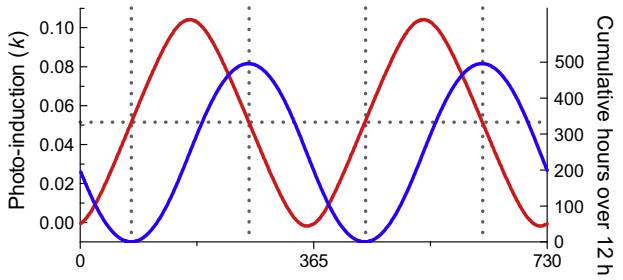

Fig. 9. Annual cycles in photo-induction and photo-inhibition in quail during two years (730 days). The red line shows changes in photo-induction (k), calculated as $k = (p \times 0.0118) - 0.09$, where p is photoperiod. The photoperiod used is that at 52°N because data on the annual cycle in testis size exist for quail at that latitude (Robinson and Follett, 1982). This is the equation derived for starlings, but it works well for quail. k is, of course, maximal at the summer solstice and minimal at the winter solstice. The blue line shows photo-inhibition, equivalent to cumulative hours in excess of 12L at 52°N. This is essentially the two broken lines in Figs. 5 and 8 re-calculated for 52°N and joined together. Photo-inhibition is lowest at the spring equinox and maximal at the autumn equinox. The vertical dotted lines show the time of the equinoxes. Net positive drive on GnRH1 secretion at any time is the difference between the two lines (when photo-induction is greater than photo-inhibition). When photo-inhibition exceeds photo-induction, drive on GnRH1 secretion is negative. It is unclear what this means, but the negative value is used to model the rate of testicular regression in Fig. 10.

2.7. Modeling annual cycles

Although I used cumulative hours in excess of 11.5 to calculate the accumulation of photorefractoriness in the fixed photoperiod case above, I revert to 12 h for modeling annual cycles. Although photo-inhibition may start gradually at about 11.5, presumably the dissipation of photo-inhibition would likewise start slowly when photoperiod decreases to 12.5L. It has to be symmetrical, otherwise birds would accumulate more photo-inhibition during the summer months that they would dissipate during the winter months.

The seasonal cycle in photo-induction and photo-inhibition in quail is shown in Fig. 9. This explains why testicular maturation starts in spring and regression starts in autumn. It also explains why in relatively photorefractory species such as quail, transferring birds at any point during testicular regression to a long photoperiod will induce renewed testicular growth; photo-inhibition never increases sufficiently to exceed photo-induction on a long photoperiod. Furthermore, it explains why the longer quail are kept on a long photoperiod, the less of a decrease in photoperiod is needed to cause regression, and conversely, why the longer birds are on short photoperiods, the smaller the increase in photoperiod is required to induce testicular maturation (Follett and Pearce-Kelly, 1990).

Using the data in Fig. 9 it is possible to calculate the net drive on GnRH1 secretion each day and hence the daily change in testicular size (Fig. 10). This results in close agreement with the annual cycle in testis size in quail. Testicular maturation starts in spring and is rapid. Regression occurs in the autumn. The result is a comparatively long period of testicular maturity which is only slightly asymmetrical with respect to photoperiod.

Exactly the same procedure can be used for starlings. Fig. 11 shows the annual cycle in photo-induction and photo-inhibition. Photo-induction is identical to that in quail, but starlings are much more sensitive to photoperiod causing photo-inhibition, represented by the stretched right vertical axis. This predicts that regression occurs in late spring and that maturation starts in autumn. It also explains why, in absolutely photorefractory species, testicular regression is spontaneous under long photoperiods and why transferring photorefractory birds to very long photoperiods does not induce renewed testicular growth – photo-inhibition far exceeds photo-induction even on 24 h of light.

Again, this data can be used to model daily changes in testis size to construct an annual cycle (Fig. 12). The predicted annual cycle closely reflects reality, and is very different to the situation in quail. Testicular maturation starts in autumn, but the rate of maturation slows as photoperiod decreases, and then increases again after the winter solstice. Regression occurs during late spring. The period of full testicular maturation, and hence the breeding season, is short.

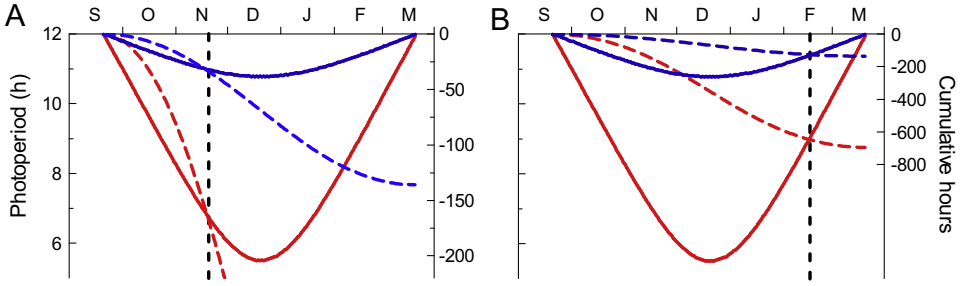

Fig. 8. The dissipation of photo-inhibition in starling (A) and quail (B). This is essentially the reverse of Fig. 5. The red lines refer to 60°N and blue to 20°N. The solid lines show change in photoperiod, decreasing from 12L at the autumn equinox to a nadir at the winter solstice. The broken lines show the dissipation of the cumulative hours over 12 h accrued during the summer months. Again the lines cross at the same point at different latitudes predicting that gonadal maturation will start from that time. For starlings this is in November and for quail in February.

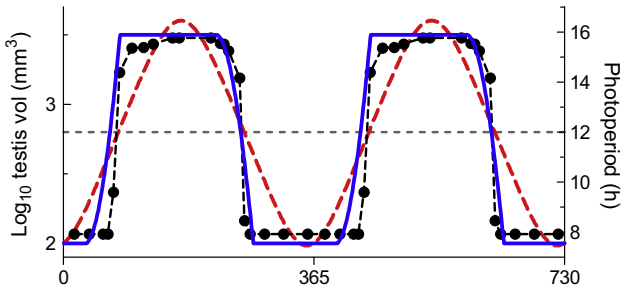

Fig. 10. Annual cycles in testicular size in quail. The blue line shows changes in testis size predicted by data in Fig. 9. The size of the testes at the start of maturation is known (Robinson and Follett, 1982). Daily increments in testis size are calculated from the daily difference between photo-induction and photo-inhibition in Fig. 9 until the testes attain maximum size. Similarly, regression is modeled using the excess of photo-inhibition over photo-induction for each day until minimum size is reached. Real data for changes in testis size are shown by the dots and black broken line (from Robinson and Follett, 1982). Photoperiod is shown by the broken red line. Quail show only weak photo-inhibition and so testicular cycles are only slightly asymmetrical with respect to photoperiod.

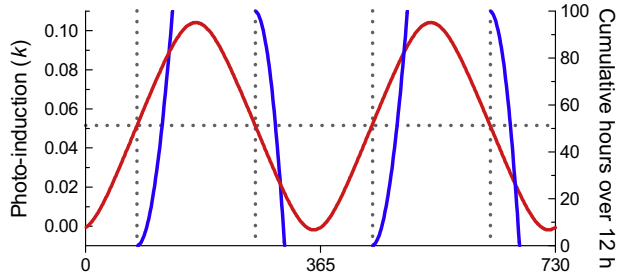

Fig. 11. Annual cycles in photo-induction and photo-inhibition in starlings during two years (730 days). The red line shows changes in photo-induction (k), calculated as $k = (p \times 0.0118) - 0.09$, where p is photoperiod. The photoperiod used is that at 52°N because data on the annual cycle in testis size exist for starlings at that latitude (Dawson, 2003). The blue line shows photo-inhibition, equivalent to cumulative hours in excess of 12L at 52°N. This is essentially the two broken lines in Figs. 5 and 8 re-calculated for 52°N and joined together. Photo-inhibition is lowest at the spring equinox but increases much more rapidly than in quail. Net positive drive on GnRH1 secretion at any time is the difference between the two lines (when photo-induction is greater than photo-inhibition). When photo-inhibition exceeds photo-induction, drive on GnRH1 secretion is negative.

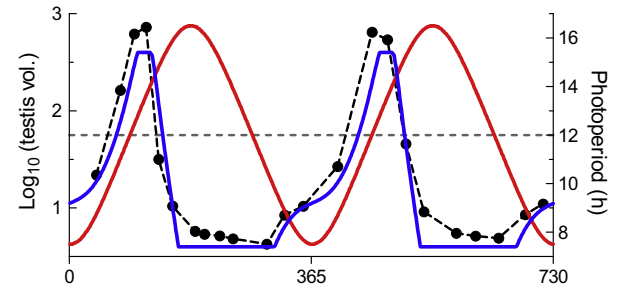

Fig. 12. Annual cycles in testicular size in starlings. The blue line shows changes in testis size predicted by data in Fig. 11. The size of the testes at the start of maturation is known. Daily increments in testis size are calculated from the daily difference between photo-induction and photo-inhibition in Fig. 11 until the testes attain maximum size. Similarly, regression is modeled using the excess of photo-inhibition over photo-induction for each day until minimum size is reached. Real data for changes in testis size are shown by the dots and black broken line (from Dawson, 2003). Photoperiod is shown by the broken red line. Starlings show strong photo-inhibition and so testicular cycles are very asymmetrical with respect to photoperiod. Maturation begins during the autumn, before the winter solstice. Photoperiod and hence photo-induction decrease after that time, so the initial rate of increase in testis size slows, and then increases as photoperiod increases after the winter solstice. Rapid regression occurs before the summer solstice.

3. Conclusions and discussion

This paper proposes a model in which two photo-neuroendocrine processes act together at all times of the year to regulate GnRH1 secretion. The first, photo-induction, affects GnRH1 secretion through a direct effect of the prevailing photoperiod. The second, photo-inhibition, is a longer term process acting through changes in GnRH1 synthesis. It progresses each day during daylight hours, but is reversed during darkness. GnRH1 secretion on any particular day is the net result of these two processes. The model can predict annual cycles in testicular maturity in two species of birds at the extremes of seasonality – the only difference that needs to be invoked between species is their sensitivity to photo-inhibition. This model can potentially explain differences in the timing and duration of breeding seasons between species, why some species become absolutely photorefractory and others only relatively photorefractory, why breeding seasons end at the same time at different latitudes within species, and why experimental protocols sometimes produce results that appear counter to what happens naturally. Nevertheless, this is just a hypothesis; it does not explain the dynamics of GnRH1, but it is testable and, hopefully, will be useful in stimulating new directions for research.

It is interesting that the only species difference required in the model is sensitivity to the photo-inhibitory process. It is this aspect of GnRH1 dynamics that appears to be most plastic; in addition to photoperiod, it can be affected by social cues, temperature and nutrition (Dawson, 1986; Dawson, 2008; Hahn and MacDougall-Shackleton, 2008; Dawson and Sharp, 2010; Stevenson et al., 2008; Visser et al., 2011).

It has been argued that photoperiodic history is important, in other words, that the effect of a particular photoperiod is not just dependent on that photoperiod, but also on the preceding photoperiods. At any latitude, there are two days during the year with the same photoperiod. I have argued that photo-*induction* is directly proportional to photoperiod (Section 2.2) and therefore not dependent on photoperiodic history. But photo-*inhibition* gradually increases under long photoperiods and decreases during short photoperiods. Although photo-induction is the same on different days with the same photoperiod, the net result of photo-induction and photo-inhibition on GnRH1 secretion is not. For example, in Fig. 9, photo-induction is the same at the spring equinox as it is at the autumn equinox, but cumulative hours, i.e. photo-inhibition, is very different, minimal at the spring equinox and maximal at the autumn equinox. Thus the net effect on GnRH1 secretion leads to testicular growth in spring but regression in autumn (Fig. 10). In that sense, photoperiodic history is important; there are no two days during the year when birds are in the same neuroendocrine state.

One prediction of the model is that relatively photorefractory species such as quail always retain the ability to respond to an increase in photoperiod – during the annual cycle photo-inhibition never exceeds the photo-induction resulting from a long photoperiod. Nor do they show spontaneous testicular regression under long photoperiods. However, if they are kept on a constant long photoperiod for a very long time, the model predicts that eventually photo-inhibition should exceed photo-induction. Quail kept on 13L for a long time do show testicular regression (Robinson, J.E. unpublished data) and in domestic poultry kept on long photoperiods to maintain egg production, production does eventually decline.

One major difference between absolute and relative photorefractoriness is that in the former hypothalamic stores of the GnRH1 peptide disappear, whereas in the latter they remain largely unchanged. In starlings under a short photoperiod, GnRH1 levels are moderately high. Following transfer to a long photoperiod they

increase for a short period even though secretion must have increased. Presumably there is an internal feedback mechanism such that synthesis increases to compensate for increased secretion. As photo-inhibition later increases dramatically (Fig. 11) and synthesis declines as a result, hypothalamic peptide stores become depleted. In quail, the decline in synthesis as a result of photo-inhibition is less dramatic and it occurs at the same time as a decrease in photoperiod and hence secretion (Fig. 9). Thus hypothalamic GnRH1 peptide may not be depleted.

The model invokes a high degree of symmetry in that the times when photo-induction and photo-inhibition are equal, the times when maturation starts and regression starts, are 6 months apart. In quail this is clearly apparent; rapid testicular maturation in spring is 6 months ahead of regression. In starlings this is true, but less clear. Maturation does indeed start in the autumn, 6 months ahead of regression, but because photoperiod is short then, the rate of maturation is slow until increasing photoperiods of spring. So in the majority of species, most gonadal maturation occurs in spring. The length of the breeding season is determined more by the timing of regression than the timing of maturation. In general, birds with predictable breeding seasons tend to start breeding in spring, some end later in spring and others continue for various periods until late summer or autumn. Opportunistic species retain greater plasticity (MacDougall-Shackleton et al., 2006; Hahn, 1998; Hahn et al., 2008).

Some species of birds, when kept on a constant equatorial photoperiod of 12L, undergo free-running cycles of testicular maturation and regression – circannual rhythms – with periodicities typically of 9–15 months. However, not all species do (Donham et al., 1983). The model can accommodate circannual rhythms if there is a degree of flexibility in the way that 12L is perceived (see Section 2.3). If a bird perceives 12L as a long photoperiod, this will cause photo-induction and maturation but also the build-up of photo-inhibition and then regression. If it then perceives 12L as a short photoperiod, this will result in the dissipation of photo-inhibition. The difference in perception could be the result of the different physiological states – e.g. birds undergoing maturation will have circulating testosterone, those undergoing regression will not. Castrated starlings do not show repeated cycles (Dawson and McNaughton, 1993; Dittami and Gwinner, 1987) and there is evidence (in mice) that testosterone can influence the circadian clock and SCN responsiveness to light (Daan et al., 1975; Butler et al., 2012). This process could result in a self-sustaining circannual clock, but only under a narrow range of constant photoperiods about 12L. If the photo-inhibitory process builds up during daylight hours and dissipates during darkness, then it would remain roughly neutral under 12L. However, the same would be true under non-24 h schedules where daylight equals darkness. Starlings held on 11L:11D photoperiods also show cycles of maturation and regression (Gwinner, 1981). Free-running circannual rhythms can occur naturally in tropical seabirds for which photoperiod and food resources remain fairly constant throughout the year (Reynolds et al., 2014).

This paper has focused on temperate zone species. What about tropical and sub-tropical species? Low latitude species will experience low amplitude annual cycles in photoperiod. Within the tropics, photoperiod does not exceed 13.5L or decrease below 10.5L. Starlings show a degree of plasticity in their interpretation of photoperiods between 11L and 13L as long or short (see Section 2.3). For tropical and sub-tropical species, photoperiod remains within these limits for most of the year. Thus a circannual cycle in how photoperiod is perceived, or a circannual clock, may become relatively more important than direct effects of photoperiod, in comparison to temperate and high latitude species (Budki et al., 2012).

The modeling in this paper also assumes that birds are resident at the same latitude throughout the year. This means that there is symmetry during the year; neuroendocrine changes during the summer months are reversed at the same rate during the winter months. Migrants present a challenge. Many species that breed in temperate and high latitudes over-winter in tropical or subtropical latitudes. In this case, over-wintering photoperiods would generally range between 11L and 13L depending on whether they were in the northern or southern tropics. These photoperiods are presumably sufficiently short to allow dissipation of photo-inhibition. Garden warblers (*Sylvia borin*) held on a 12.8L photoperiod, simulating that experienced during the "winter", do show a gradual recovery of photosensitivity which is complete by the following spring (Gwinner et al., 1988). Bobolinks (*Dolichonyx oryzivorus*), which are trans-equatorial migrants, recovered photosensitivity after 8 weeks of 12L photoperiods, whereas juncos (*Junco hyemalis*) and white-throated sparrows (*Zonotrichia albicollis*), which are not trans-equatorial migrants, did not (Engels, 1961). The situation in very long distance migrants, where wintering is as far south as breeding is north, is completely unknown.

4. Perspectives – comparison with mammals

Nicholls et al. (1988) argued that absolute and relative photorefractoriness in birds could be explained by a common but undefined mechanism causing photorefractoriness. They went on to argue that this could also potentially encompass different breeding strategies in mammals. In this review, I have argued that the net result of two processes, one related directly to prevailing photoperiod and the other which develops during daylight hours but is reversed during darkness, can explain the range of seasonalities seen in birds, from marked absolute photorefractoriness to weak relative photorefractoriness. This too could potentially offer an explanation for the spectrum of seasonalities in mammals. Mammals are often classified into long day or short day breeders. Species with short gestations, such as voles, are long day breeders; fertility starts during increasing photoperiods of spring. Species such as sheep, with longer gestations, are short day breeders; fertility starts during decreasing photoperiods in autumn. In both cases this results in young born in spring or summer. Unfortunately, terminology between mammals and birds adds confusion. Photorefractoriness to avian biologists means the process which develops during long photoperiods, refractoriness to the stimulatory effects of long photoperiods, which eventually leads to gonadal regression. To mammalian biologists, photorefractoriness means the development during short photoperiods of refractoriness to the inhibitory effects of short photoperiods. The underlying physiology may be analogous, but the terminology is the direct opposite.

In quail, gonadal maturation starts during increasing photoperiods in spring and regression occurs during decreasing photoperiods in autumn (Fig. 10). This is similar to breeding seasonality in small mammals (Paul et al., 2008) such as Siberian hamsters (*Phodopus sungorus*). Maturation starts in quail at the termination of refractoriness to long photoperiods, and in hamsters at the start of refractoriness to short photoperiods! The reverse is true for gonadal regression. So the terminology is opposite, but the actual physiological processes may be analogous. In starlings, gonadal maturation starts during decreasing photoperiods in autumn, and regression occurs during increasing photoperiods in spring. The same is true for sheep. The difference between sheep and starlings is that full gonadal maturity occurs early within this period in sheep. In castrated starlings, LH increases rapidly to maximum values during autumn (Dawson and Goldsmith, 1984). The absence of rapid gonadal maturation at this time in intact birds is presumably because gonadal steroid feedback is sufficient to largely inhibit the weak photoperiodic drive on GnRH secretion. However, some

birds, e.g. emus, do breed under short days (Blache et al., 2001; Malecki et al., 1998).

Another point that this paper emphasises is the importance of the continually changing annual cycle in photoperiod, as opposed to photoperiod per se. Experiments using constant photoperiods yield results at odds with reality. Paul et al. (2008) argued that there are two different annual time keeping mechanisms in small mammals: photoperiodic interval timers and circannual clocks. In the former, in species such as Siberian hamsters, the switch between reproductively active and inactive states occurs at fixed times after the change in photoperiod that initially induced that state. In contrast, species such as golden-mantled ground squirrels (*Spermophilus lateralis*) appear to use a circannual clock. The switches between reproductively active and inactive states occur spontaneously and repeatedly under constant photoperiods. However, these two mechanisms are manifest in experimental conditions in which animals are kept either under constant photoperiods or switched between two different constant photoperiods. Recent studies (Butler et al., 2007, 2010) emphasise the importance of naturally changing photoperiods; under such conditions the differences between these two mechanisms disappear.

Acknowledgments

This work was supported by the UK Natural Environment Research Council. I am indebted to Brian Follett, not just for useful discussions on this paper, but for 40 years of support, encouragement and advice.

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