Review

Hormones, life-history, and phenotypic variation: Opportunities in evolutionary avian endocrinology

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a r t i c l e i n f o

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Life-histories provide a powerful, conceptual framework for integration of endocrinology, evolutionary biology and ecology. This has been a commonly articulated statement but here I show, in the context of avian reproduction, that true integration of ultimate and proximate approaches has been slow. We have only a rudimentary understanding of the physiological and hormonal basis of phenotypic variation in (a) reproductive traits that contribute most to individual variation in lifetime "tness in birds (e.g. laying date, clutch size, parental effort) and (b) trade-offs that link these traits or that link reproduction to other life stages (e.g. migration, molt). I suggest that some reasons for this relative lack of progress include (a) an increasingly reductionist and centralist (upstream) focus which is more and more removed from ecological/evolutionary context, and from peripheral (downstream) mechanisms that actually determine how phenotypes work (b) a long-standing male-bias in experimental studies, even though the key reproductive traits which contribute most to variation in "tness are female-speci"c traits (e.g. onset of vitellogenesis, egg size or number). Endocrine systems provide strong candidate mechanisms for regulation of phenotypic variation in single traits, and two endocrine concepts capture the essence of life-history trade-offs: (a) hormonal •pleiotropy•, when single hormones have both positive and negative effects on multiple physiological systems and (b) hormonal con"ict between regulatory systems required for different but over-lapping or linked life-history stages. I illustrate these ideas with examples of reproductive anemia, migration-reproduction overlap, and molt-breeding overlap, to highlight some of the tremendous opportunities that exist for comparative endocrinologists to contribute to mechanistic studies of avian reproduction in an evolutionary context.

••We have for the most part contented ourselves with the "gure of one sex only, and that the male••
John Ray (1676, cited in [40])

1. Introduction

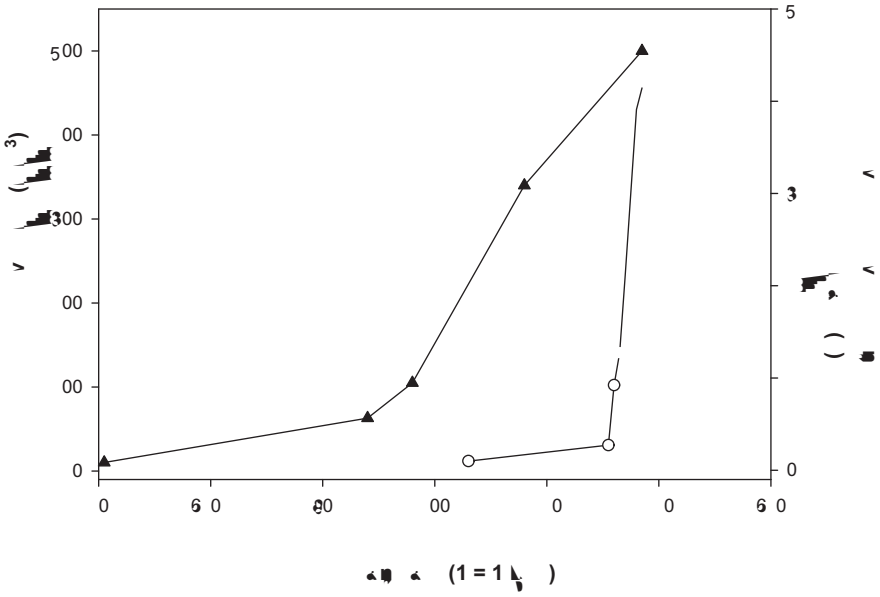
What do we know about the hormonal mechanisms underlying individual, phenotypic variation in the key reproductive life-history traits of birds? This might seem an odd question given that integration of endocrinology with ecology, and more recently with evolutionary biology, has been a commonly articulated theme over the last 20…30 years [52,53,106…108]. However, true integration of ultimate and proximate approaches has been slow in many areas of organismal •evolutionary physiology• [15,50]

research focus and (b) why we should work on female reproductive traits and individual or phenotypic variation. In the context of evolutionary endocrinology I would argue that we need to identify hormonal mechanisms for reproductive traits that contribute most to variance in life-time "tness. An increasing number of long-term, individual-based, population studies of birds and mammals are providing answers to this question, measuring empirical differences in "tness between individuals and assessing the causes of these differences [19]. These studies show that the traits most strongly correlated with individual variation in lifetime "tness (estimated as the total number of offspring recruited to the breeding population) include timing of egg-laying, clutch size, longevity, and the total number of young "edged from all breeding attempts, which is a composite of how well individuals look after their offspring, i.e. parental care [17,38,63,82]. Longevity is outside the scope of this review but, in the context of ageing and senescence, represents another fascinating area of integration of mechanism and evolutionary biology [66,67] Two of these ••key•• reproductive life-history traits, timing of egg-laying and clutch size, are clearly female-speci"c traits. Both sexes can contribute, sometimes equally, to parental care but even for this stage of breeding sex-speci"c differences in parental care are routinely attributed to differential costs of earlier reproductive decisions in females [102] again arguing for a female-speci"c research focus.

For each of these critical reproductive traits there is marked individual or phenotypic variation among females within populations (Fig. 1). If we believe that hormones play a key role in determining phenotype, as endocrinologists do we have mechanisms to explain this individual variation or phenotypic plasticity? At the outset it is important to recognize that considering individual variation forces us to ask a different set of questions with regard to endocrine mechanisms [101]. A common approach in comparative endocrinology is to treat reproduction as a qualitative, categorical trait, i.e. we analyze and compare hormonal, cellular or molecular differences between groups of individuals that are either breeding (reproductively active) or not breeding. We might "nd that non-breeding females have baseline plasma estradiol (E2) levels whereas egg-laying females have highly elevated plasma E2 and we would conclude, probably correctly, that E2 plays an important role in regulating egg production. However, to understand hormonal mechanisms underlying individual variation we need to consider reproduction as a quantitative, continuous trait or complex of traits … the way that ecologists and evolutionary biologists treat most traits. Here, the appropriate question is, • do females laying many, large eggs have higher plasma E2 than females laying few, small eggs•? In other words, does quantitative, individual variation in plasma E2 explain individual variation in reproductive phenotype? Although we all collect hormonal data from individual animals this latter approach to data analysis and interpretation is still relatively rare [101]. Next, I will review some aspects of timing of breeding, clutch size and parental care, and ask how well we understand hormonal mechanisms regulating these traits in this context of female-speci"c reproduction and individual, phenotypic variation.

3. Hormonal regulation of timing of breeding

We have had a working model for control of seasonal reproduction for 40 or more years. This model suggests that day length provides reliable •initial predictive information• for general timing of seasonal breeding, that •supplemental factors• such as temperature, food and social cues "ne-tune the actual timing of egg-laying, and that this environmental information is integrated by the hypothalamic…pituitary…gonadal (HPG) axis to regulate gonadal function [10,26,29,107]. There is substantial, experimental support for this model but, I would argue, mainly for photoperiodic control of testis maturation in male birds, and from studies of males in captivity. Male birds transferred from short-day (SD) photoperiods (8L:16D) to stimulatory long-days (LD, >13L) show an increased release of hypothalamic gonadotropin-releasing hormone (GnRH), an increase in plasma levels of the pituitary gonadotropins luteinizing hormone (LH) and follicle-stimulating hormone (FSH) and, even in captivity, this leads to testis maturation, spermatogenesis and steroidogenesis, with elevated plasma testosterone levels [26,69,107]. In contrast, females of most non-domesticated species held in standard captive conditions (small cages) will not undergo complete ovarian development: the ovary can develop to the pre-vitellogenic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just a few days in highly synchronous breeders (Fig. 4a). Multi-brooded species have a different seasonal pattern with clutch size “rst increasing with date then declining [20] , but this can be phe-notypically plastic depending on the laying environment (Fig. 4b; [90]). Any generalized endocrine mechanism must be able to ex-plain this inter- and intra-speci“c variation in clutch size, including the ••“ne-tuning•• of clutch size to the prevailing environment over time periods as short as 1…2 days, and date-independent variation or plasticity in clutch size (Fig. 4b). So do we have an endocrine mechanism to explain this individual or phenotypic variation in clutch size?

Predictive models for hormonal-regulation of clutch size deter-mination have been around for 15…20 years but appear to have been largely ignored by endocrinologists, at least experimentally. These models were derived from studies by Meijer and colleagues in Eurasian kestrels Falco tinnunculus [11,64] and similar work in small songbirds by Haywood [45,46] . Both models propose the existence of an endogenous ••inhibitory signal•• which increases seasonally and/or during egg-laying, and is associated with devel-opment of incubation behavior, but which eventually interrupts follicle development, thus terminating ovulation and determining “nal clutch size (see Fig. 5). Meijer et al. [64] and Haywood [45] speculated on the physiological nature of the controlling variable underlying their models and suggested that this was a prolactin-mediated mechanism with the ••inhibitory signal•• involving anti-gonadal effects of prolactin (see also Meijer et al. [65] and Sockman et al. [97]

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abundant literature on endocrine and neuroendocrine regulation of exercise, albeit largely from human studies of kinesiology and sports medicine [13,33,55]. Concepts such as ••over-reaching•• and ••over-training•• are associated with long-term performance decrements due to intense exercise, and as any amateur athlete knows exercise can lead to ••exhaustion and temporary suppression of vitality•• (to use Daan•s [24] words). Can this conceptual approach provide a model for physiological regulation of workload associated with chick-rearing? Numerous studies have characterized hormonal changes associated with exercise including glucocorticoids, prolactin, thyroid hormones, catecholamines, growth hormone, IGF-1, and gonadal steroids [33] so there are no shortage of candidate hormones of interest. Furthermore, parental care lends itself to experimental work since free-living birds can easily be captured, hormonally-manipulated, and released during chick-rearing without risk of nest abandonment. Finally, an intriguing suggestion in the context of female-speci“c costs of reproductive effort [102] is that females might be more susceptible to over-training than males [83]. This clearly represents an almost untouched area of integration of comparative endocrinology and ecology where hormonal studies could signi“cantly contribute to our understanding of individual variation in parental care.

6. Hormonal regulation of trade-offs between traits or life-history stages

So far I have considered single traits (phenology, clutch size) or single breeding stages (chick-rearing), but I now brie”y want to consider hormonal regulation of trade-offs and carry-over effects between different traits or different life-history stages. Two endocrine-related concepts capture the essence of evolutionary or life-history trade-offs: hormonal pleiotropy and hormonal con”ict. Hormonal pleiotropy (a term •borrowed• from evolutionary genetics) occurs where a single hormone has both positive and negative effects on different physiological systems or traits [36,43,53] and is a long-recognized hallmark of hormone action. Hormonal con”ict can occur when different regulatory systems are required simultaneously for overlapping functions or life-history stages and the regulatory mechanisms underpinning each function generate inherent hormonal (or metabolic) incompatibilities. Both these mechanisms might operate whenever birds have to do different things at the same time. Fig. 6a presents one commonly held view of life-histories, prevalent in the literature, where different stages of the life cycle are sequentially orchestrated with minimum overlap between stages (it is argued due to high energetic costs of each stage). However, Fig. 6b presents a more accurate, though still over-simpli“ed, view of life-histories with lots of interaction and overlap between successive stages. Using this framework I will describe some potential examples of hormonal control of trade-offs

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