



## Endocrinology of thermoregulation in birds in a changing climate

Suvi Ruuskanen<sup>a,\*</sup>, Bin-Yan Hsu<sup>a</sup>, Andreas Nord<sup>b</sup>

<sup>a</sup> Department of Biology, University of Turku, Finland

<sup>b</sup> Lund University, Department of Biology, Section for Evolutionary Ecology, Ecology Building, Sölvegatan 37, SE-22362, Lund, Sweden

### ARTICLE INFO

#### Keywords:

Avian  
Thermoregulation  
Thyroid hormone  
Corticosterone  
NST  
avUCP  
BMR  
Plasticity  
Climate change  
Temperature  
Weather  
Metabolic rate  
Heterothermy

### ABSTRACT

The ability to maintain a (relatively) stable body temperature in a wide range of thermal environments by use of endogenous heat production is a unique feature of endotherms such as birds. Endothermy is acquired and regulated via various endocrine and molecular pathways, and ultimately allows wide aerial, aquatic, and terrestrial distribution in variable environments. However, due to our changing climate, birds are faced with potential new challenges for thermoregulation, such as more frequent extreme weather events, lower predictability of climate, and increasing mean temperature. We provide an overview on thermoregulation in birds and its endocrine and molecular mechanisms, pinpointing gaps in current knowledge and recent developments, focusing especially on non-model species to understand the generality of, and variation in, mechanisms. We highlight plasticity of thermoregulation and underlying endocrine regulation, because thorough understanding of plasticity is key to predicting responses to changing environmental conditions. To this end, we discuss how changing climate is likely to affect avian thermoregulation and associated endocrine traits, and how the interplay between these physiological processes may play a role in facilitating or constraining adaptation to a changing climate. We conclude that while the general patterns of endocrine regulation of thermogenesis are quite well understood, at least in poultry, the molecular and endocrine mechanisms that regulate, e.g. mitochondrial function and plasticity of thermoregulation over different time scales (from transgenerational to daily variation), need to be unveiled. Plasticity may ameliorate climate change effects on thermoregulation to some extent, but the increased frequency of extreme weather events, and associated changes in resource availability, may be beyond the scope and/or speed for plastic responses. This could lead to selection for more tolerant phenotypes, if the underlying physiological traits harbour genetic and individual variation for selection to act on – a key question for future research.

### 1. Aims and motivation

Birds maintain a high and (relatively) stable body temperature ( $T_b$ ), on average  $41.02 \pm 1.29$  °C (s.d.) at rest during the active phase (Prinzinger et al., 1991), across a wide range of thermal environments via physiological, morphological and behavioural modifications, including endocrine regulation. This endothermic lifestyle enables the wide aerial, aquatic, and terrestrial distribution of birds in variable habitats across the globe (Yahav, 2015). For example, desert birds are able to live and reproduce in arid conditions where the environment can be hotter than their own bodies, while resident species in temperate regions have to deal with winter temperatures that may be 80 °C below  $T_b$ . Due to our changing climate, all organisms, including birds, are faced with potential new challenges for thermoregulation, such as more frequent extreme weather events, lower predictability of climate, and

increasing mean temperature (IPCC 2014). The key question is how they will respond and potentially adapt.

This review is aimed at both endocrinologists interested in embracing the links of their own field with thermoregulation and climate change, and for (thermal) ecologists interested in embracing the underlying endocrine regulation of thermoregulation (see glossary in Box 1). Thus, the first aim of this review is to provide an overview on thermoregulation in birds and its endocrine and molecular mechanisms, pinpointing gaps in current knowledge and recent developments. We focus on birds, because they are researched widely across the globe, and because recent work has identified their substantial vulnerability to climate change (Conradie et al., 2019; Riddell et al., 2019). However, many of the future challenges in light of climate change are not taxon-specific. We especially focus on various aspects of (across and within-generation) plasticity in thermoregulation and its underlying

\* Corresponding author. Department of Biology, University of Turku, Vesilinnantie 5, Turku, Finland.

E-mail address: [skruus@utu.fi](mailto:skruus@utu.fi) (S. Ruuskanen).

<https://doi.org/10.1016/j.mce.2020.111088>

Received 29 June 2019; Received in revised form 13 November 2020; Accepted 17 November 2020

Available online 21 November 2020

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endocrine mechanisms, because proper understanding of plasticity is a key to predicting responses to changing environmental conditions. Because current literature on endocrinology and thermoregulation is biased towards work on poultry, we have put special emphasis on studies in non-model species and wild populations to understand the generality of, and variation in, mechanisms. To our knowledge, there are no previous reviews that tie together the complexity of the endocrine basis of  $T_b$  regulation in birds with plasticity of thermoregulation under climate change and also discuss the recent advances in molecular mechanisms of thermoregulation. This summary will set the stage for the second set of goals: discussing challenges for avian thermoregulation in the face of climate change, how this may affect endocrine traits, and how these processes combine to facilitate or constrain adaptation to the changing world. Understanding the scope of physiological plasticity and potential physiological constraints may help to understand the responses at individual and even population levels. We purposely use the wording ‘climate change’ over ‘global warming’ throughout, to account for the multitude of direct and indirect changes in the environment that challenge avian thermoregulation.

## 2. A brief overview of thermoregulation in birds

The thermoregulatory system that enables maintenance of a relatively constant  $T_b$  over a wide range of environmental temperatures in birds consists of (Fig. 1, modified by Yahav, 2015): (1) a *sensory part*, which detects changes in the environment (thermo-, osmo-, and baro-receptors), (2) an *integrating part*, the thermoregulatory center at preoptic anterior hypothalamus (PO/AH), in which temperature-sensitive neurons monitor local temperature changes and temperature information received from peripheral thermoreceptors. The  $T_b$  set point is then defended by mechanisms for heat production or heat loss depending on the thermal status of the bird; and (3) the *command part*, involving neurological and endocrine signals that lead to downstream mechanisms for  $T_b$  control, viz. shivering thermogenesis (ST), non-shivering thermogenesis (NST), evaporative heat loss (including respiratory- and cutaneous heat loss), peripheral vasoconstriction or vasodilation, and behavioural changes (e.g. basking, huddling, shade/-cover seeking).

Potential mechanisms for elevating the capacity for thermogenesis in

birds include (i) increasing muscle mass devoted to ST and possibly NST (but see Milbergue et al., 2018 for recent contrasting results) (ii) increasing mass-specific aerobic enzyme capacity of muscle tissue (Bicudo et al., 2002; Liknes and Swanson, 1996; potentially also liver, see Liu et al., 2006), and (iii) increasing mitochondrial density and/or respiration rate to fuel aerobic metabolism, and (iv) possibly increasing NST (Collin et al., 2003b; Dridi et al., 2004). In ST, the birds increase the rate of heat production by increased contractile activity in the skeletal muscles, whereas, in NST, heat production is increased via metabolic energy transformations which do not include contraction of muscles. ST contributes most to thermogenesis in birds (e.g. Hohtola, 2002). NST (in muscle and liver tissue, in contrast to mammalian brown adipose tissue NST) is probably widespread in birds (e.g. Dridi et al., 2008; Mozo et al., 2005; Raimbault et al., 2001; Talbot et al., 2004; Vianna et al., 2001a; Vianna et al., 2001b), but direct evidence for a significant contribution of NST to heat production is still scarce (Teulier et al., 2010, 2014). The molecular mediators of NST in mitochondria have been heavily discussed, and may include (i) the avian homolog of mammalian uncoupling protein (avUCP) - an anion-carrier protein that, by dissipating the proton gradient across the inner mitochondrial membrane, potentially uncouples respiration from ATP synthesis (Dridi et al., 2008; Mozo et al., 2005; Raimbault et al., 2001; Talbot et al., 2004; Vianna et al., 2001a, 2001b); (ii) adenine nucleotide translocase (ANT) as the uncoupling protein (Walter and Seebacher (2009) or (iii) Ca<sup>2+</sup> slippage to produce heat via binding of the peptide sarcolipin (SLN), to Ca<sup>2+</sup>-ATPase in muscle tissue (SERCA) (reviewed in Nowack et al., 2017). In this review we focus on endocrine and molecular mechanisms underlying thermoregulation, while neuronal mechanisms in vertebrates have been reviewed elsewhere (e.g. Morrison et al., 2008).

## 3. Endocrine and molecular mechanisms of thermoregulation in birds

### 3.1. The role of the hypothalamus-pituitary-thyroid (HPT) axis in avian thermoregulation

Thyroid hormones (THs, triiodothyronine, T<sub>3</sub> and thyroxine, T<sub>4</sub>) are the most important hormones controlling thermogenesis; a link that was established already in the late 1950's (e.g. Decuyper et al., 2005;

#### Box 1

Glossary of key terminology concerning thermoregulation (following IUPS Thermal commission 2003).

**Acclimatization** physiological or behavioural changes that occur within the lifetime of an organism to reduce the strain of variation in the natural climate in the wild, such as geographic or seasonal responses to variation in ambient temperatures.

**Basal Metabolic rate (BMR)** Metabolic energy transformation calculated from measurements of heat production or oxygen consumption in a rested and awake organism that is within its thermoneutral zone and has fasted sufficiently long to be in a postabsorptive state.

**Endothermy** The pattern of thermoregulation of animals in which the body temperature depends on a high and controlled rate of endogenous heat production.

**Epigenetic temperature adaptation** Lifelong(?) thermal adaptation that is triggered during prenatal (embryogenesis) or early posthatching ontogeny, influencing the individuals' capacity to produce or dissipate heat.

**Hyperthermia** The condition of an animal where body core temperature is above its range specified for the normal active state of the species.

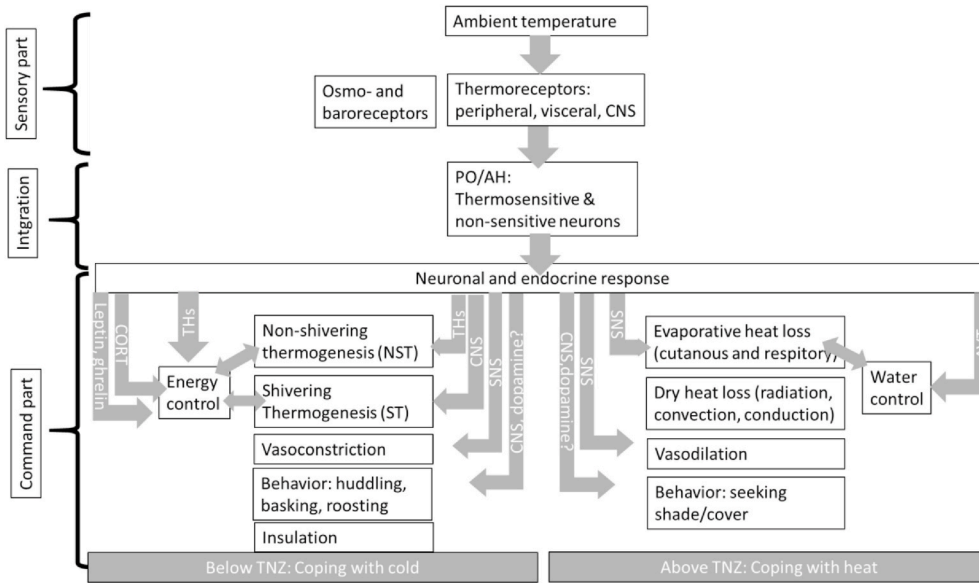
**Hypothermia** The condition of an animal where body core temperature is below its range specified for the normal active state of the species.

**Non-shivering thermogenesis (NST)** Heat production due to metabolic energy transformation by processes that do not involve contraction of skeletal muscles.

**Resting Metabolic Rate (RMR)** The metabolic rate of an animal at rest in a specified environmental context, and not necessarily in a post-absorptive state.

**Shivering thermogenesis (ST)** Heat production due to increased contractile activity of skeletal muscles (not involving voluntary movements).

**Thermoneutral zone (TNZ)** the range of ambient temperatures at which normothermic body temperature is maintained without regulatory changes in metabolic heat production or heat dissipation.



**Fig. 1.** A simplified schematic figure of the mechanisms of thermoregulation in birds and putative key endocrine and neuronal mechanisms underlying each response (modified from [Yahav, 2015](#)). PO/AH = preoptic anterior hypothalamus, THs = thyroid hormones, CORT = corticosterone, AVT = arginine vasotocin, CNS = central nervous system, SNS = sympathetic nervous system, TNZ = thermoneutral zone. See text for references.

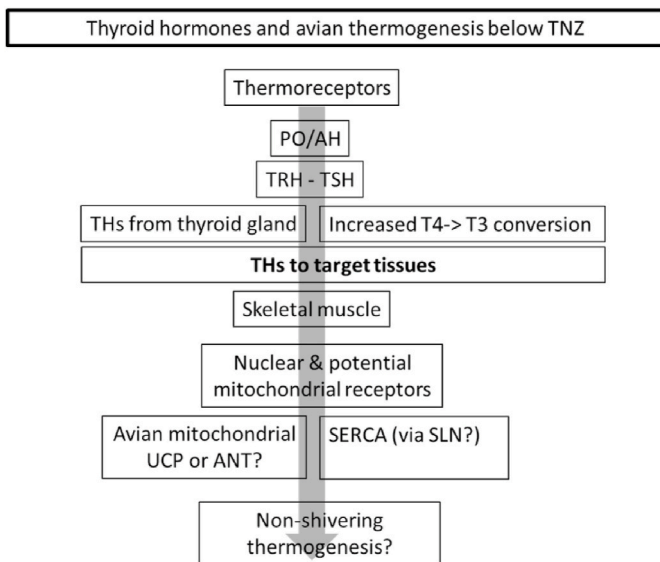
[Klandorf et al., 1981](#); [Mellen and Wentworth, 1958](#); [Mellen and Wentworth, 1962](#)). The proposed hormonal and molecular pathways with HPT-axis involvement on thermogenesis are outlined in [Fig. 2](#).

PO/AH is activated by input from thermoreceptors. PO/AH then stimulate the hypothalamic paraventricular nucleus (PVN) and leads to an increase in thyrotropin releasing hormone (TRH) synthesis and secretion ([Arancibia et al., 1996](#)). TRH stimulates the thyrotrophs in the anterior pituitary to secrete thyroid stimulating hormone (TSH), which interacts with the follicular cell membrane receptors in the thyroid gland. This results in increased thyroid hormone (mainly T4) synthesis and release. T4 is the precursor form of T3, the biologically active form. T4 is converted to T3 in tissues by deiodinase enzymes (DIO1-3). The secretion of TSH by the pituitary is (besides hypothalamic TRH) modulated by a negative feedback mechanism of T3. Alternatively, cold exposure can lead to increased conversion of T4 to T3 (and less to

inactive rT3) by deiodinase enzymes (DIO1-3) in tissues, mostly in the liver ([Collin et al., 2003a](#); [Van der Geyten et al., 1999](#)), thus leading to higher circulating T3 levels. In tissues, T3 binds to both nuclear and mitochondrial receptors and influences gene expression to modulate metabolic rates and respiration (see below).

There is experimental evidence supporting the modulatory role of THs on thermoregulation within the thermoneutral zone (TNZ), also coined 'obligatory thermogenesis': For example, experiments where birds were made hypothyroid (i.e., lacking THs) using drugs blocking TH release, or when thyroid glands were removed (thyroidectomy), reported reduced heat production in poultry (e.g. [Mellen and Wentworth, 1962](#)), whereas experimental administration of THs stimulated thermogenesis ([Arieli and Berman, 1979](#)). Experimental elevation of THs in blood increased thermogenesis also in non-model species, little buntings (*Emberiza pusilla*) ([Liu et al., 2006](#)), and positive correlations between TH concentration and thermogenesis were observed in goldfinches (*Carduelis tristis*) ([Dawson et al., 1992](#)), great tits (*Parus major*) and willow tits (*Poecile montanus*) ([Silverin et al., 1989](#)). In addition, a growing body of (mostly correlational) evidence suggests a positive association between TH concentration (T3) and basal metabolic rate (BMR) in several non-model bird species measured in free-ranging populations ([Bobek et al., 1977](#); [Chastel et al., 2003](#); [Kim, 2008](#); [Liu et al., 2006](#); [Vezina et al., 2009](#); [Zheng et al., 2014a](#)).

Thyroid hormones are important for thermogenesis below thermoneutrality (also coined 'facultative thermogenesis' see [Fig. 2](#)). They are likely involved in regulating non-shivering thermogenesis (NST), while shivering thermogenesis (ST) may be predominantly controlled directly via neuronal mechanisms ([Morrison et al., 2008](#)). Mitochondria in avian skeletal muscle have recently been found to express thyroid hormone receptors (see e.g. [Cioffi et al., 2013](#) for reviews on mammals; [Lanni et al., 2016](#); [Lassiter et al., 2018](#)). For example, an increase in potential uncoupling protein, avUCP, expression was associated with increased plasma T3 levels and heat production in chicken ([Collin et al., 2003a](#)). Furthermore, experimental elevation of T3 caused an overexpression of avUCP mRNA, while a thyroid blocker (methimazole) treatment significantly downregulated its expression ([Collin et al., 2003b](#); [Walter and Seebacher, 2009](#)), supporting the involvement of THs in avUCP regulation. The other potential uncoupling protein, adenine nucleotide translocase (ANT) may also be controlled by THs, because its expression was decreased by TH blockers ([Walter and Seebacher, 2009](#)). Finally,



**Fig. 2.** A schematic diagram of the potential pathways of THs on thermogenesis below thermoneutral zone. UCP = uncoupling protein, ANT = adenine nucleotide translocase, SERCA = Ca<sup>2+</sup> ATPase. See text for references.

THs also influenced expression of, a known metabolic mediator (PGG-1) in mammals (Walter and Seebacher, 2007, 2009), providing another potential molecular mediator for NST in birds. However, the exact molecular mechanisms underlying how THs may be controlling the uncoupling proteins and, thus, NST in birds are not understood, while in mammals, UCP and PGC1 sequences show response elements for thyroid hormone receptor (e.g. Rabelo et al., 1996; Wulf et al., 2008). Also non-genetic pathways of TH action have been characterized, mostly in rodents (reviewed in Cheng et al., 2010; Davis et al., 2016; Davis et al., 2018; Singh et al., 2018), yet some indication of non-genomic influence of THs on mitochondria e.g. glucose uptake was observed in chicken embryos (Segal and Gordon, 1977).

In most studies, associations between THs and thermogenesis are based on circulating hormone measurements. However, the final availability of THs is influenced by plasma binding proteins, conversion of THs by deiodinase enzymes (DIO1-3) in the target tissues, and the responsiveness to THs depends on receptor densities (Darras et al., 2006). As expected, ambient temperature has also been found to influence deiodination: Increased hepatic T4 to T3 conversion via increased DIO2 expression was detected in cold-exposed chickens (Ikegami et al., 2015; Rudas and Pethes, 1984, 1986), while heat stress decreased DIO2 expression (Jastrebski et al., 2017). Cold exposure also decreased hepatic DIO3 activity in domestic chicks (Collin et al., 2003a), leading to less T3 degradation. To our knowledge, variation in TH receptor expression (either nuclear or mitochondrial) has not been studied in response to temperature variation in birds. In mice and rats, mitochondrial receptor (p43) overexpression was recently found to be associated with higher  $T_b$  (Bertrand-Gaday et al., 2016). In vivo (knockout) and in vitro studies in mice revealed that p43 is associated with mitochondrial biogenesis, activity, mito-nuclear crosstalk and even muscle metabolic and contractive phenotype (reviewed in Wrutniak-Cabello et al., 2017; Wrutniak-Cabello et al., 2018), but similar data is lacking from birds.

### 3.2. The role of the hypothalamus-pituitary-adrenal (HPA) axis in avian thermoregulation

Glucocorticoids are crucial for maintaining physiological and energetic homeostasis. Following a stressor, corticosterone (CORT) - the main avian glucocorticoid - is released from the adrenals (stress-induced CORT) in response to adrenocorticotrophic hormone (ACTH) from the hypothalamus. Fast temperature changes, induced by both cooling and heating, have been shown to increase circulating CORT across captive and wild species in both correlative and experimental studies (Bize et al., 2010; de Bruijn and Romero, 2011; Frigerio et al., 2004; Jessop et al., 2016; Jimeno et al., 2018; Krause et al., 2016; Wingfield, 2015; Wingfield et al., 2017; Xie et al., 2017; Shipley et al., 2019). Both baseline and stress-induced CORT may be affected by temperature (reviewed e.g. in Jessop et al., 2016). Thus, it has been suggested that CORT may play a role in how the individual responds and adapts to rapid temperature changes. Despite repeated observations of such patterns, the exact mechanism for how CORT may be linked to thermoregulation, either physiologically or behaviourally, is not well understood. Because CORT is needed for the synthesis of glucose from liver glycogen or fat reserves, the most likely explanation is that this hormone mobilises internal energy reserves, and/or activates food searching behaviour to fuel the higher metabolic rate in cold conditions (de Bruijn and Romero, 2011; Jimeno et al., 2018). Finally, recent correlative studies also suggest an association between baseline CORT, peripheral body temperature, and slow changes in environmental temperature over winter and summer (blue tits, Jerem et al., 2018), which may indicate that CORT is not only involved in short-term heat/cold stress.

In addition to its potential role in responses to thermal stress, the HPA-axis probably also plays a role in the ontogeny of avian thermoregulation, interacting with the HPT-axis (reviewed in Debonne et al., 2008). In neonatal chicks, injection of corticotropin releasing hormone

(CRH) induced thermogenesis and increased  $T_b$  (Tachibana et al., 2004; Takahashi et al., 2005). CRH injection in chicken embryos also increased TSH and circulating T4 and T3 concentrations (Meeuwis et al., 1989), potentially via CRH-receptors, expressed in the thyrotrophs (De Groef et al., 2005), suggesting interactions between CRH and THs on thermoregulation. Interestingly, the HPT-axis became less responsive to CRH in adult chickens (Geris et al., 2003), likely due to altered interaction with the peripheral TH deiodinase enzymes (Darras et al., 2006), thus showing that such interactions between hormones on thermoregulation may vary across stages in ontogeny.

### 3.3. The role of potential other endocrine mechanisms in thermoregulation

There is a plethora of other neuropeptides and hormones that may be linked to thermoregulation, but the role of these is largely unexplored, especially beyond chicken models. It is also well understood that many hormonal axes are likely to interact (see also above). (i) Nguyen et al. (2015) discovered that hypothalamic orexigenic neuropeptide Y (NPY) responds to cold exposure in poultry. (ii) Melatonin, an indoleamine produced in the pineal gland, has been implicated to play a role in avian thermal homeostasis (John and George, 1991). Experimental melatonin administration increased  $T_b$  in chickens (e.g. Sinkalu et al., 2015), and induced improved cold resistance, thermal insulation and maximal heat production (similar to cold acclimatization) in quails (Saarela and Heldmaier, 1987; Saarela and Reiter, 1994). This is in line with melatonin showing seasonal and circadian patterns, and thus conveying information on seasonal progression in birds and mammals (Nelson and Demas, 1997; Reierth et al., 1999).

Balancing energetic needs is essential for thermoregulation: both ST and NST, and (some routes of) evaporative heat loss require energy, the availability of which often fluctuates with high/low environmental temperatures. A few key hormones are likely to be involved with energy balance during thermal stress: (iii) Leptin was found to be elevated following acute heat and cold stress in liver and plasma in chicken (Dridi et al., 2008). It has been suggested to be involved in regulating feed intake and lipid metabolism (reviewed in Zhang and Chua, 2018), which are tightly linked with thermoregulation. (iv) Ghrelin, a peptide hormone produced mainly in the gastrointestinal tract, may also be indirectly linked to thermoregulation: peripheral administration of ghrelin in turkeys increased levels of plasma CORT, glucose and T4 (Shahryar and Lotfi, 2017), suggesting indirect effects via e.g. thyroid hormones. Moreover, experimentally increased leptin and ghrelin was associated with mass gain in the coal tit (*Parus ater*, Henderson et al., 2018), which is likely to also (indirectly) influence thermoregulation during winter.

In hot and dry environments, thermoregulation is often directed towards heat dissipation which is mostly achieved by evaporative cooling. Because this is costly in terms of water (Nord and Williams, 2015; Tieleman and Williams, 1999), it is important to understand endocrine regulation of water balance. One of the key regulators is (v) arginine vasotocin (AVT), produced in the posterior pituitary gland (neurohypophysis) of the brain. In a series of experiments on starlings (*Sturnus vulgaris*), Nephew et al. (2005) showed that peripheral AVT can decrease heart rate, feeding, drinking, preening, and overall activity. AVT also reduced shivering and  $T_b$  but had no effects on blood pressure, heart rate or respiratory rate in pigeons (*Columba livia*) (Hassinen et al., 1994, 1999). Finally, AVT was further found to increase plasma levels of T4, and a decrease in that of T3 (John and George, 1992), which may be one of the mechanisms linking AVT to thermoregulation.

## 4. Conclusions and notes

The majority of the known endocrine mechanisms in thermoregulation concern thermogenesis, i.e. the production of heat below the TNZ. However, it is equally important to understand the endocrine and

neurological mechanisms of thermoregulation above the TNZ where heat needs to be dissipated to keep  $T_b$  at set point and prevent overheating. These include evaporative heat loss, facultative hyperthermia, vasodilation and multiple behavioural responses, such as seeking cooler microclimates. Heat loss, vasomotor action, as well as many of the behavioural effects are likely under neuronal control, including dopamine, as suggested in mammals (Madden and Morrison, 2019; Terrien et al., 2011). However, there are few corresponding data for birds. Filling these knowledge gaps can be important to understand responses to climate-change driven extreme weather, such as heat waves and droughts, which is expected to become more frequent in the coming years (see section §5).

Furthermore, as thermoregulation requires excess energy (in cold environments) and water (in hot environments), the associated endocrine pathways of water and energy intake should also be considered for a more complete understanding of the physiological state. Thus, the relative contributions of other hormonal controllers of organismal thermal biology beyond HPT and HPA axis on thermoregulation need to be further clarified.

## 5. Plasticity in thermoregulation and its endocrine control – five time-scales

Many bird species have a large capacity to plastically respond to varying environmental temperature on different time scales via: (i) rapid stress-induced hyperthermia (ii) short-term facultative hypo- or hyperthermia (iii) acclimatization, (iv) epigenetic temperature adaptation or (v) transgenerational plasticity, as summarized in Fig. 3.

### 5.1. Rapid stress-induced hyperthermia

Birds, like other endotherms, respond to acute ecological and psychological stressors (e.g. predation) via immediate, stress-induced hyperthermia (Cannon, 1915). The response reflects increased  $T_b$  set point regulated by the sympathetic-adrenal-medullary pathway, while CORT could also play some role (Briese and Cabanac, 1991; Yahav, 2015). For certain stimuli, the thermoregulatory changes during stress are sensitive to variation in environmental temperature (Nord and Folkow, 2018). However, it is unlikely such immediate response will be significantly altered in a climate change context. Thus, stress-induced  $T_b$  responses are not discussed further.

### 5.2. Facultative hypo- and hyperthermia

$T_b$  of birds fluctuates substantially around the normothermic mean ( $41.02 \pm 1.29$  °C (s.d.)) during the circadian cycle (Prinzinger et al., 1991). Many bird species deviate even further from normothermia over time periods ranging from a few hours to several days, referred to as facultative hypothermia ( $T_b$  below the normothermic range) and hyperthermia ( $T_b$  above the normothermic range) (reviewed in Gerson et al., 2019) (McKechnie and Lovegrove, 2002; Ruf and Geiser, 2015).

Facultative hypothermia is typically employed when birds are energy limited, either because they feed on ephemeral resources, have high energy turnover rates, or reside in challenging (e.g. resource-poor, cold) environments (McKechnie and Lovegrove, 2002; Nilsson and Nord, 2017; Ruf and Geiser, 2015). It leads to energetic savings and significant fitness benefits (Brodin et al., 2017). Facultative hyperthermia is likely rather universal in birds and varies phylogenetically (reviewed by Gerson et al., 2019). Although it has mostly been studied in desert species, where it reduces the need for evaporative cooling, and hence demand for water, species from other regions and habitats also express facultative hyperthermia when under heat stress (Gerson et al., 2019).

The molecular and endocrine mechanisms underlying facultative hypo- and hyperthermia are not well understood. CORT or testosterone could play a role in regulating facultative hypothermia. In hummingbirds external CORT increased the use of torpor (Hiebert et al., 2000). In mammals, experimental administration of testosterone has been shown to inhibit torpor (Mzilikazi and Lovegrove, 2002). A potential testosterone-dependence could also explain why, in birds, torpor was restricted to females in the Puerto Rican tody (*Todus mexicanus*) (Merola-Zwartjes and Ligon, 2000), and hypothermia was deeper in females compared to males in Eurasian skylarks (*Alauda arvensis*) and blue tits (*Cyanistes caeruleus*) (Nord et al., 2009; Powolny et al., 2016). Finally, in chicken models, low temperature combined with fasting, suppressed hepatic thyroid hormone deiodinase enzyme (DIO2) and serum T3 level (Ikegami et al., 2015), which suggest that THs may also function as a regulatory mechanism for facultative hypothermia in cold and food-restricted conditions.

### 5.3. Acclimatization

Acclimatization of thermoregulation refers to reversible physiological or behavioural changes that occur within the lifetime of an organism to allow the individual to cope with longer-term temperature variation over days or weeks. This allows maintained performance over a range of

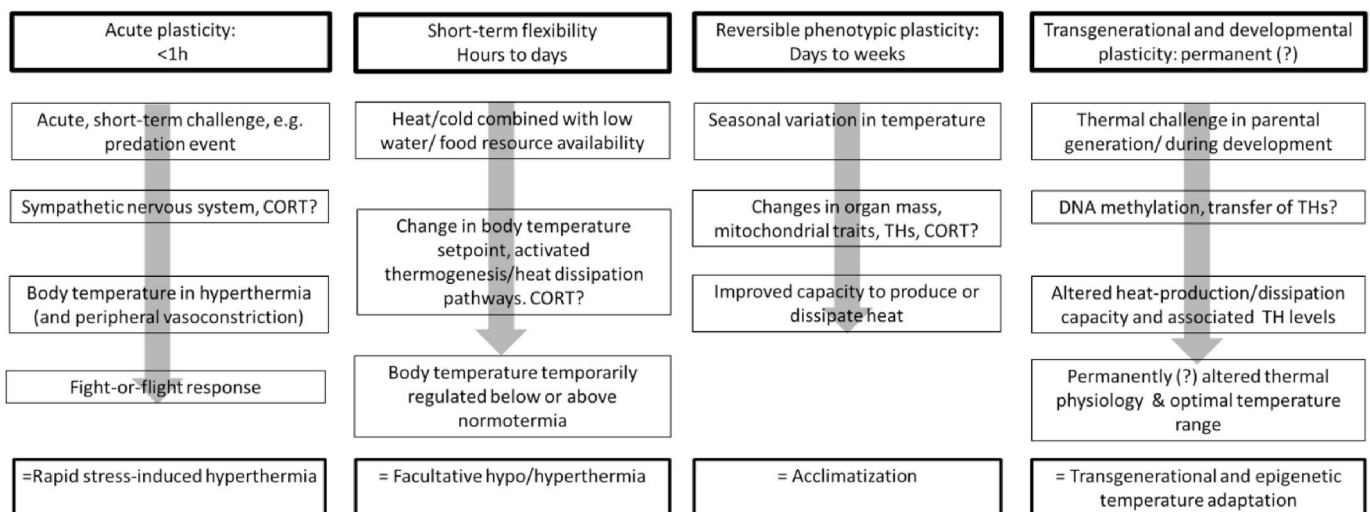


Fig. 3. Schematic diagram of plasticity in thermoregulation at different time-scales, and associated putative molecular and endocrine mechanisms. See text for references.

environmental conditions, such as between seasons and across the species' distribution range among populations (Fig. 3). There is evidence that THs play a role in seasonal acclimatization: For example, in wild migratory birds such as white-crowned sparrows (*Zonotrichia leucophrys*) and red knots (*Calidris canutus canutus*), annual cycles of THs are correlated with seasonal changes in BMR, suggesting that seasonal changes in TH could be involved in adjusting energy expenditure to environmental conditions (Jenni-Eiermann et al., 2002). In resident birds, passerines showed enhanced thermogenic capacity via seasonal increases of muscle tissue used for thermogenesis (several muscle types, Hohtola, 2002; Milbergue et al., 2018) and growth of metabolically active organs such as the liver and heart (reviewed by Swanson and Vezina, 2015). These changes were coupled to increased activity of respiratory enzymes and higher levels of T3 (Burger and Denver, 2002; Liknes and Swanson, 2011a, 2011b; Silverin et al., 1989; Smit and McKechnie, 2010; Zheng et al., 2014a, 2014b), supporting the role of THs in seasonal acclimatization.

#### 5.4. Epigenetic adaptation

Epigenetic temperature adaptation (e.g. Yahav, 2015) means that exposure to temperature variation during the ontogeny of the thermoregulatory system (and HPT- and HPA-axes) alters thermal physiology, presumably via changes to DNA methylation, which influences the capacity of the individual to respond to variation in thermal environment in early and later life. For example, chickens that are exposed to a short cold challenge exactly during the HPT and HPA axis development in the egg are better heat-producers after hatching and later in life while in contrast, chickens exposed to similar short-duration heat challenges prenatally cope better in higher ambient temperatures later in life (Kamanli et al., 2015; Loyau et al., 2015; Morita et al., 2016; Piestun et al., 2008a, 2008b, 2009, 2015; Shinder et al., 2009; Yahav et al., 2004). For non-domesticated species, Nord and Nilsson (2011) and DuRant et al. (2011), found that blue tits and wood ducks (*Aix sponsa*) that were incubated in cold temperature throughout the incubation period had higher metabolic rate close to independence, which was interpreted in the same way – i.e. a response that might improve thermogenic capacity. However, wood ducks incubated in this manner were worse, not better, at dealing with a cold challenge shortly after hatching (DuRant et al., 2012, 2013). Similar results have been obtained for chickens when incubated in chronically hypothermic conditions (Black and Burggren, 2004), which suggests that there are switch points where a thermal dose during embryogenesis transitions from being ameliorating to being constraining.

Altered temperature tolerance is assumed to be associated with altered HPT-axis function across species: chickens exposed to pre-natal heat showed reduced TH levels and altered deiodinase enzyme expression in response to a thermal challenge later in life (Loyau et al., 2014; Nassar et al., 2015; Piestun et al., 2008a, 2008b, 2009) and thermal manipulation also influenced TH levels in hatching wood ducks (although later life THs nor responses to thermal stress were not studied, DuRant et al., 2014). Unfortunately, no data on the effects of prenatal temperatures on endocrine or other physiological mechanisms is available from wild birds. Interestingly, heat exposure also altered stress-induced CORT later in life in domestic chicken (e.g. Wilsterman et al., 2015). Thermal manipulation affected the proportion of thermosensitive neurons, which can then influence the downstream neuronal and hormonal responses involved in thermoregulation (Loh et al., 2004; Tzschentke and Basta, 2002). At the cellular level, expression of heat shock proteins (HSPs, i.e. protective protein chaperones) was lower in birds that were heat-challenged in the egg compared to controls when chicks were exposed to a thermal challenge after hatching. Interestingly, this was accompanied with altered DNA methylation of the HSP promoter regions following embryonic (day 15) heat-challenge in chicken (Vinoth et al., 2018). Recent transcriptomic studies suggest that pre-natal heat exposure may further affect vascularization and

angiogenesis in the chicken, likely influencing their ability to thermoregulate (Loyau et al., 2013, 2016). The neuronal/endocrinological pathways involved in these responses have, however, not been studied.

In addition to prenatal temperatures, we believe that an important avenue for further research will be to also address if post-hatching temperature could also have similar developmental programming functions, because chicks are arguably poorly buffered against temperature variation before their thermoregulatory system fully develops. For example, pigeons and chicken raised in very hot ambient temperatures in early life were found to have improved capacity for evaporative cooling as adults (Loyau et al., 2015; Marder and Arieli, 1988). Birds differ in the timing of development of thermoregulation and maturation of the HPT-axis: in altricial species development occurs post-hatch, while in precocial species it occurs largely during the embryonic period (reviewed by Debonne et al., 2008; McNabb, 2007; Price and Dzialowski, 2018). This suggests that altricial species might be particularly susceptible to epigenetic thermal adaptation post-hatching. Here again, studies extending beyond captive models, such as the precocial chicken, has high potential to reveal important pathways present in other bird taxa. Importantly, it is currently also not understood how permanent such adaptations are, as studies in chicken hardly extend beyond 30 days.

#### 5.5. Transgenerational plasticity

Ambient temperature variation in the parental environment may also influence offspring thermoregulation and temperature tolerance, so called transgenerational plasticity, as shown recently in multiple taxa (*Drosophila*: Cavieres et al., 2019; fish: Donelson et al., 2012; reviewed in Donelson et al., 2018; marine invertebrates: Morley et al., 2017; terrestrial invertebrates: Zizzari and Ellers, 2014). Similar data from birds are lacking. However, in birds, low ambient temperature increased the transfer of THs to eggs in great tits (Ruuskanen et al., 2016c), which may have consequences for development (Hsu et al., 2017; Ruuskanen et al., 2016a; Ruuskanen and Hsu, 2018), later HPT-axis function (Hsu et al., 2017), and thus potentially also thermoregulation. Furthermore, in chicken, maternal heat stress influenced embryonic heat shock protein expression and caused global DNA hypomethylation, but also increased oxidative damage (Zhu et al., 2017), while any corresponding influence on thermoregulation and adaptive plasticity has not been studied. Transgenerational plasticity could include a multitude of mediators of parental temperature effects, such as DNA methylation/histone modification, transfer of (small)mRNAs, hormones and other components in the eggs and sperm, with possible links to (the development of) thermoregulation, as shown in other taxa (Adrian-Kalchauer et al., 2018; Kekalainen et al., 2018; Salinas and Munch, 2012; Weyrich et al., 2016a, 2016b).

#### 5.6. Conclusions and notes

What becomes clear in this section is that there are correlative links showing that expression of thermoregulatory traits (and related energy and water balance) have an endocrine basis (on five time-scales), but that the responses may be either adaptive or mal-adaptive.

### 6. Thermoregulation in a changing climate: What is changing, and what are the challenges for thermoregulation?

Climate change brings about various types of challenges for thermoregulation, the most important of which are summarized in Fig. 4. It is likely that the predicted average increase of some 1.5 °C compared to pre-industrial times by the end of the 21st century will pose, at most, a minor direct threat per se, as both the absolute increase and the slow rate of temperature change is arguably well within scope of short- and long-term adaptations (see plasticity, section §4). Yet, indirect effects are likely. However, we caution on concluding on general trends, because (i)

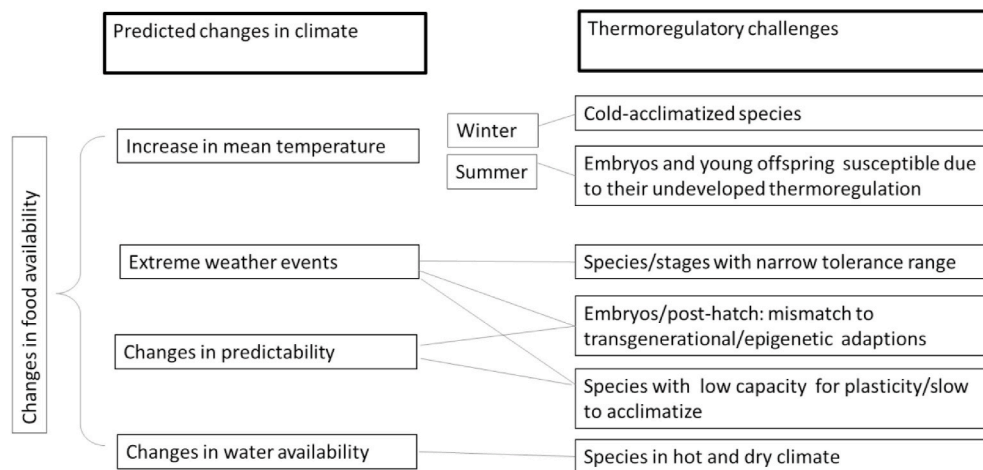


Fig. 4. Key thermoregulatory challenges in birds in changing climate. See text for references.

the changes in temperature are not likely to be uniform across the globe (e.g. warming is faster in the arctic region), and (ii) the scope for further adaptation may be limited in some species, e.g. in tropics and subtropics already enduring considerable heat load (cf. DuRant et al., 2019).

What is likely to be a more relevant challenge for thermoregulation is the predicted increases in the frequency of weather irregularities, such as heat waves (IPCC, 2014; Rahmstorf and Coumou, 2011; Stillman, 2019; Wingfield et al., 2017), which can bring negative and even lethal consequences in wild bird populations (desert populations of birds Gardner et al., 2017; McKechnie and Wolf, 2010) and for the poultry industry (Kumari and Nath, 2018). The challenge may be especially severe for species adapted to relatively narrow temperature ranges (Boyles et al., 2011a, 2011b), such as birds in the tropic (Huey et al., 2012; Khaliq et al., 2014), and those already operating close to their upper thermal limit such as desert birds (Gerson et al., 2019). However, even birds inhabiting far less extreme, thermally variable, and generally colder habitats, may suffer the negative consequences of acute heat stress (blue tits and great tits (Andreasson et al., 2018; Nord and Nilsson, 2019; Rodriguez et al., 2016).

In addition to heat waves, climate change is likely to lead to milder and more unstable winter weather (IPCC, 2014). This aspect has received much less attention than summer heat waves in both physiological and ecological research. Even moderate warming events in winter may cause problems in cold-acclimatized birds: for example Adelie penguins (*Pygoscelis adeliae*) suffered very low reproduction already at temperatures just above 0 °C and rainy conditions (Ropert-Coudert, 2014), though it is unclear if this was directly or indirectly linked to thermoregulation. Thus, relative, not only absolute, temperature changes, and the effects of changing temperature on the ecosystem, should always be considered.

Importantly, the most severe challenges may not be posed by temperature per se, but indirectly because mean temperature changes, extremes and changes in humidity are likely to influence food and water availability for birds at all trophic levels (Boyles et al., 2011a; Tieleman and Williams, 1999). We suggest that these indirect changes due to climate change can lead to challenges not only for energy acquisition, but also directly for thermoregulation for birds in hot and dry climates. Specifically, heat loss in birds is only by evaporation when ambient temperature exceeds  $T_b$ , and dry heat loss is increasingly less effective as the latter approaches the former. A modelling approach of avian evaporative water requirements and survival times revealed that the IPCC-predicted increases in maximum air temperatures will result in large 150–200% increases in water requirements relative to current values in small birds, which might severely reduce survival during extremely hot weather if water is not available (McKechnie and Wolf, 2010).

Finally, climate change also concerns production animals, as temperatures in rearing facilities cannot always be controlled. Poultry are genetically selected for a fast growth rate in a stable temperature, and express high metabolic rate which has impacted their temperature tolerance (Tickle et al., 2018), making them susceptible to both high and low environmental temperatures (Tickle and Codd, 2019). To this end, a vast amount of work has been performed to identify the physiological and behavioural responses to heat (and cold) stress to improve production strategies (reviewed in Kumari and Nath, 2018).

## 7. How to respond to climate change, and the role of endocrine control

Bird species may respond to new thermoregulatory challenges that follow climate change by three mechanisms: 1) populations may shift distributions to track their original environment (range shifts, reviewed e.g. in Devictor et al., 2008; La Sorte and Jetz, 2012); 2) individuals within a population may show phenotypic plasticity of thermal and metabolic responses to temperature, either within or across generations; 3) selection may lead to microevolutionary changes (i.e., adaptation) in temperature tolerance or plasticity and its underlying endocrinological and molecular mechanisms. Endocrine mechanisms could play a role in 2 and 3, and are thus discussed here.

### 7.1. Climate change and plasticity in thermoregulation: scope and speed, costs and constraints

Within-species plasticity of thermoregulation and associated endocrine traits were reviewed in section §4. The potential role of transgenerational plasticity in adapting to climate change has been recently addressed across taxa (Bonduriansky et al., 2012; Cavieres et al., 2019; Donelson et al., 2018; Meylan et al., 2012; Ruuskanen et al., 2016; Shama et al., 2014; Stillman, 2019). We argue that plasticity via transgenerational/epigenetic adaptation may allow birds to cope better with climate-change related higher/lower mean temperature. However, we also speculate that transgenerational effects may cause maladaptation. For example, if developmental temperature is predisposing for the thermoregulatory phenotype, this could occur if temperatures during parental/pre-natal/early postnatal stages differ from those that the individual will experience in its future environment, e.g. for birds that develop under the influence of a heat wave (cf. Burness et al., 2013). Alternatively, birds that develop during spells of extreme weather may suffer costs to normal growth and maturation, with potentially lasting negative effects on their metabolic phenotype. However, it has been recently suggested that embryos may also express considerable physiological and even behavioural plasticity in reptiles and birds (reviewed in

Du and Shine, 2015). The capacity for early-life plasticity can be crucial for survival: if extreme events would kill off young before they reach maturity, plasticity in adult individuals will be irrelevant (cf. Burggren, 2018).

Many bird species have excellent capacities to plastically acclimatize to different ambient temperatures during their lifetime (see sections 4.2, 4.3). Yet, there is also some evidence (from invertebrates) that higher basal temperature tolerance may constrain further thermal acclimatization (Gerken et al., 2015), which should be addressed in future studies. Furthermore, the speed of acclimation may be crucial in responding to unpredictable weather (see also Dubois et al., 2016; Swanson and Olmstead, 1999). There are indications that the acclimatization to increasing and decreasing temperature is not uniform: Dubois et al. (2016) found in experimental conditions that thermogenic capacity was lost at a faster rate when temperature increased, than it was gained when temperature decreased. When considering that thermogenic performance may be linked to overwinter survival (Petit et al., 2017), we propose that a reduction of thermogenic capacity during increasingly frequent spells of mild winter weather in the temperate region (IPCC 2014) could prove challenging for resident bird species.

A potential endocrine constraint for plasticity may lie in the fact that hormones have highly pleiotropic functions on physiology apart from their functions on thermoregulation and metabolism. For example, THs are critical in developmental programming, and CORT influences behavior and even memory. It is currently not understood if such pleiotropy may constrain optimal hormonal responses in temperature regulation. On the other hand, we may speculate that hormonal and associated transcriptomic changes in the above-mentioned plastic responses are likely to be relatively fast, and will not constrain the speed of acclimation. For example, there is evidence for very rapid cellular responses, such as  $\text{Na}^+\text{K}^+$ ATPase and oxygen consumption to heat treatment in non-model species (house sparrow, *Passer domesticus* Jimenez and Williams, 2014). Behavioral changes are likely to be immediate and may, at least partly, mitigate effects of acute thermal stress. For example, several studies show that the amount of physical work in birds is reduced at high ambient temperature (du Plessis et al., 2012; Powers et al., 2017), presumably to avoid somatic costs of overheating (Nilsson and Nord, 2018), though at the potential cost of reduced reproductive success (Nord and Nilsson, 2019). Beyond endocrine regulation, a major constraint on plasticity is likely to be the energetic (heat production) or water constraint (of by evaporative cooling), see §3.

All in all, while physiological and behavioural plasticity may be sufficient to respond to thermoregulatory needs to average temperature increases, inherent constraints and costs of acclimatization means that responses to weather extremes and an unpredictable climate in the future may not be sufficient in the long-term, and microevolution of thermal physiology may be needed.

### 7.2. Climate change, microevolution in thermoregulation and its underlying physiological mechanisms

A requirement for microevolutionary change in temperature tolerance is that the underlying physiological, endocrine and molecular mechanisms show genetic variation on which selection can act. Several key aspects in thermoregulation show individual and potentially genetic variation across taxa: Multiple studies in wild and captive birds report individual variation in BMR, reflected in moderate heritability and repeatability of these traits (Nilsson et al., 2009; Ronning et al., 2007; Tieleman et al., 2009). In one of the rare studies on potential heritability of heat loss mechanisms, Versteegh et al. (2008) reported that evaporative water loss also shows individual variation (repeatability). Moreover, several studies indicated that there is individual consistency in thermoregulatory reaction norms over a temperature gradient. This has been reported in ectotherms such as common lizards (*Zootoca vivipara*, Artacho et al., 2013) and Western slimy salamanders (*Plethodon albagula* Careau et al., 2014), and in endotherms such as mouse lemurs

(*Microcebus murinus*, (Vuarin et al., 2013) and zebra finches (*Taeniopygia guttata*, Briga and Verhulst (2017)). The potential genetic background of such individual plasticity in thermoregulatory reaction norms, has, however, not yet been studied.

Along with variation in metabolic rate, individual differences in hormonal and molecular responses to temperature variation is predicted, and will be a key in adapting to changing climate. To our knowledge, individual variation in responses to temperature challenges in circulating THs, the key hormones underlying thermoregulation, or associated molecular pathways (DIOs, receptors) has not been tested. However, there is some evidence on heritability of the mean levels of T3 in eggs (Ruuskanen et al., 2016b), suggesting scope for microevolutionary responses of THs to climate change. Interestingly, endocrine responses to thermal stress have been shown to vary among poultry breeds, potentially suggestive of genetic variation in temperature tolerance (Xie et al., 2018). Moreover, there is rather good evidence that individuals consistently differ in their CORT responses to standard stressor (Cockrem, 2013) as well as food stress (Lendvai et al., 2014), while CORT shows also moderate heritability (Stedman et al., 2017; Taff et al., 2018). To our knowledge, there is no data on individual plasticity in thermogenic mechanisms (ST, NST), mitochondrial function or its molecular basis (such as ANT, avUCP or SERCA expression, see §3) in birds.

Taken together, we still have very limited knowledge on the capacity for microevolution of thermoregulation and its associated endocrine and molecular mechanisms, and we currently lack direct studies on microevolution of thermal physiology in birds. However, some key thermoregulatory and endocrine traits seem to show individual variation with a genetic basis. In mammals, artificial selection for BMR led to changes in 10 generations (Sadowska et al., 2015), which suggests that a response to temperature changes could potentially be rather fast. However, even if there would be variation for selection to act on, the most important open question is which traits and mechanisms will/may be under selection in a changing climate. Is there selection on the mean values, or on scope and speed of plasticity when weather changes abruptly? What is the main selective agent? If selective agents like heat waves are rare, selective pressure is low, and microevolution likely plays a little role (Boyles et al., 2011b). However, increasing frequency of extreme events may increase the selective pressure on speed or scope of plasticity, at least if they occur frequently enough to be experienced more than once over the lifetime of an individual.

## 8. Conclusions and future directions

We conclude that while the general patterns in the endocrine regulation of thermogenesis are quite well understood, at least in poultry, the molecular and endocrine mechanisms controlling e.g. mitochondrial function need to be unveiled. In contrast, we have rather poor knowledge on the molecular mechanisms controlling heat loss. Birds express plasticity on a wide range of time scales, from transgenerational and developmental to day-to-day changes in response to environmental temperature variation, but the molecular mechanisms are also poorly described. Importantly, many of those can only be studied in non-model species that live under fluctuating temperature and experience climate change as it is, emphasizing the need for a wide range of study species to understand the scope of plasticity. Plasticity can ameliorate climate change effects on thermoregulation to some extent. However, the increased frequency of extreme weather events may be beyond the scope or speed for plastic responses, and so may potentially result in mass mortality (Gardner et al., 2017; McKechnie and Wolf, 2010) and selection for more tolerant phenotypes. To understand which traits can respond to selection, more studies on genetic variation in thermoregulation, its plasticity, and underlying endocrine and molecular mechanisms are needed. The combination of climate change related effects, that include alterations in humidity, precipitation patterns and primary production, are likely to pose even greater challenges for plastic



thermoregulatory phenotypes. The underlying endocrine and molecular mechanisms involved in such responses to the changing world, and their costs and constraints, should be studied more closely. We must also remember that even minor changes in absolute temperature can bring cascading effects to the ecosystems in which birds live, with potential negative feedback on several aspects of thermoregulation. Below, we outline some future directions:

### 8.1. Individual and population-level variation in endocrine responses to temperature

To understand if and how endocrine mechanism may adapt or constrain how populations and species may respond to changing temperatures, we need more experimental studies on the individual reaction norms on endocrine and metabolic traits, and underlying molecular mechanisms, as well as on the correlation between baseline and speed and slope of change. Data on heritability and plasticity could be acquired by measuring hormones in pedigreed populations, combined with within-subject experimental thermal exposures (relevant to climate change scenarios), measuring relevant hormones. It is especially important to not only consider the well-known endocrine pathways including THs, but extend to include other associated endocrine pathways such as CORT, melatonin and leptin.

### 8.2. Transgenerational/epigenetic adaptation and endocrine mechanisms

To understand transgenerational effects and developmental programming of thermoregulation, the molecular and endocrine mechanisms of transgenerational and epigenetic adaptation should be further addressed. This could be achieved via e.g. parental and prenatal manipulations (such as hormones) combined with post-natal/adult thermal challenges, and tracking the underlying molecular (e.g. DNA methylation) signatures.

### 8.3. More realistic experimental designs

Results of laboratory studies on acclimation are difficult to use in predicting how thermal physiology will be affected in wild populations where climate-change effects on temperature, humidity, and food availability can act synergistically on selection for thermal phenotypes. Similarly, behavioural strategies and movement that influence temperature tolerance in the field populations can only be simulated in the lab with difficulty. Following from the previous points, experimental designs testing thermal physiology should include multiple interacting factors as well as temporal stochasticity to yield more realistic estimates of how thermoregulatory and endocrine traits may be affected by climate change.

### Acknowledgements

We thank Laurence Besseau and Laura Nisembaum for invitation to the special issue, and two anonymous reviewers for comments that improved a previous version of the manuscript. SR was financially supported by the Academy of Finland. BYH was financially supported by the Academy of Finland and Ella and Georg Ehrnrooth Foundation. AN was supported by the Birgit and Hellmuth Hertz Foundation/The Royal Physiographic Society of Lund (grant no. 2017-39034).

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