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REVIEW

Endocrine Regulation of the Oxidative Metabolism in Poikilothermic vertebrates

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INTRODUCTION

Vertebrates have evolved a number of metabolic pathways to meet various requirements for successful survival. The oxidative metabolism is responsible for cellular oxidation of metabolic substrates and generation of energy which is stored in the form of ATP. The entire process of the biological oxidation (oxidative metabolism) involves a number of enzymes, co-enzymes and metabolic substrates, and consumes oxygen at the mitochondrial level. Further, the amount of energy produced is directly proportional to the amount of oxygen consumed. Therefore, the rate of oxygen consumption acts as an index of the metabolic rate/oxidative metabolism, and also reflects overall physiological status of an organism.

The metabolic rate (MR) of vertebrates depends on a number of external (environmental) and internal (physiological) factors, i.e., temperature, photoperiod, food, activity, body size and weight, age, sex, time of the day and month of the year, blood circulation and phylogeny etc. [1–3]. The neuroendocrine system plays an important role in the regulation of the metabolic rate. Hormones and neurohormones act at cellular and sub-cellular levels and alter the oxidative metabolism primarily by influencing the activities of enzymes of the oxidative pathways [4]. Central nervous system (CNS) and endocrine system interact with each other to different extents under different physiological/environmental conditions to meet the energy demand in the most suitable way [5–9].

A minimum metabolic rate operates even in the absence of physical performance, and is termed as obligatory, basal, maintenance or standard metabolic rate (SMR) [9]. SMR is essential to meet the minimum energy demand for vital processes like ventilation, circulation and the maintenance of ionic gradients across the cellular and sub-cellular membranes. Recent findings suggest that the metabolic reactions are nearly similar in all vertebrates with respect to the metabolic substrates, enzymes, co-factors, activators and inhibitors with few exceptions [10]. However, SMR is generally higher in homeotherms (birds and mammals) as compared to the poikilothermic vertebrates (fishes, amphibians and reptiles). A high homeothermic SMR is essential for maintaining relatively a constant body temperature. Poikilotherms are unable to maintain their body temperature that changes with ambient temperatures. The neuroendocrine system is involoved in metabolic regualtion in both homeotherms and poikilotherms [11–13]. In the present article, we have tried to give a brief account of the involvement of various hormones in the regulation of the oxidative metabolism of the poikilothermic vertebrates.

Thyroid hormones and the oxidative metabolism

Notwithstanding variations in morphology, location and organization of the thyroid gland in

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different classes of vertebrates, the follicular structure and function in relation to iodide metabolism and hormone synthesis as well as storage have been largely conserved. However, the same hormonal molecules—triiodothyronine (T_3) and thyroxine (T_4)—produce a wide range of diverse biological actions. Thyroid hormones play a major role in the regulation of SMR of birds and mammals [5, 11, 12, 14–17]. These hormones are also essential for the calorigenic action of the catecholamines in mammalian species [5, 7, 11, 18]. However, unlike in the homeotherms, the calorigenic function of the thyroid hormones in the poikilothermic vertebrates is not unequivocally accepted [8, 19–24].

In the last six decades, numerous attempts have been made to establish the calorigenic action of thyroid hormones in the ectothermic vertebrates. In the first few decades, a large number of investigators studied the effects of experimentally-induced hypo- and hyper-thyroidism on the metabolic rate of a large number of fishes, amphibians and reptilian species, and reported both positive and negative results. Several investigators have reported stimulation following administration of thyroid hormones and decrease in the respiratory rate following thyroidectomy (chemical/surgical/radiological) in a number of piscine species [25–45]. In contrast to these reports, an equally large number of scientists could not find any effect of thyroid hormones and thyroidectomy on the SMR of many species of fishes [46–62]. Similarly, many workers could not find any change in the SMR of amphibian species following the administration of the thyroid hormones and thyroidectomy [63–70]. Further, both endogenous and exogenous thyroid hormones reportedly have no effect on the SMR of metamorphosing amphibian larvae [69, 71–74]. Notwithstanding these negative results, the respiratory rate has been found to be stimulated by thyroid hormones in many amphibians [75–80]. As in fishes and amphibians, even in reptiles a few early attempts made to stimulate the rate of oxygen consumption by thyroid hormones were not successful [26, 47, 81]. No satisfactory explanation was provided for the contradictory reports regarding the calorigenic effect of thyroid hormones in the poikilothermic vertebrates till late fifties when, for the first time, importance of acclimation temperature came to light [82]. The information that thyroid hormones are calorigenically effective only at comparatively high (at or above 25°C) and ineffective at low (20°C or below) temperatures in the lizard, *Anolis carolinensis* reopened the issue of the possible involvement of the thyroid gland in the energy metabolism of the ectotherms, and provided a strong base for further studies including review of the previously reported negative results [8, 13, 20, 23, 24, 83].

Temperature-dependent calorigenic effect of thyroid hormones

Most of the earlier investigations were carried out under controlled laboratory conditions, but no importance was given to the acclimation temperatures. As mentioned earlier, the decisive role played by the ambient temperature became evident when the effects of thyroxine and thyroidectomy were studied on the respiratory rate of *Anolis carolinensis* at high and low temperatures [82]. Both administration of thyroxine and thyroidectomy were found to be effective in lizards maintained at high (ecritic) temperature and ineffective at low temperature. Subsequently, temperature-dependent effects of thyroid hormones and thyroidectomy on the oxidative metabolism was confirmed in *Lacerta muralis* [84], *Eumeces obsoletus* [85], *Eumeces faciatus* [86], *Sceloporus cyanogenys* [87], and *Natrix rhombifera* [88]. Similarly, Thapliyal and co-workers have reported temperature-dependent stimulatory effect of thyroxine and inhibitory effect of thyroidectomy on the respiratory rate of the whole body, liver and skeletal muscle of four tropical reptiles, i.e., *Calotes versicolor, Hemidactylus flaviviridis, Natrix piscator,* and *Ptyas mucosus* [83, 89, 91–100].

When acclimation temperature was considered, even in amphibians the temperature-dependent effect of thyroid hormones and thyroidectomy on the metabolic rate became evident. Thyroxine administration and thyroidectomy were found to be effective at higt temperatures and ineffective at low ambient temperatures in influencing the SMR of a number of amphibian species [101–106]. Recently, both *in vivo* and *in vitro* treatments with T_3

and T_4 did not stimulate the oxygen consumption rate of liver and muscle tissues of the Indian streaked frog, *Rana limnocharis* exposed to a natural temperature of 15°C (during the hibernation phase) and 21°C (during the post-hibernation phase) [107]. However, both T_3 and T_4 stimulated tissues respiration of the toad, *Bufo melanostictus* during the post-hibernation phase (25°C) but not during the hibernation phase (15°C) [108]. These findings suggest that the calorigenic effect of the thyroid hormones may vary with the species and temperatures.

A strong correlation has been reported between the thyroid activity and the oxygen consumption rate in an air breathing siluroid fish, *Heteropneustis fossilis* [45]. In the same study, thyroxine administration increased and treatment with thiouracil decreased the rate of whole body oxygen consumption under both surfacing-allowed and surfacing-not allowed conditions. In a recent study, *in vivo* and *in vitro* administrations of T_3 and T_4 did not stimulate the respiratory rate of liver and skeletal muscle tissues of an air breathing fish, *Clarias batrachus* maintained at 16°C [109]. This report again confirms the ineffectiveness of thyroid hormones at low temperature.

It is, thus, apparent from the studies conducted at different temperatures that thyroid hormones are involved in the regulation of the oxidative metabolism of the ectotherms exposed to high (favourable) temperatures. Moreover, the calorigenic ineffectiveness is conspicuous in all most all studies conducted at low ambient temperatures or during the hibernation phase (cold winter months). So far there is no information about the mechanism by which temperature influences the sensitivity of the oxidative pathways for thyroid hormones in the ectothermic vertebrates. Since temperature regulates a number of seasonal events (including activity, molt and reproduction) in poikilotherms, therefore, the calorigenic action of thyroid hormones in these vertebrates might be related to energy dependnet annual events [83, 110].

In vitro stimulation of oxygen uptake of various tissues of fishes, amphibians and reptiles strongly suggests that thyroid hormones stimulate oxidative processe by acting directly at cellular and/or subcellular levels [8, 83]. This suggestion is further supported by a number of recent reports indicating that both T_3 and T_4 stimulate a wide range of mitochondrial enzymes in a number of tissues (skeletal muscle, cardiac muscle, liver, gills, brain, kidney etc.) of fishes [40, 43, 111–113], amphibians [114–118], and reptiles [119–130]. Thyroid hormones are also involved in the regulation of the intermediary metabolism of the cold-blooded vertebrates [20, 23, 24, 89, 90, 105, 131–146]. Involvement of these hormones in both intermediary and oxidative metabolisms seems to suggest that even the metabolic function of the thyroid gland has been conserved to a greater extent during the complex course of evolution.

After monitoring the circulating level of thyroxine following administration of 200 ng T_4/g body weight in a lizard (*Dipsosaurus dorsalis*), John-Alder [119] concluded that in most of the earlier studies, pharmacological doses of thyroid hormones were used, therefore, the physiological significance of those studies remained doubtful. Further, these studies were conducted under simulated high and/or low temperatures, and no attempt was made to study the metabolic effects of thyroid hormones under natural climatic conditions with reference to the annual activity cycle. Therefore, we tried to understand the physiological importance of thyroid hormones in the oxidative metabolism of the Indian garden lizard. Effects of thyroidectomy and administration of T_4 were studied on the respiratory rate of the whole body and tissues (liver, skeletal muscle, kidney and brain) of the euthyroidic and thyroidectomized lizards maintained under natural temperatures and photoperiods during every month (January to December) of the year [83]. We also used a comparatively low dose of T_4 (66 ng/g body weight) which has been found recently to be well within the physiological range [124–126, 130]. Findings of the study clearly suggest that the calorigenic effect of thyroid hormones is associated with the annual activity cycle, and seems to be independent of the annual rhythm of temperature. Further, different tissues exhibit different annual sensitivity rhythms for thyroxine [83].

At present it is difficult to explain the exact mechanism responsible for the changes in sensitiv-

ity of tissues for T_4 with the changing seasons. However, changes in the nuclear and/or mitochondrial receptors, alterations in the binding sites on the receptor proteins, degree of saturation of receptors with the endogenous thyroid hormones, metabolic state of tissues, availability of substrates and energy demand, separately or in combination(s), might be responsible for the annual changes in tissue sensitivity for thyroxine. So far there is no information about the annual rhythms of tissues sensitivity for T_3. Significant stimulation by a physiological dose of thyroxine and marked decrease following thyroidectomy in the respiratory rate of the whole body and tissues (liver and skeletal muscle) during the entire active phase (post-hibernation to pre-hibernation), when energy demand for various purposes (foraging, offence/deffence, courtship etc.) is high [147], strongly favour a physiological role of thyroid hormones in regulation of the oxidative metabolism of the lizard as suggested in earlier studies. Thyroidectomy is found to increase the tissue sensitivity for T_4 [83]. This is further supported by the fact that thyroxine increases the SMR of the thyroidectomized but not of the euthyroidic lizards [124].

Implantation of T_4 to release physiological dose in the field active lizards has been reported to stimulate SMR, Vo_2max, running endurance and a number of oxidative enzymes [122, 123, 125, 130]. However, the same dose of implanted thyroxine could not increase the SMR of the captive lizards significantly. This might be probably due to the difference between the natural thermal range (23.6°C to 40.8°C) and the thermal range (19.3°C to 34.6°C) provided under captivity. Alternatively, stress-induced starvation and/or decreased muscular activity under captive condition might be responsible for the insignificant increase in the SMR. Starvation has been reported to decrease the thyroid hormone receptors in mammals [17]. Further, starvation also blocks the stimulatory effects of T_3 and T_4 on the respiratory rate of liver and skeletal muscle in a frog, *Rana limnocharis* maintained at 28°C (Gupta, unpublished). Thus, in addition to other factors, low temperatures and stress-induced starvation/anorexia also can negate the calorigenic effect of thyroid hormones in poikilothermic vertebrates. Now attempts should be made to define the mechanism that regulates sensitivity of the metabolic pathways for thyroid hormones in cold-blooded vertebrates.

Mechanisms of calorigenic action of thyroid hormones

Thyroid hormones have multiple sites of action within the body, even within a cell, and multiple mechanisms of action [17]. Three basic mechanisms are reportedly involved in the calorigenic action of thyroid hormones in mammals [7, 17]. These are (i) rapid stimulation of mitochondrial respiration under coupled state [148–159], (ii) an increase in the total mitochondrial content of tissues [160–163], and (iii) an increase in the activity of membrane-bound enzyme Na^+-K^+-ATPase [164–171]. These mechanisms are not delineated, therefore, may operate simultaneously. Further, it is also difficult to assess the relative contribution of each mechanism in mammalian calorigenesis. Thyroid hormones rapidly stimulate respiratory rate by acitng at mitochondria [17, 157]. This suggestion is further supported by dissociation between nuclear receptor concentration and stimulation of oxygen consumption in the neonatal rat brain, and also by the absence of mitochondrial thyroid hormone recptors in brain, testis and spleen—the tissues in which oxygen consumption is not stimulated by thyroid hormones [156]. Nuclear receptors of thyroid hormones are supposed to be involved in anabolic effects (growth, differentiation, maturation and cell maintenance) and prolonged calorigenesis [17, 157]. So far only a few attempts have been made to investigate the calorigenic mechasnisms of thyroid hormones in the ectothermic vertebrates. In the Indian garden lizard a single dose of thyroxine produces early (within 1 hr) and delayed (after 24 hr) stimulatory effects on the respiratory rate of the liver and skeletal muscle tissues [89]. While the early stimulatory effect of T_4 is blocked by ouabain (an inhibitor of Na^+-K^+-ATPase), manitol (an inhibitor of Na^+ transport), and chloromphenicol (an inhibitor of mitochondrial protein synthesis), the delayed stimulatory effect of thyroxine on the tissues respiration is inhibited by

actinomycin D (an inhibitor of nuclear transcription) [172, Sharan, Gupta and Thapliyal, unpublished]. These findings suggest that even in the lower vertebrates, thyroid hormones stimulate the oxidative metabolism mainly by acitng at cell membrane, mitochondrial and nuclear levels. Now attempts should be made to find out the relative importance of these mechanisms in the cold-blooded animals with special reference to the ambient temperature and the phases of the annual activity cycle.

Calorigenic action of gonadal hormones

Gondal steroids play a major role in the development, growth, maturation and maintenance of the primary and secondary sex organs and sex characters in all vertebrates. In addition, sex hormones have also been reported to influence metabolism of some other tissues of homeotherms. In poikilotherms, gonadal hormones seem to be involved in the regulation of the metabolic rate [8, 83, 107]. Administration of testosterone in males and estradiol in females is followed by stimulation of respiratory rate in a number of fishes [50, 60, 109, 173–175]. In some of the fishes, hypophysectomy resulted in decreased metabolic rate, presumably due to decreased levels of gonadal hormones [33, 176–178].

In a series of *in vivo* and *in vitro* experiments, Thapliyal and co-workers have reported significant increase in respiratory rates of vital tissues following administration of gonadal hormones, and reduction in the respiratory rates following castration/ovarietomy in a number of reptilian species [92, 93, 95, 96, 98, 99, 129, 179, 180]. We have studied the role of testicular hormones in the regulation of oxidative metabolism of the Indian garden lizard in detail. In this extensive study, effects of testosterone and thyroxine administration in intact, castrated and thyroidectomized lizards were recorded on the respiratory rates of the whole body and tissues (liver, muscle, kidney and brain) during all the twelve months of the year under natural climatic conditions [83]. In general, testosterone was found to be calorigenically more effective at low temperatures (during hibernation and early post-hibernation phases) where thyroxine was calorigenically ineffective. The annual sensitivity rhythms of the tissues for testosterone were found to be different. Castration decreased the respiratory rate of the whole body, liver and brain significantly during the post-hibernation phase, and of the skeletal muscle and kidney during all the months except December and January (hibernation phase). Testosterone administration invariably reversed the ill-effect of castration on the oxygen consumption rate of the whole body and the tissues. Testosterone also reversed decrease in the respiratory rate following thyroidectomy. Stimulation of respiratory rates of the whole body and tissues of the thyroidectomized lizards by testosterone strongly suggests a direct role of gonadal hormones in the energy metabolism of the lizards during the cold and non-breeding phase. Testosterone has been found to be calorigenic even at the simulated low temperature (15°C) where thyroxine failed in stimulating the oxygen uptake rate [95]. Calorigenic effect of gonadal hormones seems to be determined by the energy demand of various tissues. Recently testosterone has been reported to stimulate a number of mitochondrial enzymes in the liver of the Indian garden lizard [129].

In vivo and *in vitro* administration of testosterone and estradiol in low doses also stimulate the respiratory rate of liver and skeletal muscle tissues of both the sexes of the Indian streaked frog, *Rana limnocharis* [107]. Testosterone is found to be more potent in males and estradiol in females. Gonadal hormones stimulate tissues oxygen consumption during both hibernation and active phases at low temperatures where both T_3 and T_4 do not stimulate respiration of the tissues following both *in vivo* and *in vitro* administration [107]. Similarly, estradiol has been found to be calorigenic in the liver and muscle tissues of the female toad, *Bufo melanostictus* following both *in vivo* and *in vitro* treatments irrespective of season/activity phase [108]. Gonadal hormones have also been reported to exert significant effect on the intermediary metabolism and erythropoiesis of the pokilothermic vertebrates [181–189]. Gonadal steroids are also involved in the calorigenic mechanism of birds [14–16, 190]. The mechanism of calorigenic action of gonadal hormones remains

to be worked out. Involvement of gonadal steroids in the oxidative metabolism of lower vertebrates, especially at low ambient temperatures, might be of great significance for the survival and successful breeding of these cold-blooded animals under diverse climatic conditions [8, 83].

Calorigenic action of catecholamines

Catecholamines (norepinephrine and epinephrine) are synthesized and stored in the sympathoadrenomedullary (SAM) system, and released under diverse physiological conditions, i.e., stress (emotional, physical and physiological), emergency, danger etc. In addition to their effect on the cardio-vascular system, catecholamines invariably accelerate tissue respiration (non-shivering thermogenesis) in mammals. Both norepinephrine (NE) and epinephrine (EP) play a major role in the regulation of the oxidative metabolism of mammals exposed to low temperatures (191–201). The release of NE and EP is regulated as per erquirement by the central nervous system (hypothalamus) [5, 11, 202].

In poikilothermic vertebrates, calorigenic function of the catecholamines is not well studied [203]. Both EP and NE are reported to play a significant role in the regulation of the intermediary metabolism of fishes [204–211], amphibians [212–217] and reptiles [139, 218–220]. In a limited number of earlier studies, catecholamines inhibited the whole body oxygen consumption of fishes [221–223]. However, notwithstanding these reports, *in vivo* and *in vitro* treatments with comparatively low doses of EP and NE stimulated significantly the respiratory rate of liver and skeletal muscle tissues of the fish, *Clarias batrachus* [109]. Similarly, adrenaline administration in the fish *Anabas testiduneus* produces time- and dose-dependent stimulatory effect on activities of a number of oxiadtive enzymes in the liver tissue [224]. Catecholamones are also reported to stimulate the oxidative metabolism of amphibians [106–108, 225, 226] and reptiles [95, 203, 227]. In amphibians the relative potencies of EP and NE seem to vary with the species, tissues and seasons. In the Indian garden lizard, intra-muscular injection of adrenaline was found to be calorigenically more potent than noradrenaline in the euthyroidic lizards, however, both the hormones were equipotent in the thyroidectomized animals [227]. Furthre, the presence of thyroid is a pre-requisite for the calorigenic action of adrenaline in the garden lizard during the breeding phase [203].

Recently, administration of EP and NE have been reported to inhibit the resting metabolic rate (RMR) of *Alligator mississipiens* for about two hours and thereafter to stimulate the RMR which remains increased for many hours [220]. A large amount of ATP formed immediately after the intra-muscular injections of the catecholamines as a result of glycolysis has been found to be responsible for the transient inhibition of the RMR. A similar mechanism might be responsible for the reported inhibition of fish oxygen consumption [221–223]. It is noteworthy that the calorigenic action of the catecholamines in fishes, amphibians and reptiles is independent of ambient temperature and needs a very short lag period [107–109, 203, 220]. Due to these reasons, catecholamines are supposed to act as emergency hormones for the regulation of the energy metabolism in the poikilothermic vertebrates [8, 13, 107–109, 203, 227].

The mechanisms of calorigenic action of catecholamines is well studied in mammals [200]. The catecholamines act throught α- and β-adrenergic receptors [228]. The binding of catecholamines to the adrenergic receptors stimulates production of cAMP which activates a protein kinase. The cAMP-stimulated protein kinase phosphorylates and stimulates lipase which accelerates mobilization of free fatty acids (FFA). Then FFA are converted to acyl carnitine and acyl Co A. The increased levels of FFA stimulate proton conductance in the inner mitochondrial membrane. Finally, the increased proton conductance accelerates oxidation of acyl carnitine and results in increased rate of mitochondrial oxygen consumption. Thus, the catecholamines stimulate oxidative metabolism of mammals by accelerating mobilization as well as oxidation of FFA and involve cAMP as a second messenger. As in mammals, EP and NE also mobilize FFA in the ectothermic vertebrates [139, 204, 229–232]. Indirect evidences suggest that even in poikilotherms the catecholamine hormones stimulate oxygen

consumption by stimulating oxidation of FFA [203]. In addiition, EP and NE may also influence energy metabolism by their action on glycogenolysis and electron transport chain [203, 211, 218, 224].

Calorigenic effects of corticosteroids

The adrenal gland has evolved both structurally and functionally during the course of evolution. In homeotherms, corticosteroids are functionally divided into two groups—glucocorticoids and mineralocorticoids. However, a functional classification of the cortical hormones is not possible in the lower vertebrates where various corticoids share overlapping functions [233]. Glucocorticoids are reported to stimulate the oxidative metabolism in mammals by their action on oxidative enzymes and the electron transport directly. They also support the calorigenic action of other metabolic hormones [5, 11, 234, 235]. An intact and normally functioning adrenal cortex is essential for normal thermoregulation at low temperatures [5, 236]. Unlike in mammals, there is scarcity of information regarding the calorigenic function of corticoid hormones in lower vertebrates [233]. In the Indian garden lizard, corticosterone administration had no effect on the whole body oxygen consumption in any season. However, depending on the season (activity phase) and thyroidal status, it may stimulate, inhibit or produce no effect on respiratory rate of liver, muscle, kidney and brain [203]. Corticosterone seems to be calorigenic in aquatic (fish) and semiaquatic (amphibians) vertebrates where corticosteroids are involved in the energy-dependent osmoregulatory mechanisms. Corticosterone stimulates respiratory rate of liver and muscle tissues of a fish [109], a frog [107] and a toad [108]. Hydrocortisone stimulates cytochrome oxidase activity in the liver of *Calotes versicolor* in a dose-dependent manner [237]. The calorigenic effect of corticoid hormones has been found to be rapid and temperature independent. There are many reports which suggest that adrenocortical hormones play an important role in the regulation of intermediary metabolism in cold-blooded vertebrates [238–244]. Corticosterone-induced increase in the respiratory rate might be associated with energy consuming anabolic processes, mobilization and oxidation of FFA, and osmoregulation. The question, whether corticosteroids influence the oxidative metabolism of ectothermic vertebrates directly or indirectly by playing a permissive role to support the calorigenic actions of other hormones (as reported in mammals), remains to be answered. Further attempts should be made to study effects of corticosteroid hormones on activities of enzymes associated with the oxidative pathways and energy-dependent osmoregulatory process.

Melatonin in calorigenesis

It has been suggested that photoperiod, acting via pineal gland, is capable of influencing energy metabolism and thermoregulation in mammals and birds [9]. Short photoperiods and melatonin administration increase the behavioral thermoregulation as well as non-shivering thermogenesis, and also improve the thermogenic capacity of homeotherms [245–253]. However, it is not clear whether melatonin affects the oxidative metabolism directly or potentiates the calorigenic effects of catecholamines [247, 254, 255]. There are a few indications that melatonin can influence metabolic rate of mammals also by affecting thyroid activity [256]. There is scarcity of information on the role of pineal and melatonin in the energy metabolism of the ectothermic vertebrates. There is practically no information on the metabolic effect of melatonin in any fish. The role of the pineal complex in thermoregulation of amphibians and reptiles is not clear [255, 256]. Effect of parietalectomy in the frog, *Rana temporaria* reported at different temperatures is inconsistent [257, 258]. Some of the findings suggest that interactions between temperature and photoperiod, depending on the circannual phase, produce varied effects on the amphibian metabolic rate [257, 259, 260]. However, there is complete lack of information regarding effects of melatonin and pinealectomy on the oxidative metabolism and/or thermo-regulation of amphibians.

The pineal complex, however, seems to be involved in the thermoregulation of lizards [261–263]. In general, parietalectomy is reported to

increase [264] and pinealectomy to decrease the body temperature of lizards [265–267]. These reports suggest that the pineal complex mediates both behavioral and physiological thermoregulation through the CNS (hypothalamus) involving neuroendocrine mechanisms [268]. The precise role of the pineal complex in the regulation of the reptilian energy metabolism remains to be investigated. In a series of reports, photoperiod has been found to influence the body temperature of the European green lizard, *Lacerta viridis* [269–272]. In this lizard, short photoperiods and administration of melatonin reduce body temperature. Since the environmental light-dark cycle not only acts as a "Zeitgeber" (time keeper) but also provides the main source for behavioral thermoregulation, therefore, the thermoregulatory effect of photoperiod via pineal complex is supposed to be of adaptive significance. The precise mode of melatonin action in the poikilothermic metabolism remains practically unknown.

Summary and Conclusions

The oxidative metabolism of poikilothermic vertebrates is regulated by a complex neuroendocrine mechanism. Hormones of thyroid, gonads, adrenal and pineal are actively involved in the regulatory process. Recent reports, based on carefully designed experiments, unequivocally and strongly suggest that thyroid hormones play a leading role in the metabolic regulation of lower vertebrates during the active phase. The calorigenic action of thyroid hormones seems to depend largely on the energy demand associated with physical and/or physiological activities of the animals. In addition to temperature, hormones associated with feeding and fasting seem to influence the calorigenic action of these hormones. The precise mechanism, which regulates metabolic action of T_3 and T_4 at cellular level, remains unknown. Indirect evidences suggest that the calorigenic action of these hormones might be regulated by receptors of thyroid hormones and/or inducibility of the oxidative enzymes which are influenced by a number of external and internal factors.

The involvement of gonadal steroids and the pineal complex, which are controlled mainly by the circadian and circannual variations in day-lenghth (photoperiod), seems to synchronize and couple the annual cycles of energy metabolism and breeding phase to ensure successful and timely reproduction. Stimulation of the respiratory rate by gonadal steroids at low temperatures probably ensures timely emergence from hibernation, and also helps in calorigenesis during the active phase in the event of sudden drop in environmental temperature.

Adrenal hormones in general and catecholamines in particular act as emergency hormones for the regulation of calorigenesis in ectotherms. Catecholamines (NE & EP) accelerate respiratory rate of all tissues instantly irrespective of seasons and environmental temperatures. There is scarcity of information on the calorigenic action of corticosteroid hormones in lower vertebrates.

On the basis of the available information, it can be concluded that the oxidative metabolism of pokilothermic vertebrates is regulated by a neuroendocrine mechanism having three major components—(i) thyroid hormones (regulated primarily by temperature), (ii) Hormones of gonads and pineal (controlled mainly by environmental light), and (iii) sympatho-adrenomedullary system (regulated by the central nervous system). While the first two components govern strategies for long term energy production associated with the annual events, the third component seems to ensure rapid but transient energy production under emrgency/life-threatening situations. It is, thus, apparent that the cold-blooded vertebrates possess an endogenous mechanism which regulates energy metabolism efficiently according to energy demand and ensures successful survival even at the ever-changing body temperature.

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REFERENCES

1 Prosser, C. L. (1973) In "Comparative Animal

Physiology". Ed. by C. L. Prosser, W. B. Saunders Co., Philadelphia, London, Toronto, 3rd Ed., pp. 165–206.

2 Coulson, R. A. (1986) Comp. Biochem. Physiol., **84A**: 217–229.

3 Bennet, A. F. (1988) Amer. Zool., **28**: 699–708.

4 Darnell, J., Lodish, H. and Baltimore, D. (1986) In "Molecular Cell Biology". Scientific American Books, Inc., New York, pp. 859–910.

5 Gale, C. C. (1973) Ann. Rev. Physiol., **35**: 391–430.

6 Horwitz, B. A. (1979) Fed. Proc., **38**: 2147–2149.

7 Himms-Hagen, J. (1983) In "Mammalian Thermogenesis". Ed. by L. Girardier and M. Stock, Chapman and Hall, London, pp. 141–177.

8 Thapliyal, J. P. and Gupta, B. B. P. (1983) In "Recent Trends in Life Sciences". Ed. by A. Gopalakrishna, S. B. Singh and A. K. Saxena, Manu Publication, Kanpur, pp. 260–275.

9 Heldmaier, G., Klaus, S., Wiesinger, H., Friedrich, U. and Wenzel, M. (1989) In "Living in the Cold II". Ed. by A. Malan and B. Canguilhem, John Libbey Eurotext Ltd., pp. 347–358.

10 Coulson, R. A., Herbert, J. D. and Coulson, T. D. (1989) Amer. Zool., **29**: 921–934.

11 Jansky, L. (1973) Biol. Rev., **48**: 85–132.

12 Thapliyal, J. P., Lal, P., Gupta, B. B. P. and Sharan, S. (1983) Gen. Comp. Endocrinol., **49**: 333–343.

13 Gupta, B. B. P. (1987) In "Proc. First Congress of the Asia and Oceania Society for Comparative Endocrinology". Ed. by E. Ohnishi et al., Nagoya University Corporation (Printing Section), Nagoya, Japan, pp. 265–266.

14 Thapliyal, J. P., Pati, A. K. and Gupta, B. B. P. (1982) Gen. Comp. Endocrinol., **48**: 84–88.

15 Thapliyal, J. P., Lal, P., Pati, A. K. and Gupta, B. B. P. (1983) Gen. Comp. Endocrinol., **51**: 444–453.

16 Gupta, B. B. P. and Thapliyal, J. P. (1984) Gen. Comp. Endocrinol., **56**: 66–69.

17 Ingbar, S. H. (1985) In "Williams Text Book of Endocrinology". Ed. by J. D. Wilson and D. W. Foster, W. B. Saunders Co., Philadelphia, pp. 682–815.

18 Fregly, M. J., Field, F. P., Katovich, M. J. and Barney, C. C. (1979) Fed. Proc., **38**: 2162–2169.

19 Gorbman, A. (1969) In "Fish Physiology". Ed. by W. S. Hoar and D. J. Randal, Academic Press, New York, Vol. 2, pp. 241–274.

20 Lynn, W. G. (1970) In "The Biology of the Reptilia". Ed. by C. Gans and T. S. Pearsons, Academic Press, New York, Vol. 3, pp. 201–234.

21 Sage, M. (1973) Am. Zool., **13**: 899–905.

22 Leibson, L. G. and Plisetskaya, E. M. (1973) In "Intern. Encycloped. Pharm. Therap". Ed. by M. Michelson, Sect. 85, pp. 625–685.

23 Eales, J. G. (1979) In "Hormones and Evolution". Ed. by E. J. W. Barrington, Academic Press, New York, Vol. I, pp. 341–436.

24 Rosenkilde, P. (1979) In "Hormones and Evolution". Ed. by E. J. W. Barrigton, Academic Press, New York, Vol. I, pp. 437–491.

25 Smith, H. W. (1930) J. Biol. Chem., **88**: 97–130.

26 Haarman, W. (1936) Arch. Exptl. Pathol. Pharmakol. Naunyn-Schmiedberg's **180**: 167–182.

27 Smith, D. C. and Mathews, S. A. (1947) Anat. Record **99**: 593.

28 Smith, D. C. and Mathews, S. A. (1948) Am. J. Physiol. **153**: 215–221.

29 Jones, R. W., Gibson, W. C. and Nicholls, C. Jr. (1951) Anat. Record **111**: 509.

30 Osborn, P. E. (1951) Progr. Fish Cult., **13**: 75–80.

31 Zaks, M. G. and Zamkova, M. A. (1952) Dokl. Akad. Nauk. USSR., **84**: 1101–1103.

32 Müller, J. (1953) Z. Vergl. Physiol., **35**: 1–12.

33 Chavin, W. and Rossmoore, H. W. (1956) Anat. Record **125**: 599.

34 Hopper, A. F. (1959) Growth **23**: 13–19.

35 Mohsen, I. and Godet, R. (1960) Nature (London) **185**: 108.

36 Pritchard, A. W. and Gorbman, A. (1960) Biol. Bull., **119**: 109–119.

37 Wolf, E. C. and Wolf, J. (1964) In "The Thyroid Gland". Ed. by R. Pitt-Rivers and W. R. Trotter, Butterworths, London, Vol. I, p. 237.

38 Sage, M. (1965) Gen. Comp. Endocrinol., **5**: 706–707.

39 Sage, M. (1968) Gen. Comp. Endocrinol., **10**: 304–309.

40 Massey, B. D. and Smith, C. L. (1968) Comp. Biochem. Physiol., **25**: 241.

41 Ruhland, M. (1969) Experientia **25**: 944–945.

42 Ruhland, M. (1971) Can. J. Zool., **49**: 423–425.

43 Leray, C., Bonnel, A., Febreve, A., Vallet, F. and Pic, P. (1970) Ann. Endocrinol., **33**: 567–572.

44 Gabos, M., Pora, E. A. and Race, L. (1973) Studii se Cercetâri de Bioogie **25**: 39–43.

45 Pandey, B. N. and Munshi, J. S. D. (1976) J. Endocrinol., **69**: 421–425.

46 Horton, F. M. (1934) J. Exp. Biol., **11**: 257–261.

47 Drexler, E. and von Issekutz, B. (1935) Arch. Exp. Pathol. Pharmakol., **177**: 435–441.

48 Root, R. W. and Etkin, W. (1937) Proc. Soc. Exp. Biol. Med., **37**: 174–175.

49 Etkin, M. N., Root, R. W. and Mofshin, B. D. (1940) Physiol Zool., **13**: 415–429.

50 Hasler, A. D. and Meyer, R. K. (1942) J. Exp. Zool., **91**: 391–404.

51 Smith, D. C. and Everett, G. M. (1943) J. Exp. Zool., **94**: 229–240.

52 Punt, A. and Jungbloed, J. (1945) Arch. Nearl. Zool., **7**: 1–15.

53 Leach, W. J. (1946) Physiol. Zool., **19**: 365–374.

54 Mathews, S. A. and Smith, D. C. (1947) Physiol. Zool., **20**: 161–164.

55 Matty, A. J. (1954) J. Marine Biol. Assoc. U. K., **33**: 689–697.

56 Matty, A. J. (1957) J. Endocrinol., **15**: 1–8.

57 Baraduc, M. M. (1954) c. r. hebd. Seane. Soc. Biol. Paris **151**: 393–405.

58 Suhrmann, R. (1955) Biol. Zbl., **74**: 432–448.

59 Fromm, P. O. and Reineke, E. P. (1956) J. Cell Comp. Physiol., **48**: 393–404.

60 Hoar, W. S. (1958) Can. J. Zool., **36**: 113–121.

61 Thornburn, C. C. and Matty, A. J. (1963) Comp. Biochem. Physiol., **1**: 1–12.

62 Klieka, J. (1965) Physiol. Zool., **38**: 177–179.

63 Gayda, T. (1922) Arch. Physiol.,**20**: 267–285.

64 von Issekutz, B., Tukats-Leinzinger, M. and von Issekutz, A. M. (1943) Arch. Exptl. Pathol. Pharmakol. Naunyn-Schmiedberg's **201**: 334–345.

65 Lynn, W. G. and Wachowsky, H. E. (1951) Quart. Rev. Biol., **26**: 123–168.

66 Galton, V. A. and Ingbar, S. H. (1962) Endocrinology **70**: 622–632.

67 Dodd, J. M. and Matty, M. J. (1964) In "The Thyroid Gland". Ed. by R. Pitt-Rivers and W. R. Trotter, Butterworths, London, Vol. I, pp. 303–356.

68 Thornburn, C. C. and Matty, A. J. (1964) J. Endocrinol., **28**: 213–217.

69 Marusic, E., Martinez, R. and Torretti, J. (1966) Proc. Soc. Exp. Biol. Med., **122**: 164–167.

70 Tayler, R. E. and Barker, S. B. (1967) Gen. Comp. Endocrinol., **9**: 129–134.

71 Etkin, W. (1934) Physiol. Zool., **7**: 129–148.

72 Fletcher, K. and Mayant, N. B. (1959) J. Physiol., **145**: 353–368.

73 Lewis, E. J. C. and Frieden, E. (1959) Endocrinology **65**: 273–282.

74 Funkhouser, A. and Mills, K. S. (1969) Physiol. Zool., **42**: 22–28.

75 Taylor, A. (1939) J. Exp. Zool., **81**: 135–146.

76 Warren, M. R. (1940) J. Exp. Zool., **83**: 127–159.

77 Barch (1954–1955) Cited in "Comparative Endocrinology". Ed. by A. Gorbman, John Wiley and Sons, Inc., New York/London, 1959, p. 279.

78 Donoso, A. O. and Trivelloni, J. C. (1958) Rev. Soc. Arg. Biol., **34**: 46–52.

79 Jankowsky, H. O. (1960) Z. Vergl. Physiol., **43**: 392–410.

80 Jenkin, P. M. (1962) In "Animal Hormones—A Comparative Survey", Oxford-Pergamon Press, Part I, pp. 167–185.

81 Scott, A. H. (1935) Am. J. Physiol., **III**: 105–117.

82 Maher, M. J. and Levedahl, B. H. (1959) J. Exp. Zool., **140**: 169–189.

83 Gupta, B. B. P. and Thapliyal, J. P. (1985) Gen. Comp. Endocrinol., **58**: 20–27.

84 Maher, M. J. (1961) Amer. Zool., **1**: 461.

85 Maher, M. J. (1964) Endocrinology **74**: 994–995.

86 Maher, M. J. (1965) Gen. Comp. Endocrinol., **5**: 320–325.

87 Wilhoft, D. C. (1966) Gen. Comp. Endocrinol., **7**: 445–451.

88 Turner, J. E. and Tipton, S. R. (1972) Gen. Comp. Endocrinol., **18**: 98–101.

89 Thapliyal, J. P. (1980) In "Hormones, Adaptation and Evolution". Ed. by S. Ishii, M. Wada and T. Hirano, Japan Sci. Soc. Press, Tokyo/Springer-Verlag, Berlin, pp. 241–250.

90 Thapliyal, J. P. (1980) In "Proc. Natl. Acad. Sci. India, Golden Jubilee Comemmoration Volume", pp. 531–540.

91 Chandola, A., Kumar, D. S. and Thapliyal, J. P. (1973) VII Conf. Euro. Comp. Endocrinol., Budapest (Abstract).

92 Chandola, A., Kumar, D. S. and Thapliyal, J. P. (1974) J. Endocrinol., **61**: 285–291.

93 Thapliyal, J. P., Chandola, A. and Kumar, D. S. (1974) J. Endocrinol., **63**: 419–420.

94 Gupta, B. B. P. (1982) Ph. D. Thesis, Banaras Hindu University, Varanasi, India.

95 Thapliyal, J. P. and Gupta, B. B. P. (1984) Indian J. Exp. Biol., **22**: 179–181.

96 Chandola, A., Kumar, D. S. and Thapliyal, J. P. (1974) J. Endocrinol., **63**: 191–199.

97 Kumar, D. S., Oommen, O. V. and Thapliyal, J. P. (1974) In "Proc. Internatl. Union Physiol. Sci. XI", New Delhi (Abs.).

98 Thapliyal, J. P., Kumar, D. S. and Garg, R. K. (1974) Gen. Comp. Endocrinol., **22**: 308–311.

99 Thapliyal, J. P., Kumar, D. S. and Oommen, O. V. (1975) Gen Comp. Endocrinol., **26**: 100–106.

100 Kar, I., Thapliyal, J. P. and Singh, R. (1989) Arch. Biol. (Bruxelles) **100**: 1–6.

101 Maher, M. J. (1967) Copeia **1967(2)**: 361–365.

102 McNabb, R. A. (1969) Gen. Comp. Endocrinol., **12**: 276–281.

103 Packard, G. C., Packard, M. J. and Stiverson, R. K. (1974) Gen. Comp. Endocrinol., **22**: 195–198.

104 Packard, G. C. and Packard, M. J. (1975) Gen. Comp. Endocrinol., **27**: 162–168.

105 Rosenkilde, P. (1981) In "Proc. 9th Gunma Symp. Endocrinology", Maebashi, Japan.

106 Gupta, B. B. P. and Chakrabarty, P. (1988) In

"Proc. Symp. Recent Trends in Comparative Endocrinology", March 11–13, Varanasi, India.
107 Gupta, B. B. P. and Chakrabarty, P. (1990) Indian J. Exp. Biol., **28**: 23–26.
108 Gupta, B. B. P. and Deka-Borah, H. (1991) Indian J. Exp. Biol., (In press).
109 Gupta, B. B. P. (1988) In "Proc. Natl. Symp. Current Status of Gen. Comp. Endocrinol.". November, 25–27, Delhi, India, pp. 70–71.
110 Barrington, E. J. W. (1975) In "An Introduction to General and Comparative Endocrinology". Clarendon Press, Oxford, pp. 147–185.
111 Shivakumar, K. and Jayaraman, J. (1984) Arch. Biochem. Biophys., **233**: 728–735.
112 Leloup, J. and Luze, A. D. (1985) In "The Endocrine System and the Environment". Ed. by B. K. Follett, S. Ishii and A. Chandola, Springer-Verlag, Berlin, pp. 23–32.
113 Peter, S. M. C. and Oommen, O. V. (1987) J. Reprod. Biol. Comp. Endocrinol., **7**: 53–60.
114 Lagerspetz, K. Y. H., Harri, H. N. E. and Okslarti, R. (1974) Gen. Comp. Endocrinol., **22**: 169–176.
115 Brucker, R. F. and Cohen, P. P. (1976) J. Biol. Chem., **251**: 6161.
116 Lagerspetz, K. Y. H. (1977) J. Therm. Biol., **2**: 223–231.
117 Goto, Y., Amuro, N. and Shukuya, R. (1982) Biochim. Biophys. Acta **719**: 102–109.
118 Sutharam, K. K., Peter, S. M. C. and Oommen, O. V. (1990) Gen. Comp. Endocrionol., **79**: 246–252.
119 John-Alder, H. B. (1983) Amer. J. Physiol., **244**: R659–R666.
120 John-Alder, H. B. (1984) J. Comp. Physiol., **154B**: 409–419.
121 John-Alder, H. B. (1984) J. Exp. Biol., **109**: 175–189.
122 John-Alder, H. B. (1986) Amer. Zool., **26**: 23A.
123 John-Alder, H. B. (1988) Amer. Zool., **28**: 43A.
124 John-Alder, H. B. (1990) Gen. Comp. Endocrinol., **77**: 52–62.
125 John-Alder, H. B. (1990) Physiol. Zool., **63**: 600–614.
126 John-Alder, H. B. and Joos, B. (1991) Gen. Comp. Endocrinol., **81**: 120–132.
127 Begum, K. A., Behera, H. N. and Patnaik, B. K. (1984) Gen. Comp. Endocrinol., **53**: 402–409.
128 Sham, J. S. K., Chiu, K. W. and Pang, P. K. T. (1987) Amer. Zool., **27**: 121–131.
129 Oommen, O. V. and Sreedeviamma, K. K. (1988) Gen. Comp. Endocrinol., **69**: 391–398.
130 Joos, B. and John-Alder, H. B. (1990) Physiol. Zool., **63**: 873–885.
131 Hanke, W. (1974) In "Chemical Zoology" Ed. by M. Florkin and B. T. Scheer, Academic Press, New York and London, pp. 123–159.
132 Thapliyal, J. P., Gupta, S. C. and Garg, R. K. (1974) J. Endocrinol., **60**: 517–524.
133 Narayansingh, T. and Eales, J. G. (1975) Comp. Biochem. Physiol., **52**: 407.
134 Packard, G. C. and Randall, J. G. (1975) J. Exp. Zool., **191**: 365–370.
135 Thapliyal, J. P. and Gupta, S. C. (1975) Endocrinol. Exptl., **9**: 73–76.
136 Gupta, S. C., Thapliyal, J. P. and Garg, R. K. (1975) Gen. Comp. Endocrionol., **27**: 223–229.
137 Eales, J. G. (1990) In "Progress in Comparative Endocrinology", Willey-Liss, Inc., pp. 415–420.
138 Chiu, K. W. (1982) In "Phylogenetic Aspects of Thyroid Hormone Action", Ed. by Inst. Endocrinol., Gunma University, Acad. Publ., Tokyo, pp. 107–122.
139 Gupta, S. C. and Thapliyal, J. P. (1982) J. Endocrinol., **94**: 333–338.
140 Plisetskaya, E., Woo, N. Y. S. and Murat, J. C. (1983) Comp. Biochem. Physiol., **74A**: 179–187.
141 Kühn, E. R. Darras, V. M. and Gevaerts, H. (1985) In "The Endocrine System and the Environment". Ed. by B. K. Follett, S. Ishii and A. Chandola, Japan. Sci. Soc. Press, Tokyo/Springer-Verlag, Berlin, pp. 55–69.
142 Chiu, K. W., Sham, J. S. K., Maderson, P. F. A. and Zucker, A. (1986) Comp. Biochem. Physiol., **84A**: 345–351.
143 Dickhoff, W. W. and Sullivan, C. (1987) Amer. Fish Soc. Symp., **1**: 197–210.
144 Gapp, D. A. (1987) In "Fundamentals of Comparative Vertebrate Endocrinology", Ed. by I. Chestro-Jones, P. M. Ingleton and J. G. Phillips, Plenum Press, New York, pp. 623–660.
145 Hilton, J. W., Plisetskaya, E. M. and Leatherland, J. F. (1987) Fish Physiol. Biochem., **4**: 113–120.
146 Chiu, K. W. and Woo, N. Y. S. (1988) J. Therm. Biol., **13**: 179–184.
147 Gupta, B. B. P. and Thapliyal, J. P. (1986) Proc. Indian Acad. Sci. (Animal Sci.), **95**: 403–410.
148 Buchanan, J. and Tapley, D. F. (1966) Endocrinology **79**: 81–89.
149 Buchanan, J., Primack, M. P. and Tapley, D. F. (1970) Endocrinology **87**: 993–999.
150 Buchanan, J. L.., Primack, M. P. and Tapley, D. F. (1971) Endocrionology **89**: 534–537.
151 Primack, M. P., Tapley, D. F. and Buchanan, J. (1972) Endocrinology **91**: 840–844.
152 Primack, M. P. and Buchanan, J. L. (1974) Endocrinology **95**: 619–620.
153 Sterling, K. and Milch, P. O. (1975) Proc. Natl. Acad. Sci. U. S. A., **72**: 3225–3229.
154 Tata, J. R. (1975) Nature **257**: 18–23.
155 Sterling, K., Milch, P. O., Brenner, M. A. and

Lazaras, J. H. (1977) Science **197**: 996–998.
156 Sterling, K., Lazaras, J. H., Milch, P. O., Sakurada, T. and Brenner, M. A. (1978) Science **201**: 1126–1129.
157 Sterling, K., Brenner, M. A. and Sakurada, T. (1980) Science **210**: 340–342.
158 Shears, S. B. (1980) J. Theor. Biol., **82**: 1–13.
159 Triandafillou, J., Gwilliam, C. and Himms-Hagen, J. (1982) Can. J. Biochem., **60**: 530–537.
160 De Leo, T., De Meo, S., Barletta, A., Martino, G. and Goglia, F. (1976) Pflügers Arch., **366**: 73–77.
161 Nishiki, K., Erecinska, M., Wilson, D. F. and Cooper, S. (1978) Amer. J. Physiol., **235**: C212–C219.
162 Courtright, J. B. and Fitts, R. H. (1979) Horm. Metabol. Res., **11**: 304–306.
163 Wooten, W. L. and Cascarano, J. (1980) J. Bioenergetics Biomembranes **12**: 1–12.
164 Ismail-Beigi, F. and Edelman, I. S. (1970) Proc. Natl. Acad. Sci. U.S.A., **67**: 1071–1078.
165 Ismail-Beigi, F. and Edelman, I. S. (1971) J. Gen. Physiol., **57**: 710–722.
166 Edelman, I. S. (1974) New Engl. J. Med., **290**: 1303–1308.
167 Edelman, I. S. (1976) Fed. Proc., **35**: 2180–2184.
168 Folke, M. and Sestoft, L. (1977) J. Physiol., **269**: 407–419.
169 Biron, R., Burger, A., Chinet, A., Clausen, T. and Dubois-Ferriére, R. (1979) J. Physiol., **297**: 47–60.
170 Ismail-Beigi, F., Bissell, D. M. and Edelman, I. S. (1979) J. Gen. Physiol., **73**: 369–383.
171 Smith, T. J. and Edelman, I. S. (1979) Fed. Proc., **38**: 2150–2153.
172 Sharan, S. S. (1983) Ph. D. Thesis, Banaras Hindu University, Varanasi, India.
173 Raffy, A. and Fountaine, M. (1930) c. r. Soc. Biol. (Paris) **104**: 287–288.
174 Stanley, L. L. and Tescher, G. L. (1931) Endocrinology **15**: 55–56.
175 Mann, H. (1939) Zool., Anz., **127**: 315–318.
176 Hanson, R. C. and Stanley, J. G. (1970) Comp. Biochem. Physiol., **33**: 871–879.
177 Johanson, P. H. and Gomery, J. D. (1973) Can. J. Zool., **51**: 1289–1291.
178 Chan, D. K. O. and Woo, N. Y. S. (1978) Gen Comp. Endocrinol., **35**: 160–168.
179 Oommen, O. V. (1980) J. Anim. Morphol. Physiol., **27**: 124–131.
180 Oommen, O. V. (1981) Comp. Physiol. Ecol., **6**: 95–98.
181 Rao, C. A. P. and David, G. F. X. (1967) Gen. Comp. Endocrinol., **9**: 27.
182 Follett, B. K. and Redshaw, M. R. (1968) J. Endocrinol., **40**: 436.
183 Kaur, R. J. and Thapliyal, J. P. (1975) Gen. Comp. Endocrinol., **27**: 203–208.
184 Thapliyal, J. P., Gupta, S. C. and Garg, R. K. (1975) J. Endocrinol., **65**: 333–339.
185 Wiegand, M. D. and Peter, R. E. (1980) Comp. Biochem. Physiol., **66A**: 323.
186 Lone, K. P. and Matty, A. J. (1980) Gen. Comp. Endocrionol., **40**: 409–424.
187 Sinha, R. C. (1982) Experientia **38**: 1066–1067.
188 Pati, A. K. and Thapliyal, J. P. (1984) Gen. Comp. Endocrinol., **53**: 370–374.
189 Sreedeviamma, K. K. and Oommen, O. V. (1987) J. Reprod. Biol. Comp. Endocrinol., **7**: 13–20.
190 Feurbacher, I. and Prinzinger, R. (1981) Comp. Biochem. Physiol., **70A**: 247–250.
191 Cannon, B. (1971) Eur. J. Biochem., **23**: 125–135.
192 Horowitz, J. M. (1972) In "Bioenergetics". Ed. by R. E. Smith, J. P. Hannon, J. C. Shields and B. A. Horwitz, Proc. Internatl. Symp. Environ. Physiol., Dublin, pp. 115–121.
193 Leblanc, J., Lafrance, L., Villemaire, A., Roberge, C., Valliéres, J. and Rousseau, S. (1972) In "Bioenergetics". Ed. by R. E. Smith, J. P. Hannon, J. C. Shields and B. A. Horwitz, Proc. Internatl. Symp. Environ. Physiol., Dublin, pp. 71–76.
194 Leblanc, J., Labrie, A., Lupien, D. and Richard, D. (1982) Can. J. Zool., **60**: 783–787.
195 Himms-Hagen, J. (1975) In "Handbook of Physiology". Ed. by S. R. Geiger, Amer. Physiol. Soc., Washington D. C., Sect. 7, Vol. VI, pp. 637–665.
196 Himms-Hagen, J. (1976) Ann. Rev. Physiol., **38**: 315–351.
197 Himms-Hagen, J. (1990) FASEB J., **4**: 2890–2898.
198 Steiner, G. (1973) In "The Pharmacology of Thermoregulation". Ed. by P. Lomax and E. Schönbaum, Karger, Basel, pp. 42–46.
199 Horwitz, B. A. (1979) Fed. Proc., **38**: 2170–2176.
200 Nicholls, D. G. and Locke, R. M. (1984) Physiol. Rev., **64**: 1–64.
201 Ellison, G. T. H. and Skinner, J. D. (1990) Comp. Biochem. Physiol., **97A**: 23–26.
202 Marques, P. R., Illner, P., Williams, D. D., Green, W. L., Kendall, J. W., Davis, S. L., Johanson, D. G. and Gale, C. C. (1981) Am. J. Physiol., **241**: E420–E427.
203 Gupta, B. B. P. and Thapliyal, J. P. (1983) J. Endocrinol., **99**: 211–216.
204 Larssons, A. L. (1973) Gen. Comp. Endocrinol., **20**: 155–167.
205 Dashow, L. and Epple, A. (1983) J. Comp. Physiol., **152**: 35–41.
206 Ottolenghi, C., Puviani, C., Gavioli, E. and Brighenti, L. (1985) Gen. Comp. Endocrinol., **59**:

219–229.

207 Janssens, P. A. and Lowrey, P. (1987) Amer. J. Physiol., **252**: R653–R660.

208 Perry, S. F. and Vermette, M. G. (1987) J. Exp. Biol., **128**: 235–253.

209 Sheridan, M. A. (1987) Endocrinology **120**: 2234–2239.

210 Mommsen, T. P., Walsh, P. J. Perry, S. F. and Moon, T. W. (1988) **70**: 63–73.

211 Danulat, E. and Mommsen, T. P. (1990) Gen. Comp. Endocrinol., **78**: 12–22.

212 Danforth, W. H., Helmreich, E. and Cori, C. F. (1962).

213 Herman, C. A. (1977) Gen. Comp. Endocrinol., **32**: 321–329.

214 Farrar, E. S. and Frye, B. E. (1979) Gen. Comp. Endocrionol., **39**: 372–380.

215 Janssens, P. A., Caine, A. G. and Dixon, J. E. (1983) Gen. Comp. Endocrinol., **49**: 477–484.

216 Janssens, P. A., Kleineke, J. and Caine, A. G. (1986) J. Endocrinol., **109**: 75–84.

217 Janssens, P. A. and Griggs, J. A. (1984) Comp. Biochem. Physiol., **77**: 403–408.

218 Coulson, R. A. and Hernandez, T. (1979) Comp. Bichem. Physiol., **64C**: 115–121.

219 Coulson, R. A. and Hernandez, T. (1983) In "Alligator Metabolism: Studies on Chemical Reactions in vivo", Pergamon Press, Oxford.

220 Coulson, R. A. and Hernandez, T. (1986) Comp. Biochem. Physiol., **84A**: 673–676.

221 Smith, D. C. and Mathews, S. A. (1942) Am. J. Physiol., **137**: 553.

222 Banerjee, S. and Joshi, S. C. (1981) Indian J. Exp. Biol., **19**: 982–983.

223 Akbarsha, M. A. (1984) J. Reprod. Biol. Comp. Endocrinol., **4**: 34.

224 Ignatius, J. and Oommen, O. V. (1987) Indian J. Exp. Biol., **25**: 613–617.

225 Harri, M. and Hadenstam, R. (1972) Comp. Biochem. Physiol., **41A**: 409–419.

226 Farrar, E. S. and Frye, B. E. (1977) Gen. Comp. Endocrinol., **33**: 76–81.

227 Gupta, B. B. P. and Thapliyal, J. P. (1985) Indian J. Exp. Biol., **23**: 241–243.

228 Mohell, N., Nedergaard, J. and Cannon, B. (1983) Euro. J. Pharmacol., **93**: 183–193.

229 Farkas, T. (1967) Progr. Biochem. Pharmacol., **3**: 314.

230 Farkas, T. (1969) Acta Biochim. Biophys. Acad. Sci. (Hung.) **4**: 237–249.

231 Lipshaw, L. A., Patent, J. G. and Foa, P. P. (1972) Horm. Metab. Res., **4**: 34.

232 Perrier, H., Perrier, C., Gudefin, Y. and Gras, J. (1972) Comp. Biochem. Physiol., **43A**: 341.

233 Hanke, W. (1990) In "Progress in Comparative Endocrinology", Wiley-Liss, Inc., pp. 445–452.

234 Bottoms, G. and Goetsch, D. D. (1968) Endocrinology **10**: 310–314.

235 Deavers, D. R., Musacchia, X. J. and Meininger, G. A. (1978) J. Therm. Biol., **3**: 93.

236 Deavers, D. R. and Musacchia, X. J. (1979) Fed. Proc., **38**: 2177–2181.

237 Prasannakumar, K. and Oommen, O. V. (1988) Indian J. Exp. Biol., **26**: 125–128.

238 Inui, Y and Yokote, M. (1975) Bull. Jpn. Soc. Sci. Fish., **41**: 973–981.

239 Callard, I. P. and Callard, G. V. (1976) In "General, Comparative and Clinical Endocrinology of the Adrenal Cortex". Ed. by I. Chester-Jones and I. W. Henderson, Academic Press, London/New York, Vol. II, pp. 291–418.

240 Chan, D. K. O. and Woo, N. Y. S. (1978) Gen. Comp. Endocrionol., **35**: 205–215.

241 Leach, G. J. and Taylor, M. H. (1982) Gen. Comp. Endocrionol., **48**: 76–83.

242 Barton, B. A., Schreck, C. B., Ewing, R. D., Hammingson, A. R. and Patino, R. (1985) Gen. Comp. Endocrionol., **59**: 468–471.

243 Vijayan, M. M. and Leatherland, J. F. (1989) Can. J. Zool., **67**: 2746–2750.

244 Hanke, W., Lange, C. and Weindel, K. (1990) In "Biology and Physiology of Amphibians". Ed. by W. Hanke, G. Fischer Verlag, Stuttgart/New York, (in press).

245 Ralph, C. L., Firth, B. T. Gern, W. A. and Owens, D. W. (1979) Biol. Rev., **54**: 41–72.

246 Heldmaier, G., Steinlechner, S., Rafel, J. and Vsiansky, P. (1981) Science **212**: 917–919.

247 Heldmaier, G., Steinlechner, S., Ruf, T., Wiesinger, H. and Klingenspor, M. (1989) J. Biol. Rhythms **4**: 251–265.

248 Hall, E. S. and Lynch, G. R. (1985) Life Sci., **37**: 783–788.

249 Holtorf, A. P., Heldmaier, G., Thiele, G. and Steinlechner, S. (1985) J. Pineal Res., **2**: 393–403.

250 Heldmaier, G. and Lynch, G. R. (1986) Pineal Res. Rev., **4**: 97–139.

251 McElroy, J. F. and Wade, G. N. (1986) Physiol. Behav., **37**: 307–311.

252 Saarela, S. and Heldmaier, G. (1987) J. Comp. Physiol., **157B**: 625–633.

253 Puchalski, W., Bulave, S. J., Lynch, C. B. and Lynch, G. R. (1988) Physiol., Behav., **42**: 173–177.

254 McElroy, J. F., Mason, P. W., Hamilton, J. M. and Wade, G. N. (1986) Amer. J. Physiol., **250**: R383–R388.

255 Viswanathan, M., Hissa, R. and George, J. C. (1986) Life Sci., **38**: 73–79.

256 Lewinski, A., Webb, S. M. and Reiter, R. J.

(1987) Pineal Res. Rev., **5**: 69–94.

257 Kashbohm, P. (1967) Halgol. Wissen. Meeruntersuch., **16**: 157–178.

258 Chugunov, Y. D. and Kispov, K. A. (1969) Doklady Biol. Sci., **187**: 596–597.

259 Hutchison, V. H. and Kohl, M. A. (1971) Z. Vergl. Physiol., **75**: 367–382.

260 Dunlop, D. G. (1989) Comp. Biochem. Physiol., **94A**: 519–523.

261 Hutchison, V. H. and Kosh, R. J. (1974) Oecologia **16**: 173–177.

262 Engbretson, G. A. and Hutchison, V. H. (1976) J. Exp. Zool., **198**: 29–38.

263 Bartholomew, G. A. (1982) In "Biology of Reptilia". Ed. by C. Gans and F. H. Pough, Academic Press, London/New York, Vol. 12, pp. 167–212.

264 Stebbins, R. C. (1960) Copeia **1960**: 276–286.

265 Roth, J. J. and Ralph, C. L. (1976) J. Exp. Zool., **198**: 17–28.

266 Ralph, C. L., Firth, B. T. and Turner, J. S. (1979) Am. Zool., **19**: 273–293.

267 Firth, B. T., Mouldin, R. E. and Ralph, C. L. (1988) Physiol. Zool., **61**: 176–185.

268 Phillips, J. A. and Howes, K. A. (1988) Physiol. Behav., **42**: 103–108.

269 Rismiller, P. D. and Heldmaier, G. (1982) Oecologia **53**: 222–226.

270 Rismiller, P. D. and Heldmaier, G. (1985) Physiol. Zool., **58**: 71–79.

271 Rismiller, P. D. and Heldmaier, G. (1987) J. Therm. Biol., **12**: 131–134.

272 Rismiller, P. D. and Heldmarier, G. (1988) Oecologia **75**: 125–131.

Gupta, B B P and Thapliyal, J. P. 1991. "Endocrine Regulation of the Oxidative Metabolism in Poikilothermic vertebrates." *Zoological science* 8, 625–638.

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