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Role of catecholamines and corticosteroids in regulation of the oxidative metabolism in male *Clarias batrachus*

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***In vivo* and *in vitro* effects of norepinephrine (NE), epinephrine (EP), corticosterone, cortisol, cortisone and metapyrone were studied on the rate of tissue (liver, muscle, kidney and brain) respiration of the male air-breathing fish, *Clarias batrachus* exposed to natural climatic conditions during winter and summer. Both NE and EP stimulated the respiratory rate of all the tissues irrespective of the season/water temperature and the mode of treatment. EP in muscle and NE in liver were comparatively more potent. Both the catecholamines were found to be more effective in stimulating tissue respiration during winter than during summer. Similarly, the corticosteroid hormones increased the respiratory rate of all the four tissues both *in vivo* and *in vitro* experiments irrespective of the season. Only cortisone had no significant effect on brain tissue respiration during summer. The corticosteroid hormones were also more effective in stimulating fish tissue respiration during winter than during summer. *In vivo* administration of metapyrone significantly reduced the rate of respiration of all the tissues. The inhibitory effect of metapyrone was reversed by *in vivo* administration of the corticoids. These findings suggest that the catecholamines and the corticosteroids are directly involved in the regulation of tissue respiration of *C. batrachus*. Due to their temperature-independent calorigenic action, these hormones might be acting as emergency hormones for the regulation of fish respiration.**

THE adrenal gland in mammals is a discrete and compact organ. However, in teleost fishes, the catecholaminergic (CT) and corticosteroidogenic (CSG) tissues do not constitute a well-defined adrenal gland. These tissues are scattered separately in the form of islets in the anterior part of the kidney[1,2]. The catecholamines are produced from the CT and as a neurotransmitter from the sympathetic nerve terminals[3–6]. As in mammals, the CSG tissue of the fish secretes corticoids like corticosterone, cortisol and cortisone[7]. The catecholamines and the corticoster-

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oids have been reported to be involved in a number of metabolic pathways of vertebrates[8]. Due to their multi-directional physiological actions, these hormones play a major role during the emergency conditions and in general adaptations against the adverse effect of a new environment[6,8,9].

The catecholamine hormones have been reported to induce non-shivering thermogenesis in cold acclimated mammals[10–16]. These hormones have been reported to stimulate oxygen consumption in a number of mammalian species[13,15–17], reptiles[18,19] and amphibians[20–23]. Due to their rapid and temperature-independent calorigenic actions, catecholamines act as emergency hormones for regulation of the oxidative metabolism in reptiles and amphibians[18,23–27]. However, unlike in reptiles and amphibians, there is scarcity of information on the involvement of catecholamines in fish respiration[28]. So far no attempt has been made to study in detail the calorigenic role of catecholamines in fish at the tissue respiration level to eliminate the stress factor which might be affecting the whole body oxygen consumption.

The metabolic influence of corticosteroids on the oxidative metabolism in mammals seems to depend on tissues and the length of hormonal treatment[29]. Hydrocortisone, predinisolone and corticosterone separately increased liver oxygen uptake in mammals[29,30]. Glucocorticoids have been reported to have a direct influence on mitochondrial transcription and respiratory enzyme synthesis in mammals[31,32]. In reptiles and amphibians, the corticosteroids seem to have a direct and temperature-independent effect on the rate of tissue respiration[18,23,26,27,33]. They also increased the activity of oxidative enzymes in reptilian liver in a dose-dependent manner[25]. In amphibians also, these hormones have been reported to increase the activity of respiratory enzymes[24,34]. However, unlike in other vertebrates, there is practically no information on the calorigenic action of the corticosteroid hormones in any fish species. Therefore, keeping in view the scarcity of information on the calorigenic role of catecholamines and corticosteroids and the phylogenic importance of fish, we decided to investigate in detail the role of these hormones in regulation of the oxidative metabolism of the air breathing fish, *Clarias batrachus* maintained at natural climatic conditions during winter and summer/rainy seasons.

Materials and methods

Adult male *C. batrachus* (length, 18–22 cm; weight, 70–80 g) were purchased from the local market and acclimatized for 15 days under natural climatic conditions before the experiment was started. Fishes were maintained in earthen pots and acclimatized at least for 15 days in the laboratory under natural climatic conditions at Shillong (latitude, 25.30°N, longitude, 91.52°E; altitude, 1450 asl; minimum water temperature, 4°C and maximum water temperature, 22°C). During acclimatization, the fishes were fed daily with minced earthworms *ad libitum*. The water was changed frequently to avoid infections. *In vivo* and *in vitro* experiments were conducted during both winter and summer/rainy months as per the experimental protocol shown in Table 1.

In vivo experiments

The *in vivo* experiments were conducted during both winter and summer/rainy seasons. After acclimatization, fishes were divided into different groups (four in each

Table 1. Experimental protocol

Expt. no.	Treatment	*In vivo*/ *in vitro*	Month (temperature in °C)	Dose	Duration of treatment (days)
(A)	Saline	*In vivo*	January (9.6)		4
	Epinephrine		September (20)	2 µg/fish/day	4
	Norepinephrine			2 µg/fish/day	4
	Corticosterone			2 µg/fish/day	4
	Cortisol			2 µg/fish/day	4
	Cortisone			2 µg/fish/day	4
(B)	Control	*In vitro*	January (9.6)		
	Epinephrine		July (20)	1 µM	
	Norepinephrine			1 µM	
	Corticosterone			1 µM	
	Cortisol			1 µM	
	Cortisone			1 µM	
(C)	Saline	*In vivo*	August (20)		4
	Metapyrone			1 µg/g/day	4
	Corticosterone			2 µg/fish/day	4
	Cortisol			2 µg/fish/day	4
	Cortisone			2 µg/fish/day	4
	Metapyrone + corticosterone			1 µg/g + 2 µg/fish/day	4
	Metapyrone + cortisol			1 µg/g + 2 µg/fish/day	4
	Metapyrone + cortisone			1 µg/g + 2 µg/fish/day	4

group) for treatment with different hormones. The hormones and metapyrone were injected intramuscularly on the lateral side of the dorsal fin at an interval of 24 h between 10 and 11 am for four consecutive days. Details about doses of hormones and metapyrone, duration of treatment, water temperature and the month are mentioned in Table 1. The doses of hormones used in the present investigation are based on our earlier studies on poikilotherms, and fall under the physiological range[23]. Water temperature was recorded daily at 10.30 am. Twenty-four hours after the last injection, fishes were decapitated, the tissues (liver, muscle, kidney and brain) rapidly removed, rinsed in ice-cold fish buffer saline and stored in the ice-chamber (– 15°C) in a refrigerator. The rate of tissue respiration was measured within 15 days of storage. When the tissues were stored in the refrigerator no significant alteration was found in the rate of tissue respiration up to one month. For the measurement of tissue respiration, the tissues were first blotted, weighed and homogenized in a loose-fitting all-glass homogenizer (Remi Homogenizer, Remi Equipments, Bombay) in ice-cold fish buffer saline (9 : 1) solution (pH 7.4). One ml of homogenate was added to 4 ml of fish buffer saline solution and placed into the incubation chamber of the oxygen electrode for measuring the rate of tissue respiration.

In vitro experiments

In vitro effects of the selected hormones on the rate of tissue respiration were also conducted during both winter and summer/rainy seasons. Four adult male fishes were first weighed and then decapitated. The tissues (liver, muscle, kidney and brain) were quickly removed separately, rinsed in ice-cold fish buffer saline and stored in a freezer as mentioned earlier. The tissues were used to study the *in vitro* effects of hormones within 15 days. For *in vitro* treatments, the tissues were blotted, weighed and homogenized in a loose-fitting all-glass homogenizer in ice-cold fish buffer saline solution (pH 7.4). One ml of homogenate was added to 3.9 ml of fish buffer saline and incubated with 0.1 ml of hormone solution having the desired concentration (for details, see Table 1). The tissue homogenates treated with corticosterone, cortisol and cortisone were pre-incubated at 4°C for one hour prior to the measurement of the rate of oxygen consumption. This incubation was necessary to allow the binding of these hormones to the tissues. Norepinephrine (NP) and epinephrine (EP) were added to the homogenates in the incubation chamber 15 min before measuring the rate of respiration. Then the rate of tissue respiration was measured with the help of an oxygen electrode.

Measurement of tissue respiration

The rate of oxygen consumption of each tissue (liver, muscle, kidney and brain) was measured with the help of an oxygen electrode (Digital Oxygen System, Model 10; Rank Brothers Ltd, England). For measuring the rate of respiration, the polarizing voltage was kept at 0.6 V and fish buffer-saline (pH 7.4) was used as the polarizing medium. The rate of oxygen consumption of tissue homogenates was measured at 25°C by circulating water at 25°C in the water jacket of the incubation chamber using the thermostatic water circulator. The homogenates were incubated in the chamber for 20 min before recording the readings. Readings were recorded at an interval of 5 min for half an hour in the linear range of oxygen consumption. The rate of tissue respiration was expressed as µl O_2/mg wet weight tissue/h. The data were analysed statistically with the help of Student's t test[35]. A $P < 0.05$ was considered as significant.

Results

In vivo and in vitro effects of EP and NE

The data are presented in Tables 2 and 3. Both *in vivo* and *in vitro* administration of EP and NE increased significantly the respiratory rate of all the tissues (liver, muscle, kidney and brain) during winter as well as during the summer/rainy months. Comparatively, EP was more potent than NE in muscle, while NE was more potent than EP in stimulating liver tissue respiration. Irrespective of the mode of treatment, the degree (% increase) of stimulation of the liver, muscle and kidney respiration by EP and NE was higher during winter when compared to that of the summer/rainy months (Tables 2 and 3).

In vivo and in vitro effects of corticosteroids and metapyrone

The data are presented in Table 4. *In vivo* and *in vitro* treatments of corticosterone, cortisol and cortisone had significantly increased the respiratory rate of all the four tissues (liver, muscle, kidney and brain) during both winter and summer/rainy seasons, except during summer/rainy seasons where *in vivo* administration of cortisone had no effect on the respiratory rate of the brain tissue (Table 2). As in the case of catecholamines, the responses of the tissues to the corticosteroids were higher during winter when compared to that of summer/rainy seasons.

Unlike in mammals, fishes lack a well-defined adrenal gland. Therefore adrenalectomy was not possible. In order to assess the role of corticoids in the oxidative metabolism of fish, metapyrone (an inhibitor of 11β-hydroxylase) was used to block the synthesis of corticoid hormones. The data are presented in Table 4. Administration of metapyrone significantly decreased the respiratory rate of all the tissues (liver, muscle, kidney and brain) when compared to the control group. When metapyrone

was administered together with the corticoids, the hormones reversed the ill-effects of metapyrone (Table 4).

Discussion

The earlier reports on the effect of catecholamines on the whole body oxygen consumption in fish seem to be contradictory to each other[36–38]. The measurement of the rate of the whole body oxygen involves confinement of the fish leading to unspecific stress, which might be responsible for the equivocal results. There is very limited information on the calorigenic role of corticoids in fish[33]. To the best of our knowledge, this might be the first study of its kind in which the calorigenic role of catecholamines (NE and EP) and major corticosteroids have been investigated in the respiration of a number of vital tissues using both *in vivo* and *in vitro* experiments conducted under natural climatic conditions of both winter and summer/rainy months.

In general, *in vivo* and *in vitro* administration of catecholamines and corticoids significantly stimulated the respiratory rate of all the tissues during both winter and summer/rainy months. The only exception was the brain tissue respiration, which was not stimulated by *in vivo* administration of cortisone during summer/rainy months (Table 2). These findings suggest that both catecholamines and corticoids are actively involved in fish tissue

Table 2. *In vivo* effect of adrenal hormones on the rate of tissue respiration of male *Clarias batrachus* during winter (average water temperature: 9.6°C) and summer (average water temperature: 20°C)

Treatment	Rate of tissue oxygen consumption (µl oxygen/mg/h)			
	Liver	Muscle	Kidney	Brain
Winter				
Saline (control)	2.88 ± 0.07	1.12 ± 0.05	2.69 ± 0.08	5.26 ± 0.09
Epinephrine	4.14 ± 0.11^{c}	2.63 ± 0.03^{c}	3.69 ± 0.05^{c}	5.87 ± 0.05^{b}
Norepinephrine	4.33 ± 0.09^{c}	2.24 ± 0.11^{c}	3.78 ± 0.07^{c}	5.90 ± 0.04^{c}
Corticosterone	3.78 ± 0.03^{c}	2.37 ± 0.06^{c}	3.88 ± 0.14^{c}	5.61 ± 0.07^{a}
Cortisol	3.88 ± 0.16^{b}	2.53 ± 0.08^{c}	4.17 ± 0.03^{c}	5.74 ± 0.08^{b}
Cortisone	3.53 ± 0.12^{b}	2.21 ± 0.10^{c}	3.72 ± 0.08^{c}	5.52 ± 0.04^{a}
Summer				
Saline (control)	4.71 ± 0.08	2.72 ± 0.05	4.10 ± 0.05	4.30 ± 0.07
Epinephrine	5.68 ± 0.05^{c}	3.72 ± 0.08^{c}	4.97 ± 0.05^{c}	5.04 ± 0.07^{c}
Norepinephrine	5.84 ± 0.07^{c}	3.59 ± 0.12^{c}	4.81 ± 0.07^{c}	4.91 ± 0.14^{b}
Corticosterone	5.39 ± 0.08^{c}	3.46 ± 0.04^{c}	4.59 ± 0.09^{b}	4.68 ± 0.03^{b}
Cortisol	5.58 ± 0.03^{c}	3.85 ± 0.10^{c}	4.94 ± 0.03^{c}	4.87 ± 0.05^{c}
Cortisone	5.45 ± 0.11^{b}	3.56 ± 0.05^{c}	4.62 ± 0.08^{b}	4.33 ± 0.09

All values are expressed as mean ± Standard Error (S.E.); $n = 4$.
^{a,b} ^{c}Differ from respective controls: $P < 0.05$, 0.01 and 0.001, respectively.

Table 3. *In vitro* effect of adrenal hormones on the rate of tissue respiration of male *Clarias batrachus* during winter (average water temperature: 9.6°C) and summer (average water temperature: 20°C)

Treatment	Rate of tissue oxygen consumption (µl oxygen/mg/h)			
	Liver	Muscle	Kidney	Brain
Winter				
Control	2.69 ± 0.06	0.89 ± 0.08	2.53 ± 0.05	5.01 ± 0.04
Epinephrine	4.17 ± 0.07^{c}	2.01 ± 0.05^{c}	3.66 ± 0.07^{c}	5.81 ± 0.09^{c}
Norepinephrine	4.33 ± 0.06^{c}	1.95 ± 0.03^{c}	3.88 ± 0.11^{c}	5.84 ± 0.06^{c}
Corticosterone	3.88 ± 0.09^{c}	1.34 ± 0.04^{b}	3.94 ± 0.09^{c}	5.74 ± 0.11^{c}
Cortisol	3.98 ± 0.12^{c}	1.44 ± 0.07^{b}	4.26 ± 0.03^{c}	5.71 ± 0.03^{c}
Cortisone	3.49 ± 0.03^{c}	1.15 ± 0.06^{a}	3.62 ± 0.07^{c}	5.32 ± 0.07^{b}
Summer				
Control	4.81 ± 0.12	2.85 ± 0.02	4.01 ± 0.03	5.07 ± 0.07
Epinephrine	5.29 ± 0.06^{a}	3.85 ± 0.06^{c}	5.36 ± 0.05^{c}	5.97 ± 0.07^{c}
Norepinephrine	5.55 ± 0.05^{b}	3.46 ± 0.04^{c}	5.10 ± 0.07^{c}	5.87 ± 0.05^{c}
Corticosterone	5.45 ± 0.07^{b}	3.21 ± 0.09^{a}	3.88 ± 0.05	5.52 ± 0.10^{a}
Cortisol	5.39 ± 0.03^{b}	3.43 ± 0.05^{c}	4.81 ± 0.09^{c}	5.77 ± 0.08^{c}
Cortisone	5.71 ± 0.11^{b}	3.55 ± 0.08^{c}	4.78 ± 0.04^{c}	5.74 ± 0.06^{c}

All values are expressed as mean ± Standard Error (S.E.); $n = 4$.
^{a,b} ^{c}Differ from respective controls: $P < 0.05$, 0.01 and 0.001, respectively.

Table 4. *In vivo* effects of corticosteroids and metapyrone on the rate of tissue respiration of male *Clarias batrachus* during summer (average water temperature: 21°C)

Treatment	Rate of tissue oxygen consumption (µl oxygen/mg/h)			
	Liver	Muscle	Kidney	Brain
Saline (control)	4.84 ± 0.09	2.98 ± 0.10	3.82 ± 0.05	5.20 ± 0.07
Corticosterone	5.26 ± 0.05[a]	3.53 ± 0.07[b]	4.52 ± 0.02[c]	5.61 ± 0.05[b]
Cortisol	5.48 ± 0.05[c]	3.98 ± 0.09[c]	4.87 ± 0.04[c]	5.81 ± 0.03[c]
Cortisone	5.45 ± 0.07[a]	3.62 ± 0.05[b]	4.62 ± 0.08[c]	4.81 ± 0.07
Metapyrone	4.49 ± 0.04[a]	2.50 ± 0.07[b]	3.43 ± 0.08[b]	4.84 ± 0.05[b]
Corticosterone + metapyrone	5.10 ± 0.02[a,d]	3.27 ± 0.03[a,e]	4.20 ± 0.03[c,f]	5.42 ± 0.02[a,d]
Cortiso + metapyrone	5.23 ± 0.03[b,g]	3.69 ± 0.06[c,g]	4.55 ± 0.07[c,h]	5.45 ± 0.07[a,h]
Cortisone + metapyrone	5.16 ± 0.06[a,k]	3.33 ± 0.04[a,k]	4.36 ± 0.05[c,k]	4.97 ± 0.05

All values are expressed as mean ± Standard Error (S.E.); $n = 4$.
[a,b,c]Differ from the saline-treated control group: $P < 0.05$, 0.01 and 0.001, respectively.
[d,e,f]Differ from the value of the group treated with corticosterone: $P < 0.05$, 0.01 and 0.001, respectively.
[g,h]Differ from the value of the group treated with cortisol: $P < 0.05$ and 0.01, respectively.
[k]Differ from the value of the group treated with cortisone: $P < 0.05$ and 0.01, respectively.

respiration. Further, these hormones were found to be calorigenic during summer as well as winter. However, they were more effective in stimulating the respiratory rate of tissues during winter when compared to summer. Therefore, low temperature seems to increase the sensitivity of tissues to the adrenal hormones. *In vitro* stimulation of the respiratory rate of all the tissues by these hormones (Table 3) indicates that both catecholamines and corticoids stimulate the respiratory rate of fish tissues directly without involving any other hormones. On the basis of the present findings, therefore, it may be concluded that the catecholamines and the corticosteroid hormones play a major role in the regulation of the energy metabolism of the fish. Further, catecholamines and corticoids, which are secreted under stressful conditions[25], might be of adaptive importance for ensuring basal metabolic rate required for survival of the fish, at low temperature. Administration of metapyrone (an inhibitor of corticosteroid synthesis) significantly reduced the respiratory rate of all the tissues, and combined treatment of metapyrone and corticoids reversed the inhibitory effect of metapyrone on tissue respiration (Table 4). Thus, these findings suggest that indigenous corticoids are actively involved in the regulation of tissue respiration in *C. batrachus*. The direct and temperature-independent calorigenic action of the adrenal hormones suggests that both catecholamines and corticoids might be acting as emergency hormones for the regulation of respiration in *C. batrachus*. These hormones have also been reported to act as emergency hormones for the regulation of the oxidative metabolism in amphibians and reptiles[18,23,26,27,33,39–41]. As observed in this study, the rate of tissue respiration has been reported to remain significantly elevated after 24 h of treatment with adrenal hormones also in reptiles[18] and amphibians[23,26,27]. In agreement with our findings, it has been reported that catecholamines and corticoids increase the liver oxygen uptake in mammals[29,30,32], and activities of enzymes and tissue respiration in poikilotherms[24,34,42,43]. There are a few reports stating that the calorigenic response of mammalian and reptilian tissues to NE is decreased by hypothyroidism and increased by hyperthyroidism[44–46]. Thyroid hormones have also been reported to potentiate the calorigenic action of catecholamines in mammals at low temperature[39,47–50], reptiles[33] and amphibians[23]. However, there is no information on potentiation of catecholamine action by thyroid hormones in any piscine species. Catecholamines have been reported to increase the non-shivering thermogenesis and oxygen consumption involving both α- and β-adrenergic receptors in mammals[51,52] and amphibians[53]. The catecholamines might also be stimulating the rate of fish tissue respiration through a similar mechanism.

On the basis of the present findings it may be concluded that catecholamines and corticoids are directly involved in the regulation of the oxidative metabolism in the fish as emergency hormones. The catecholamines and the corticosteroids seem to be of adaptational importance for maintaining basal metabolic rate of the fish in order to ensure survival under stressful conditions, particularly at low temperature[54]. The proposed direct calorigenic action of corticoids in fish (present study), amphibians[23] and reptiles[18] has been strongly supported by a recent study in which corticoids directly increased the activities of mitochondrial oxidative enzymes in mammals[32].

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