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Research Article

Effects of Urotensin-II and Somatostatin -25 on carbohydrate and lipid metabolism of frog *Rana pipiens*

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Abstract

The study was conducted to find out the metabolic activity of lipid and carbohydrate which is involves the nerve impulse transmission, energy storage, hormone regulation and fat-soluble nutrient transportation in human bodies. Does dependent significant hypoglycaemia caused by Urotensin II and Somatostatin-25 were studied and observed increase in plasma free fatty acids which diminished with time and increased liver glycogen contents. Higher doses of Urotensin-II (5ng/g body weight for 6hr) and Somatostatin-25 (10 ng/g body weight for 6 hr) were more potent to increase plasma protein and plasma total lipids, whereas weaker doses of both did not show any type of effect. These results show that partial homology and partial analogy of Urotensin-II and Somatostatin-25 contribute towards similar type of effect on carbohydrate and lipid metabolism in frog *Rama pipiens.* Based on the current study, the more specific work can be done in context of the metabolic syndrome, in diabetes and in cardiovascular disease.

Keywords: Blood Plasma; Carbohydrate; Corticosteroids; Freefattyacid; Lipid; Metabolism; Palmaglucasse; Somatostatin-25; Urotensin-II

Introduction

The metabolism of vertebrates gains profound effect of Mammalian Somatostatin-25 (somatotropin release-inhibiting factor (SRIF)) aside from its well-known inhibitory action on the release of various pancreatic and pituitary hormones **[1]**. In mammals, it was observed that the administration of Somatostatin-25 generally increased FFA (plasma free fatty acids) and decreased plasma glucose **[2, 3]**. Urotensin-II (U-II) secreted by candal neurosecretory system of teleost fish that can be described as Somatostatin -25

because of partially analogous characteristics [4]. Due to the structural similarity between Somatsetatin-25 and Urotensin-II where identical amino acids in five positions, it is called partially homology [5], Metabolic effects of Urophysial-II are less but well characterized. The elevated plasma glucose level in snakehead fish were observed after 4-6 hours of injecting Urophysial extract [6]. Synthetic Gillichthys Urotensin-II stimulated fatty acid release from Coho Salmon liver incubated *in vitro* [7].

Keeping in view the "partial homology" and "partial analogy" of Urotensin-II and Somatostatin-25, the present study made undertaken on liver, glycogen, total lipids, and proteins, glucose and on plasma free fatty acids of frog, *Rana pipiens.*

Materials and Methods

Adult frogs *Rana pipiens* of both male and female sexes were captured from ponds near Fatime Town, Sultan during July, August and September 2022 and maintained at room temperature (27 °C) in the laboratory.

Frogs were divided into control and experimental groups. The control frogs (5 animals) were injected with distilled water, intraperitoneal region at the dose of 10 ul/g body weight. After pitting the animals were dissected for blood and tissue sampling after 4 and 6 hours. The animals of the treated group were divided into four groups and each treated groups were further divided into two batches of 5 animals each. The animals of each batch of treated group were given injection of Urotensin-II low dose (2.5 ng/g in volume of 10 ul/g body weight), high dose (5 ng in volume 10 ul/g body weight), Somatultation-25 low dose (5 ng in volume of 10 ng body weight), high dose (10ng in volume 10ul/g body weight) intraperitoneally. Treys were pithed and dissected for blood and tissue after 4 and 6 hours.

The blood plasma was used for the estimation of glucose [8]. Glucose was also measured with the method of [9] and free fatty acids [10], and total lipids [11]. and protein [12]. The hepatic glycogen content was estimated [13]. The statistical analysis of the results was carried using t-tests (SPSS 2023).

Results and Discussion

The (Table 1) shows the effects of Urotensin-II and Somatostatin-25 in low and high dose, on the various biochemical components. A significant dose-dependent decrease in plasma glucose, increases in plasma free fatty acids, total lipids, elevated liver glycogen and protein had been observed.

In this study, our results indicate that both Urotensin-II and Somatostatin-25 have profound effects an plasma free fatty acids plasma total lipids, plasma glucose, liver glycogen and plasma protein of frog *Rana pipiens.* Somatostatin in the present study at low and high doses showed a significant dose dependent decrease in plasma glucose. This hypoglycemic effect was similar to that reported in mammals [14].

The potent inhibitor of the secretion of growth hormone in men during insulin-induced hyperglycemia is known as synthetic GRIH (growth release inhibiting hormone). In contrary, neither the basal values nor the normal increase in the secretion of prolactin and corticosteroids during hypoglycemia are influenced [14]. Since LH and FSH were also released after the synthetic gonadotropin-releasing hormone was influenced by GRIH, there must be more than one of pituitary-cell receptor connected with FSH release. While this hypothalamic regulatory hormone seemed to be directed at inhibiting GH release without any influence to the basal secretion of other anterior-pituitary hormones, it did not interfere with some of the actions of TRH (thyroid releasing hormone). Animal Studies indicated that it acted directly upon the anterior pituitary cells [15]. In birds the decrease of Somatostatin caused the decrease of plasma glucose [16]. Our results are contradicted to those reported [17] in eel fish and in lower mammals [18].

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Table 1: Change in mean plasma free fatty acids, total lipids, plasma glucose, liver glycogen, and proteins of frog *Rana pipiens* after administration of Urotensin- II **and Somatostatin -25 at the dose of 2.5ng/g body weight (low dose) and 10 ng/g body weight (high dose)**

	Times in Hours	Control (n=5)	Urotensin-II		Somatostatin -25	
			Lower dose (n=5)	Higher dose (n=5)	Lower dose (n=5)	Higher dose (n=5)
Free fatty acids (meq/ l)	4	0.59 ±0.01	1.30 ± 0.04**	0.83 ± 0.01*	1.18 ± 0.05*	0.76 ±0.04**
	6	0.57 ± 0.02	0.99 ± 0.02**	0.86 ± 0.02*	0.85 ± 0.06*	0.77 ± 0.01**
Plasma Total lipids (mg /100 ml)	4	143.00 ±21.09	149.00 ± 27.23	264.40 ±52.71**	149.00 ± 27.23	264.40±55.70**
	6	139.00 ±22.15	133.00 ± 23.71	226.00 ± 27.57*	133.00 ± 23.71	226.00±27.57*
Plasma glucose (mg /100 ml)	4	11.07 ±0.85	8.63 ± 1.7**	7.19 ± 0.89**	8.33 ± 0.98*	7.37 ± 0.72**
	6	10.24 ± 0.82	7.38 ± 1.41*	5.84 ± 0.55**	7.33 ± 0.52*	5.93 ± 0.36**
Liver glycogen (mg /100 ml)	4	1.33 ±0.31	1.9 I 0.16*	2.52 ± 0.08**	1.9 ± 0.16*	2.52 ± 0.08**
	6	1.22 ±0.22	2.23 ± 0.16*	2.33 ±0.036**	2.23 ± 0.16*	2.33±0.036**
Plasma protein (mg /100 ml)	4	0.50 ± 0.03	0.50 ± 0.10	0.58 ± 0.01**	0.50 ± 0.10	0.56 ± 0.09*
	6	0.46 ± 0.03	0.52 ± 0.11*	0.61 ± 0.06**	0.53 ± 0.05*	0.69 ± 0.06**

* P < 0.02; **P< 0.01

We observed the increase in free fatty acids, total lipids and liver glycogen in frog *Rana pipiens* after the induction of low and high doses of somatostatin urotensin-II. Again we reached at a contradiction of results that were reported by [7] in coho-salmon and those obtained in mammals [2] as both reported SRIF-25 and SRIF-28. However, similar suppressed basal insulin levels despite the duration of suppression prolonged after SRIF-25 infusion. These results contrast with mammalian studies, in which, SRIF-28 appears to be a more potent and longer-lasting inhibitor of insulin release [19] and may suggest a difference in the control of avian pancreatic function. However, it is possible that the greater inhibitory effect of SRIF-25 on duck insulin concentrations may merely reflect the higher dose at which it was infected. The increase in the immune reactive plasma glucagon concentration in ducks infused with SRIF-25 also contrasts with its inhibitory effects in mammalian species [19]. The increase in glucagon like immune-reactivity in ducks was previously shown to occur in response to an SRIF-induced fall in the plasma-free fatty acids levels [16].

The results of this study are related with the results reported [7] where urotensin -II showed caused lipolysis in salmon liver and showed increased plasma total lipids. Beside the direct influence on lipid mobilization by somatostatin it was further indicated by decreased *in vitro* acetate incorporation into mouse liver [20]. Somatostatin and urotensin- II stimulatid hyperlipidemia can be also mediated by several observations. *In vivo* administration, insulin to cyclostomes and fish generally resulted in a reasonable reduction of plasma-free fatty acids based on several factors, including dole and method of administration [21]. The fatty acids release in mammals is directing influenced by insulin through reesterification and by inhibiting lipylysis. A decrease in adenylate cyclase activity and increase in phopho-diesterase activity resulted in antilipolytic effect of insulin [22].

Somatostatin in low and high dress at 4 hours was not significant but at 6 should cause a significant increase in plasma protein. Urotensin-II in low and high dose like somatostatin was ineffective at 4 hours but at 6 hours caused a significant increase in plasma protein values.

Conclusion

These results show that partial analogy and partial homology of somatostatin and urotensin-II contribute towards a similar effect on the lipid and carbohydrate metabolism of frog *Rana pipiens.*

Authors' contributions

Conceived and designed the experiment: M Zafar, Performed the experiments: MM Malik, M Zafar, MT Sajjad, S Ali, M Talal, S Bibi, I Mushtaq & Z Batool. Analysed the data: MM Malik & S Masud, Contributed materials/ analysis/ tools: M Zafar, MS Noor, S Masud & MH Latif, Wrote the paper: M Zafar.

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