International Journal of Medicine and Biomedical Research
Volume 4 Issue 2 May – August 2015
www.ijmbr.com

Original Article Open Access

Human growth hormone alters carbohydrate storage in blood and liver in both genders of an Indian bird, *Acridotheres tristis* (Linn.)

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Received: 16.02.15; Accepted: 27.05.15; Published: 02.06.15

ABSTRACT

Background: Growth hormone (GH) is a peptide hormone that plays vital roles in cell growth and metabolism. **Aim:** The study investigates the effect of GH on carbohydrate metabolism using Indian bird, *Acridotheres tristis.* **Methods:** Three different doses (0.4, 0.6, and 0.8mg/100g body weight) of human growth hormone (HGH) given once to both genders of a bird *Acridotheres tristis* to observe the effect on blood glucose and hepatic glycogen content in the body. Glucose and glycogen were quantitatively assayed. **Results:** Their effect was recorded for different time intervals (1, 4, 12, 24, 72, 96, and 144 h). Hypoglycaemic condition was recorded within an hour of hormone treatment in male and female birds. The lowest dose (0.4mg/100g body weight) was more effective than other two doses. Simultaneous depletion of hepatic glycogen was also recorded, although initially increase in glycogen level was also noticed in both genders. It was noticed that the highest dose (0.8mg/100g body weight) was most responsive. **Conclusion:** The effect of human growth hormone was not dose and time dependent in both male and female birds. HGH is thus hypoglycaemic and hepatic glycogenolytic in nature in *A. tristis.*

Key words: Human growth hormone, blood glucose, hepatic glycogen, hypoglycaemia glycogenolysis, bird

INTRODUCTION

The pancreas plays a central role in carbohydrate metabolism. Glucagon-secreting α-cells predominate in the islets of Langerhans of granivorous species, such as the chicken and duck.[1] Insulin appears to be more dominant in carnivorous avian species.[1] The roles of growth hormone (GH) in regulation of carbohydrate metabolism in Indian birds have scantly been studied. GH is a polypeptide hormone found in all vertebrate lineages.[2] It is the key hormone to maintain the glucose homoeostasis, but the effect of GH on glucose homeostasis is tissue-specific.[3] Whatever data is available, there is disagreement among

the result of different investigators. Von Euler stated that injection of purified porcine GH in birds brought no alteration in carbohydrate metabolism.[4] King and Scanes noticed that removal of anterior pituitary gland in growing chickens greatly increases hepatic glycogen level.[5] They also noticed that GH injection in small and large doses is without any effect on carbohydrate and lipid reserves in adult chicken. Adenohyphophysectomy of adult chicken have been reported to cause marginal decrease in plasma glucose level.[6]

The effects of GH on carbohydrate metabolism are more complicated and may be mediated indirectly via the antagonism of insulin action.[7] Ahsan and Ahsan noticed the hypoglycaemic and glycogenolytic role of GH in an Indian teleost, *Clarias batrachus*.[8] GH treatment reduces glycogen synthetase activity and decreased hepatic glycogen levels in tilapia.[9] The effect of GH on carbohydrate reserves have been studied in other vertebrate and to a large extent in mammal. Several studies have shown that GH elevates hepatic glucose production by glycogenolysis in rodents and human.[7,10] However, it has also been reported that no effect of GH on gluconeogenesis.[11] Studies in humans and animal models show that chronic excess GH has an anti-insulin effect on carbohydrate and lipid metabolism.[12] Most of the information regarding mammals shows that GH is hyperglycaemic in nature[13] but Baxter *et al.*[14], Yakar *et al.*[15] and Jezova *et al.*[16] found the growth hormone to be hypoglycaemic. Giuffrida *et al.*[17] could not find any effect on blood glucose level of human, following the GH therapy.

In view of the foregoing facts and lack of adequate data and conflicting reports of the effect of growth hormone on carbohydrate regulation in Indian birds, the present investigation was carried out to study the effect of HGH on blood glucose and liver glycogen content of a common Indian bird species *Acridotheres tristis.*

METHODOLOGY

The present investigation was conducted on the hormone related physiology of the common Indian bird, *Acridotheres tristis* (linn.), locally called as “Myna” in India. *A. tristis* belonging to class- Aves, order Passeriformes and family- Stunidae, in an extensively distributed birds species of the Indian sub-region. The International Union for Conservation of Nature (IUCN) declared in 2000 this myna as one of the only three birds among the world’s 100 worst invasive species. Although, *A. tristis* show unmistakable preference to insects as food items, yet it exploits diverse source of food including vegetable dish. The study was approved by the Ethical Committee of P.G. Research Laboratory of Animals of the university.

Collection of birds and their maintenance

Apparently healthy looking adult specimens of both genders of *A. tristis,* weighing 75 to 95g, were obtained through local bird’s supplier. Collections were made during the pre-breeding season beginning from November to February. The experiment was conducted according to the guidelines of “Committee for the Purpose of Control and Supervision on Experiments on Animals” (CPCSEA 2003), India.[18]

The birds were kept in wire cages (4’x 4’x 2’) in a batch of four. Before autopsy the birds were allowed to recover from asphyxia suffered during transport and to acclimate to the laboratory condition for seven days. During the period and also during the entire course of the experiment, the birds were fed daily on small piece of earthworm, insects, seeds of gram and gram flour *ad libitum.* During the whole period, birds were provided with adequate water. The initial control, the experimental birds and the experimental controls were kept under similar conditions.

Collection of blood and autopsy

Before autopsy, the birds were weighed to the nearest gram. A batch of 10 birds from each gender was sacrificed to establish the normal value of blood glucose and hepatic glycogen of *A. tristis* during the pre-breeding season. For collection of blood, the birds were caught with a soft and taken out the cage with minimum possible disturbance and were use of anaesthetized with either by putting the chloroform soaked cotton near the nostril. Immediately, after giving anaesthesia, the feather from underneath surface of the left wing were carefully cleaned. Blood for blood glucose analysis was collected with the help of a glass syringe by cutting the left branchial artery with a help of sharp scalpel, the collected blood was immediately transferred to glass centrifuge tube and was left for 30

minutes in order to obtain serum. The whole process of blood collection and their transference was undertaken with great care as to avoid the chances of haemolysis. Immediately after the collection of blood, autopsy was done. The abdomen was opened and small piece of liver (Approximately-100mg) was taken from the right lobe of birds liver. Blood and liver piece were collected between 8am to 10am only, as to avoid any error due to possible diurnal.

Blood glucose estimation

The quantitative estimation of blood glucose followed a procedure described by Kumari and Ahsan,[19] from serum by the o-toluidine method.[20]

Liver glycogen estimation

The quantitative estimation of glycogen followed a procedure described by Kumari *et al.*[21] Isolated homogenized hepatic tissues were heated at 100°C for 30 minutes.[21] Samples were supplemented with two vol. 95% ethanol and incubated overnight.[21] After the addition of two or three drops of Na_2SO_4 and centrifugation process, glycogen was precipitated.[21] The glycogen pellets were washed with 66% ethanol and then completely dried.[21] The glycogen level (expressed as mg/g) was analyzed in 0.2% anthrone reagent dissolved in H_2SO_4 using a spectrophotometer.[21]

Treatment with growth hormone

Human growth hormone (HGH) was obtained from Bethesda, USA, and was injected intramuscularly in bird with 1ml tuberculine syringe and gauges, 24-size needle. The hormone was supported in birds' saline made after the formula of Benzienger and Krebs as mentioned in Sturkie.[6] A batch of 320 birds of both genders was used for HGH treatment. It was then divided into 3 smaller batches consisting of 80 birds in each batch. These three batches were selected for the treatment with 3 different doses of growth hormone. Each smaller batch was sub-divided into 8 groups consisting of 10 birds in each group. The groups were used for hourly sampling of glycogen and glucose levels at a particular dose. The three different doses selected for the treatment were 0.4, 0.6 and 0.8mg/100gm body weight. Sampling was made after 1, 4, 12, 12, 24, 48, 72, 96 and 144h after the hormone injection. Along the experiment, a batch of 5 birds were injected with similar volume of birds' saline intramuscularly and autopsied with the experimental ones. Similar procedure of sampling and estimation of glucose and glycogen value was adopted as were used for experimental ones.

Statistical analysis

Data were expressed as mean ± standard deviation (SD). Analysis of variance (ANOVA) was used to test for significance across groups. $P<0.05$ was used as the significance level.

RESULT

Initial value (normal value)

Blood glucose: Blood glucose analysis of *A. tristis* during pre-breeding period gave different values in males and female birds. The normal blood glucose content of male ranged from 194.56 mg to 220.5 mg/dl of blood with an average value of 207.33 ± 12.67 mg/dl of blood, whereas those of female bird ranged from 164.3 to 185.5 mg/dl of blood with an average of 176.66 ± 10.5mg/dl of blood. The difference between male and female in this parameter of carbohydrate content was significant ($P< 0.001$) with male bird exhibiting higher value.

Liver glycogen: Quantitative estimation of liver glycogen content of *A. tristis* during pre-breeding period of the bird gave different values in male and female birds. The normal liver glycogen content of male bird ranged from 73.63 to 101.73 mg/g with an average value of 93.75 ± 6.12 mg/g where as those of female bird ranged from 65.31 to 90.3 mg/g with an average value of 78.43 ± 6.78 mg/g. The difference between male and female bird in the parameter of carbohydrate reserves was significant ($P<0.001$) with male bird exhibiting higher value.

After treatment

Blood glucose assay: Human growth hormone treatment of *A. tristis* at three dose levels of 0.4, 0.6 and 0.8mg/100 g body wt in the bird gave a generally consistent result in both genders. Dose dependent hypoglycaemia was noticed 1 and 4h after HGH injection in both genders at all the three dose levels. The decrease in blood glucose as compared to control specimens, was significant ($P<0.001$) in both genders at all the dose levels. Lower

dose of 0.4 mg was more hypoglycaemic than the higher doses of 0.6 mg and 0.8 mg. Although, during 12h to 144h treatment the HGH injected birds maintained hypoglycaemic condition, the result was not always dose dependent. The blood glucose level reached to a minimum in 48 h post treatment in both genders after that a recovery trend was noticed. At the end of 144h, the treated specimens though could not achieve the normoglycaemic level, exhibited a definite recovery (tables 1 and 2).

In short, HGH has an immediate effect on blood glucose level in *A. tristis* resulting in hypoglycaemia, which is not always dose dependent. The lower dose of 0.4 mg/100g body weight of bird was consistently more hypoglycaemic in effect in males at the sampling from 1 to 144h in females, though the same trend was noticed but at 48h sampling middle dose of 0.6mg caused more pronounced hypoglycaemia.

Liver glycogen assay: Birds treated with single injection of HGH at three different dose levels of 0.4, 0.6 and 0.8mg/100g of body wt of the bird showed an initial glycogenesis in both genders at all the three dose levels after an hour of treatment. This increase was dose dependent in both genders with highest dose of 0.8mg/100g body weight caused more glycogenesis as compared to lower doses. Liver sample of both genders analysed after 12 h of treatment except at 0.8mg dose level when significant ($P<0.001$) hepatic glycogenesis was noticed, at other lower doses the glycogen level was achieved normal level. After that, depletion in glycogen level was noticed at all dose levels in both genders (Table: 3 & 4). A recovery was noticed at the end of experiment but even after 144h of hormone injection normal glycogen level could not be achieved.

HGH is thus, initially and transiently glycogenetic with sharp and highly significant ($P<0.001$) dose dependent increase in liver glycogen, but later on, the hormone caused a slow and sustained glycogenolytic effect for up to 96 h of initial treatment. Only after this, a recovery in liver glycogen store towards normal was seen as the effect of hormone appeared to wane.

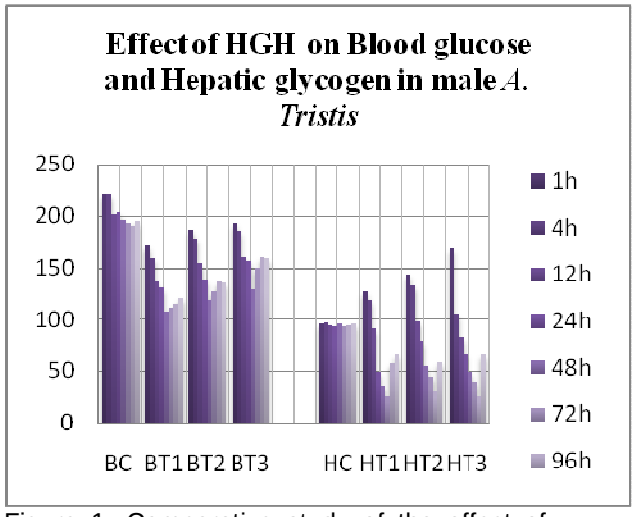

Figure 1: Comparative study of the effect of human growth hormone on blood glucose and hepatic glycogen in Male *A. tristis.*

BC: Blood glucose content in control; BT1: Blood glucose content in treated with 0.4/100gm body wt.; BT2: Blood glucose content in treated with 0.6/100gm body wt.; BT3: Blood glucose content in treated with 0.8/100gm body wt.; HC: Hepatic glycogen content in control; HT1: Hepatic glycogen content in treated with 0.4/100gm body wt.; HT2: Hepatic glycogen content in treated with 0.6/100gm body wt.; HT3: Hepatic glycogen content in treated with 0.8/100gm body wt.

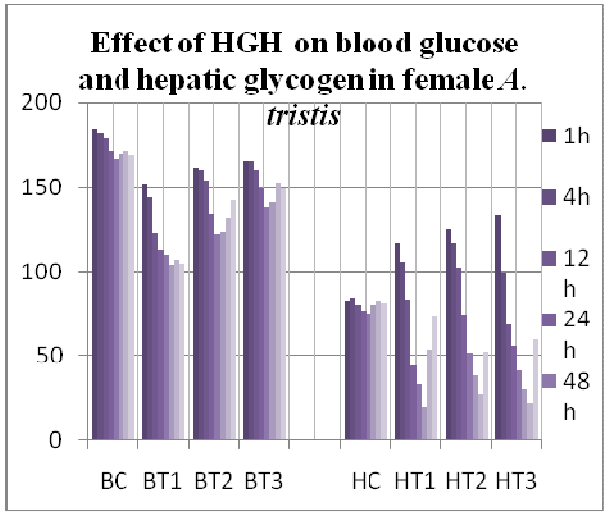

Figure 2: Comparative study of the effect of human growth hormone on blood glucose and hepatic glycogen in female *A. tristis*

BC: Blood glucose content in control; BT1: Blood glucose content in treated with 0.4/100gm body wt.; BT2: Blood glucose content in treated with 0.6/100gm body wt.; BT3: Blood glucose content in treated with 0.8/100gm body wt.; HC: Hepatic glycogen content in treated with control; HT1: Hepatic glycogen content in treated with 0.4/100gm body wt.; HT2: Hepatic glycogen content in treated with 0.6/100gm body wt.; HT3: Hepatic glycogen content in treated with 0.8/100gm body wt.

Table 1: Effect of HGH on blood glucose level (mg/dl) of male *A. tristis*

Time of assay	MC	MT1	MT2	MT3
1h	221.86±9.46^{d}	170.61±10.75^{b}	188.26±15.25^{bc}	180.25±10.73^{b}
4h	220.86±13.49^{d}	159.51±12.63^{b}	178.35±10.53^{bc}	185.32±9.65^{bc}
12h	200.86±9.76^{d}	136.31±9.86^{ab}	154.21±13.76^{b}	160.63±8.92^{b}
24h	203.51± 8.96^{d}	130.78±12.66^{ab}	138.66±11.96^{ab}	156.26±9.69^{b}
48h	196.39± 9.82^{d}	107.85±8.15^{a}	118.42±11.96^{a}	129.35±9.76^{a}
72h	192.51±10.69^{cd}	111.33±7.91^{a}	127.33±10.92^{a}	150.15±12.13^{b}
96h	190.71± 6.98^{cd}	115.26±10.84^{a}	136.52±14.68^{ab}	161.01±10.96^{b}
144h	195.12± 10.19^{d}	121.36±12.54^{a}	135.85±10.96^{ab}	159.61±9.76^{b}

Mean value with S.D, MC: male in control; MT1: male treated with 0.4mg/100gbody weight; MT2: male treated with 0.6mg/100gbody weight; MT3: male treated with 0.8mg/100gbody weight. Results in each value with different superscript letters were significantly different (P<0.05).

Table 2: Effect of HGH on blood glucose level (mg/dl) of female *A. tristis*

Time of assay	FC	FT1	FT2	FT3
1h	184.21±11.68^{d}	151.01±13.21^{ab}	161.31±8.31^{b}	165.01±12.61^{b}
4h	182.31±12.68^{d}	143.62±9.96^{ab}	160.01±8.63^{b}	165.67±8.93^{b}
12h	179.58±13.85^{d}	122.32±10.62^{a}	154.61±10.91^{ab}	160 02±11.76^{b}
24h	171.01±14.61^{d}	113.01±10.74^{a}	134.36±12.61^{b}	149.22±11.12^{ab}
48h	166.51±14.95^{d}	109.61±6.61^{a}	122.12±8.96^{a}	138.32±10.62^{ab}
72h	169.61±15.22^{d}	103.86±11.95^{a}	122.98±11.91^{a}	140.61±9.81^{ab}
96h	171.51±13.51^{d}	106.72±8.95^{a}	131.96±8.96^{a}	151.73±8.96^{ab}
144h	168.81±15.01^{d}	104.81±6.78^{a}	142.33±7.54^{ab}	148.83±10.12^{ab}

Mean value with S.D, FC: female in control; FT1: female treated with 0.4mg/100gbody weight; FT2: female treated with 0.6mg/100gbody weight; FT3: female treated with 0.8mg/100gbody weight. Results in each value with different superscript letters were significantly different (P<0.05).

Table 3: Effect of HGH on hepatic glycogen level (mg/g) of male *A. tristis*

Time of assay	MC	MT1	MT2	MT3
1h	96.15±5.69^{d}	**126,78±8.97^{b}**	**143.35±10.21^{a}**	**168.32±11.96^{a}**
4h	97.12±6.79^{d}	**118.20±9.91^{b}**	**132.82±9.12^{b}**	**105.61±11.65^{b}**
12h	94.02±7.51^{d}	91.45±8.83^{d}	88.35±5.66^{cd}	82.31±6.18^{cd}
24h	93.12±6.61^{d}	48.81±7.91^{a}	78.52±10.12^{b}	66.58±8.65^{b}
48h	95.58±6.69^{d}	36.12±8.19^{a}	55.01±6.19^{ab}	48.96±6.92^{a}
72h	93.01±7.18^{d}	24.85±8.92^{a}	43.86±7.61^{a}	39.31±7.91^{a}
96h	94.66±7.15^{d}	58.01±9.62^{ab}	30.12±8.62^{a}	25.63±5.78^{a}
144h	95.36±5.81^{d}	66.13±8.71^{ab}	58.51±5.91^{ab}	66.21±6.91^{b}

Mean value with S.D, Bold letters are showing increased value of glycogen level. MC: male in control; MT1: male treated with 0.4mg/100gbody weight; MT2: male treated with 0.6mg/100gbody weight; MT3: male treated with 0.8mg/100gbody weight. Results in each value with different superscript letters were significantly different (P<0.05).

Table 4: Effect of HGH on hepatic glycogen level (mg/g) of female *A. tristis*

Time of assay	FC	FT1	FT2	FT3
1h	82.61±6.75^{d}	**116.37±7.81^{b}**	**124.81±8 .98^{a}**	**133.86±10.96^{a}**
4h	84.23±5.68^{d}	**105.61±10.43^{b}**	**116.52±8.95^{b}**	**99.12±7.81^{cb}**
12h	80.11±9.13^{d}	**83.43±7.31^{d}**	72.81±9.16^{cd}	68.51±6.91^{c}
24h	76.52±6.51^{d}	44.16±5.61^{ab}	74.63±5.69^{cd}	56.12±7.61^{c}
48h	74.95±7.97^{d}	33.31±8.91^{a}	51.45±9.61^{ab}	41.65±6.39^{ab}
72h	80 67±6.91^{d}	20.08±5.81^{a}	38.01±8.91^{a}	30.01±7.19^{a}
96h	82.39±7.97^{d}	53.96±8.21^{c}	27.11±5.81^{a}	21.81±8.61^{a}
144h	81.13±7.51^{d}	73.8 ±6.52^{b}	52.32±9.81^{c}	59.86±10.91^{c}

Mean value with S.D, Bold letters are showing increase in glycogen level. FC: female in control; FT1: female treated with 0.4mg/100gbody weight; FT2: female treated with 0.6mg/100gbody weight; FT3: female treated with 0.8mg/100gbody weight. Results in each value with different superscript letters were significantly different (P<0.05).

DISCUSSION

Hayashida[22] used immunochemical technique proposed similarities between mammalian and avian GH. His finding was based upon the immunologic cross-reaction between rat and chicken pituitary extract. Farmer *et al.*[23] also mentioned that GH from turtle, duck and human all revealed close similarities for contents of histidine, arginine, aspartic acid, threonine and isoleucine. In view of these, there should not be any hesitation in accepting the effectiveness of HGH in birds and that generalisation can be made about the role of GH in birds by using purified HGH when purified avian GH is not available.

HGH is intact *A. tristis* produced hypoglycaemia within an hour of hormone administration. There was simultaneous loss of liver glycogen, although initially there was in increase. The effect on carbohydrate store of blood glucose and liver is not complementary. In both, HGH stimulates the utilization of carbohydrate reserve. The effect is similar and generally proportional in both males and females (figures 1 and 2). The effect revealed that the lower dose of 0.4mg was more potent in its effects as compared to the other higher doses of 0.6mg and 0.8 mg/100g of body weight. On the contrary during the initial part of the experiment higher doses were more effective is causing hepatic glycogenesis.

Among few pikilothermals studied for the effect of GH on carbohydrate reserves, Higgs *et al.*[24] noticed stimulation of β- cells of islet of Langerhans on bovine GH treatment in Coho salmon suggesting the hypoglycaemic effect of GH. Ahsan and Ahsan[9] also found that bovine GH produces hypoglycaemia and increase in hepatic and muscles glycogen level in *Clarias batrachus*. However, Penserat *et al.*[25] found no significant differences in glucose level in GH transgenic and non-transgenic Coho salmon.

The data regarding the effect of HGH are still scantly available in birds. Injection of purified porcine GH brought no alteration in carbohydrate metabolism,[4] and injection of purified bovine GH into young chick neither altered the blood glucose level nor the liver glycogen content. Removal of anterior pituitary gland is growing chickens leads to profound increase in hepatic glycogen level, suggesting a possible hepatic glycogenetic role of GH.[5] GH injection in young fed chicks produced slight hypoglycaemia and lowering of hepatic glycogen level one hour after injection.[26] These works suggest that avian carbohydrate shows fluctuating sensitivity to HGH. Comparison of present finding will be made with those of mammalian studies. In mammals, GH has been reported to produce both diabetogenic and anti-diabetogenic effects. It has been reported to be lypolytic by some and antilypolytic by others. Mollar *et al.*[27] and Lu *et al.*[3] have reported a hyperglycaemic and

diabetogenic effect of GH in human and mice respectively.

In the present study, HGH causes a distinct depletion of carbohydrate reserves in both blood and liver. In birds, therefore, HGH has a hypoglycaemic effect and reduces hepatic glycogen reserve. Glucose removal from blood stream is apparently not utilized for glycogen synthesis. It may be suggested that this reserve is used to meet the increased metabolic needs of the animal consequent to GH treatment. A possible utilization of other energy reserves cannot be ruled out. Lipolysis consequents to GH treatment have been reported by Sakharova *et al.*[28] but their mechanism was not conformed. Although, Quantitative estimation of lipid content has not been made in the present work, it appears that besides carbohydrate, GH also stimulate lipid reserve to meet the energy requirement of the bird consequent to GH treatment. The main role of HGH in *A. tristis* appears to promote the carbohydrate store and to some extent also, the lipid reserves. It is obvious that such an action would favour the conservation of protein reserve. This action of HGH in bird appears to spare amino acids which would have otherwise been utilized for energy production. This suggestion is further strengthened by the fact that HGH has a primary protein anabolic effect in all vertebrates and naturally any process which favour retention of protein reserve will be beneficial to the animal. GH promotes positive nitrogen balance, lowers blood non protein nitrogen and increase protein synthesis.[6]

Dose effect has been noticed in the present study in both genders. The lower dose of 0.4mg/100g body wt is more effective in producing hypoglycaemia as compared to higher doses (figures 1 and 2). This may be due to the possibility that lower dose is more close to the physiological dose and the higher doses effect being of pharmacological nature. The hypoglycaemia produced by HGH in *A. tristis* may partially be due to greater insulin production on GH treatment, as has been found in Coho salmon.[24] This study clearly shows that the main utilizable carbohydrate reserve is blood glucose and liver glycogen.

As the study was conducted on intact bird a possible synergistic effect with other endogenous hormones cannot be ruled out. Since, the dose selected was on a slightly higher side, it would be reasonable to assume that the endogenous HGH production by *A. tristis* would be almost completely blocked. However, the bird's interregnal hormones and other pituitary hormones may have been active in synergism with the exogenous hormone. Undoubtedly, carbohydrate metabolism is controlled by a well-regulated and co-ordinated interaction of various hormones, but evidence from present study suggests that GH of the bird functioning like HGH produces anti-diabetic and glycogenolytic effect, which help in promoting protein anabolism, an apparently important biological action of GH in all vertebrates.

In conclusion, in *A. tristis*, HGH has been shown to exert definite control over carbohydrate utilization. It is, unlike in mammals, hypoglycaemic and probably part of the blood glucose is carried to liver for glycogenesis. This effect later fades out and a glycogenolytic pattern is seen in later sampling for glycogen in liver. The pattern of changes is occurring in carbohydrate reserves in both genders is similar. This suggests the HGH is equally effective in both male and female and responsible to alteration in carbohydrate storage in blood and liver. Depletion of blood glucose level alters the hepatic glycogen reserve to meet the increased metabolic needs of the animal consequent to HGH treatment.

REFERENCES

1. Pollock C. Carbohydrate regulation in avian species. In: Seminars in Avian and Exotic Pet Medicine 2002;11:57-64. WB Saunders Publishers.
2. Kawauchi H, Sower S.A. The dawn and evolution of hormones in the adenohypophysis. Gen Comp Endocr 2006;148:3-14.
3. Lu C, Kumar PA, Sun J, Aggarwal A, Fan Y, Sperling MA, Menon RK. Targeted deletion of growth hormone (GH) receptor in macrophage reveals novel osteopontin-mediated effects of GH on glucose homeostasis and insulin sensitivity in diet-induced obesity. J Biol Chem 2013;288:15725-15735.
4. von Euler U.S. Chromaflin Cell Hormones. Comp Endocrinol 2012;1:258.

5. King D.B, Scanes C.G. Effect of mammalian growth hormone and prolactin on the growth of hypophysectomized chickens. Proceedings of the Society for Experimental Biology and Medicine (New York, NY) 1986;182: 201-207.
6. Sturkie P.D. Sturkie's Avian Physiology-/ed. by G. Causey Whittow. Academic Press 2000.
7. Vijayakumar A, Novosyadlyy R, Wu Y, Yakar S, LeRoith D. Biological effects of growth hormone on carbohydrate and lipid metabolism. Growth Horm IGF Res 2010;20:1–7.
8. Ahsan J, Ahsan S.N. Role of growth hormone in regulation of blood glucose and glycogen level of an Indian teleost *Clarias batrachus* (Linn.). Comp Physiol Ecol 1984;9:178–182.
9. Leung T.C, Ng T.B, Woo N.Y.S. Metabolic effects of bovine growth hormone in the tilapia *Oreochromis mossambicus*. Comp Biochem Physiol A 1991;99:633-636.
10. Dubowski K.M. An o-toluidine method for body fluid glucose determination. Clin Chem 1962;8:215–235.
11. Cho Y, Ariga M, Uchijima Y, Kimura K, Rho J.Y, Furuhata Y, Hakuno F, Amanouchi K, Nishihara M, Takahashi S. The novel roles of liver for compensation of insulin resistance in human growth hormone transgenic rats. Endocrinology 2006;147:5374–5384.
12. Davidson M.B. Effect of growth hormone on carbohydrate and lipid metabolism. Endocrine Reviews 1987;8:115-131.
13. Costa C, Solanes G, Visa J, Bosch F. Transgenic rabbits over expressing growth hormone develop acromegaly and diabetes mellitus. The FASEB Journal 1998;12:1455-1460.
14. Baxter R.C, Holman S.R, Corbould A, Stranks S, Ho P.J, Braund W. Regulation of the insulin-like growth factors and their binding proteins by glucocorticoid and growth hormone in non-islet cell tumor hypoglycaemia. J Clinic Endocr Metab 1995;80:2700-2708.
15. Yakar S, Setser J, Zhao H, Stannard B, Haluzik M, Glatt V, LeRoith D. Inhibition of growth hormone action improves insulin sensitivity in liver IGF-1–deficient mice. J Clin Invest 2004;113:96-105.
16. Jezova D, Radikova Z, Vigas M. Growth hormone response to different consecutive stress stimuli in healthy men: Is there any difference? Stress (Amsterdam, Netherlands) 2007;10:205–211.
17. Giuffrida F, Berger K, Monte L, Oliveira C.H, Hoff A.O, Maciel R, Vieira J.G.H. Relationship between GH response and glycaemic fluctuations in the glucagon stimulation test. Growth Horm IGF Res 2009;19:77-81.
18. Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Government of India. CPCSEA guidelines for laboratory animal facility. Indian Journal of Pharmacology 2003;35:257–274.
19. Kumari B, Ahsan J. Acute exposure of arsenic tri-oxide produces hyperglycaemia in both sexes of an Indian teleost, *Clarias batrachus* (Linn.). Arch Environ Contam Toxicol 2011;61:435-442.
20. Dubowski K.M. An o-toluidine method for body fluid glucose determination. Clin Chem 1962;8:215–235.
21. Kumari B, Ahsan J, Kumar V. Comparative studies of liver and brain glycogen content of male and female *Clarias batrachus* (L.) after exposure of different doses of arsenic. Toxicol Environ Chem 2012;94:1758-1767.
22. Hayashida T. Comparative immunochemical studies of pituitary growth hormones. Growth and Growth Horm 1972;1:25-37.
23. Farmer S.W, Papkoff H, Hayashida T. Purification and properties of avian growth hormones. Endocrinology 1974;95:1560-1565.
24. Higgs D.A, Sutton J.N, Kim H, Oakes J.D, Smith J, Biagi C, Devlin R.H. Influence of dietary concentrations of protein, lipid and carbohydrate on growth, protein and energy utilization, body composition, and plasma titres of growth hormone and insulin-like growth factor-1 in non-transgenic and growth hormone transgenic coho salmon , *Oncorhynchus kisutch*(Walbaum). Aquaculture 2009;286: 127-137.
25. Panserat S, Kamalam B.S, Fournier J, Plagnes-Juan E, Woodward K, Devlin R.H. Glucose metabolic

gene expression in growth hormone transgenic coho salmon. Comp Biochem Physiol Part A: Molecular & Integrative Physiology 2014;170:38-45.
26. Scanes C.G, Braun E. Avian metabolism: its control and evolution. Front Biol 2013;8:134-159.
27. Moller, Niels, and Jens Otto Lunde Jørgensen. Effects of growth hormone on glucose, lipid, and protein metabolism in human subjects. Endocrine Reviews 2009;2:152-177.
28. Sakharova A, Horowitz J.F, Surya S, Goldenberg N, Harber M.P, Symons K, Barkan A. Role of growth hormone in regulating lipolysis, proteolysis, and hepatic glucose production during fasting. J Clinic Endoc Metab 2008;93:2755-2759.

doi: http://dx.doi.org/10.14194/ijmbr.4.2.1
How to cite this article: Hassan N, Kumari B and Ahsan J. Human growth hormone alters carbohydrate storage in blood and liver in both genders of an Indian bird, *Acridotheres tristis* (Linn.). Int J Med Biomed Res 2015;4(2):63-71

Conflict of Interest: None declared