Insulin And Thyroid Hormones Changes In Broiler And Native Saso Chicken Serum After Induced Infectious Runting And Stunting Syndrome (IRSS) 1-Hormonal Disturbance

Hesham A.A. Bayoumie*; Reayid A.M. El-Koshnia; Tahia S. Ismail**; Hosam H. Allam*; Kaied I.H. Galhoom*.**

**Animal Health Res. Inst. Zagazig; ** Radio Immunoassay Central Lab., Zagazig Univ. Hospitals.*

ABSTRACT

The effect of induced infectious runting and stunting syndrome (IRSS) on insulin and thyroid hormones in both broiler and native Saso chicks was investigated. Both types of chicks were inoculated with crude intestinal homogenate from birds suffering IRSS immediately after hatching. Sera samples were collected at 7, 14, 21 and 28 days post-inoculation (PI). Significant increase in T_3 ($P<0.05$) was the only recorded in inoculated Native Saso chicks 7 days PI. Significant increase in T_4 ($P<0.05$) was recorded in inoculated broilers and native Saso 7 days PI. On the other hand insulin level was found significantly lower ($P<0.05$) in inoculated broilers at 14, 21, 28 days PI, and in native Saso at 7, 14, 21 day (PI). Significant weight reduction was recorded in both breeds during the experimental period. Positive correlation was found between body weight and mean ELISA insulin at 14, 21 and 28 days of age in both breeds PI. These findings can correct the understanding of underlying data concerning IRSS.

Three different Reoviruses were isolated from the three crude intestinal homogenate used as evidenced by AGPT and neutralization against S-1133 hyperimmune serum. Their titers were $10^{5.1}$, $10^{4.9}$, $10^{4.75}$ EID_{50}/0.2 ml respectively.

INTRODUCTION

The effect of Malabsorption Syndrome on thyroid function after inoculating one day old broilers with intestinal homogenate, from naturally infected chickens with Malabsorption syndrome was investigated and it was concluded that thyroid function is one of the early targets of the IRSS *(1)*.

Growth retardation in birds suffering from infectious runting and stunting syndrome (IRSS) is due to pancreatic duct obstruction., which led to pancreatic enzymes deficiency., causing maldigestion., with consequent growth retardation *(2)*.

The pathological lesions were described to affect the pancreatic secretions in the coarse of IRSS due to individualization and necrosis in the acinar epithelium and islets of Langerhans beside necrosis and individualization in the ductal epithelium *(3)* which suggests hormonal involvement beside enzymatic deficiency previously described *(2)*.

The present study was aimed to investigate the effet of IRSS on insulin T_3 and T_4 levels in broilers and native saso chicks.

MATERIAL AND METHODS

Material

Specimens

Specimens were obtained from three field cases suffering from IRSS and submitted to Animal Health Research Institute, Zagazig. The proventriculus, pancreas and small intestine from each case were collected separately. These specimens were subjected to viral and bacterial isolation trials and were used for preparation of crude intestinal homogenate used as viral inocula.

Chicks

Forty and sixty recently hatched broilers and Native Saso chicks respectively were used in the present study. Experimental design is summarized in Table 1.

Table 1. Summarizes the experimental design.

Group	Breed	No. of birds	Dose & route	Inocula	No. of sera samples examined	Remarks
1	Broilers chicks	10	0.5 ml orally	Int. homog. a	4	Body wt was recorded at 7, 14, 21 & 28 days of age and sera samples were collected simulta-neously. Sera samples from native Saso chicks were collected through slaughtering at 7 days only then via wing vien
2		10		Int. homog. b	4	
3		10		Int. homog. c	4	
4 (c)		10		-	4	
1	Native Saso chicks	15	0.5 ml orally	Int. homog. a	4	
2		15		Int. homog. b	4	
3		15		Int. homog. c	4	
4 (c)		15		-	4	

4 (c) = control group Int.homog. = Intestinal homogenate
a,b,c are first, second and third inocula from field cases respectively.

Fertile chicken eggs

Fertile chicken eggs from Native Saso breeders was used for virus isolation, titration and virus neutralization.

Reovirus reference strain S-1133

It was obtained from "Vet. Serum and Vaccine Res. Inst. Abbassia, Cairo" its titer was 10^6 EID_{50}/0.2 ml.

Reovirus precipitating antigen and antisera.

Reovirus precipitating antigen (S-1133) and antiserum against S-1133, were obtained form the International Marketing Center, Cairo, Egypt.

Negative serum

Negative serum was kindly obtained form "Dr. M. Abd-El Gany, "Vet Serum and Vaccine Res. Inst. Abbassia, Cairo, Egypt.

Bacteriological media

Blood agar, MacConkey Agar, Nutrient Agar and broth (oxid) were used for bacterial isolation from the crude intestinal homogenate and for detection of bacterial sterility. Media were prepared as instructed *(4)*.

T_3 ELISA kits

T_3 enzyme immunoassay test kit biocheck, inc. catalog number BC-1005 was used *(5)*.

T_4 ELISA kits

T_4 enzyme immunoassay test kit. Biocheck, Inc. catalog number BC-1007 was used *(6,7)*.

Insulin ELISA kits

Insulin enzyme immunoassay test kit. Biosource Europe S.A. catalog number KAP 1251 was used *(8)*.

Methods

Preparation of crude intestinal homogenate

Crude intestinal homogenate was prepared *(9)*.

Bacterial isolation

A loopful from intestinal homogenate was streaked onto blood agar and MacConkey agar. The inoculated plates were incubated for 24 and 48 hrs under aerobic and anaerobic conditions, the later was obtained through the use of (oxid) gas generating kit. Bacterial identification and differentiation was done by gram staining and biochemical testing *(10)*.

Specimen preparation for virus isolation

Crude intestinal homogenate was centrifuged at 3000 rpm for 15 min. The supernatant fluid was collected, antibiotic treated with mixture of penicillin 10,000 IU + streptomycin 5 mg/ml and left at room temp. for 30 min, then membrane filtered through 200 nm filter and examined for bacterial sterility through streaking a loopful on nutrient and MacConkey agar. Bacteria free suspension is used for viral assay and part of it was preserved at -40°C to be used in experimental trials *(3)*.

Viral isolation and titration

Reovirus isolation and titration was performed as previously described *(3)*.

Agar gel precipitation test (AGPT)

AGPT was performed for preliminary Reovirus identification and for judgment of specific chicken embryo mortality due to Reovirus *(11)*.

Virus neutralization

Alpha-virus neutralization was performed *(11).*

Productive performance

Birds in both breeds were weighed weekly and compared to their controls. Birds in both groups were fed ad libitum of pelleted broiler ration "Kairo Poultry Company".

Statistical analysis

Data were statistically analysed *(12)* using MSTAT-C computer program. F-test was used to compare four groups.

RESULTS

Clinical picture and P.M lesions in field cases suffering IRSS

Flock (a)

Flock (a) was (5000 Hubbard broilers 35 days old) reared in an open sided house in Menia El-Kameh; showing signs of IRSS, manifested by presence of 3 sizes of chickens, runted birds is about 10% of the reared flock, P.M. lesion encountered was paleness of the carcass and easily broken long bones, runting, short and severely thickened proventriculus, enteritis, maldigested food materials and bad feathering.

Flock (b), (c)

They are back yard reared Native Saso chickens showing clinical sings of IRSS with varying intensities.

Results of bacterial isolation

Non hemolytic E.coli was isolated form flock (a) while Enterococcus faecalis was isolated form flock (b), (c).

Results of virus isolation

Inoculated eggs via CAM route showed severe thickening of CAM, and greenish liver with marginal necrosis. The mortality pattern, number of passages for each isolate and the results of AGPT are all shown in Table 2-a.

Results of viral titration and neutralization

Results are shown in table (2-b), (2-c)

Pathogenicity of the isolated Reovirus

The mortality % (PI), the clinical symptoms observed and the P.M lesion for both breeds are all shown in Table (3).

Table 2. Shows virological assay.

Sub table	Item	Passages		AGPT for Reo virus	Viral titer **	Neutralizing titer	Neutralization index
		P_1	P_2				
2-a	Viral isolation from sample a	1/5	4/5	+			
	b	4/5	ND	+			
	c						
2-b	Viral titration Reo a				$10^{5.1}$		
	Reo b				$10^{4.9}$		
	Reo c				$10^{4.75}$		
	Reo S-1133				10^{6}		
2-c	Virus neutralization						
	* S-1133 + negative serum					$10^{5.7}$	-
	* S-1133 + hyper immune serum					$10^{3.67}$	$10^{2.03}$
	* Reo 1 + hyperimmune serum					$10^{3.1}$	$10^{1.7}$
	* Reo 2 + hyperimmune serum					$10^{2.7}$	$10^{1.9}$
	* Reo 3 + hyperimmune serum					$10^{2.5}$	$10^{1.95}$

** Viral titer is expressed as EID_{50}/0.2 ml ND : not done

Table 3.Mortality, clinical symptoms and P.M lesions in experimentally infected broilers and Native Saso chicks.

Breed	Group	Inocula	Route	Mort. % *	Observed clinical symptoms				P.M lesion observed at 7 & 14 days P.I
					7 day	14 day	21 day	28 day	
Broilers	1	Int. homg. a	Oral	-	VP	-	-	S	Proventriculitis, enteritis
	2	Int.homg. b	Oral	-	VP	-	-	S	Proventriculitis, pale viscera (PV)
	3	Int. homg. c	Oral	10%	VP	-	-	S	Proventriculitis,(PV),Femur necrosis
	4 (c)	-	-	-	-	-	-	-	-
Native saso	1	Int. homg. a	Oral	-	-	-	-	S	Proventriculitis
	2	Int.homg. b	Oral	-	VP	-	-	S	Proventriculitis
	3	Int. homg. c	Oral	-	VP	-	-	S	Proventriculitis
	4 (c)	-	-	-	-	-	-	-	-

Int.homog. = intestinal homogenate S = stunting 4 (c) = control group.
VP = Vent pasting PV = Pale viscera
* Mortality percent int the first 2 weeks.

Performance

Inoculated groups from both chicken breeds showed significant weight reduction at 7, 14, 21 and 28 days of age. The intensity of weight reduction was severe in groups receiving intestinal homogenate (c). The plateu of weight progression was similar for inoculated and control birds although control showed significantly higher body weight but Native Saso breed usually have a lower body weight than broilers (Table 4 and Fig.1,2).

Hormonal assay

T_3

The mean ELISA T_3 expressed as ng/ml showed non significant difference when measured at 7, 14, 21 or 28 day old in broiler chicks. While it was significantly different at 7 days of age in Native Saso breed (Table 4 & Fig. 3,4).

T_4

The mean ELISA T_4 expressed as ng/ml showed only significant difference higher than control when measured at 7 days of age in both chicken breeds (Table 4 & Fig. 5, 6).

Insulin

The mean ELISA insulin expressed as µIu/ml was significantly lower in inoculated broilers at 14, 21 and 28 days and it was significantly lower in inoculated Native Saso when measured at 7, 14 and 21 days of age (Table 4 & Fig. 7,8).

One way analysis of mean ELISA insulin level for inoculated birds against their age didn't show significant difference from one week to another, while positive significant increase was recorded from the 1st week moving to the 4th week of age in broiler and Native Saso control chicks. Statistical correlation between mean ELISA insulin level and body weight at different intervals revealed positive correlation at 14, 21 and 28 days age for both chicken breeds.

Table 4. Body weight, insulin, T_3 and T_4 levels in broilers and native saso at different intervals.

Examined parameters	Broilers									Native saso							
	Group	7 days	Reduction%	14 days	Reduction%	21 days	Reduction%	28	Reduction%	7 days	Reduction%	14 days	Reduction%	21 days	Reduction%	28	Reduction%
Body weight in (gms)	1	104.6 ± 5.54 b	29.1	244 ± 10.18 b	32.1	485.2 ± 18.4 b	25.5	748.8 ±18.9 b	25.2	67.27± 2.44 b	26.2	144.71± 9.34 c	25	272.71± 10.4 b	19.2	476.71± 12.8 c	26
	2	108.6 ± 2.57 b	26.4	256.2 ± 6.9 b	28.7	489.4 ± 13.3 b	24.9	685.2 ± 20.5 c	31.5	62.81± 2.11 b	31.1	148.28± 3.8 bc	23	252.71± 12.8 b	25.1	558± 10.4 b	13.4
	3	103.6 ± 4.27 b	29.8	253.9 ± 10.33 b	29.4	532.3 ± 17.4 b	18.4	800.5 ± 22.6 b	20.0	61.9± 3.15 b	32.1	164.71± 5.38 b	14.7	280± 10.04 b	17.1	558± 10.4 b	13.4
	Control	147.6 ± 2.96 a *	-	359.8 ± 9.95 a *	-	652.4 ± 13.7 a *	-	1001.7 ± 22.5 a*		91.27± 2.07 a *	-	193.14± 5.5 a *		337.8± 6.8 a*	-	645.0± 10.17 a*	-
Mean ELISA T_3 (ng/ml)	1	3.6 ± 0.9		2.22 ± 0.4		1.86 ± 0.2		1.73 ± 0.1		3.2 ± 0.37 b		3.7± 1.1		3.23± 0.6		3.79± 1.12	
	2	2.7 ± 0.6		1.18 ± 0.7		3.22 ± 0.9		1.17 ± 0.5		4.25± 0.6 a		4.2± 0.8		3.21± 1 .		2.8± 0.9	
	3	1.17 ± 0.7		1.79 ± 0.4		0.95 ± 0.12		1.49 ± 0.09		3.65± 0.12 ab		2.55± 0858		2.5± 0.2		3.72± 1.15	
	Control	2.46 ± 0.48 n.s		2.97 ± 0.42		1.922 ± 0.2 n.s		1.922 ± 0.2 n.s		4.37± 0.42 a *		4.4± 0.64 n.s		4.22± 0.6 n.s		3.75± 0.6 n.s	
Mean ELISA T_4 (ng/ml)	1	4.982 ± 0.691 a		2.63 ± 0.7		2.18 ± 0.3		1.606 ± 0.672		3.5± 0.18 a		3.19± 0.32		1.87± 0.34		2.3±0.4	
	2	3.408 ± 0.52 ab		2.72 ± 0.4		1.53 ± 0.09		1:722 ± 0.33		3.53± 0.51 a		4.04± 0.87		2.7± 0.81		1.01± 0.09	
	3	3.962 ± 1.09 ab		1.73 ± 0.7		1.46 ± 0.09		1.112 ± 0.06		3.87± 0.68 a		4.04± 1.02		2.94± 0.53		1.91± 0.32	
	Control	1.579 ± 0.14 b *		1.91 ± 0.4 n.s		0.981 ± 0.04 n.s		1.579 ± 0.376n.s		1.53± 0.18 b*		2.4± 0.26 n.s		1.37± 0.19 n.s		1.46± 0.19 n.s	
Mean ELISA insulin (uIU/ml)	1	9.67 ± 1.33	29	8.182 ± 0.17 b	70	8.277 ± 0.11 b	56	9.55 ± 0.7 b	39	7.33 ± 0.58 b	45	9.23 ± 0.66 b	46	10.36 ± 0.31 b	34	9.59 ± 0.0.35	41
	2	8.442 ± 1.91	38	9.627 ± 0.84 b	66	9.05 ± 0.27 b	51.9	10.96 ± 0.64 b	30.9	6.96 ± 0.56 b	48	11.05 ± 0.92 b	36	10.87 ± 0.63 b	31	16.06 ± 0.3.4	1.7
	3	8.977 ± 0.742	34	4.79 ± 1.63 b	83	8.14 ± 0.12 b	56	10.05 ± 0.59 b	36.6	8.573 ± 0.88 b	35	9.6 ± 0.52 b	44	10.77 ± 0.78 b	32	9.97 ± 0.69	39
	Control	13.632 ± 1.62 n.s	-	28.435 ± 1.88 a *	-	18.85 ± 0.2.5a *	-	15.87 ± 2.93 a *	-	13.387 ± 0.1.4 a *	-	17.3 ± 2.97 a *	-	15.87 ± 2.4 a *	-	16.35 ± 2.12 n.s	

n.s = non significant * Means followed by different letters were significantly different and the letter "a" is given for the highest value.

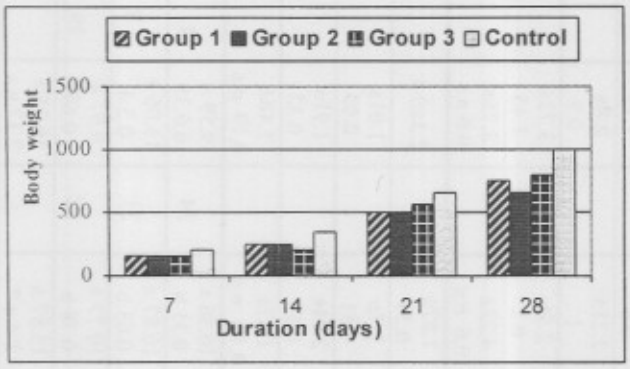

Fig. 1 . Body weight of boiler chickens PI in gms.

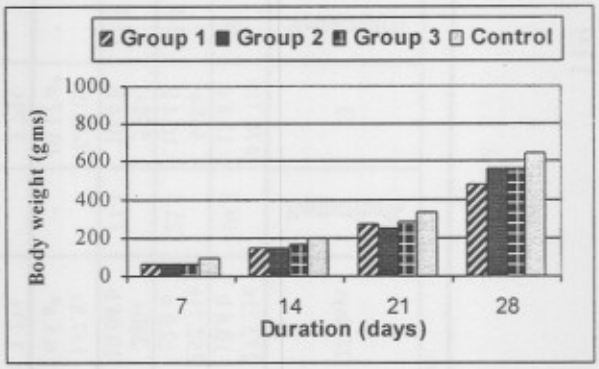

Fig. 2. Body weight of Saso chickens PI in gms

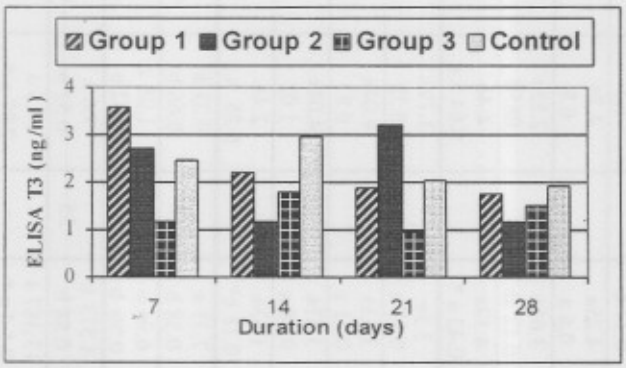

Fig. 3 . T3 ng/ml of broilers PI .

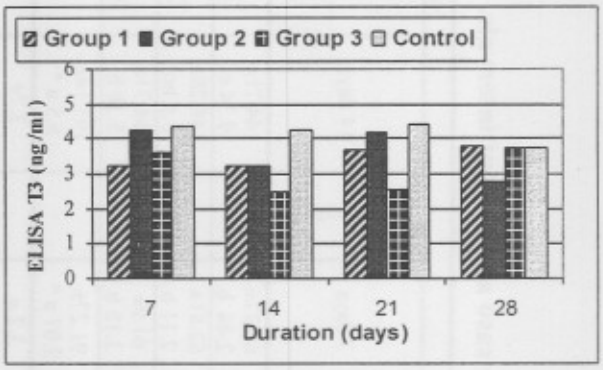

Fig. 4 . T3 ng/ml of native saso PI .

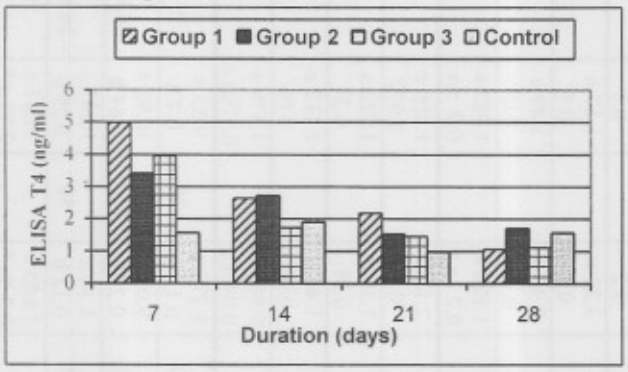

Fig. 5 . T4 ng/ml of broilers PI .

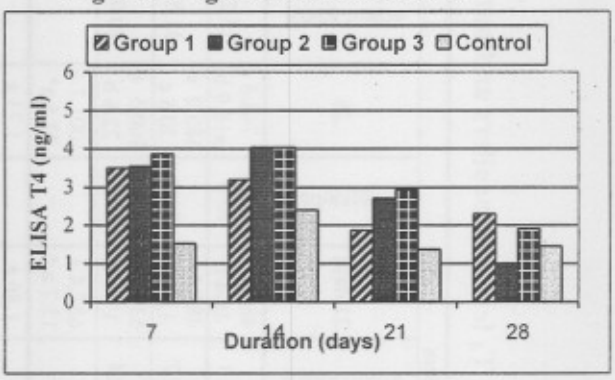

Fig. 6 . T4 ng/ml of native saso PI .

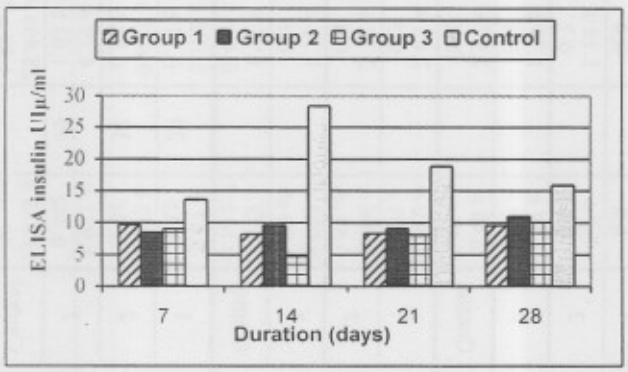

Fig. 7 . Insulin UIμ/ml of broilers PI.

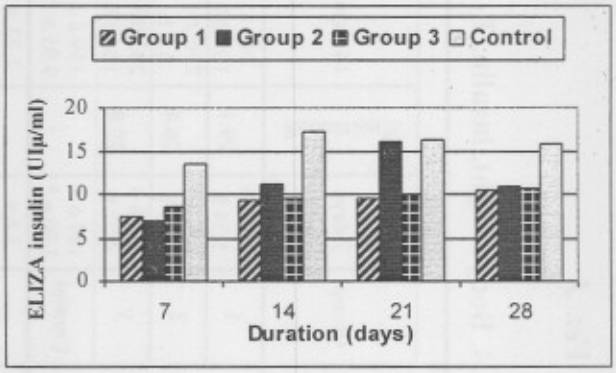

Fig. 8 . Insulin UIμ/ml of native saso PI.

PI = Post inoculation

DISCUSSION

Clinical signs and P.M lesion recorded in the present study are similar to those recorded by several investigators *(3,13- 16).*

Viral isolation trials revealed successful isolation of three viral agents, they were preliminary identified as reoviruses using AGPT, similar findings were previously recorded *(17,18)* virus neutralization using alpha neutralization technique for tentative viral identification was performed., titers of isolated viruses were $10^{5.1}$, $10^{4.9}$ and $10^{4.75}$ EID_{50}/0.2 ml respectively. The neutralization indices using S-1133 reovirus hyperimmune serum were $10^{1.7}$, $10^{1.9}$ and $10^{1.95}$ respectively. *(19)* stated that in the constant serum varying virus procedure a 1.5 $\log_{10}$ neutralization index indicates significant neutralization; thus isolated viruses were reoviruses. Five reoviruses were isolated from IRSS affected flocks and they were related to S-1133 *(20).* Eight reoviruses were isolated from IRSS affected broiler and Native Saso flocks, and they were related to S-1133. *(3).* Reoviruses were isolated from 3 different chicken breeds in USA suffering IRSS *(16).*

The isolated reoviruses *(3,20)* and those isolated in the present study were having different titers and were variably neutralized with S-1133 hyperimmune serum and they had different viral pathogenicity this may be due to the antigenic overlap between reovirus serotypes *(15).* The difference in pathogenicity is initially strain dependent., but it is also helped by other experimental conditions such as the nature of inocula, maternally derived immunity, age, inoculation route and the breed of the inoculated bird *(3).*

In the present study non hemolytic E.coli and Enterococcus bacteria were isolated, similar results were cited by several authors *(16,21,22).* It has been proved that bacteria are not the causative agent of stunting syndrome (23); in the present study the inocula used was antibiotic treated then membrane filtered to insure that only bacteria free inocula are used for experimental infection.

Severe hypothyroidism few days (PI); T_3 level was significantly lower 2 days earlier before T_4 began to drop, the early changes detectable in thyroid function was a decrease in liver 5'-deiodinase activity *(1).* This may be the sequella of coagulative liver necrosis caused by IRSS *(3).* T_3 serum level returned to normal ten day PI but neither T_4 nor 5'deiodinase returned to normal *(1).* The previous authors found that T_4 is not the point at which the syndrome affects thyroid function because during the 1st five days PI no difference could be demonstrated between infected or control birds; thus they concluded that IRSS affects thyroid function via the conversion of T_4 into T_3. The return of T_3 level to normal 10 days PI without the return of T_4 or 5'-deiodinase suggests another mechanism for T_3 formation. T_3 could be formed by the condensation of monoiodotyrosine and diiodotyrosine beside the conversion route of T_4 into T_3 via 5'-deiodination *(24).* In the present study T_3 level PI showed no significant difference at 7, 14, 21 or 28 day in broilers; while it was significantly different at 7 days in Native Saso chicks. T_4 level showed significantly higher levels than control at 7 days in both chicken breeds and was non significant at 14, 21 and 28 days this was in agreement with *(25).* Thus we could conclude that thyroid hormones is not incriminated in the pathogenesis of IRSS

This paper is the first to deal with insulin deficiency as a possible candidate in the pathogenesis of IRSS. Insulin hormone is involved in several biochemical pathways involves carbohydrate, protein, and lipid metabolism thus nearly most of the occurring clinical symptoms can be attributed to this deficiency "symptoms of diabetus mellitus" *(26).* In the present study the mean ELISA insulin expressed as UIu/ml was significantly lower in the inoculated broilers at 14, 21 and 28 days of age; and it was significantly lower in inoculated Native Saso too at 7, 14 and 21 days PI. The reduced body weight in both chicken breeds is acceptable in view of insulin

deficiency., and it should be noted that, although insulin measurement was done at vast intervals; but the sustained weight reduction during the experimental period can prove the continued insulin depression; this insulin depression was correlated with the depressed body weight., and it didn't show any significant changes form one week to another when one way analysis was performed. Circulating plasma insulin in 6 week old chickens is 40-60 UIu/ml and it was 60-90 UIu/ml at 10-24 week old adult chickens *(27)*. The observed extended insulin depression can be interpreted in view of the previously reported extended viral shedding period which may be 14 days *(17)* or 28 days as previously reported by *(3,28-30).* Several authors *(22,31-33,)* stated that the pancreatic lesion is of prime importance in the pathogenesis of IRSS. The degree of pancreatic damage and its repair would determine the degree and duration of growth retardation *(2)*, it worse to mention that they were only referring to the exocrine pancreas. Results obtained in the present study justifies further examination for the clinico-chemical parameters in the collected sera samples.

REFERENCES

1-Rudas P, Salyi G and Szabo J (1986): Decreased thyroxine, triiodothyronine and 5'-deiodination levels in malabsorption syndrome "runting or stunting syndrome" in artificially inoculated broilers. Avian Dis. 30: 293-297.

2-Martland MF and Farmer H (1986): Pancreatic duct obstruction in a stunting syndrome of broiler chickens. Vet. Rec. 118: 531-534.

3-Bayoumie HAA (2004): Studies on Reovirus infection in chickens. Ph. D. Thesis Poult. Dis., Fac. Vet. Med., Zag. Univ.

4-Bridson EY (1990): Cultural media in oxoid manual, 6th Ed. Unipath. Limited, Wade Road, Basingstoke

5-Walker WHO (1977): Introduction : An approach to Immunoassay. Clin. Chem. 23: 384.

6-Wistom GB (1976): Enzyme immunoassay. Clin. Chem. 22: 1243.

7-Schuurs AHWM and Van Weeman BK (1977): Review, Enzyme-Immunoassay. Clin. Chem. Acta. 81:1.

8-Temple PC, Clark PM and Hales CN (1992): Measurement of insulin secretion in type 2 diabetes: Problems and pitfalls. Diabetic Med. 9: 503-512.

9-Kowvenhoven B, Verlommen M, Goren E (1988): Investigation into the role of Reovirus in the malabsorption syndrome. Avian Path. 17: 879-892.

10-Wolfgang B, Gunter A (1988): Gram-negative, aerobic or micro-aerophilic or facultatively anaerobic rods, with simple cultural requirements. Pp. 160-214 in Farbatlas Zur Dignose bakterieller infektionserreger der Tiere. Verlag Panl Parey. |Berlin und Hamburg.

11-Beard CW (1980): Serologic procedure in isolation and identification of Avian pathogens. Pp. 131. AAP. Dept. of Vet. Microbiol. Texas A & M Univ. College Station Texas.

12-Snedecor GW and Cochran WG (1967): Statistical Methods. 6th ed., Iowa State Univ. Press. Ames., Iowa, USA.

13-Mcferran JB and McNulty MS (1993): The runting stunting syndrome. General assessment : in virus infection of birds. El Sevier Science Publishers B.V. Pp. S19.

14-Ghanem IA. and Abd-Allah AAM (1998): Observation on runting stunting syndrome in Native breed chickens. Clinico-biochemical studies. 4th Vet. Med. Congress, Zag. (26-28 August, 1998) in Hurghada.

15-Mcnulty MS, Jones RC and Gough R (2002): Reoviridae: in poultry disease edited by Jordan F, Patison M, Alexander D, Faragher T.

16-Zarala G and Barbosa T (2006): Runting and stunting in broiler chickens. Apinco-Facta, May 2006.

17-Kibenge FSB, Dhillon AS (1986): A comparison of the pathogenicity of four Avian Reoviruses in chickens. Avian Dis. 31: 39-42.

18-Vanderhide L, Lutticken D and Horzinek M (1981): Isolation of Avian Reovirus as a possible etiologic agent of osteoporons "Brittle borne disease, femoral head necrosis in broiler chickens. Avian Dis. 25 : 847-856.

19-Hanson RP (1975): Newcastle disease virus in : Isolation and identification of Avian pathogens. Pp. 63-66 Texas A & M Univ. College Station, Texas 77843.

20-Bekhit BA (1988): Studies on Reovirus in commercial broiler farms. Ph. D. Thesis "Poult. Dis.", Fac. Vet. Med., Alex Univ.

21-Songserm Th, Pol JMA, Van Roozelaar D, Kok GL, Waganaar F and Ter Huurne AAHM (2000): Acomparative study of the pathogenesis of malabsorption syndrome in broilers. Avian Dis. 44: 556-567.

22-Songserm Th, Zekarias B, Van Roozeloar DJ, Kok RS, Pol JMA and Pijpers A (2002): Experimental reproduction of malabsorption syndrome with different combinations of reovirus, Escherichia coli and treated homogenates obtained from broilers. Avian Dis. 46: 87-94.

23-Sell JL, Reynolds DL, Jeffrey M (1992): Evidence that bacteria are not causative agents of stunting syndrome in poults. Poult. Sci. 71: 1480-1485.

24-Phillis JW (1975): Vet. Physiology. Edited by Phillis JW. Dept. of Physiology College of Medicine. Univ. of Sashatchewan. Bristol. : Wrigw-Scientechnica.

25-Stojevic Z, Milinkovic-Tur S, Curcija K **(2002):** Changes in thyroid hormones concentrations in chicken blood plasma during fattening. Veterinarski Archiv 70: 31-37.

26-Lumeij JT (1994): Endocrinology. In Avian Medicine: principles and Application. Edited by Ritchie BW, Harrison GJ, Harrison LR. Wingers Publishing Inc.

27-Hazelwood RL (1976): Carbohydrate metabolism in Avian physiology. Pp. 210. 3rd Ed. Edited by Sturkie PD. Springer-Verlag New York Heidelberg Berlin.

28-Jones RC and El-TAher AMM (1985): Reisolation of Avian Arthrotropic Reovirus R_2 from chicks inoculated as embryos. Avian Path., 14: 373-382.

29-Kibenge FSB, Gwaze GE, Jones RC, Chapman AF and Savage CE (1985): Experimental Reovirus infection in chickens: observation on early viraemia and virus distribution in bone marrow, liver and enteric tissues. Avian Path. 14: 87-98.

30-Kheir El-Din AMW and El-Sanousi A (1986): Infections stunting and runting syndrome "Helicopter disease". 1. Clinical investigation, isolation and identification of the possible aetiologic agents. J. Egypt. Vet. Med. Assoc. 46: 288-294.

31-Sinclair AJ, Embury DH, Smart IJ, Barr DA, Reece RC, Hooper PT and Gould JA (1984): Pancreatic degeneration in broilers with runting and stunting syndrome. Vet. Rec. 115: 485-488.

32-Salyi G and Szabo J (1985): Observation on the pathogenesis of "Malabsorptoin syndrome" of chicken. 1- STudy on the digestive enzymes and digestive coefficients. Magyar Allatorvosok Lapja. 40: 323-327.

33-Frazier JA, Farmer H and Martland MF (1986b): A Togavirus like agent in the pancreatic duct of chickens with infections stunting syndrome. Vet. Rec. 119 : 208-209.

الملخص العربى

التغيرات الحادثة فى هرمون الأنسولين وهرمونات الغدة الدرقية فى سيرم كتاكيت التسمين والبلدى الساسو بعد العدوى الصناعية بمتلازمة التقزم واعاقة النمو
١- الإختلال الهرمونى

هشام أحمد عبد البديع محمد بيومى* ، رياض على محمد الخشنيه،**
تحيه شفيق اسماعيل ، حسام حسن علام* ، قايد ابراهيم حسن جلهوم***
*معهد بحوث صحة الحيوان بالزقازيق – وحدة الدواجن والباثولوجيا الاكلينيكية- **المعمل المركزى للنظائر المشعة – مستشفيات جامعة الزقازيق

أجريت هذه الدراسة لمعرفة تأثير متلازمة التقزم واعاقة النمو على هرمون الأنسولين والغدة الدرقية لما لهم من علاقة مباشرة على الهضم والتمثيل الغذائى. فى هذه الدراسة تم استعمال ٤٠ كتكوت تسمين و ٦٠ كتكوت بلدى ساسو. قسم كل نوع إلى أربع مجموعات بحيث يتم حقن المجموعات الثلاث الأولى بالمزيج المعوى من كتاكيت مصابة حقليا بمتلازمة التقزم واعاقة النمو وذلك بعد فقسها مباشرة. وتبقى المجموعة الرابعة كضابط للتجربة. أمكن من خلال هذه الدراسة عزل ثلاث معزولات لفيروس الريو من المزيج المعوى المحضر من ثلاث حالات بها إصابة بمتلازمة التقزم واعاقة النمو. أمكن التعرف عليهم بعد اجراء اختبار الترسيب فى الآجار ضد الانتجين والمصل المرجعى لعترة الريو S-1133 وكذلك بعد اجراء اختبار التعادل المصلى فى أجنة البيض. وكذلك أمكن فى هذه الدراسة عزل بكتريا القولون (Non hemolytic E.coli) من المزيج المعوى للقطيع (A) وكذلك عزل ميكروب الانتيروكوكس (Enterococcus) من المزيج المعوى (c) , (b) وقد تم معاملة السائل الطافى للمزيج المعوى بمخلوط مضاد حيوى من البنسلين والاستربتومايسين وتمرير هذا السائل من خلال المرشح الغشائى(membrane filter) سعة (200 µm). وتم اختبار هذه السوائل بالزرع على المزروعات البكترية واستعملت فى تجارب العدوى بعد أن ثبت خلوها من البكتريا. تم تجميع عينات السيرم من الكتاكيت عند ٧ ، ١٤، ٢١ و ٢٨ يوم بعد الحقن. وقد استلزم ذلك ذبح عينات من الطيور البلدى فقط عند عمر ٧ أيام بعد ذلك أمكن جمع العينات من الجناح. تم فحص عينات السيرم وقياس هرمون الأنسولين وهرمونات الغدة الدرقية T_4, T_3 فى الأعمار السابق ذكرها.

أظهرت النتائج اختلافا معنويا فى مستوى هرمون T_3 ($P<0.05$) فى سيرم الكتاكيت البلدى المحقون عند عمر ٧ أيام. وكذلك لوحظ وجود ارتفاع معنوى فى هرمون T_4 ($P<0.05$) فى سيرم الكتاكيت البلدى والتسمين المحقون بالمزيج المعوى عند عمر سبعة أيام. ومن ناحية أخرى فقد أظهرت النتائج انخفاضا معنويا ($P<0.05$) فى مستوى هرمون الأنسولين عند عمر ١٤ ، ٢١ و ٢٨ يوم فى الكتاكيت التسمين وقد وجد أن هذا الانخفاض كان فى عمر ٧ ، ١٤ و ٢١ يوم بعد الحقن فى الكتاكيت البلدى الساسو وقد وجد أن هذا الانخفاض كان مرتبطا بانخفاض متوسط الوزن الملاحظ طول فترة التجربة فى نوعة الكتاكيت المحقونة. وقد لوحظ عند تحليل هذه النتائج باستخدام "One way analysis" التحليل فى جهة واحدة "العمر" ان مستوى هرمون الانسولين لم يتغير معنويا من أسبوع لآخر فى الكتاكيت المحقونة فى حين أنه كان يتغير تغيرا معنويا من أسبوع لآخر فى ضابط التجربة البلدى الساسو والتسمين.

هذا البحث يعتبر الأول الذى يشير إلى الدور الذى يلعبه انخفاض هرمون الأنسولين فى متلازمة التقزم وسوف يؤدى إلى فهم كثير من المشاهدات الخاصة بمتلازمة التقزم واعاقة النمو . وقد وجد أن النتائج التى تم الحصول عليها تستدعى اجراء الاختبارات البيوكيميائية على عينات السيرم لكشف النقاب عن حقائق علمية أخرى.