vet.Med.J., Giza. Vol.48, No.4. (2000): 539-555.

SERO-EPIDEMIOLOGICAL, HAEMATOLOGICAL AND HISTOPATHOLOGICAL STUDIES ON TUMOR VIRUS INFECTIONS IN BROILER BREEDERS AND COMMERCIAL BROWN LAYER CHICKENS.

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Received: 2.7.2000. Accepted: 3.8.2000.

SUMMARY

Results of seroepidemiological, haematological and pathological studies of 5 breeds of broiler breeders raised on 12 farms and one breed of commercial brown layers on 7 farms suffering from visceral and bone tumor formation were suggestive of infection with the novel avian leukosis subgroup-J (ALV-J). These infection was associated with reticuloendotheliosis virus (REV) and Marek's disease virus (MDV) exposure.

The impact of coinfection with these viruses and some aspects of control were discussed.

NTRODUCTION

Service tetrovirus of chickens that has emerged in the UK (Panye, 1989; Panye et al.,

1991) and the USA (Fadly, 1998) and can cause significant disease losses in meat type breeders and progeny. Disease caused by ALV-J can be neoplastic often myeloid leukosis (ML), or non-neoplastic. The exact nature and impact of non-neoplastic disease caused by ALV-J is not completely understood; however, selective immunosuppression, increased mortality, increased morbidity, decreased weight gain and uneven growth have been attributed to ALV-J in commercial settings (Spackman et.al., 1999).

Avian leukosis viruses (ALVs) from chickens are now classified serologically into six subgroups (A,B,C,D,E and J) by their viral envelope antigens (Payne and Fadly,1997). Unlike the acute avian leukemia viruses, exogenous ALVs, belonging to subgroups A,B,C,D, and J and endogenous ALVs (subgroup E) are not genetically defective but lack host oncogenes. Exogenous ALVs induce a variety of neoplasms, but endogenous

ALVs are rarely oncogenic.

In Egypt, Ahmed et al. (1999) first reported ALV-J infection in imported broiler parent chickens based on gross pathological lesions and antibody detection to ALV-J.

During the last few years, the existance of unusual tumor formations on bone surfaces on autopsy pathology and high incidence of tumor mortality were observed in broiler breeder and commercial brown layer flocks, which resulted in severe economic consequences. In the present study, results of gross pathology and histopathological, haematological and serological investigations are reported, which covered 19 of such farms located in 7 governorates in Egypt.

MATERIALS AND METHODS

History of examined chicken farms:

A total of 19 chicken farms were investigated during 1997 - 2000. They included 12 broiler breeder farms of 5 breeds (3 local and 2 imported breeds) and 7 commercial brown layer farms of the one breed located in 7 governorates . Further details about the history of examined farms are given elswhere (Table 1 and 5).

Samples for histopathological studies:

Post-mortem examination was performed on variable numbers of live and freshly dead birds . Gross lesions were recorded and organs with

gross tumor lesions, mainly visceral and bone tumor formation, were collected for histopathological studies as follows:

(1) Soft tissues:

Samples from liver, kidney, intestine, proventriculus, heart, lung, spleen ,ovaries and testicles were fixed in 10 % neutral formaline.

(2) Bones:

Samples from sternum, ribs, keel bone, pelvic bone, and vertebrae were fixed in 10 % neutral formaline for 2 to 5 days then immersed in large quantity of decalcifying solutions (Soln. A: 50 gm sodium citrate in 125 ml dist. water and soln. B: 125 ml of 90 % formic acid in 125 ml dist. water. After immersing the bone specimens in soln. A and B for 5 - 14 days, they were washed by tap water over night.

The washed soft tissues and decalcified bones were dehydrated in different concentration of alcohols, cleared in xylol and embedded in paraffin. Sections of 5 micrometer were then cut and stained with haematoxylin and eosine (H & E) stain according to Lillie (1984).

Haematological studies: -

Blood was collected by heart puncture into clean dry bottles containing the anticoagulant ethylene diamine-tetra-acetic acid (EDTA) for the following tests:

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(A) Total erythrocytic and leucocytic counts:

These were performed according to Natt and Henrick (1952).

(B) Haemoglobin estimation:

Haemoglobin was determined by the acid hematin method and the values were multiplied by the factor 0.91 (Duckes and Schwarts, 1931).

(C) Diffrential leucocytic count:

This was done by the standard method given by Schalm et al. (1975).

Serological examination:

Serum samples from the investigated farms were screened by ELISA for antibodies to ALV - J and REV, using commercial ELISA Kits supplied by IDEXX laboratories, Inc., Westbrook, ME 04092. Application and interpretation of the test were according to the instructions of the kits producer.

Statistical analysis:

Data of haematological studies were analyzed by the students t - test after Steel and Torrie (1960) to determine the significance of differences between treatments and controls.

RESULTS

(A) Epidemiological features of ALV- J infected farms:

During the period 1997 - 2000, 19 ALV - J suspected farms were investigated. They were located in 7 governorates and involved 12 adult broiler breeder farms of 5 breeds (3 locally produced and 2 imported breeds) and 7 adult commercial brown layer farms of one breed as shown in tables (1 and 5). The examined birds suffered from tumor mortality of 1.3 - 4.9 % in locally produced and 1.4 - 3.2 % in imported broiler breeder breeds , and 0.4 - 3.3 % in commercial brown layers per week . In addition, all farms showed lower egg prodution levels (43 - 69 % and 63 - 69 % in broiler breeders respectively and 52 - 82 % in layers) and 4 - 39% and 6 - 7% lower hatchability in locally produced and imported broiler breeder breeds, respectively.

(B) Gross lesions:

Postmortem examination of dead and sacrificed birds of broiler breeders and commercial brown layers revealed moderately to markedly emaciated carcasses, unusual tumor bone formations, congested and / or enlarged liver, spleen, lung, kidney, heart, gonads and mesentry with diffuse or nodular whitish infiltrations (Figs. 5 and 6). Very characteristic lesions were creamy and friable or chessy, and diffuse or nodular tumors, often at the costocondral junction of the ribs, over the

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Fig (1): ML, 40-WK-old broiler breeder, showing pale, yellow tumors over the sternum under the breast muscles.

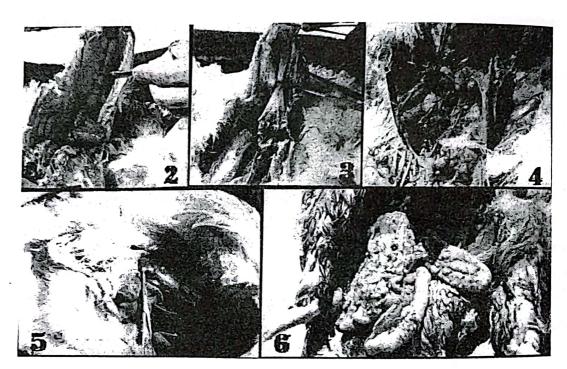


Fig (2): ML, 36-WK-old broiler breeder, showing soft, pale, yellow tumors associated with bones typical of ML. The inner sternum and the costocondral junction of the ribs were common sites for tumor formation.

Fig (3): 35-WK- old broiler breeder, showing creamy and friable nodular tumors at the inner surface of keel bone.

Fig (4): ML, 49 - WK-old broiler breeder. Tumors associated with the flat bones of the pelvis around the hip joint.

Fig (5): ML, 49-WK-old broiler breeder, showing enlargement of liver with nodular whitish infiltration. Fig (6): ML, 48-WK-old brown commercial layer, showing creamy nodules on the mesentery.

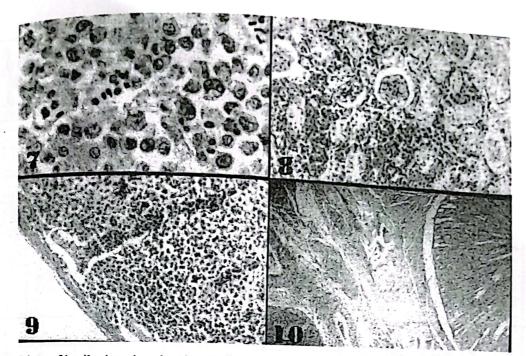


Fig (7): Liver of broiler breeder, showing myelocytes with marked granulations (H&E) X 1000.

Fig (8): Spleen of commercial layer, showing infiltration with myelocytes [H&E] X 250].

Fig (9): Kidney of commercial layer, showing proliferation of myelocytes in between degenerated renal tubules [H&E] x 250]

Fig (10): Proventriculus of commercial layer, showing proliferation and infiltration of lymphoid tumor cells [H&E] X 40].

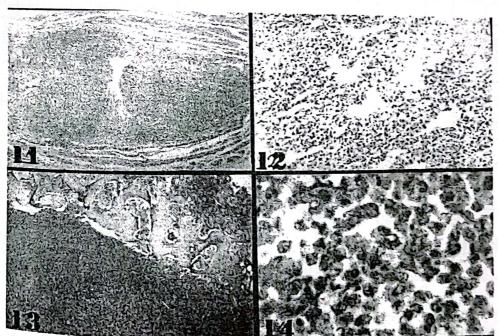


Fig (11): Heart of broiler breeder, showing degeneration of myocardial cells with myelocytic infiltration in between [H&E X 40].

Fig (12): Lung of commercial layer showing myelocytic infiltration throughout the lung parenchyma [H&E X 2501

Fig (13): Keel bone of broiler breeder, showing massive proliferation of mylocytes with errusion of the trabeculae in bone marrow [H & E X 40].

Fig (14): Tumor on pelvic bone commercial layer, showing granular myelocytes [H&E X 1000].

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Table (1): History of examined locally produced and imported broller breeder farms.

Valiabia		Kallobia A 10.000 36	A 10.000 36 65 40 Not recorded	A 10.000 36 65 40 Not recorded	A 10.000 36 65 277 40 Not recorded Not recorded
		· >	A 10.000 36 Not recorded 44 Not recorded	A 10.000 36 65 Not recorded Not 40 Not recorded Not	A 10.000 36 65 277 40 Not recorded Not recorded Not ercorded Not
3	3.000 3.000		3.000 3.000	3.000 3.000 49	3.000 3.000 49 66
The second secon	3.000 3.000 3.000 3.000 3.000 10.000 10.000		49 35 57 0 31 0	49 66 35 68 57 69 31 64	49 66 494 35 68 820 57 69 310 31 64 478



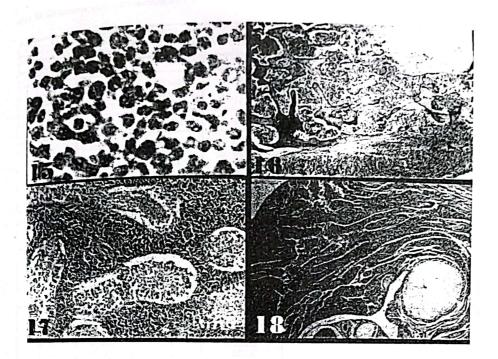


Fig (15): Myelocytosis in bone marrow of broiler breeder [H&E X 1000].
Fig (16): Rib bone of broiler breeder, showing massive proliferation of myelocytes accompanied with trabecular errusion [H & E X 40].

Fig (17): Testicles of broiler breeder, showing infiltration of myelocytes with atrophy and degeneration of

reginered of total street, showing initiation of inference with anophy and degeneration of semineferous tubules [H & E X 100].

Fig (18): Ovary of commercial layer, showing massive aggregation of myeloid tissue, reticuloendothelial cells and lymphocytes [H & E X 40].

Table (2): History of examined commercial brown layers.

Examined	Governorate	Governorate Housing House Age/ Egg	Egg	Mortality / We			
Date	os i dinorate	System	Capacity	Wks.	production % wk	No.	%
15/8/99	Sharkia	Floor	9.000	28	56	300	3.3
19/3/98	Sharkia	Floor	12.000	25	68	100	0.8
12/7/98	Kaliobia	Floor	20.000	45	76	120	0.6
7/5/99	Kaliobia	Floor	20.000	38.	82	400	0.4
6/3/99	Kaliobia	Floor	11.000	55	55	75	0.7
5/11/99	Kaliobia	Floor	13.000	60	52	130	1
15/2/2000	Kaliobia	Floor	7.200	48	78	120	0.8
	Date 15/8/99 19/3/98 12/7/98 7/5/99 6/3/99 5/11/99 15/2/2000	15/8/99 Sharkia 19/3/98 Sharkia 12/7/98 Kaliobia 7/5/99 Kaliobia 6/3/99 Kaliobia 5/11/99 Kaliobia 15/2/2000 Kaliobia	Date Sovembrate System 15/8/99 Sharkia Floor 19/3/98 Sharkia Floor 12/7/98 Kaliobia Floor 7/5/99 Kaliobia Floor 6/3/99 Kaliobia Floor 5/11/99 Kaliobia Floor 15/2/2000 Kaliobia Floor	Date Governorate System Capacity 15/8/99 Sharkia Floor 9.000 19/3/98 Sharkia Floor 12.000 12/7/98 Kaliobia Floor 20.000 7/5/99 Kaliobia Floor 20.000 6/3/99 Kaliobia Floor 11.000 5/11/99 Kaliobia Floor 13.000 15/2/2000 Kaliobia Floor 7.200	Date System Capacity Wks. 15/8/99 Sharkia Floor 9.000 28 19/3/98 Sharkia Floor 12.000 25 12/7/98 Kaliobia Floor 20.000 45 7/5/99 Kaliobia Floor 20.000 38 6/3/99 Kaliobia Floor 11.000 55 5/11/99 Kaliobia Floor 13.000 60 15/2/2000 Kaliobia Floor 7.200 48	Date Governorate Reservation of the second	Date Governorate Housing System House Capacity Age/Wks. production % wk No. 15/8/99 Sharkia Floor 9.000 28 56 300 19/3/98 Sharkia Floor 12.000 25 68 100 12/7/98 Kaliobia Floor 20.000 45 76 120 7/5/99 Kaliobia Floor 20.000 38 82 400 6/3/99 Kaliobia Floor 11.000 55 55 75 5/11/99 Kaliobia Floor 13.000 60 52 130 15/2/2000 Kaliobia Floor 7.200 48 78 120

 $W_{K_S} = M_{\text{ortalities}}$.

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Table (3): Results of antibody detection to avian leukosis subgroup - J and reticuloendotheliosis viruses by ELISA

Breeds/ Origin	Farm	No. of	Examined	POS. NO. / I	Ab- detection	by ELISA	
Origin	No.	houses	Date	ALV-J*	REV**	Precentage	of inciden
	1	I	7/2/99	18/37	W 40 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	Vr.1.	REV.
1	. 1		4/3/99	19/23	20/20 22/22	48.6	1000
			1/4/99	30/34	20/20	82.60	100.0
	and and		Total	67/94	62/52	88.20	100.0
Ī	2	6	20/8/99	20/20	0232	71.2	100
	1 E		20/0/37	20/20 20/20	10/20	100.0	73.8
- 6		- 46	y	18/20	12/22	100.0	50.0 65.0
	1 1			18/20	12/20	90.0 90.0	60.0
			F* .	18/20 16/20	12/22	90.0	60.0
1	* 1		Total	110/120	16/20	80.0	60.0 80.0
ł	3	1	15/12/99		74/120	91.66	61.5
ŀ	4	de la constitución de		17/20	18/20	85.0	90.0
	4	3	19/10/99	16/20	18/20	80.0	
				20/20 20/20	16/20	100.0	90.0 80.0
	5	6	Total		16/20	100.0	80.0
I marall	7 7	and the state of		56/60	50/60	93.3	83.3
Locally produced			31/8/99	16/20	15/20	80.0	75.0
breeds	1 - 1			18/20 20/20	16/20 15/20	90.0	80.0
1				20/20	17/20	100.0	75.0
				20/20 20/20	18/20	100.0	85.0 90.0
			Total		20/20	100.0	100.0
	6	4	8/4/2000	114/120	101/120	95.0	64.2
		1	8/4/2000	10/12 4/14	11/20	83.3	91.67
				10/14	8/14 14/14	28.57	57.14
	J	i.	te construction	7/12	12/12	71.42 58.3	100
	7		Total	31/52	45/52	59.6	85.5
	\	2	13/12/98 14/6/98	15/20	18/20	75.0	90.0
			Total	18/20	20/20	90.0	100.0
	8	3	2/1/99	33/40	38/40	82.5	95.0
	, i		2/1/99	16/20 13/20	18/20	80.0	90.0
				12/20	14/20 12/20	65.0 60.0	70.0 60.0
	100		Total	41/60	44/60	68.3	73.3
	9	E I I I	5/2/98	10/20	14/20	50.0	70.0
	10	2	25/2/2000	17/20		1111	
				20/20	10/20 11/20	85.0 100.0	50.0 55.0
			Total	37/40	21/40	92.5	52.5
	11	1	26/7/98	14/20	20/20	70.0	100.0
	12	15	22/8/98	4/10	6/10	40.0	60.0
				4/10	5/10	40.0	50.0
				5/10 4/10	7/10	50.0	70.0 70.0
		4.1	1	5/10	7/10 - 6/10	40.0 50.0	60.0
			Total	22/50	31/50	44.0	62.0
			9/11/97	2/10	4/10	20.0	40.0 50.0
1.50		+		3/10	5/10	30.0	40.0
Imported	1		1 122 111	1/10 3/10	4/10 4/10	10.0 30.0	40.0 30.0
Breeds	9	W 1		2/10	3/10	20.0	40.0
		7 - 1	19254	2/10 1/10	4/10 3/10	20.0 10.0	30.0
		1	1	3/10	4/10	30.0	40.0
	the same of	Dec 1	- be-	2/10	4/10	20.0 20.0	40.0
1			Total	2/10	4/10	21.0	39.0
Total	12	47		21/100	39/100	2000	71
and the second	1 T	-7/	9/11/97	573/786	557/784	72	

[•]ALV - J= Avian leukosis virus subgroup - J.
•• REV = Reticuloendotheliosis virus.

Table (4): Results of antibody detection of avian leukosis subgroup - J and Reticuloendotheliosis viruses by ELISA test in commercial brown layers.

Farm No.	No. of houses	Examined Date	Ab- detection (Pos./ex	by ELISA am.)	Precentage	of incidence
			ALV-J*	REV**	ALV-J	REV
1	1	15/8/99	9/10	10/10	90.0	100.0
2	1	19/3/98	6/10	8/10	60.0	80.0
3	1	12/7/98	13.20	16/20	65.0	80.0
4	5	7/5/99	35/100	60/100	35.0	60.0
5	1	6/3/99	3/10	5/10	30.0	50.0
6	1	5/11/99	4/10	8/10	40.0	80.0
7	2	15/2/2000	16/20	20/20	80.0	100.0
Total	12	3/98-2/2000	86/180	127/180	47.8	70.6

^{*} ALV - J= Avian leukosis virus subgroup - J.
** REV = Reticuloendotheliosis virus.

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Table (5): Characteristics of avian leukosis subgroup - J infection in examined chicken farms.

r	The second						777	100	_	_	
	Commercial layers				parents	B Bill				de	Breed
	Locally produced breed			Imported breeds			Dreeds	produced		mgr.o	Breed
	Brown commercial layers	Total range	ш	D	Total	C	В	۵		variety	Bird
	25-60	29-38	29-38	38	28-65	28-55	35	31-65	244	range /	Age
	52-82	63-69	53	69	43-69	61-68	68	43-69		rang %	Egg prod.
27.10	0.4-3.3	1.4-3.2	1.4-2.0	3.2	1.3-4.9	1.3-4	2.7	2.4-4.9		range /Wk	Mortality
	•	73-74	73	74	41-76	69-74	41	66-76		rang%	Hatchahility
Linda	86/180	57/170	43/150	14/20	516/626	177/232	17/20	322/374	ALV-J*	1.67	
the section of the section	127/180	90/170	70/150	20/20	467/594	191/232	18/20	258/342	REV**	exam. no.	Ab-detection by
	47.8	33.52	28.66	70.0	82.43	76.29	85.0	86.10	ALV-J	Incidence	on by ELISA
	70.6	52.94	46.66	100.0	78.62	82.33	90.0	75.44	REV	ncidence precentage	
					Visceral	-12				mortem	Post-
				- Reticulo-endothelial cells.	myeloblasts Pleomor- phic and / or small	Myelocytes and/or				Histopathological	

Histopathological findings for Marek's disease recorded at 25-40 weeks of age of examined birds.
 *ALV - J = Avian leukosis virus subgroup - J.
 * REV = Reticuloendothellosis virus.



Table (6): Haematological changes in broiler breeder chickens (strain A) during ALV-J outbreaks.

1			and the					Differential leukocytic count (%)	ocytic count (%)	
no.	houses	sample		(gm%)	(gm%) count	Heterophils	Heterophils	Lymphocytes	Monocytes	Eosinophils	Basophils
	pin	no.	(10 ⁶ /ml) Mean ± sd	Mean±sd	(10³/ml) Mean±sd	(band) Mean±sd	Mean±sd	Mean±sd	Mean±sd	Mean±sd	Mean±sd
: ::::::::::::::::::::::::::::::::::::	1	20	2.40± 0.840	9.90±* 0.250	46.500±* 0.520	1.30±* 0.120	27.60±* 0.565	66.67±* 3.440	2.03±* 0.10	2.2± 0.04	0.65± 0.12
10		20	2.3± 0.660	9.67± 0.110	48.630±* 0.690	1.69±* 0.190	28.14±* 0.780	65.38±* 4.890	2.740±* 0.30	2.6± 0.08	0.70± 0/04
Control**	1	٥.	2.60± 0.350	9.24± 0.150	26.75± 0.652	0.00	21.76± 1.150	63.43± 1.080	11.34± 0.240	2.30± 0.270	1.00± 0.140
*: Significantly different from control at P< 0.001	y different fr	rom control at	P< 0.001	cally negat	tive for ALV-J	and REV by I	ELISA testing a	*: Significantly different from control at P< 0.001 *: Significantly different from control at P< 0.001	ative for postn	nortem and	

**: Control: Broiler breeder chickens were serologically liego histopathological examination for tumor formations.

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sternum under the breast muscles and inner surface of the keel bone (Figs.1,2,3,4). Whitish nodular tumors were found on synsarcum and vertebrae. Thickening of proventriculus mucosa was associated with wall enteritis.

(C) <u>Histopathological examination</u>:

In all cases of the broiler breeders and commercial brown layers, the liver, spleen, lung, kidney, heart, proventriculus, ovaries, and testicles were infiltrated with myeloblasts and/or myelocytes. In some cases, these organs and the isciatic nerves were infiltrated with pleomorphic and/or small lymphocytes and in others, the proventriculus, heart and ovaries were infiltrated with reticuloendothelial cells (Figs. 7 - 12 and 17, 18) The bone marrow of ribs,keel and pelvic bones were massively proliferative with myelocytes and /or myeloblastes in between erroded trabeculae in all cases of examined farms (Figs. 12 - 15).

(D) Serological results:-

The results of serological examination of serum samples from locally produced and imported broiler breeder farms are shown in tables (3,5). From these tables, it is evident that the 3 locally produced breeds (A,B,C) reacted positively for ALV-J and REV antibodies with higher percentages than the 2 imported breeds D&E (averages 82.43 % and 78.62 %, respectively, versus 33.52 % and 52.94% respectively). Concerning the

bird variety, strain A showed higher incidence for ALV-J antibodies (86.10 %) than the other 4 strains, while strain D gave higher incidence for REV antibodies (100.00 %) than the other 4 strains.

In addition, in farm No.(1) antibodies to ALV-J and REV were detected 3 times at monthly intervals with constant high incidence for REV antibodies (average 100 %), while ALV-J antibody incidence showed a rising pattern (average 48.6,82.6 and 88.2 %).

In commercial brown layer chickens, the incidence of REV antibodies which averaged 70.60 % was higher than for ALV-J antibodies which averaged 47.80% (Tables 4,5).

(E) Haematological results:

The results of haematological changes of blood samples from farm (1) and (10) are shown in table (6). From this table, it is obvious that the total WBCs count revealed a significant increase in both farms as compared with control normal birds.

In addition, there were significant differences in the differential leucocytic count precentages in heterophils band between farm (1) and farm (10) as compared with normal birds, but in heterophils mature the differences were less than heterophils

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band. On the other hand, there were significant differences in lymphocytes and monocyts counts in both farms.

DISCUSSION

Avian leukosis is a complex group of disease conditions caused by a variety of tumor- inducing viruses, which adversely affect breeder flock performance (Payne and Fadly, 1997) and the production performance of broilers from breeders with ALV-J tumors as well (Goodwin et al., 1998b).

ALV-J is an example of a new avian retrovirus which has emerged over the past decade to become a serious problem to the poultry industry. Several reports from different countries have been published during the last few years on an increased incidence of tumors, mainly of the myeloid type and other types of neoplasms (Fadly, 1998; Fadly and Smith, 1999; Goodwin et al., 1998 a; Payne et al., 1991; Ahmed et al., 1999; Arshad et al., 1999).

In the present study, we investigated 12 broiler. breeder farms of 5 breeds (3 locally produced and 2 imported breeds) in seven different governotessuffering from tumor mortality which ranged between 1.3-4.9% in locally produced and 1.4 -

3.2 % in imported breeds, with lower egg production levels (43 - 69 % in locally produced and 63 - 69 % in imported breeds) than target.

Moreover, all 12 farms suffered from lower hatchability by 4 - 39 % in locally produced and 6 - 7 % in imported breeds . Farm No. 3 in particular suffered severe drop in hatchability (39 %) and it was observed that males had unusual habit of mating each other, which may explain the role of males infected with ALV-J in lowered fertility and hatchability as well as in the epidemiology of the disease. Histopathological alterations in the testicles, which included infiltration with myelocytes and degeneration and atrophy of the semineferous tubules may support our findings. The ovaries also revealed infiltrations with myeloid reticuloendothelial cells as well as lymphocytes. On the other hand, we also investigated 7 commercial brown layer farms of one breed in 2 governorates suffering from tumor mortality ranging between 0.4 - 3.3 % and lower production levels (ranged from 52 - 82 %) than the targets.

The gross pathological lesions in all examined farms revealed markedly emaciated carcasses and soft, pale, yellow or creamy tumors at the flat bones of the pelvis around the hip joint, over the sternum under the breast muscles, the inner surface of sternum and the costocondral junctions of the ribs. Visceral organs revealed enlargement

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with diffuse or nodular whitish infilteration and in some cases creamy nodules on the mesentry. Histopathologically the internal organs were infiltrated with myeloblasts and /or myelocytes. Also, the bone marrow of ribs, keel and pelvic bones were massively infiltrated with proliferative myelocytes and /or myeloblastes in between erroded trabeculae. Similar observations were made by Payne et al. (1993).

These pathological changes were strongly suggestive of ALV-J infection and agreed with the findings of Arshad et al. (1997b). In some cases, the isciatic nerves were infiltrated with pleomorphic and / or small lymphocytes suggestive of Marek's disease virus infection, and in other cases, proventriculus, heart and ovaries were infiltrated with reticuloendothelial cells suggesting reticuloendotheliosis virus (REV) infection. The results of serological screening for ALV-J antibodies revealed variable incidence (10 % - 100 %) among the twelve broiler breeder farms, which averaged 72 % and was higher in locally produced than imported breeds (82.43 % versus 33.52 %). Considering the bird variety, it is obvious that strain A has higher incidence of ALV-J infection (86.10%) than other strains. In general, a high incidence of ALV-J specific antibodies were detected in all 12 broiler breeder farms located in 7 different governorates which confirmed a previous report by Ahmed et al . (1999) of exposure of such flocks to ALV-J infection, most probably through horizontal transmission from immune tolerant-viremic chickens (Payne, 1998). In seven commercial brown layer farms of one breed located in 2 governorates, the results of serological examination for ALV-J antibodies also revealed variable incidence (30 - 90 %) which averaged 47.8 % and was lower than in broiler breeder farms which confirmed exposure of the flocks to ALV-J infection.

This is considered the first report of ALV-J infection in layers in Egypt. Arshad et al. (1997a) reported that ALV-J had oncogenic properties for Brown leghorn chickens (BrL).

The most remarkable serological finding was the simultineous detection of serum antibodies also against REV in all examined farms with variable incidence, which averaged 71 % in broiler breeder farms and 70.6 % in commercial brown layer farms. REV - infection in broiler breeders in Egypt has been recorded previously on the basis of serological (Ahmed, 1991) and histopathological evidences (El-Sawy, 1994). Mixed infections with MD, REV, lymphoid leukosis (LL) and infectious chicken anaemia (ICA) viruses have also been diagnosed on serological basis (Ahmed, 1991). This significantly high incidence of REV field infection might result from the use of Marek's disease and fowl pox contaminated vaccines

(Jackson et al., 1977; Witter and Johnson, 1985; Ahmed, 1991), or from mosquito transmission (Sinkovic, 1983; Motha et al., 1984), while a low frequency of congenital transmission has been documented in laboratory-infected, viremiatolerant dams (Bagust et al., 1981; Witter et al., 1981).

Furthermore, the impact of REV on ALV-J tumor incidence and virus shedding is still not clear and has been discussed by Payne (1998) and Ahmed et al. (1999). On the other hand, there is another speculations about the impact of coinfections of ALV-J and REV one of the two viruses plays as a trigger and immunosuppressive agent to the other . Payne (1998) speculated the role of intercurrent infections by immunosuppressive viruses or vaccines as predisposing factors in the field which may influence ALV-J disease incidence which give our support for this mentioned proposal. There is no doubt that chickens under poor management either are heavily exposed to virus or are more susceptible to infection by virtue of the extra stress they receive. Haematological examination revealed significant differences in the total WBCs count, heterophilis (band,mature) and monocytes which were associated with the recorded viral infections and might be of additional dignostic value. Payne (1998) mentioned that the HPRS-103 virus replicated in blood monocytes that tissue tropism may relate to their ability o cause myeloid leukosis. The significant increase in lymphocytes may refer to the viral multicausal agents of tumor formation.

In conclusion, more work is needed to study the relationship or interaction between ALV-J and other retroviruses, the role of vaccines in transmitting the infection and genetic resistance to ALV-J infection and tumor formation. Moreover, the existing evidence of high mutation rate of ALV-J may dictate that diagnostic and eradication procedures would consider variant viruses (Payne, 1998). Also, control programs might be needed in the future to impose import restrictions on infected breeder strains.

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