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Detection of granulocyte macrophage colony stimulating factor in quails exposed to thermal stress and treated with effective microorganism

M.M. Younis and H.B. Al-Sabaawy

Department of Pathology and Poultry Diseases, College of Veterinary Medicine, University of Mosul, Mosul, Iraq

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Correspondence:
H.B. Al-Sabaawy
hadeelbasim2006@gmail.com

Abstract

The study investigated the histopathological effects of the liver of quail birds prone to heat stress and treated with effective microorganisms. Forty-one-day-old broilers were erratically distributed into 4 groups, with ten birds for each group, as follows: first group as a control, second group treated with effective microorganisms, third group exposed to heat stress, and the fourth group treated with effective microorganisms and exposed to heat stress. The birds were placed in the animal house and left for 10 days to adapt. On the eleventh day, the experiment was started. For thirty days, the animals were sacrificed. On the fifteenth day and the last day of the experimentation, microscopic lesions were identified as hemorrhage, congestion, vacuole degeneration, cloudy swelling, infiltration of inflammatory cells, and necrosis. Pathological lesions were analysed statistically between the first and second slaying. They were highest in the third group and during the two. slayings. The severity of the pathological changes in the 2nd group was less than in the first, third, and fourth groups, and a significant correlation between lesions was more predominant in disturbance of circulation as hemorrhage. The intensity of protein expression represented by a granulocyte-macrophage colony stimulating factor (GMCSF) was highest in the first slaying, second, and third groups compared to the intensity of it in the fourth group at the second kill. We conclude from the results that living organisms can decrease pathological changes and be used as a protective protocol in poultry farming fields.

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Introduction

Heat stress is the product of an adverse equilibrium between the net amount of force streaming from the trunk to its surrounding ecological area and the amount of thermic force produced by the animal's body (1,2). This lopsidedness may be caused by the alteration of a combination of ecological agents like heat irradiation, the temperature of the air, sunlight, humidity (3), and animal characteristics like species, rate of metabolism, and mechanisms of thermoregulatory system (4), due to the negative effects of various environmental pressures, such as transportation, density, nature of food, heat stress and others, which significantly affect animal agriculture, there has become a sign of awareness and a cause for concern on the part of breeders regarding these environmental pressures (5). All kinds of birds are considered sensitive to environmental stresses related to heat stress. Elewa (6) mentions that the best production of broiler chickens is between 18-22ºC; high temperatures lead to a decrease in production and ultimately cause stress associated with heat stress (7). Birds, including ducks, geese, and quails, differ from other mammals because they do not have sweat glands, and most of their body parts are covered with feathers. Thus, such environmental conditions have caused the chicken's difficulty heating out their body ecologically during the hot conditions (8,9). Heat stress affects the regulatory ability of different body systems, and the response to heat stress causes complicated responses to maintain a steady state of the body (10). One of these responses is increased heart rate and blood flow to the body's

muscles, brain, and heart (11,12). This may cause congestion in the liver and kidneys. The liver is a vital organ in the body that affects metabolic activity. It is represented by cellular functions containing the equilibrium force metabolism, synthesis of vitamins and minerals, and detoxification (13). High blood flow transfers from the hepatic zone to the respiratory system and other body tissue to accelerate heat loss and reduce the temperature under thermal stress; the liver is the most delicate organ to thermic fever (14,15). This fever causes impaired liver function in the broiler and accumulation of fat and inflammation (16,17). Effective microorganisms (EM) are useful organisms composed of 70-80 different types of organisms. They are used as food additives, enhance growth performance (18), modify the gut microbiota, stimulate the immunity system, and curtail the detrimental effects of thermal stress by safeguarding the digestive system from illness and fostering nutrient digestion (19,20). In livestock breeding, these organisms have been widely used as feed additives that regulate the function of the intestinal system by stabilizing the balance of the intestinal microbiota and maintaining it from Pathogens and microbes. EM also contributes to the intestinal digestion of carbohydrates, proteins, and fats, stimulating the necessary enzymes for the body and removing toxins (21). The beneficial effects of Effective microorganisms in birds are evident through increasing daily weight, increasing production performance, improving feed absorption, and reducing mortality rates (22). GMCSF were classified as a hematopoietic outgrowth factor and play a central role in the mature regulation of a myeloid population cell under both inflammation and homeostatic conditions (23,24).

This work aimed to assess the effects of heat stress and effective microorganisms on the quail liver and evaluate immunohistochemistry markers' expression.

Materials and methods

Experimental animal

Forty-one-day-old quails obtained from a private hatchery were placed in the animal house of the College of Veterinary Medicine at the University of Mosul to be subjected to the experiment protocol. The experiment was conducted by the Law for the Care and Use of Animals for Scientific Experiments, in which the Ethics Committee approved it, were affiliated to the University of Mosul at authorization number UM.VET.2023.033.

Experimental design

Into four groups, the birds were split as follows, TI (control N=10), T2 treated with Effective Microorganism N=10 (EM, 1000 PPM in drinking) (22), T3 (N=10) were rear under heat stress (38ºC for six h) and T4 (n=10) were giving EM and exposed to thermal stress, during the experimental period all groups received the same ration of food, the pre-trial period consisted of an acclimation period of 10 days for quails, the birds followed over thirty days, during which the birds were euthanized. Liver sampling was gathered during the 15 and 30 days of the experiment.

Histopathological protocol

At the end of the experiment, the birds were sacrificed, and liver specimens were taken from all the groups (control and treated groups); the samples were placed in 10% formalin solution, and for 24 hours, the specimens were fixed and used for histopathology and immunocytochemical studies. Dehydrated in ascending level of alcohol, cleared by xylol. In paraffin wax, the tissues were embedded routinely, prepared paraffin blocks were cut at 6 micrometers thickening and placed in the routine stain (hematoxylin and eosin) for histopathological studies (25,26).

Immunocytochemistry of GMCSF

The expression of GMCSF in the liver of quails was detected by staining of IHC with the anti-rabbit GMCSF (suit 216, Houston, Texas E-AB-15694). Blocks of paraffin were used, and the liver quail's section at 6µ were firm on charged slide. In the oven at 60ºC for 1 hour, the slides were heated and then washed with xylene; the obtained sections were dehydrated in a descending level of alcohol, washed, and rinsed with buffered saline for five minutes. The antigen retrieval protein blocking solution section was immersed after putting it in the oven for 20 M at 93ºC. For antigen-restricted immunoglobulin or cytokeratin, enzymatic retrieval is used, followed by heat treatment. The section slides were chilled at room temperature and rinsed for 5 minutes with phosphate-buffered slain. Anti GMSCF was applied and incubated overnights at 37ºC, directly rinsing in phosphate-buffered slain then generator color with DAB was used, incubated for 25-30 min, rinsing and washing and for 4-5 minutes, slides were stained with hematoxylin and rinsed with water, at last, the section dehydrated with descending level of alcohol and xylene. Abandoned to droughty at room temperature for 18-20 min, finally DPX and coverslip (27,28).

Statistical analysis

Statistical data analysis was accomplished utilizing the graphing prism pad five. the parameters were analyzed by one- and two-way ANOVA tests accompanied by Duncan's test at $P<0.05$, which was deemed to show a notable difference for all compared data at mean±SEM (29).

Results

Histopathology findings in quail liver

Histopathology findings noticed in various liver sections were subordinated to consideration. Histopathological section of the quail liver of all groups at 15 days post-treatment in the first killing where take, the control group showed standard architecture of liver represented by

hepatocytes, sinusoids, and central vein (Figure 1), cloudy swelling of hepatocytes (Figure 2), exhibited congestion, dilation of the blood vessel, infiltration of inflammatory cell l with necrosis. Vacuolar degeneration was observed in T2 during the second killing (Figure 3), exemplifying standard hepatocytes architecture (Figure 4), vacuolar degeneration (Figure 5), infiltration of inflammatory cells in addition to disturbance of circulation was observed in T3 and T4 exemplify by congestion, vacuolar degeneration and severe necrosis (Figures 6-8).

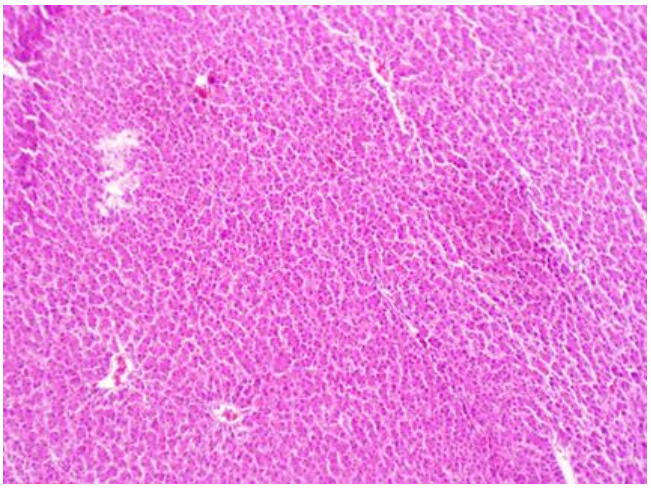

Figure 1: Micrograph section of liver, 1st, control group, H&E, 10X.

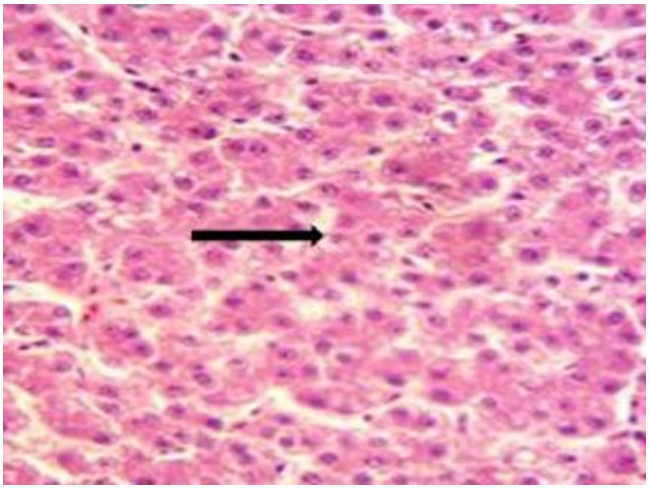

Figure 2: Micrograph section of liver, 1st, EM, cloudy swelling. H&E, 40X.

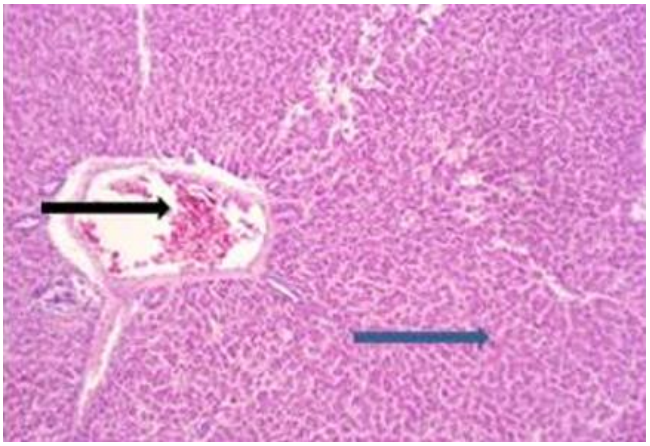

Figure 3: Micrograph section of liver, 1st, H, necrosis blue arrow & congestion black arrow. H&E, 10X.

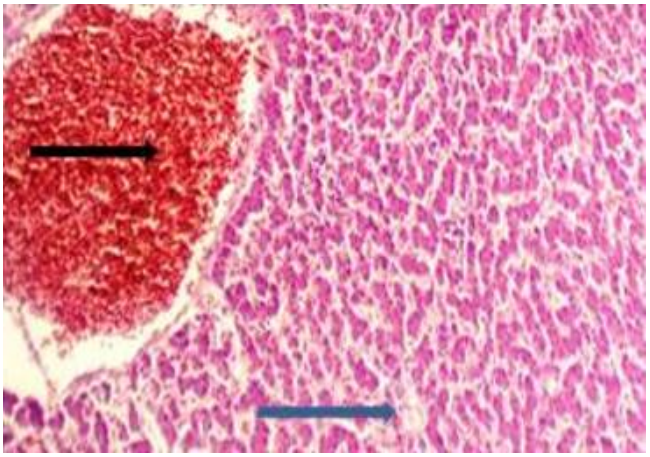

Figure 4: Micrograph section of liver, 1st, M, congestion black arrow, necrosis blue arrow. H&E, 40X.

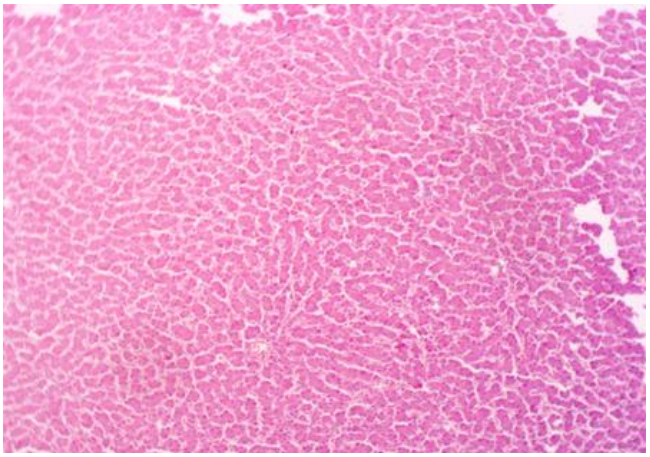

Figure 5: Micrograph section of liver, 2nd, control group, H&E, 10X.

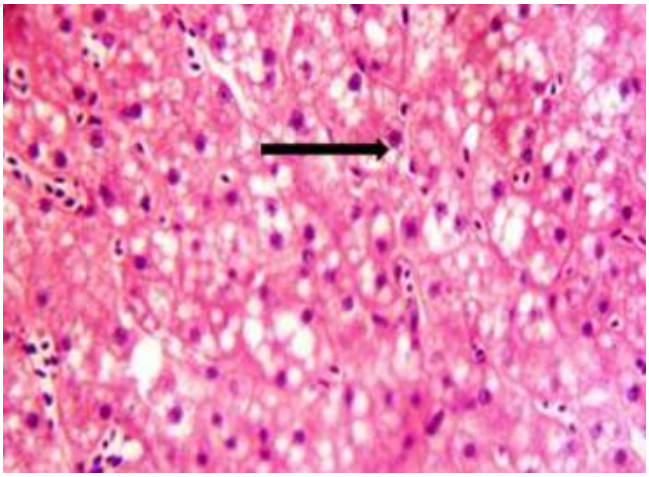

Figure 6: Micrograph section of liver, 2nd, EM, vacuolar degeneration black arrow. H&E, 40X.

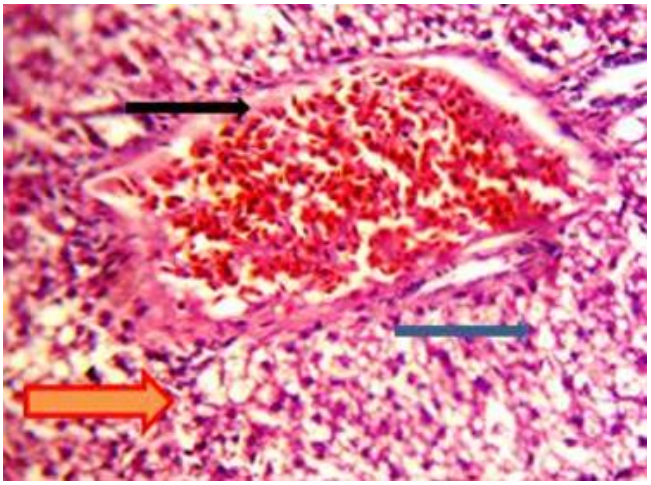

Figure 7: Micrograph section of liver, 2nd, H, congestion black arrow, vacuole degeneration blue arrow, inflammatory cell orange arrow, H&E, 40X.

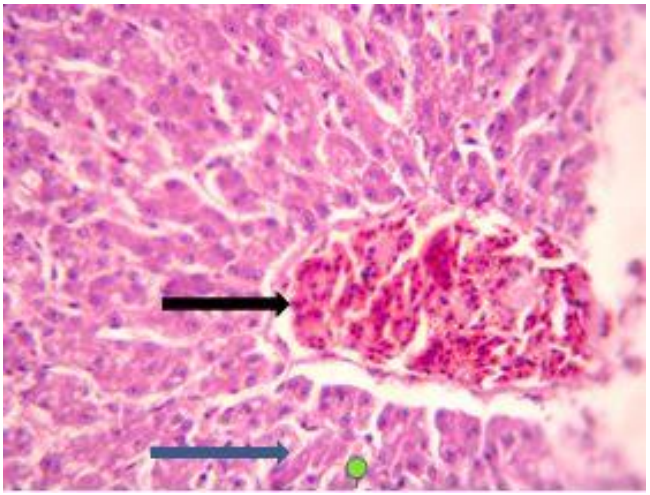

Figure 8: Micrograph section of liver, 1st, H+M, congestion black arrow, necrosis blue arrow. H&E, 40X.

Histological scoring

Statistical analysis outcome showed that there was a significant variance 11±7.55 in hemorrhagic lesions in the thermal group at P<0.05. In contrast, tissue congestion in the third group at the first killing had a significant variation 23.60±1.732 compared to the second and fourth groups in the second killing 10.00±3.46 and 16.00±3.464 and the second group at the first killing, which did not show significant differences between them. The heat Stress group also showed a significant difference in vasodilation lesions and hemorrhage in the third group at the first killing 10.00±3.46 and 16.00±3.464. In contrast, the fourth group, supplemented with live microorganisms and exposed to heat stress, showed a significant difference during the first killing 4.67±1.155. Table 1 and 2 showed the liaison coefficient between the first and second killing traits.

Table 1: The main Histopathological lesion during the first and second killing

Killed	Groups	Hemorrhage	Congestion	Dilation	Inflammation	Thrombus
1st	1	0.00±0.00 b	5.00±4.58 bc	2.00±3.46 b	0.00±0.00 c	0.00±0.00 b
	2	0.00±0.00 b	16.00±3.46 ab	4.00±3.46 b	0.00±0.00 c	0.00±0.00 b
	3	0.00±0.00 b	23.00±1.73 a	10.00±3.46 a	1.67±0.57 b	16.00±3.46 a
	4	0.00±0.00 b	10.00±3.46 abc	4.00±3.46 b	4.67±1.15 a	4.00±3.46 b
2nd	1	0.00±0.00 b	2.00±3.46 c	0.00±0.00 b	0.00±0.00 c	0.00±0.00 b
	2	0.00±0.00 b	18.00±12.00 ab	0.00±0.00 b	0.00±0.00 c	0.00±0.00 b
	3	11.00±7.55 a	12.00±6.00 abc	3.00±0.00 b	1.33±1.52 bc	2.00±3.46 b
	4	0.00±0.00 b	18.00±12.00 ab	4.00±3.46 b	1.00±0.00 bc	0.00±0.00 b

Significant variation between groups represented vertically at P<0.05.

Table 2: Liaison coefficient between first and second killing traits

First killing	Second killing				
	Hemorrhage	Congestion	Dilation	Inflammation	Thrombus
Hemorrhage	-				
Congestion		0.679*			
Dilation of blood vessels			0.506^{NS}		
Inflammation				0.387^{NS}	
Thrombus					0.308^{NS}

* Refer to variables at P<0.05. NS refers to a non-significant correlation between variables.

Immunocytochemistry finding

Immunocytochemistry findings confirm previous results. At the first and second killing, the control group showed negative nuclear expression of the GMCSF marker (Figure 9-14). Meanwhile, the severe expression of GMCSF in the third group that was exposed to thermal stress was found in (Figure 11-15), and mild expression (Figure 12-16). Figure 17 summarizes the IHC results and the correlation between the first and second killing and the GMCSF protein marker. Figure 17 shows the average value between 1st and second kill to reaction with immunohistochemistry protein markers of GMSCF in stress quails 1st second and third groups showed the most significant difference in the intensity of the protein interaction of GMSCF in the first killing in contrast to the intensity with the second killing, while the 4th group in the second killer showed the most significant variance at P<0.05 in the intensity of the interaction compared with the intensity of it in the first killer.

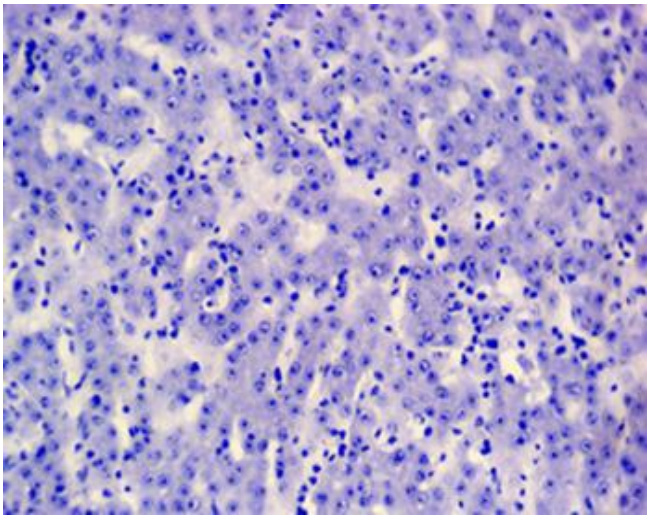

Figure 9: Light microscopic section of control group, 1st kill, negative GMCSF nuclear expression, 40X.

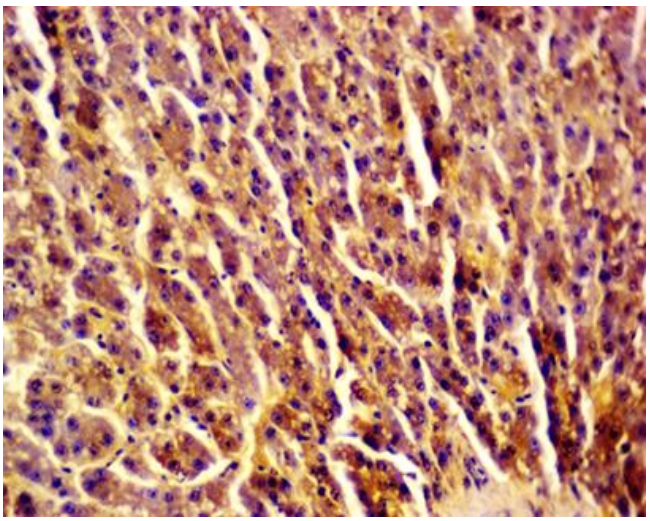

Figure 10: Light microscopic section of EM group, 2nd kill, moderates GMCSF nuclear expression, 40X.

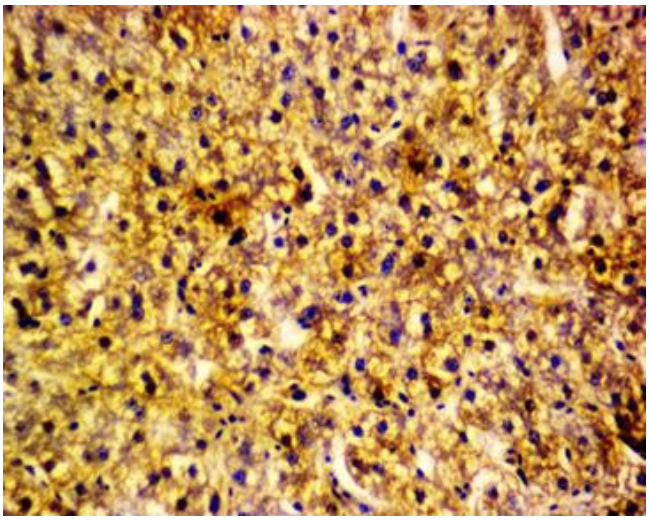

Figure 11: Light microscopic section of H group, 1st kill, sever GMCSF nuclear expression, 40X.

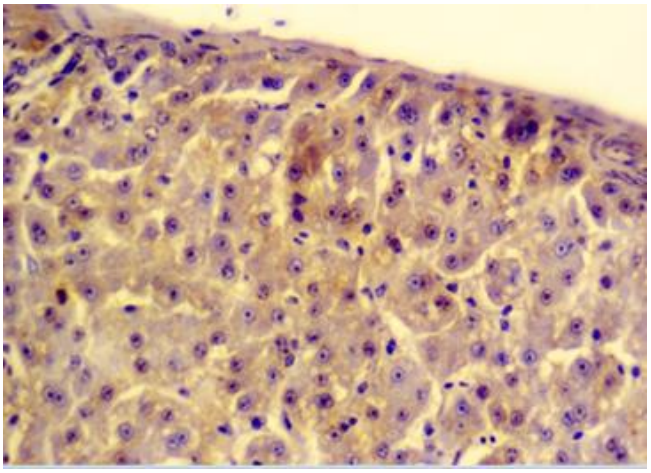

Figure 12: Light microscopic section of H+EM group, 1st kill, mild GMCSF nuclear expression, 40X.

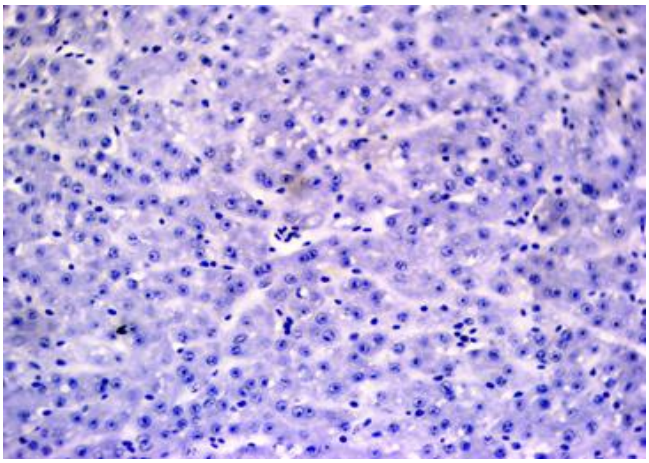

Figure 13: Light microscopic section of control group, 2nd kill, negative GMCSF nuclear expression, 40X.

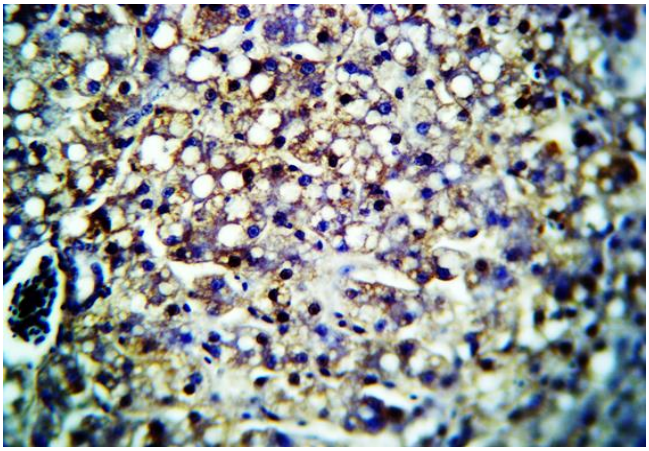

Figure 14: Light microscopic section of EM group, 2nd kill, moderates GMCSF nuclear expression, 40X.

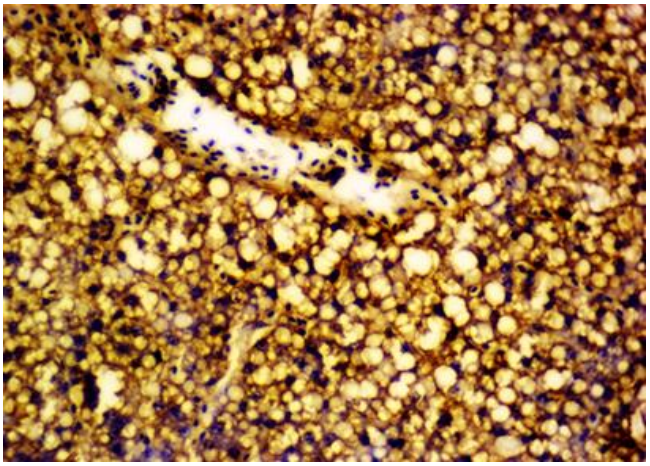

Figure 15: Light microscopic section of H group, 2nd kill, sever GMCSF nuclear expression, 40X.

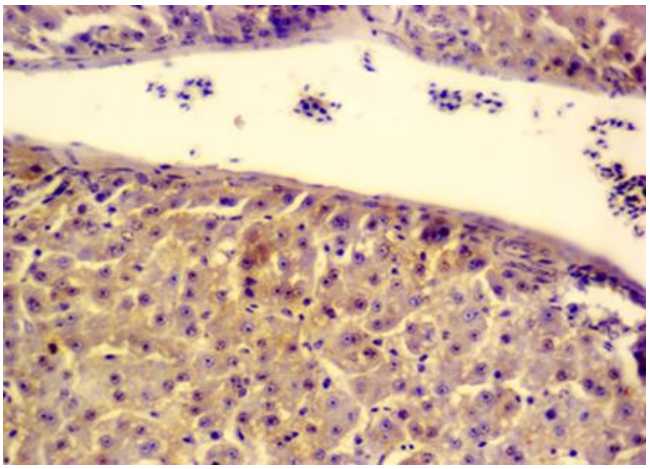

Figure 16: Light microscopic section of H+EM group, 2nd kill, mild GMCSF nuclear expression, 40X.

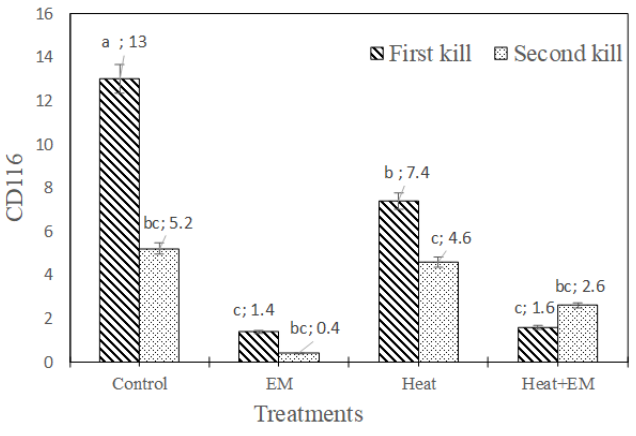

Figure 17: Correlation with GMSCF between 1st and 2nd kill.

Discussion

The liver is the big appendix gland of the bird's digestive system and immediately lies caudal to the lung and heart complex (30). Exposure of animals to ecological thermal stress increases the portal content of venous radicals, leading to cellular hypoxia of hepatocytes (31). The liver of quail birds is exposed to heat stress. Histopathological portion revealed hepatic tissue with infiltration of inflammatory cells depending on heat load severity, limited supply of nutrition, and high temperature also causes organ damage, particularly liver and intestine, which impacts the normal function of broiler (32,33) thermal stress inducing the infiltration of inflammatory cell in the liver of thermal group quails. As the hugest digestive system organ in the chicken, injury of the liver may influence the digestive system and absorption activity of the body, which may clarify why chicken growth performance is reduced to a specific range after thermal stress. Although the relation between infiltration of the inflammatory cells of the chicken and the susceptibility to thermal stress is not entirely understood, hepatocyte congestion, hemorrhage, and dilution of the blood vessels, in addition to the focal area of necrosis in the thermal stress groups, could be due deficiency of oxygen in the central lobular zone of the hypoxic liver (34,35). Alteration of the bacteria lumen, production of it, and changes the balance of reactive oxygen species production (36,37).

In a consequence, hepatocytes inflammation has been observed in animals exposure to heat stress (38,39), and burden for heat stress negative effects, hepatocytes degeneration experiencing cloudy swelling, microscopic examination revealed the granular cytoplasm this alteration occurs when water accumulated in the cytoplasm and absorbed by cytoplasmic organelles which causing mitochondria swelling and enlargement of the smooth and rough endoplasmic reticulum accompanied by ribosomes loss (40). Necrosis of hepatocytes with different size and shape were also detected decreased lipid droplet number, shrinkage of the nucleus and peripheral pallor of the cytoplasm (41). GMCSF is produced by various cells such as fibroblasts, smooth muscle, endothelial cells, and macrophages; GMCSF expression levels have been increasing in several conditions and illnesses, inclusive of inflammation, cancer, and stress factors (42). GMCSF expression is produced by pro-inflammatory chemokines and cytokines like IL-12, IL-4, and TNF-α; in inflammation sites, GMCSF has pro-inflammatory impacts through myeloid cell recruitment and improves their surveillance and activation (43). In murine out immunity disease and inflammation, blocking GMCSF causes reduced monocyte levels and recruitment of neutrophils with the pacification of disease severity. At the same time, in vivo GMSCF administration results in monocyte mobilization from the bone marrow to the bloodstream (44); GMCSF stimulates the output of CCL2 and CCL3 by macrophages and neutrophils (45,46). These observations are distinct from the role of GMCSF homeostatic; GMCSF induces macrophage survival but also helps their discrimination toward a pro-inflammatory type.

Conclusion

Liver samples collected from quail show a spectrum of Histopathological lesions exemplified by disturbances of circulation, inflammation, cell swelling, vacuolar degeneration, and necrosis. Protein marker expression was high in the ^{first} kill in contrast to its level in the second kill, and these may have contributed to the strengthening of the body's immune response, which is associated with advanced age and the bird's large size. The increase in GMCSF expression reflects the grade of thermal stress in quails.

Conflict of interest

No probable conflicts interest about this research, the authors pronounced.

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تحديد العامل المحفز لمستعمرات الخلايا البلعمية والمحببة في طيور السمان المعرضة للإجهاد الحراري والمعالجة بالمتعضيات الحية

مروة ميسر يونس و هديل باسم ذنون

فرع الأمراض وأمراض الدواجن، جامعة الموصل، كلية الطب البيطري، الموصل، العراق

الخلاصة

تناولت هذه الدراسة التأثيرات النسجية لكبد طائر السمان المعرضة للإجهاد الحراري والمعاملة بالكائنات الحية تم توزيع أربعين فرخا بعمر يوم واحد عشوائيا على أربع مجاميع وبواقع عشرة طيور لكل مجموعة وعلى النحو التالي: المجموعة الأولى كمجموعة سيطرة، المجموعة الثانية عوملت بالكائنات الحية الدقيقة الفعالة، المجموعة الثالثة المعرضة للإجهاد الحراري، والمجموعة الرابعة عوملت بالكائنات الحية الدقيقة الفعالة والمعرضة للإجهاد الحراري. وضعت الطيور في بيت الحيوانات وتركت لمدة ١٠ أيام لتتكيف. وفي اليوم الحادي عشر بدأت التجربة لمدة ثلاثين يوما تم التضحية بالحيوانات في اليوم الخامس عشر واليوم الأخير من بدء التجربة تم تصنيف الآفات المرضية المجهرية كالنزف والاحتقان والتنكس الدهني والتورم وارتشاح الخلايا الالتهابية والنخر ،وتم تحليل الآفات المرضية إحصائيا بين القتلة الأولى والثانية وكانت أعلاها في المجموعة الثالثة وفي كلا اليومين، كانت شدة التغيرات المرضية في المجموعة الثانية أقل منها في المجموعة الأولى والثالثة والرابعة ، كان الارتباط المعنوي بين الآفات اكثر وضوحا باضطرابات الدورة الدموية مثل النزف وكانت شدة التعبير هي الأعلى في القتلة الأولى في المجموعة الأولى والثانية والثالثة مقارنة بشدته في المجموعة الرابعة عامل تحفيز مستعمرات الخلايا البلعمية و المحببة وبالقتلة الثانية والتي أظهرت ارتفاعا معنويا في المجموعة الرابعة نستنتج من النتائج الى أن استخدام الكائنات الحية يمكن أن يقلل من التغيرات المرضية ويمكن استخدامه كبروتوكول وقائي في حقول تربية الدواجن.