Seasonal changes of antioxidant enzymes activity and some physiological parameters in sheep

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ABSTRACT

Twenty dry, nonpregnant ossimi ewes were used to examine how several physiological parameters were affected by a oneday exposure to heat stress.Animals were split equally into two groups, with ea ch group consisting of two experimental ewes, each of which was aged 3–4 years and weighed 35–40 kg (NRC 1988). The trial began on July 15, 2019, and ended on December 1. On the first day of the e xperiment, the first group was subjected to heat stress for a whole day. The control group was kept in the shadow until the experiment's conclusion.The levels of creatinine, haemoglobin (HB), glutathione peroxidase, total antioxidant capacity, glutathiones transferase, (ALT & AST), and body temperature were measured.The findings demonstrated that although the control group underwent no changes, th ose exposed to just one day of summertime direct heat experienced an increase in oxidative stress enz ymes. That also happened when people were exposed to direct cold for one day during the winter, wh ich increased their levels of oxidative enzymes in comparison to the control group.This is referring to e xposure to direct heat or direct cold that affects an animal's immunity and causes an increase in enzy mes as a defence mechanism the animal uses to withstand pressure.

Keywords: Heat stress; skin temperature; rectal temperature; respiration rate; cortisol hormone.

INTRODUCTION

Antioxidant defence is the process of using antioxidants to stop, slow down, or postpone t he oxidation of molecules including proteins, li pids, carbohydrates, and DNA in live cells that may be subjected to oxidation.

Antioxidants are chemicals that slow down or stop free oxygen radicals from causing tissu e damage.

The two types of antioxidants are enzymati c and nonenzymatic.

Superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx) are enzyme-based antioxidants, whereas vitamins E, C, A, s elenium (Se), transferrin, and lactoferrin are no n-enzymatic antioxidants.

Antioxidants can sometimes be found outsi de of cells.

The tumour cells' sequestration of antioxida nts as well as their sweeping up of lipid peroxi des may be the cause of the decrease in circulat ing antioxidant levels.

The drawback of ROS production is that it c an create different cancers that are resistant to exogenous growth on their own.

As an illustration, the endogenous abundan ce of antioxidants that are detoxifying and whi ch are ROS scavengers, such as leukaemia and CAT, makes the multidrug-resistant strain HL-60 resistant to the accumulation of ROS.

By turning on nuclear factor erythroid 2-related factor 2 (NRF2) and maintaining the im pact, a number of oncogene-induced cancer cells increase the antioxidant ac tivity.

ROS levels enable pro-tumorigenic signalling pathways to be activate d without inducing cell death.

Moreover, GSH levels that actively prevent cell death also appear to actively defend agains t ROS-inducing therapy in the event of an increase in GSH levels.

A vast network of molecules make up the antio xidant defence system, which destroys free rad icals and prevents the generation of ROS.

There are endogenous antioxidant defence mechanisms to counteract ROS-welded damage. By chelating intracellular RO S activity and redox balance, these systems con tinue to function as designed.

Glutathione peroxidase is an enzyme that c atalyses the interaction between hydrogen per oxide or lipid peroxides and the reduced form of glutathione (GSH), helping to detoxify these molecules by forming a glutathione bridge wit h another glutathione molecule (GSSG) form.

Catalase and glutathione peroxidase detoxif y H2O2.

The reduction of intracellular hydroperoxid es is largely dependent on the glutathione redo x cycle. GPx is a member of the selenocysteine

chemical class since it binds four selenium ato ms and has glutathione peroxidase's enzymatic activity. As a co-
substrate, glutathione is required.
Cysteine, glutamic acid, and glycine are the thr ee amino acids that make up the tripeptide glu tathione. The sulfhydryl (-SH) group and the - glutamyl linkage are two structural features of GSH. The physiological role of GSH as an anti oxidant against ROS and free radicals in the de toxification of xenobiotic substances is well-known.

Certain cell death may occur if the cell can no longer protect the GSH content.

The most significant internal antioxidant m olecule, GSH, performs a variety of physiologi cal tasks, including transporting amino acids, detoxifying xenobiotics, maintaining the reduc ed state of sulfhydryl groups in proteins, and f unctioning as a coenzyme in some enzymatic a ctivities.
When reacting with hydrogen peroxides or lipi d peroxides, glutathione converts from its redu ced form (GSH) to its oxidised form (GSSG) by forming a disulfide bridge with another glutat hione molecule. This reaction is catalysed by th e GPx enzyme and aids in the detoxification of these compounds.GSSG needs to be changed b ack into its reduced form in order to keep cells' processes of free radical detoxification operati ng normally.

NADPH is employed in a process that trans forms GSSG into reduced glutathione form wit h the GR enzyme.By using reduced glutathion e, glutathione peroxidase catalyses the detoxifi cation of lipid and H2O2 peroxides. As a result , it guards against peroxide oxidation of haemo globin and membrane lipids.

Moreover, the detoxification of xenobiotics involves GSH-Px.

The most crucial line of defence against the peroxidative deterioration of biological membr anes in mammalian cells is the antioxidant enz yme system.
These enzymes combine to produce the glutath ione peroxidase, catalase, and superoxide dism utase system, which works to defend the cell fr om oxidising agents. Oxidative stress is caused by the form of lipid peroxidation, which is the outcome of molecular oxygen conversion to R OS with numerous environmental conditions, i ncluding smoking, drinking, UV rays, and oth er oxidants.
As a result, a multistage carcinogenesis process begins, and cells may develop diseases as a res ult of the breakdown of the equilibrium betwe en lipophilic and enzymatic antioxidants, whic h together make up the skin's antioxidant capa city and ROS. Amain antioxidant defence syste m called glutathione peroxidase activity is cruc ial to the overall defence mechanisms and tacti cs used by biological systems. (Pemble et al., 1996, Strange and Fryer, 1999, Autrup, 1999)

MATERIALS AND METHODS

Study Site:

This study was carried out in Animal Farm. Department of Animal Production, Faculty of Agriculture, Al-Azhar University, Cairo, Egypt which provided standard laboratory chemicals and equipment required for this study. at F.O.P (Faculty of pharmacy).

Experimental animals and management: Th e Egyptian sheep used in this study initially ra nged in age from 3 to 4 years old and weighed 35 to 40 kg. The animals were kept in farms un der similar management conditions, with ambi ent temperatures of 28 to 35 C in the summer a nd between 15 and 24 C in the winter. A 12-hour light-dark cycle was maintained.

According to the animal farm's feeding met hod, the animals were fed in groups (individua lly) and received the necessary supplies, includ ing tap water. All of the animals were sound a nd clinically disease-free.

Experimental Outline:

The experiment was conducted in the sum mer (June 2019 till first of August 2019). and in the winter (December 2019 until the
first of February 2020).
Three months after receiving the animals for th e experiment, it was launched.

As soon as the animals were received, they were sheared.

Rectal temperature was measured througho ut every experiment alongside measurements of physiological indicators at regular intervals.

Summer is the experimental season (First ex periment)

A total of 20 animals were divided into two groups of ten Egyptian sheep each during the s ummer as follows:

G1 Regular group (Control).

Animals G2 Heat Exposed.

On June 1, 2019, the animals are exposed to heat stress for one day.

Following exposure, the animals were obser ved for three months to determine how quickl

y the physiological acclimation measures chan ge.

Winter (second experiment): A total of 20 a nimals were divided into two groups, each of which had 10 Egyptian sheep. The assignment s were made as follows:

G1 Regular group (Control).

G2 animals exposed to cold.

On January 12, 2019, the animals are subject ed to a day of cold stress.

Following exposure, the animals were obser ved for three months to determine how quickl y the physiological acclimation measures chang.

Measurements:

On Animals: Rectal temperature

On Blood: Glutathione peroxidase, Total antioxidant capacity, Glutathione – s transferase, (ALT & AST) , Createnin , Hemoglobin (HB)

Climate measurements:

Measuring the air temperature at a distance of 1 meter from the surface of the land.

Measuring soil temperature at a depth of 1.5 cm in the ground

Humidity

Blood samples: In order to get blood sampl es from sheep, an injection was used to extract blood from the orbital venous plexuses.

Samples were taken on two separate dates.

First times for HB and PCV analysis.

Serum can be obtained by centrifuging a sec ond sample for 20 minutes at 3000 rpm.

Serum was put into an Eppendorf tube and kept at - 20 Co until further testing.

Blood is drawn three times per day at vario us intervals.

Animal measurements:

Measurement of respiration rate per minute

Rectal temperature

Statistical Analysis:

Data was subjected to analysis of variance u sing the SPSS software package's General Line ar Models method (SPSS, 2020, version 23.0). B efore performing an ANOVA, all percentages were first converted to arcsine and then exami ned to simulate a normal distribution. Moreov er, Duncan's multiple range test (Duncan, 1955) was used to establish the significance of the d ifferences between means at the 5% level. To e valuate the impact of the days within each seas on, i.e., the exposure to heat or cold stress, two -way analysis of variance was performed.

The statistical model was as wased:

$Xijk = \mu + \alpha i + \beta j + \alpha\beta ij + eijk$

Were,

μ = is the mean of each trait.

αi = is the effect of days within season on each trait.

βj = is the effect of exposure to heat or cold stress within season.

$\alpha\beta ij$ = is the interaction between days within season and exposure to stress.

eijk = is the experimental error

RESULTS AND DISCUSSION:

Summer season

Glutathione peroxidase

Table 1's findings revealed a significant diff erence (p 0.05) in the amount of glutathione pe roxidase in the blood serum between the expos ed and control groups on experiment day 0.

While the exposed group experienced a dis cernible increase from the 30th, 60th, and 90th days.

Table 1's findings also revealed a significant difference (p 0.05) between the exposed and co ntrol groups on day 30 of the trial in the amou nt of glutathione peroxidase in the blood seru m.

But there was a sizable increase in the expo sed group between the 60th and 90th day.

On days 60 and 90 of the experiment, it was evident from the same Table (Table1) that ther e was a significant difference (p 0.05) in the am ount of glutathione peroxidase in the blood ser um between the exposed and control groups.

From the earlier findings, it is clear that sub jecting ewes to heat stress caused a considerabl e rise in the amount of glutathione peroxidase i n their blood serum.

After 30, 60, and 90 days of the trial, this lar ger rise was observed in the heat stress group on day 0 compared to the control group.

According to earlier findings, it was determ ined that subjecting ewes to heat stress increas ed their blood serum levels of glutathione pero xidase on the first day of the experiment. How

ever, this effect only persisted for 30 days after the ewes were exposed to heat stress, and it dis appeared during the subsequent periods (days 60 and 90) of the exposure to heat stress.

Rathwa, S. D. was similar to this outcome (2 017).

discovered that in native sheep, ambient te mperature had a greater impact on THI than re lative humidity. THI is a sensitive biomarker o f heat stress.

Much higher GPx levels are related to highe r THI.

Xing and Wang et al., (2015) located to

Catecholamines are released more readily d uring heat stress, which raises the levels of sup eroxide free radicals and hydrogen peroxide. E xcessive reactive oxygen species synthesis har ms the body's antioxidant system and causes o xidative stress.

Total Antioxidant Capacity

According to Table 2's findings, there was a significant difference in the blood serum level of total antioxidant capacity between the expos ed and control groups on Day 0 of the trial (p 0 .05). While the exposed group experienced a di scernible increase from the 30th, 60th, and 90th days.

The findings in Table (2) also demonstrated a significant difference (p 0.05) between the ex posed and control groups on day 30 of the trial in the amount of total antioxidant capacity in t he blood serum.

But there was a considerable rise in the exp osed group compared to the 60th and 90th day.

On days 30, 60, and 90 of the experiment, it was evident from the same Table (Table2) that there was a significant difference (p 0.05) in the level of Total Antioxidant Capacity in the bloo d serum between the exposed and control grou ps.

Even though the control group does not sig nificantly differ from it on different days.

The preceding findings make it clear that su bjecting ewes to heat stress resulted in a signifi cant rise in the amount of total antioxidant cap acity in their blood serum.

After 30, 60, and 90 days of the trial, this lar ger rise was observed in the heat stress group on day 0 compared to the control group.

According to earlier findings, it was determ ined that subjecting ewes to heat stress increas ed their blood serum's Total Antioxidant Capa city on the first day of the experiment. Howeve r, this effect only persisted for 30 days after ex posure to heat stress, disappearing in the subse quent periods (days 60 and 90) of heat stress ex posure.

This result was similar to : (Mujahid et al. 2007).
revealed that the creation of too many reactive oxygen species (ROS), which can lead to oxidat ive injury such as lipid peroxidation in membr anes and oxidative damage to proteins and DN A/RNA, can be promoted by oxidative stress b rought on by hyperthermal stress.

Harmon et al. (1997) reported that heat-stressed, mid-
lactating Holstein cows had lower plasma anti oxidant activity.

Shi, L. et al (2020) discovered that heat stress may have reduced t he antioxidant enzyme activity by causing oxid ative stress, which may be related to the enviro nment's increased generation of ROS.

Meanwhile, lamb's immune and antioxidan t systems may suffer long-
term damage from persistently high levels of h eat stress.

In particular, there was a strong correlation between all of these findings and the longer du ration and intensity of heat stress.

Glutathione – s Transferase

According to Table 3's findings, there was a significant difference in the blood serum level of glutathione-
s transferase between the exposed and control groups on Day 0 of the trial (p 0.05).

While the exposed group experienced a dis cernible increase from the 30th, 60th, and 90th days.

The data in Table 3 also revealed that, on da y 30 of the trial, there was a significant differen ce in the blood serum level of glutathione-
s transferase between the exposed and control groups (p 0.05).

But there was a considerable rise in the exp osed group compared to the 60th and 90th day.

On days 30, 60, and 90 of the experiment, it was evident from the same Table (Table3) that there was a significant difference (p 0.05) in the amount of glutathione -
s transferase in the blood serum between the e xposed and control groups.

It is clear from the previous data that the ex posure of ewes to heat stress revealed a large i

ncrease in the amount of glutathione - s transferase in the blood serum of the expose d ewes, even if there is no statistically significa nt difference between the control group on diff erent days.

After 30, 60, and 90 days of the trial, this lar ger rise was observed in the heat stress group on day 0 compared to the control group.

Based on earlier findings, it was determine d that subjecting ewes to heat stress increased t he amount of glutathione - s transferase in their blood serum on the first day of the trial.This result was similar to :

Xing and Wang et al., (2015) located to

Catecholamines are released more readily d uring heat stress, which raises the levels of hyd rogen peroxide and superoxide free radicals.

The body's antioxidant system is harmed b y excessive generation of reactive oxygen speci es, which also causes oxidative stress.

ALT

Table (4)'s findings revealed that there was a significant difference in the amount of ALT i n the blood serum between the exposed and co ntrol groups on day 0 of the trial (p 0.05).

While the exposed group experienced a dis cernible increase from the 30th, 60th, and 90th days.

Findings in Table (4) also demonstrated a si gnificant difference (p 0.05) between exposed a nd control groups in the amount of ALT in the blood serum on days 30, 60, and 90 of the trial.

While the exposed group showed no signifi cant changes after 30, 60, and 90 days.

On days 30, 60, and 90 of the experiment, it was evident from the same Table (Table 4) that there was a significant difference (p 0.05) betw een the exposed and control groups in the amo unt of ALT in the blood serum.

Even though the control group does not sig nificantly differ from it on different days

From the earlier findings, it is clear that sub jecting sheep to heat stress caused a significant rise in the amount of ALT in their blood serum .

After 30, 60, and 90 days of the trial, this lar ger rise was observed in the heat stress group on day 0 compared to the control group.

The foregoing findings led to the conclusio n that the exposure of ewes to heat stress raise d the amount of ALT in the exposed ewes' bloo d serum on the first day of the experiment.

This outcome was comparable to that obser ved in rats and goats, which both had non-significant diurnal patterns of GOT and GPT in their blood plasma (Ali, 2000). (Abd El-Hamed, 2004).

The diurnal fluctuations in plasma GOT an d GPT in rats were corroborated by Ali (2004), but he also showed that during the summer, th ey were substantially higher at 4.0 than at 12.0 midday.

AST

Findings in Table (5): revealed a significant difference (p 0.05) between exposed and contro l groups in the amount of AST in the blood ser um on days 0 and 30 of the trial.

In contrast to the 60th and 90th days, there was a considerable increase in the exposed gro up.

The findings in Table (5) also demonstrated a significant difference (p 0.05) between the ex posed and control groups on days 60 and 90 of the experiment in the amount of AST in the blo od serum.

However, there was no difference between the exposed group at 60 and 90 days.

While there is no discernible difference bet ween the control group on different days in the same Table (Table 5),

The preceding findings clearly demonstrate that subjecting sheep to heat stress resulted in a significant rise in the level of AST in the bloo d serum of the exposed ewes.

Compared to the control group, this larger r ise occurred on days 0 and 30 after exposure to heat stress.

The prior findings led to the conclusion that subjecting lambs to heat stress increased the a mount of AST in their blood serum on days 0 a nd 30 of the trial.

This outcome was comparable to that obser ved in rats and goats, which both had non-significant diurnal patterns of GOT and GPT in their blood plasma (Ali, 2000). (Abd El-Hamed, 2004).

The diurnal fluctuations in plasma GOT an d GPT in rats were corroborated by Ali (2004), but he also showed that during the summer, th ey were substantially higher at 4.0 than at 12.0 midday.

Createnin

The data in Table (6) revealed that there wa s no difference in the level of creatinine in the b

lood serum between the exposed and control g roups (p 0.05).

Findings in Table (6) also indicated that the re was no change between exposed groups that was statistically significant (p 0.05).

There is no discernible difference between t he control group on different days in the same table (Table 6).

Similar findings were made in rats and goat s, according to Ali (2000), who discovered that plasma creatinine levels were higher in the su mmer and winter at midday than they were at four in the morning (Ali, 2004).

Hemoglobin (HB)

The results in Table (7) demonstrated that t here was no difference in the level of HEMOG LOBIN (HB) in the blood serum between the e xposed and control groups (p 0.05).

Findings in Table (7) also indicated that the re was no change between exposed groups that was statistically significant (p 0.05).

Similar Table (Table 7):

Even though the control group does not sig nificantly differ from it on different days

This outcome was comparable to that of Sh oukry (1981), who discovered that rams' Hb co ntent was much lower in the autumn (8.6 g/dl). He linked these findings to a lack of iron, cop per, or mean corpuscular haemoglobin as a res ult of extended exposure to extreme heat in the summer.

The same outcomes were observed with Ba rki ewes (Abd-El-Bary et al., 1982). El Nouty et al. (1989) discovered that Hb was mu ch greater in the summer than in the spring in goats (11.3 vs. 8.8 g/l).

On the other side, it has been demonstrated that higher ambient temperatures result in a re duction in sheep haemoglobin (Da Silva et al., 1992).

Although Hassanin et al. (1996) found that heat stress raised the blood Hb of goats.

Moreover, Kume et al. (1998) observed that heat stress raised the blood Hb of heifers.

Yet, after heat stress in the same trial, newb orn calves' haemoglobin levels were lower.

Thermal stress had no effect on haemoglobi n (Hb) in Omani sheep, but it decreased Hb in Merino sheep, according to Srikandakumar et al. (2003).

According to Maurya et al. (2013), cold-stressed lambs had significantly (P0.05) higher Hb concentrations than lambs who were not ex posed to cold stress.

The explanation for the rise in Hb in the G1 lambs may be related to a rise in RBC and Hb s ynthesis to preserve homeostasis.

According to Maurya et al. (2013), cold-stressed lambs had considerably (P0.05) greate r Hb concentrations than lambs who had been given protection.

Rectal Temperature

The data in Table (8) revealed that there wa s no difference in the level of rectal temperatur e in the blood serum between the exposed and control groups (p 0.05).

Findings in Table (8) also indicated that the re was no change between exposed groups that was statistically significant (p 0.05).

There is no discernible difference between t he control group on different days in the same table (Table 8).

This outcome resembled

According to the research, sheep's average r ectal temperatures can range from 38.3 to 41 °C (Swenson and Reece, 1993; Shafie et al., 1994 a nd Shalaby et al., 1996).

Many animals have records of their internal temperatures rising in response to environme ntal stress (Ingram et al., 2002).

According to Parrott et al. (1999), sheep and goat management strategies are evaluated for their ability to cause stress-induced hyperthermia.

Beatty et al. (2006) recently observed that ha rsh environmental conditions cause elevated c ore body temperatures, decreases in feed intak e, panting, acid-base and plasma electrolyte imbalances, and el evated core body temperatures.

Glutathione peroxidase

The data in Table (9) revealed that there wa s a significant difference (p 0.05) in the amount of glutathione peroxidase in the blood serum between the exposed and control groups on da y 0 of the trial.

While the exposed group experienced a dis cernible increase from the 30th, 60th, and 90th days.

Findings in Table (9) also demonstrated a si gnificant difference (p 0.05) between exposed a nd control groups on day 30 of the trial in the a

mount of glutathione peroxidase in the blood s erum.

While there was a considerable rise in the g roup exposed compared to the 60th and 90th d ay.

On days 60 and 90 of the experiment, it was evident from the same Table (Table 9) that the re was a significant difference (p 0.05) in the a mount of glutathione peroxidase in the blood s erum between the exposed and control groups.

From the earlier findings, it is clear that sub jecting ewes to heat stress caused a considerabl e rise in the amount of glutathione peroxidase i n their blood serum.

After 30, 60, and 90 days of the trial, this lar ger rise was observed in the heat stress group on day 0 compared to the control group.

According to earlier findings, it was determ ined that subjecting ewes to heat stress increas ed their blood serum levels of glutathione pero xidase on the first day of the experiment. How ever, this effect only persisted for 30 days after the ewes were exposed to heat stress, and it dis appeared during the subsequent periods (days 60 and 90) of the exposure to heat stress.

Total Antioxidant Capacity

The Total Antioxidant Capacity level in the blood serum was significantly different betwee n the exposed and control groups on day 0 of t he experiment, according to the results in Tabl e (10) (p 0.05).

While the exposed group experienced a dis cernible increase from the 30th, 60th, and 90th days.

The data in Table (10) also revealed that, on day 30 of the trial, there was a significant diffe rence in the amount of total antioxidant capacit y in the blood serum between the exposed and control groups (p 0.05).

But there was a considerable rise in the exp osed group compared to the 60th and 90th day.

On days 30, 60, and 90 of the experiment, it was evident from the same Table (Table 10) tha t there was a significant difference (p 0.05) in t he level of Total Antioxidant Capacity in the bl ood serum between the exposed and control gr oups.

It is clear from the previous data that the ex posure of ewes to heat stress exhibited a massi ve rise in the level of Total Antioxidant Capacit y in the blood serum of the exposed ewes, even though there is no statistically significant diffe rence between the control group on different d ays.

After 30, 60, and 90 days of the trial, this lar ger rise was observed in the heat stress group on day 0 compared to the control group.

According to earlier findings, it was determ ined that subjecting ewes to heat stress increas ed their blood serum's Total Antioxidant Capa city on the first day of the experiment. Howeve r, this effect only persisted for 30 days after ex posure to heat stress, disappearing in the subse quent periods (days 60 and 90) of heat stress ex posure.

Glutathione – s Transferase

The data in Table (11) revealed that there w as a significant difference (p 0.05) in the amoun t of glutathione s transferase in the blood seru m between the exposed and control groups on day 0 of the trial.

While the exposed group experienced a dis cernible increase from the 30th, 60th, and 90th days.

The findings in Table (11) also demonstrate d a significant difference (p 0.05) between the e xposed and control groups on day 30 of the tri al in the amount of glutathione s transferase in the blood serum.

But there was a considerable rise in the exp osed group compared to the 60th and 90th day.

On days 30, 60, and 90 of the experiment, it was evident from the same Table (Table 11) tha t there was a significant difference (p 0.05) in t he amount of glutathione - s transferase in the blood serum between the e xposed and control groups.

It is clear from the previous data that the ex posure of ewes to heat stress revealed a large i ncrease in the amount of glutathione - s transferase in the blood serum of the expose d ewes, even if there is no statistically significa nt difference between the control group on diff erent days.

After 30, 60, and 90 days of the trial, this lar ger rise was observed in the heat stress group on day 0 compared to the control group.

Based on earlier findings, it was determine d that subjecting ewes to heat stress increased t he amount of glutathione - s transferase in their blood serum on the first day of the trial.

ALT

Table (12) results revealed that there was a significant difference in the amount of ALT in t

he blood serum between the exposed and contr ol groups on day 0 of the trial (p 0.05).

While the exposed group experienced a dis cernible increase from the 30th, 60th, and 90th days.

The data in Table 12 also revealed that there was a significant difference (p 0.05) in the amo unt of ALT in the blood serum between the ex posed and control groups on days 30, 60, and 9 0 of the trial.

While the exposed group showed no signifi cant changes after 30, 60, and 90 days.

On days 30 and 60 and 90 of the experiment , it was evident from the same Table (Table12) t hat there was a significant difference (p 0.05) b etween the exposed and control groups in the l evel of ALT in the blood serum.

Even though the control group does not sig nificantly differ from it on different days

From the earlier findings, it is clear that sub jecting sheep to heat stress caused a significant rise in the amount of ALT in their blood serum .

After 30, 60, and 90 days of the trial, this lar ger rise was observed in the heat stress group on day 0 compared to the control group.

It was determined from the earlier findings that subjecting lambs to heat stress increased t he amount of ALT in their blood serum on the first day of the trial.

ALT

The data in Table 13 demonstrated that ther e was a significant difference in the amount of AST in the blood serum between the exposed a nd control groups on days 0 and 30 of the trial (p 0.05).

In contrast to the 60th and 90th days, there was a considerable increase in the exposed gro up.

Table 13's findings also revealed a significa nt difference (p 0.05) between the exposed and control groups on days 60 and 90 of the trial in the amount of AST in the blood serum.

However, there was no difference between the exposed group at 60 and 90 days.the identi cal Table (Table 13):

Even though the control group does not sig nificantly differ from it on different days

The preceding findings clearly demonstrate that subjecting sheep to heat stress resulted in a significant rise in the level of AST in the bloo d serum of the exposed ewes.

Compared to the control group, this larger r ise occurred on days 0 and 30 after exposure to heat stress.

The prior findings led to the conclusion that subjecting lambs to heat stress increased the a mount of AST in their blood serum on days 0 a nd 30 of the trial.

Createnin

The data in Table (14) revealed that there w as no difference in the level of creatinine in the blood serum between the exposed and control groups (p 0.05).

Findings in Table (14) also indicated that th ere was no change between exposed groups th at was statistically significant (p 0.05).

Table 14 in the same table:

Even though the control group does not sig nificantly differ from it on different days

Hemoglobin (HB)

The results in Table (15) demonstrated that there was no difference in the level of HEMOG LOBIN (HB) in the blood serum between the e xposed and control groups (p 0.05).

There was no significant difference (p 0.05) between the exposed groups, according to Tabl e 15's results.

Table 15 in the same table:

Even though the control group does not sig nificantly differ from it on different days

Rectal Temperature

The data in Table (16) revealed that there w as no difference in the level of rectal temperatu re in the blood serum between the exposed an d control groups (p 0.05).

Findings in Table (16) also indicated that th ere was no change between exposed groups th at were statistically significant (p 0.05).

the identical Table (Table 16):

Even though the control group does not sig nificantly differ from it on different days.

CONCLUSION

The findings demonstrated that although th e control group underwent no changes, those e xposed to just one day of summertime direct h eat experienced an increase in oxidative stress enzymes.

That also happened when people were exp osed to direct cold for one day during the wint

er, which increased their levels of oxidative en zymes in comparison to the control group.

This is referring to exposure to direct heat o r direct cold that affects an animal's immunity and causes an increase in enzymes as a defence mechanism the animal uses to withstand press ure.

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Table 1: Means ± standard errors of Glutathione peroxidase in blood serum of two groups of ewes exposed to heat stress:

Exposure to heat stress	Exposed		Control	
Days	Mean±SE	Dun	Mean±SE	Dun
Day 0	395±0.92	Aa	269±0.90	Ab
Day 30	333±0.27	Ba	250±0.23	Ab
Day 60	285±0.71	Ca	265±0.60	Ab
Day 90	289±0.32	Ca	263±0.32	Ab

S.E = stander error

Dun= Duncan's Multiple Range test between groups

Mean within each row with similar letters are not significant different at p >0.05

** significant (p<0.01) * significant (p<0.05)

Table 2: Means ± standard errors of Total Antioxidant Capacity in blood serum of two groups of ewes exposed to heat stress:

Exposure to heat stress	Exposed		Control	
Days	Mean±SE	Dun	Mean±SE	Dun
Day 0	3.27± 3.74	Aa	1.87± 2.57	Ab
Day 30	2.27±5.16	Ba	1.59±4.98	Ab
Day 60	2.17±3.93	Ba	1.69±4.14	Ab
Day 90	2.11±4.22	Ba	1.57±4.53	Ab

S.E = stander error

Dun= Duncan's Multiple Range test between groups

Mean within each row with similar letters are not significant different at p >0.05

** significant (p<0.01) * significant (p<0.05)

Table 3: Means ± standard errors of Glutathione – s Transferase in blood serum of two groups of ewes exposed to heat stress:

Exposure to heat stress	Exposed		Control	
Days	Mean±SE	Dun	Mean±SE	Dun
Day 0	380±0.17	Aa	185.11±.23	Ab
Day 30	275±0.22	Ba	190±.17	Ab
Day 60	280±0.09	Ba	188±.11	Ab
Day 90	269±.13	Ba	179±.11	Ab

S.E = stander error

Dun= Duncan's Multiple Range test between groups

Mean within each row with similar letters are not significant different at p >0.05

** significant (p<0.01) * significant (p<0.05)

Table 4: Means ± standard errors of ALT in blood serum of two groups of ewes exposed to heat stress:

Exposure to heat stress	Exposed		Control	
Days	Mean±SE	Dun	Mean±SE	Dun
Day 0	47±0.08	Aa	32.07±.067	Ab
Day 30	22.88±0.08	Ba	22.97±0.06	Ab
Day 60	23.28±.050	Ba	22.28±.0.03	Ab
Day 90	22.90±.054	Ba	22.95±.054	Ab

S.E = stander error

Dun= Duncan's Multiple Range test between groups

Mean within each row with similar letters are not significant different at p >0.05

** significant (p<0.01) * significant (p<0.05)

Table 5: Means ± standard errors of AST in blood serum of two groups of ewes exposed to heat stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	96.30±0.26	Ab	99.06± 0.20	A
Day 30	96.33± 0.26	Aa	96.06± 0.21	Aa
Day 60	94.64± 0.10	Ba	94.27± 0.12	Aa
Day 90	94.68± 0.16	Ba	94.53±.12	Aa

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Table 6: Means ± standard errors of blood (Createnin) of two groups of ewes exposed to heat stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	0.94±0.26	Aa	0.97± 0.20	Aa
Day 30	0.97± 0.26	Aa	0.93± 0.21	Aa
Day 60	0.94± 0.10	Aa	0.98± 0.12	Aa
Day 90	0.98± 0.16	Aa	0.99±.12	Aa

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Table 7: Means ± standard errors of Hemoglobin (HB) in blood of two groups of ewes exposed to heat stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	12.93±0.68	Aa	12.93±0.23	Aa
Day 30	12.43±0.19	Aa	12.04±0.20	Aa
Day 60	12.40±0.27	Aa	12.43±0.24	Aa
Day 90	12.56±0.18	Aa	12.07±0.21	Aa

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Table 8: Means ± standard errors of Rectal Temperature (°C) of two groups of ewes exposed to heat stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	39.62±.083	Aa	39.54±0.04	Aa
Day 30	39.39±.030	Aa	39.56±.050	Aa
Day 60	39.45±.031	Aa	39.55±.046	Aa
Day 90	39.39±.044	Aa	39.51±.0.03	Aa

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Winter season (cold stress):

Table 9: Means ± standard errors of Glutathione peroxidase in blood serum of two groups of ewes exposed to cold stress:

Exposure to heat stress	Exposed		Control	
Days	Mean±SE	Dun	Mean±SE	Dun
Day 0	393±0.91	Aa	265±0.90	Ab
Day 30	330±.26	Ba	250±.23	Ab
Day 60	280±.70	Ca	263±.60	Ab
Day 90	284±.30	Ca	260±.31	Ab

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Table 10: Means ± standard errors of Total antioxidant capacity in blood serum of two groups of ewes exposed to cold stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	3.20± 3.70	Aa	1.85± 2.50	Ab
Day 30	2.25±5.15	Ba	1.59±4.98	Ab
Day 60	2.14±3.91	Ba	1.68±4.13	Ab
Day 90	2.12±4.21	Ba	1.55±4.52	Ab

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Table 11: Means ± standard errors of Glutathione – s Transferase in blood serum of two groups of ewes exposed to cold stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	378±0.16	Aa	184±.23	Ab
Day 30	273±0.22	Ba	189±.17	Ab
Day 60	280±0.09	Ba	187±.11	Ab
Day 90	265±.13	Ba	177±.10	Ab

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Table 12: Means ± standard errors of ALT in blood serum of two groups of ewes exposed to cold stress:

Exposure to heat stress	Exposed		Control	
Days	Mean±SE	Dun	Mean±SE	Dun
Day 0	45±0.07	Aa	31.06±.066	Ab
Day 30	22.80±0.08	Ba	22.95±0.06	Aa
Day 60	23.25±.050	Ba	22.29±.0.03	Aa
Day 90	22.70±.053	Ba	22.93±.053	Aa

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Table 13: Means ± standard errors of AST in blood serum of two groups of ewes exposed to cold stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	96.28±0.26	Aa	99.03± 0.20	Aa
Day 30	96.32± 0.26	Aa	96.03± 0.21	Aa
Day 60	94.60± 0.10	Ba	94.25± 0.12	Aa
Day 90	94.63± 0.16	Ba	94.50±.12	Aa

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Table 14: Means ± standard errors of blood **(** Createnin **)** of two groups of ewes exposed to cold stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	0.92±0.26	Aa	0.95± 0.20	Ab
Day 30	0.94± 0.26	Aa	0.91± 0.21	Ab
Day 60	0.93± 0.10	Aa	0.92± 0.12	Ab
Day 90	0.97± 0.16	Aa	0.98±.12	Ab

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

Table 15: Means ± standard errors of Hemoglobin (HB) in blood of two groups of ewes exposed to cold stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	12.91±0.68	Aa	12.90±0.23	Aa
Day 30	12.40±0.19	Aa	12.02±0.20	Aa
Day 60	12.41±0.27	Aa	12.42±0.24	Aa
Day 90	12.54±0.18	Aa	12.03±0.21	Aa

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.

Table 16: Means ± standard errors of **Rectal Temperature** (°C) of two groups of ewes exposed to cold stress:

Exposure to heat stress Days	Exposed		Control	
	Mean±SE	Dun	Mean±SE	Dun
Day 0	39.21±.04	Aa	39.38±.05	Aa
Day 30	39.22±.03	Aa	39.34±.04	Aa
Day 60	39.30±.03	Aa	39.22±.03	Aa
Day 90	39.34±.04	Aa	39.17±.04	Aa

S.E = stander error
Dun= Duncan's Multiple Range test between groups
Mean within each row with similar letters are not significant different at p >0.05
** significant (p<0.01) * significant (p<0.05)

الإختلافات الموسمية في نشاط الإنزيمات المضادة للتأكسد وبعض القياسات الفسيولوجية في الأغنام

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الملخص العربى

تم استخدام عشرين نعجة اوسيمي تم دراسة تأثير التعرض للإجهاد الحراري ليوم واحد على بعض المقاييس الفسيولوجية واهما انزيمات التأكسد. قسمت الحيوانات إلى مجموعتين بالتساوي ، كل مجموعة تتراوح أعمارها بين 3-4 سنوات ووزنها 35-40 كجم ، تم تغذية جميع النعاج على نظام غذائي مكتمل حسب (NRC 1988).بدأت التجربه في 15 يوليو 2019 وانتهت في 1 ديسمبر2019. المجموعة الأولى تعرضت للإجهاد الحراري لمدة يوم واحد في اليوم الأول من التجربة. بقيت المجموعة الضابطة تحت الظل من بداية التجربه حتى نهايتها. تم قياس انزيمات التأكسد وانزيمات الكبد والكلي ، الهيموجلوبين في مصل الدم. تم قياس درجة حرارة المستقيم. أظهرت النتائج أن التعرض للحرارة المباشرة ليوم واحد فقط في فصل الصيف أدى إلى زيادة إنزيمات الإجهاد التأكسدي ، بينما لم تحدث أي تغييرات في المجموعة الضابطة. كما يحدث عند التعرض لمدة يوم واحد للبرد المباشر في فصل الشتاء مما أدى إلى زيادة الإنزيمات المؤكسدة مقارنة بالمجموعة الضابطة. يشير هذا إلى التعرض للحرارة المباشرة أو البرودة المباشرة التي تؤثر على مناعة الحيوان مما يؤدي إلى ارتفاع الإنزيمات كجزء من المناعة التي يستخدمها الحيوان لمواجهة الضغط عليه.

الكلمات الاسترشادية: الإجهاد الحراري، درجة حرارة الجلد، درجة حرارة المستقيم، معدل التنفس، هرمون الكورتيزول.